Handbook of Aerospace and Operational Physiology, 2\textsuperscript{nd} Edition

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October 2016
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# Handbook of Aerospace and Operational Physiology, 2nd Edition

## Abstract

This handbook is an update to AFRL-SA-WP-SR-2011-0003 and is designed to be a reference for aerospace physiologists and technicians in the U.S. Air Force. It contains information about physiologic principles and application of those principles to Air Force flight operations. While it is not designed to be a clinical resource, references to clinical materials are provided to allow further study.

## Subject Terms

Circulation, respiration, aerospace physiology, hypoxia, trapped gas, oxygen toxicity, decompression sickness, acceleration, personal equipment, HUD, HMD, human effectiveness, human factors, spatial disorientation.
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FOR THE SECOND EDITION

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FOR THE FIRST EDITION

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PREFACE

TO THE SECOND EDITION

In the summer of 2011, the first edition of the Handbook of Aerospace and Operational Physiology (HAOP) was publicly released. Feedback from the field was overwhelmingly positive and helped to identify areas that were missing or that needed to be expanded. As a result, many chapters in this second edition have been extensively reworked (respiratory physiology, neural physiology, hypoxia, trapped gas, helmet mounted displays, laser awareness training, chamber training, FAQ sections for chamber and centrifuge training) and several new chapters added (vestibular physiology, somatosensory physiology, space physiology, crew resource management/maintenance resource management, radiation, centrifuge training, spatial disorientation training, aircraft identification, flight principles and terminology). One goal of this update was to improve the HAOP for use as a study reference for certification in Aerospace Physiology offered through the Aerospace Medical Association (certification objectives are included in Appendix 10). A few chapters have been combined (alveolar gas equation now in gas laws; aviation psychology and stresses of flight now in human performance optimization) or rearranged (physiological responses to thermal environments and physiological effects of noise moved to mission-imposed effects, exercise physiology and nutrition split into separate chapters) to improve the organization of the document. In addition, an attempt has been made to improve the consistency of values and formatting throughout the text.

As with the first edition, the second edition of the HAOP is not intended to be a clinical resource, nor to replace other more in-depth texts and references, but rather to serve as a consolidated reference for many of the topics relevant to the Aerospace and Operational Physiology field, as well as for other related specialties. Readers are encouraged to continue to stay actively engaged in the current research and literature. It is our goal that the Handbook of Aerospace and Operational Physiology continues to be a living document that will be periodically updated as current technologies evolve and new areas of interest arise.

Lt Col Ryan W. Maresh and Lt Col (Ret) Andrew D. Woodrow

TO THE FIRST EDITION

The purpose of this document is to provide a resource for information about physiologic principles and application of those principles pertinent to aerospace physiologists and technicians in the U.S. Air Force. While it is not designed to be a clinical resource, many references to clinical texts and articles are provided to allow further study.

The scope will likely challenge those without some familiarity with crewmember personal equipment capabilities and limitations or without any human physiology background. Without such a challenge, it would be of little use to many who need a reference relevant to their work.

The sequence of sections is meant to introduce pertinent human physiology, the atmosphere, and effects of the atmosphere on human physiology. The section on human performance enhancement requires an understanding of human physiology
covered earlier in the document and of human factors, which is emphasized in this and following sections. The next sections describe how the various Air Force aircraft systems, personal equipment, and missions interact with the individual to both limit and enhance optimal function. Background information is provided on aircraft systems and personal equipment design, function, and limitations. Aerospace physiologist training, evaluation, activities, and resources complete the body of this reference document.

Appendices 1 and 2 altitudes vs. pressure and oxygen levels as well as conversion tables are relevant to several sections, and their placement is meant to provide a common reference and avoid duplication. Appendix 8 contains URLs for internet resources that are followed by the subject description and last date checked for viability. Organizations of potential interest to aerospace physiologists follow with questions on each section. The last appendix contains short biographies of the contributors to this document.

The objectives listed at the beginning of most sections are for AFI 11-403 Original Course instructor use in developing presentation materials and background study.

Lt Col Andrew D. Woodrow and James T. Webb, Ph.D.
HISTORY

Since the 17\textsuperscript{th} century, many findings and inventions have allowed discoveries of the properties of the atmosphere and space. The problems of hypoxia, trapped gas expansion, decompression sickness, and ebullism were discovered, endured, addressed, and sometimes ignored with unfortunate consequences. To understand the problems associated with reduced atmospheric pressure, many physiologists, physicists, and physicians labored to quantify properties of the atmosphere. Their formulation of gas laws and descriptions of atmospheric characteristics enabled physiologists, physicians, inventors, and designers to investigate atmospheric effects and conceive mechanisms to enable safe passage into these realms. Better designs of air and spacecraft, personal protective equipment, and procedures to protect the crew and passengers have saved many lives and enhanced mission effectiveness and capabilities. Establishment of the Physiological Research Laboratory at Wright Field, OH, on 29 May 1935 ushered in an era of historic research and achievements, largely through the efforts of Captain Harry G. Armstrong. Highlights of some of these and other achievements in aerospace physiology are presented in many sections.
1. HUMAN PHYSIOLOGY

Organ systems of the human body are susceptible to the effects of aerospace environmental stressors. Knowledge of these systems is necessary to understand how changes in environmental conditions can produce serious hazards to human performance and survival. Using the basic knowledge of physiology presented here and of the aerospace environment, researchers, engineers, physicians, and aerospace physiologists have played critical roles in the design of aircraft systems, personal protective equipment, and procedures. The results of their efforts have reduced or eliminated most environmental threats to aircrew by innovations and improvements in aircrew equipment and procedures.

1.1. Cardiovascular Physiology

Paul W. Fisher, Col, USAF (Ret), BSC, Ph.D.

The human circulatory system is a closed, continuous loop system that can be divided into two subdivisions or circuits: the pulmonary circulation and the systemic circulation. It is composed of the blood, the heart, and the conducting vessels: arteries, arterioles, capillaries, venules, and veins. Its primary function is to serve as the transportation system within the body, but it also contributes to other functions such as thermoregulation; water, electrolyte, and pH balance; and defense against pathogens.

1.1.1. The Blood

The blood is the transport vehicle of the cardiovascular system.

The blood transports nearly everything that must be carried from one place to another within the body (Marieb and Hoehn, 2007). The blood is the only fluid tissue in the body and is composed of both cellular and liquid components. If a blood sample is spun in a centrifuge, the heavier formed elements will be packed at the bottom of the centrifuge tube with the less dense fluid plasma at the top (Figure 1.1.1-1). The red mass at the bottom of the tube is primarily erythrocytes, the red blood cells responsible for transporting oxygen. Red blood cells (RBCs) are the most abundant cell type in the body and make up approximately 45% of the total blood volume. Each mL of blood contains 5,200,000 ± 300,000 RBCs (Guyton and Hall, 2000). An average man with a total blood volume of about 5 L has approximately 26 billion RBCs circulating throughout his body. The normal percentage of RBCs, the hematocrit, varies somewhat between men and women, with males having a value of 47% ± 5% and females 42% ± 5% (Marieb and Hoehn, 2007). Hematocrit also varies with environmental and physiologic stressors such as prolonged exposure to altitude. Above the RBCs in the centrifuge tube is a thin whitish layer that contains leukocytes, white blood cells involved in the body’s immune response, and platelets, cell fragments critical to the blood clotting process. Leukocytes and platelets contribute less than 1% of blood volume, with plasma making up the remaining 55%.
Plasma, the fluid matrix of the blood, is 90% water but contains over 100 dissolved solutes (Marieb and Hoehn, 2007). Plasma proteins, most of which are produced by the liver, are the most abundant plasma solutes. They perform many functions, but of particular interest to us is their contribution to the blood’s osmotic pressure, which is critical in maintaining the water balance between the blood and the tissues. Other solutes of interest include by-products of cellular respiration, such as urea, uric acid, and creatinine; nutrients absorbed from the digestive tract including glucose, amino acids, fatty acids, cholesterol, and vitamins; electrolytes such as sodium, potassium, calcium, chloride, phosphate, and bicarbonate; the respiratory gases, oxygen, carbon dioxide, and nitrogen; and hormones.

1.1.2. Erythrocytes

Erythrocytes are of particular interest to the aerospace physiologist because of their role in the transport of respiratory gases. RBCs are small, about 7.5 µm in diameter, biconcave disc-shaped cells (Marieb and Hoehn, 2007); that is, they are flattened discs with depressed centers (Figure 1.1.2-1). Although RBCs assume this basic shape, they are highly deformable, allowing them to squeeze through small blood vessels without rupturing. When mature, human RBCs lack a nucleus and other internal organelles. They are essentially bags of hemoglobin, the protein responsible for gas transport and the protein that gives them their red color. Because mature RBCs lack a nucleus and other internal organelles, they are unable to synthesize new proteins for growth, repair, or cell division. As a result they age rapidly. Within 100 to 120 days they become rigid, their hemoglobin begins to degenerate, and they are removed from the circulation. Most of their proteins and other constituents are broken down and recycled. New RBCs are produced by division of stem cells in the bone marrow in a process called erythropoiesis at an incredible rate of more than 2 million per second in healthy individuals. The balance of RBC production and destruction is
closely regulated, as too few RBCs leads to tissue hypoxia (oxygen deprivation) while too many increases blood viscosity, impeding circulation.

![Blood Cells](image)

Figure 1.1.2-1. Blood Cells Seen Through an Electron Microscope

1.1.3. Hemoglobin

Hemoglobin is a tetrameric protein made up of four subunits: two alpha chains and two beta chains. Each subunit contains a heme pigment with a single iron atom. Each iron atom can reversibly bind a single divalent oxygen molecule (O\(_2\)); as a result, each hemoglobin molecule can bind and transport four oxygen molecules (Marieb and Hoehn, 2007).

A single RBC contains about 250 million hemoglobin molecules, so each cell is capable of transporting about 1 billion oxygen molecules. If we consider whole blood, typical hemoglobin concentration is about 34 gm/dL of RBCs (Guyton and Hall, 2000). With a normal hematocrit of 45%, whole blood contains an average of 15 gm of hemoglobin per deciliter (normal adult males have 14 – 16 grams hemoglobin in 100 mL of blood; adult females have 12 – 15 g hemoglobin/100 mL of blood) (Des Jardins, 2013). Each gram of hemoglobin is capable of binding 1.39 mL of oxygen; however, because a small fraction of the iron on the heme sites is in the Fe\(^{3+}\) state (vs. the Fe\(^{2+}\)) and cannot bind O\(_2\), a value of 1.34 mL O\(_2\) is typically used (Hlastala and Berger, 2001). As a result, each deciliter of blood is capable of carrying roughly 20 mL of oxygen in combination with hemoglobin (See also Section 1.2.6).

In addition to its oxygen-binding capability, hemoglobin can also bind and transport carbon dioxide (Marieb and Hoehn, 2007). Carbon dioxide binds to amino acids in the globin portion of the molecule rather than the heme groups. And as is true for most proteins, hemoglobin is an excellent acid-base buffer and is responsible for most of the buffering capacity of whole blood (Guyton and Hall, 2000).

1.1.4. Blood Abnormalities

Anemia is an abnormally low oxygen-carrying capacity of the blood (Marieb and Hoehn, 2007). It is usually a sign of an underlying disorder rather than a disease state in and of itself. Anemia may be due to an insufficient number of RBCs, low hemoglobin content, or abnormal hemoglobin. An abnormally low RBC count can be caused by acute or chronic blood loss (hemorrhagic anemia); excessive RBC destruction (hemolytic anemia) possibly due to hemoglobin abnormalities, certain bacterial or
parasitic infections, and some toxins; or an inhibition of RBC formation in the bone marrow by certain drugs, chemicals, radiation, or viruses. Low content of normal hemoglobin in erythrocytes is typically linked to nutritional deficiencies, such as a lack of iron or vitamin B$_{12}$ in the diet, whereas production of abnormal hemoglobin usually has a genetic link. Sickle-cell anemia, for example, is due to a mutation in the gene that codes for the hemoglobin beta chain, resulting in abnormal RBC morphology and excessive RBC destruction, especially at low oxygen tensions.

In contrast to anemia, polycythemia is an abnormal excess of RBCs resulting in increased blood viscosity. Bone marrow cancer, chronic altitude sickness, and blood doping by athletes can cause abnormal polycythemia. Physiologic polycythemia is a normal response of the body to prolonged hypoxia, such as that experienced by individuals living at high altitude (Rainford and Gradwell, 2006; Guyton and Hall, 2000). Reduced oxygen delivery to the tissues stimulates RBC production as an adaptation to increase the oxygen-carrying capacity of the blood.

1.1.5. The Heart

The heart, about the size of a fist, lies in the mediastinum, the central cavity of the thorax behind the sternum (Marieb and Hoehn, 2007). This hollow, cone-shaped organ weighs less than a pound, yet provides the main propulsive force to circulate blood throughout the body (Figure 1.1.5-1).

The heart has four chambers: two atria and two ventricles. The atria are receiving chambers for blood returning to the heart from the circulation. They are small, thin-walled chambers that do not contribute to the propulsive pumping of the blood. When they contract, they push blood into the ventricles, priming the main pumping chambers. The ventricles are much more massive than the atria and make up most of the volume of the heart; when the ventricles contract, blood is propelled out of the heart into the circulation. The right ventricle pumps blood into the pulmonary circuit while the left ventricle pumps blood into the systemic circuit. The heart can be thought of as two side-by-side pumps, each serving a separate subdivision of the circulation (or circuit). The right side of the heart is the pump for the pulmonary circulation (or circuit), which carries blood to and from the lungs for gas exchange. The left side of the heart propels blood through the systemic circulation (or circuit), which carries blood to and from all body tissues.

Although equal volumes of blood are pumped by the right and left sides of the heart, the work load of the two ventricles is very different. The pulmonary circuit is a short, low-pressure, low-resistance circulation. The systemic circuit is longer, distributing blood through the entire body. As a result, it encounters five times as much resistance to blood flow and operates at a higher pressure. Consequently, the left side of the heart is a far more powerful pump, with walls three times thicker than those of the right ventricle, and is capable of generating five times greater work output (Guyton and Hall, 2000). The heart is dependent on oxidative metabolism of fatty acids and, to a lesser extent, other nutrients such as lactate and glucose to provide the chemical energy to power the cardiac muscle (Guyton and Hall, 2000).
1.1.6. Pattern of Circulation

Blood returning to the heart from the body enters the right atrium and passes into the right ventricle. The right ventricle pumps the blood to the lungs via the pulmonary arteries. In the lungs, the blood off-loads carbon dioxide and picks up oxygen, then returns to the left atrium via the pulmonary veins. Blood passes from the left atrium into the left ventricle, which pumps it into the aorta. The aorta branches into smaller systemic arteries, which distribute blood to all body tissues where gasses, nutrients, and waste products are exchanged. The blood, once again depleted of oxygen and loaded with carbon dioxide, returns through the systemic veins to the right side of the heart, entering the right atrium via the superior and inferior venae cavae (Figure 1.1.6-1).
1.1.7. Heart Valves

This one-way flow of blood from atria to ventricles and out the great arteries is enforced by four valves that open and close in response to differences in blood pressure on their two sides. The atria and ventricles are separated by atrioventricular (AV) valves, which prevent backflow of blood into the atria when the ventricles contract (indicated as the tricuspid valve and mitral valve in Figure 1.1.5-1).

When the heart is relaxed, the AV valve flaps hang limply into the ventricles, which allows blood to flow from the atria, filling the ventricles. When the ventricles contract, the intraventricular pressure increases, which forces blood against the valve flaps, causing them to close. The flaps of the valves are supported by collagen cords, the cordae tendineae, which anchor the flaps in the closed position and prevent them from being inverted upward into the atria. The aortic and pulmonary semilunar (SL) valves prevent backflow from the aorta and pulmonary artery trunk into the respective
ventricles. In the case of the SL valves, when the ventricles contract, the intraventricular pressure rises above the pressure in the aorta and pulmonary arteries, which forces the cusps of the SL valves to flatten against the walls of the arteries, allowing blood to flow by. When the ventricles relax, blood starts to flow backward toward the ventricles, filling the cusps and closing the valves. There are no valves between the venae cavae and pulmonary vein and the right and left atria. When the atria contract, there is some backflow into these vessels; however, the amount is minimal because as the atria contract they compress and collapse the venous entry points.

1.1.8. Heart Sounds

When you listen to someone’s heart, the typical lub-dub-pause heart sounds are caused by closing of the valves during the cardiac cycle. The first sound (lub) is caused when the AV valves close, indicating the point when ventricular pressure exceeds atrial pressure. The second sound (dub), is heard when the SL valves snap shut at the beginning of ventricular relaxation, preventing backflow from the aorta and pulmonary artery trunk. The pause is the period between ventricular contractions when the heart is refilling with blood. Abnormal heart sounds, known as heart murmurs, can be indicative of valve problems. An incompetent valve, one that does not close properly, produces a swishing sound as blood backflows through the partially closed valve. A stenotic valve, one that has become stiff and does not open completely, produces a high-pitched sound or click. Like any pump, the heart can function with defective valves so long as the impairment is not too great. An incompetent valve forces the heart to pump the same blood over and over due to the backflow, while valve stenosis forces the heart to contract more forcefully to propel blood through the narrowed opening. Both problems increase the heart’s workload and can be corrected through surgical repair or replacement of the defective valve.

1.1.9. The Cardiac Cycle

The cardiac cycle includes all of the events associated with blood flow through the heart as it alternates between periods of contraction (systole) and relaxation (diastole) and is marked by a series of pressure and blood volume changes in the heart.

If we start when the heart is relaxed, we can trace these changes through a single heart beat. During diastole, pressure in the heart is low; the SL valves are closed, preventing backflow from the aorta and pulmonary arteries, and blood flows passively through the atria and open AV valves into the ventricles (Figure 1.1.9-1).
Figure 1.1.9-1. Pattern of Blood Flow through the Heart

About 80% of ventricular filling occurs during this phase. At this point the atria contract (atrial systole) compressing the blood in their chambers, increasing atrial pressure, and propelling blood out of the atria into the ventricles. This injection of blood from the atria accounts for the remaining 20% of ventricular filling and brings the ventricles to the maximum volume of blood they will contain during the cycle. This volume is known as the end diastolic volume. After priming the ventricles, the atria relax and the ventricles begin to contract. Pressure in the ventricles rises sharply, closing the AV valves. For a split second the ventricles are completely closed chambers, with both the AV and SL valves shut tight. This is known as the isovolumetric contraction phase. Once pressure in the ventricles exceeds the pressure in the aorta and pulmonary artery trunk, the SL valves open and blood is expelled from the ventricles in the ventricular ejection phase. At the end of ventricular contraction, a small quantity of blood remains in the ventricles and is referred to as the end systolic volume. As the ventricles relax, intraventricular pressure drops rapidly and blood backflows from the great arteries toward the heart, closing the SL valves. During ventricular systole, the atria have been in diastole, filling with blood, and atrial pressure has been rising. Once atrial pressure exceeds ventricular pressure, the AV valves open and the ventricles begin to refill, completing the cycle. With a heart rate of 75 beats/minute, the cardiac cycle is about 0.8 second long, with atrial systole lasting 0.1 second, ventricular systole 0.3 second, and all chambers relaxed during the remaining 0.4 second. It is important to note that blood flow through the heart is controlled entirely by pressure changes and that blood flows down a pressure gradient through any available opening. The pressure changes reflect the alternating contraction and relaxation of the heart muscle and cause the heart valves to open and close, which keeps blood flowing in the proper direction. The right and left sides of the heart display a synchronous pattern of systole and diastole during the cardiac cycle.
Both sides eject the same volume of blood with each heart beat, but the right side of the heart operates at a lower pressure than the left. Typical pulmonary artery systolic and diastolic pressures are 24 mmHg and 8 mmHg compared to aortic pressures of 120 mmHg and 80 mmHg.

1.1.10. Cardiac Output

Cardiac output (CO) is the volume of blood pumped by each ventricle in 1 minute. It is the product of heart rate (HR) and stroke volume (SV), where SV is the volume of blood ejected by each ventricle with each contraction. Using normal resting values, average adult cardiac output can be calculated as:

\[
CO = HR \times SV
\]

\[
CO = \frac{75 \text{ beats}}{\text{min}} \times \frac{70 \text{ ml}}{\text{beat}} = 5,250 \text{ mL/min}
\]

\[
CO = 5.25 \text{ L/min}
\]

Normal adult blood volume is about 5 L; therefore, the entire blood volume passes through both circuits of the cardiovascular system each minute when at rest. Because cardiac output is directly proportional to HR and SV, an increase in either the number of beats per minute or the amount of blood ejected with each beat will increase CO. CO is highly variable and responsive to demands such as exercise and exposure to altitude. The difference between resting and maximal CO is called the cardiac reserve. In non-athletes, cardiac reserve is typically four to five times resting CO (20 – 25 L/min), but elite athletes may reach cardiac outputs of 35 L/min.

1.1.11. Stroke Volume

Stroke volume is the difference between end diastolic volume (EDV) and end systolic volume (ESV). At rest, normal EDV is 110 – 120 mL (Guyton and Hall, 2000). At the end of ventricular contraction, there is normally 40 – 50 mL of blood remaining in the ventricle, giving a stroke volume of 70 mL. The fraction of the EDV that is ejected into the vasculature is called the ejection fraction, and is normally about 60%. Three primary factors influence these values: 1) preload, which affects EDV, 2) contractility, and 3) after-load. Contractility and after-load both affect ESV (Marieb and Hoehn, 2007). Preload (the tension on the heart muscle [Guyton and Hall, 2000] or the degree of stretching of the muscle cells just before contraction) is the most critical factor determining stroke volume (Marieb and Hoehn, 2007). Cardiac muscle displays a length-tension relationship, so stretching of the cardiac muscle results in an increase in contractile force. As venous return (the amount of blood returning to the heart) increases (such as during exercise), the EDV and end diastolic pressure increase, stretching the heart muscle. As a result, contractile force is increased and allows the heart to eject a greater volume of blood into the arteries, thereby increasing SV. This ability of the heart to adapt to changing volumes of inflowing blood is called the Frank-Starling mechanism (Guyton and Hall, 2000). Within physiological limits, it allows the heart to pump all of the blood that comes to it. It also balances the cardiac output of the right and left sides of the heart. Because the pulmonary and systemic circuits are in
series, if one side of the heart begins to pump more blood than the other, the increased venous return to the opposite side forces it to increase its SV and maintain an equal CO (Marieb and Hoehn, 2007).

Contractility is the contractile force generated by the heart muscle at a given muscle cell length and is independent of muscle stretch and EDV (Marieb and Hoehn, 2007). An increase in contractility results in ejection of more blood from the ventricles, increasing ejection fraction and SV by decreasing ESV to as little as 10 – 20 mL. Contractility of the heart muscle is increased by sympathetic nervous system stimulation, increased extracellular Ca\(^{2+}\), and hormones such as glucagon, thyroxin, and epinephrine. It is decreased by acidosis, elevated extracellular K\(^{+}\), and calcium channel blocker drugs. By increasing EDV through increased venous return, and decreasing ESV through increased contractility, SV can be as much as doubled (Guyton and Hall, 2000).

Finally, cardiac afterload, the load the heart muscle must contract against, essentially the pressure in the artery leading away from the ventricle, is not a major determinant of SV in healthy individuals (Marieb and Hoehn, 2007). However, hypertension reduces the ability of the ventricles to eject blood into the arteries, increasing ESV and lowering SV. Left ventricular CO is not significantly affected until mean aortic pressure exceeds 160 mmHg (Guyton and Hall, 2000).

1.1.12. Heart Rate

Heart rate is the other factor determining CO. The basic rhythmic contraction of the heart is established by an internal electrogenic system that generates rhythmical impulses to cause rhythmical contraction of the heart muscle (Guyton and Hall, 2000). Some cardiac fibers are self-excitatory, producing automatic rhythmical action potentials. The portion of this system that displays the greatest self-excitation is the fibers of the sinus node (also known as the sinoatrial or SA node) located in the wall of the right atrium. The sinus node generates impulses about 75 times per minute. Because this rate is faster than any other part of the internal electrogenic system of the myocardium, the sinus node sets the pace, or sinus rhythm, for the heart as a whole (Marieb and Hoehn, 2007). Sinus rhythm varies with age, gender, physical fitness, and body temperature. In addition, several factors influence HR, including hormone levels and extracellular ion balance, but the most important factor is autonomic neural input. Sympathetic stimulation increases HR and contractility, while parasympathetic stimulation decreases HR and has a small inhibitory effect on contractility. The heart receives continuous tonic inputs from both the sympathetic and parasympathetic systems, and it is the relative strength and balance of inputs that determine if sinus rhythm is increased or decreased. Strong sympathetic stimulation can increase HR and contractility enough to increase CO two- to threefold, while strong parasympathetic stimulation can actually stop the heart for several seconds then keep the HR depressed to about 40% of normal (Guyton and Hall, 2000).

Exercise increases HR primarily through sympathetic neural stimulation; however, an abnormally fast HR (over 100 beats/min), known as tachycardia, can be caused by high body temperature, stress, drugs, or heart disease (Marieb and Hoehn, 2007). Bradycardia, or an HR below 60 beats/min, can be caused by low body temperature, drugs, or parasympathetic neural stimulation; however, it is also a desirable consequence of endurance training. Cardiovascular conditioning causes hypertrophy of the heart, which increases SV, allowing resting HR to be lower and still
provide sufficient CO. It is not uncommon for well-conditioned athletes to have a resting HR as low as 40 beats/min.

1.1.13. The Vasculature

The blood vessels form a closed system that begins and ends at the heart, together composing the vasculature. Arteries conduct blood away from the heart, while veins return blood toward the heart. The arterial and venous sides of the circulation are connected by capillaries, where exchange of fluids, nutrients, gases, hormones, ions, and other substances occurs between the blood and the interstitial fluid (Guyton and Hall, 2000). The walls of all blood vessels, except for capillaries and the smallest arterioles, have three distinct layers (Marieb and Hoehn, 2007). The inner blood-conducting lumen of the vessel is lined by the endothelium, a layer of flat, closely fitting cells that provide a smooth inner surface to the vessel. Outside of the endothelium is a layer of smooth muscle and elastin (Figure 1.1.13-1). Contraction of the smooth muscle results in a reduction in lumen diameter (vasoconstriction), while relaxation allows expansion of the lumen (vasodilation). Small changes in lumen diameter have a large effect on blood flow. As a result, they are critical to blood distribution and regulation of blood pressure and are influenced by sympathetic neural regulation, hormones, and local tissue conditions. Elastin is a protein that can stretch and recoil, giving the vessel elastic properties. The relative abundance of these components primarily determines the characteristics of the vessel. The outer layer of the vessel wall is composed largely of collagen fibers that protect and anchor the vessel to surrounding structures.

![Figure 1.1.13-1. Vascular Anatomy](image)

1.1.14. Arteries

Large arteries, such as the aorta and its major branches, are thick walled with high elastin content and relatively little smooth muscle (Marieb and Hoehn, 2007). They receive blood from the heart at high pressure, but their large diameters make them low-resistance vessels. The high elastin content allows these vessels to serve as pressure reservoirs. When the ventricle ejects blood into the aorta, it stretches then recoils while the heart refills. This smooths both the pressure pulse and blood flow. As the aorta stretches, it dampens the ejection pressure and then maintains blood flow.
as it recoils during systole. Downstream the elastic arteries branch into smaller, more muscular distributing arteries. These have relatively more smooth muscle and less elastin and are therefore more active in vasoconstriction. Arterioles, the smallest arteries, have primarily smooth muscle in their middle layer with few elastin fibers. The very smallest arterioles immediately adjacent to the capillaries may be little more than a single layer of smooth muscle cells wrapped around the endothelium. Vasoconstriction and dilation of the arterioles control blood flow into individual capillary beds and, therefore, perfusion of specific tissues (Figure 1.1.15-1).

1.1.15. Capillaries

Capillaries are the smallest blood vessels, with an average length of 0.3 – 1 mm and a diameter of 8 – 10 µm, just large enough for RBCs to pass through single file (Guyton and Hall, 2000; Marieb and Hoehn, 2007). Capillaries are constructed of a single endothelial layer that has intercellular clefts, or pores, that allow movement of fluid and small molecules across the capillary wall. In the brain, these pores are absent, the endothelial cells forming continuous tight junctions. This is the structural basis of the blood brain barrier. Lipid soluble molecules, such as oxygen and carbon dioxide, can diffuse directly through the endothelial membranes without having to go through the pores, providing a much larger surface area for exchange of these molecules than for ions and other molecules that must pass through the openings between cells. Blood pressure within the capillaries tends to force water and dissolved substances out of the capillary into the interstitial space. Colloidal osmotic pressure, due to blood cells and plasma proteins that are too large to pass through the pores, causes osmotic fluid movement back into the capillary. The balance between these forces usually prevents excessive loss of fluid from the blood; however, a small net movement of fluid into the interstitium is normally observed, with the fluid being returned to the circulation via the lymphatic system. If capillary pressure increases, as, for example, due to gravitational hydrostatic pressure as discussed below, the balance can be disrupted with rapid loss of fluid from the blood and formation of tissue edema (Guyton and Hall, 2000).

Capillaries tend to form interconnected networks of 10 – 100 capillaries called capillary beds. Blood flow through the capillary bed is called the microcirculation and is regulated by vasoconstriction of the terminal arteriole and a bundle of smooth muscle fibers at the root of each capillary called the precapillary sphincter. When the precapillary sphincters are relaxed, blood flows through the capillaries and exchange occurs between the blood and the tissue cells. When the sphincters are constricted, blood bypasses the tissue cells (Figure 1.1.15-1). Blood flow through any particular capillary bed is intermittent and is regulated by various hormones, including vasoconstrictors such as norepinephrine, angiotensin, and vasopressin, and vasodilators, including bradykinin and histamine, as well as local tissue chemical conditions, including the concentrations of oxygen, carbon dioxide, and adenosine. In this way, blood flow is closely matched to the metabolic demands of the tissue (Guyton and Hall, 2000). Under conditions of hypoxemia, reduced oxygen content of arterial blood, whether due to altitude exposure, pneumonia, anemia, or carbon monoxide poisoning, is marked by an increase in tissue blood flow in an effort to maintain normal tissue oxygen levels and make up for the decreased amount of oxygen in the blood.
1.1.15 - Precapillary Control of Blood Flow

Veins

The capillaries empty into venules. The smallest venules are structurally little more than large capillaries and are very leaky. Fluids and white blood cells freely move across their walls. As the venules merge and become larger, their walls acquire smooth muscle and collagen layers and become less porous. Venules continue to converge, eventually forming veins with all three layers of typical blood vessels. Veins of any given size have thinner walls with less smooth muscle and elastin and larger lumens than arteries of comparable diameter. The veins have a large potential volume, though they are normally only partially filled, and are called capacitance vessels, serving as blood reservoirs. Normally more than 60% of all the blood in the circulatory system is in the veins (Guyton and Hall, 2000). If blood is lost from the body, sympathetic stimulation of the veins causes venous vasoconstriction, which reduces venous reservoir volume, making up for as much as a 20% loss of total blood volume.

The large lumen of veins offers little resistance to blood flow, facilitating the movement of blood back toward the heart in the low-pressure venous circulation. Another adaptation that facilitates unidirectional flow toward the heart in this low-pressure circuit is venous valves that prevent backflow of blood. Venous valves are formed from folds of the endothelial layer and are similar in structure to the semilunar valves of the heart (Figure 1.1.16-1).

Figure 1.1.15-1. Precapillary Control of Blood Flow
Valves are most prominent in the veins of the limbs where blood must flow upward against the pull of gravity. You can demonstrate the effect of these valves by collapsing a vein on the back of your hand or in your wrist. Let your hand hang down so the veins become engorged with blood. Place a finger across a vein on the back of your hand or on the inside of your wrist, then run another finger or your thumb up the vein toward your heart. The vein will stay collapsed until you remove your finger because the valves prevent blood from flowing backward into the collapsed vein.

1.1.17. Blood Flow

Blood flow through any part of the circulation is described by Ohm’s Law (Guyton and Hall, 2000):

\[ \dot{Q} = \frac{\Delta P}{R} \]

where:
- \( \dot{Q} \) = blood flow (mL/min)
- \( \Delta P \) = pressure difference from one end of a vessel to the other (mmHg)
- \( R \) = resistance

Blood flow is simply the volume of blood flowing through a vessel, tissue, organ, or the entire circulation in a given time (mL/min). For the entire circulation, flow is equivalent to cardiac output. Blood pressure is the force exerted by the blood against the walls of the vessel and is usually expressed in mmHg. It is the difference in blood pressure, the pressure gradient from one end of the vessel to the other, that provides the driving force for blood flow. Resistance is the impediment to blood flow. It cannot be measured directly but is estimated by measuring the blood flow and pressure difference between two points in a vessel. Vascular resistance is often expressed in peripheral resistance units (1 PRU = a flow of 1 mL/s with a 1 mmHg pressure gradient). The total resistance of the systemic circulation is about 1 PRU. Because the pulmonary circuit operates at a lower pressure but has the same flow as the systemic circuit, its total resistance is calculated to be about 0.14 PRU. Conductance, the blood flow through a vessel for a given pressure difference, is the reciprocal of resistance. Slight changes in a vessel’s diameter have a large effect on its conductance. This is due to the fact that in accordance with Poiseuille’s Law, conductance is proportional to
the fourth power of the diameter of the vessel (Guyton and Hall, 2000). Therefore, a fourfold increase in the diameter of a vessel decreases resistance and increases flow through the vessel 256-fold if ΔP is maintained.

Systemic arterial blood pressure is pulsatile due to the rhythmic ejection of blood into the aorta by the left ventricle. Systolic pressure \((P_{systolic})\), the peak pressure achieved during ventricular contraction, is about 120 mmHg at the level of the heart in healthy adults (Marieb and Hoehn, 2007). Diastolic pressure \((P_{diastolic})\), the lowest pressure reached during ventricular relaxation, is about 80 mmHg. The difference between systolic and diastolic pressures is called the pulse pressure. Mean arterial pressure (MAP) is the average pressure in the vessel throughout the cardiac cycle and the driving force that propels blood through the vasculature. At high heart rates, MAP is closer to the arithmetic average of systolic and diastolic pressure (i.e. 100 mmHg). MAP is approximated by the following equation:

\[
MAP = P_{diastolic} + \frac{1}{3} \left( P_{systolic} - P_{diastolic} \right)
\]

Under normal conditions then,

\[
MAP = 80 \text{ mmHg} + \frac{1}{3} (120 \text{ mmHg} - 80 \text{ mmHg}) = 93 \text{ mmHg}
\]

Both MAP and pulse pressure decrease with increasing distance from the heart (Figure 1.1.17-1). The greatest drop in MAP and pulse pressure occurs in the arterioles, so that by the time blood reaches the terminal arterioles, MAP is about 35 mmHg and blood flow is steady with pulse pressure equal to zero. This is due to the high resistance of the small-diameter arterioles and their inelasticity. Pressure at the arterial end of a systemic capillary is about 35 mmHg but drops to 10 – 15 mmHg at the venous end, with an average functional pressure across the capillary bed of about 17 mmHg (Guyton and Hall, 2000). Low capillary pressures are essential due to their fragile structure and porous nature.

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**Figure 1.1.17-1.** Drop in Blood Pressure across Vasculature
Blood pressure in the venous circulation is nonpulsatile, and the pressure gradient between the venules that receive blood from the capillary beds (10 – 15 mmHg) and the vena cava where it empties into the right atrium, called central venous pressure (0 mmHg), is too small to propel blood back to the heart despite the low resistance to flow in veins and valves to prevent backflow. Three systems help facilitate venous return (Marieb and Hoehn, 2007). The respiratory pump is due to pressure changes during ventilation. When we inhale, pressure in the abdomen increases, squeezing veins within the abdomen and propelling blood toward the heart. During exhalation, abdominal pressure decreases, allowing the veins to expand and refill with blood. Chest pressure follows an opposite pattern, decreasing during inhalation, allowing thoracic veins to expand, and increasing during exhalation. The muscular pump results from contraction and relaxation of skeletal muscles surrounding deep veins of the limbs. As the muscles contract, they compress the veins, propelling blood toward the heart (Figure 1.1.16-1). The final mechanism is contraction of smooth muscle in the walls of the veins in response to sympathetic neural stimulation.

The pressures described above are referenced to the level of the heart and are a close approximation throughout the body for an individual who is lying down. But in an upright person, gravity has a large effect on blood pressure throughout the vasculature.Gravitational or hydrostatic pressure occurs in the vascular system due to the weight of the blood (Guyton and Hall, 2000). In the upright person, pressures at the level of the heart remain as described above with MAP equal to 100 mmHg and right atrial pressure equal to 0 mmHg, but the addition of hydrostatic pressure causes MAP in the feet to rise to about 190 mmHg and venous pressure to 90 mmHg. Pressures at other levels of the body below the heart lie proportionately between these extremes, and pressures in regions above the heart will be proportionately decreased. For example, venous pressure in the hand rises to about 35 mmHg, while pressure in the veins of the neck drops to zero, at which time they are collapsed by atmospheric pressure. Collapse prevents creation of negative pressure in the veins of the neck, but within the skull, which forms a noncollapsible chamber, negative pressures can exist within the dural sinuses. High venous pressures in the lower extremities make functioning of the venous valves and muscular pump very important. The efficiency of these adaptations allows the venous pressure in the feet of a walking adult to remain below 25 mmHg, but in their absence, that is for an individual standing still, venous pressure rises to 90 mmHg within 30 seconds. As venous pressure rises, capillary pressure also rises, and transduction of fluid out of the capillaries into the tissues increases. This movement of fluid out of the vasculature causes the legs to swell and blood volume to decrease. Indeed, 10% - 20% of blood volume can be lost from the circulation during 15 minutes of standing at attention. These hydrostatic effects are exaggerated in hyper-G environments and essentially disappear in the microgravity of space flight.

Arterial blood pressure is closely and rapidly regulated. Pressure varies directly with CO, vascular resistance, and blood volume (Marieb and Hoehn, 2007). A change in any of these variables is quickly compensated by the others to maintain pressure homeostasis. For example, if blood pressure should start to fall due to hemorrhage, baroreceptors (pressure receptors) in the carotid sinuses of the internal carotid arteries, which provide the major blood supply to the brain, and in the aortic arch detect the pressure drop, sending signals to the central nervous system (CNS), stimulating increased sympathetic and decreased parasympathetic neural output. Sympathetic stimulation causes system-wide vasoconstriction, increasing arteriolar resistance. This increased resistance elevates arterial pressure but also decreases downstream
capillary pressure, allowing a net flow of fluid into the capillaries from the interstitial fluid and adding to blood volume (Rainford and Gradwell, 2006). Venous vasoconstriction moves blood out of the venous reservoir, increasing CO. Sympathetic nervous stimulation also increases both heart rate and contractility, increasing stroke volume and further increasing CO. All of these responses work together to maintain MAP in the face of decreased blood volume (Guyton and Hall, 2000). In the face of elevated blood pressure, an opposite response is seen, with an inhibition of sympathetic and stimulation of parasympathetic neural output, resulting in generalized vasodilation with decreased resistance, a shifting of blood into the venous reservoir, and a decrease in heart rate and contractility, all contributing to a lowering of MAP. Neural regulation of blood pressure is very rapid and compensates for the changes that occur during daily activities. For example, when you stand up after lounging on the couch, blood pressure above your heart drops rapidly. The pressure drop is detected by the carotid baroreceptors, stimulating a strong sympathetic discharge that minimizes the decrease in blood pressure and preserves brain perfusion. Longer term regulation of blood pressure is mediated through hormones that control blood volume via the kidneys. This close regulation of blood pressure during a wide variety of physiological and psychological stressors helps to ensure perfusion of body tissues.

1.1.18. Summary

This chapter attempts to select and summarize key concepts of cardiovascular physiology primarily from three respected physiology texts that are of particular importance and interest to the aerospace physiologist. For further study of fundamental cardiovascular physiology, I refer you to either Guyton and Hall (2000) or Marieb and Hoehn (2007) for a detailed treatment of any of the topics touched on in this chapter and many more. For a detailed examination of the cardiovascular consequences of the stressors associated with the aerospace environment, including exercise, hypoxia, anti-G straining maneuver, and positive pressure breathing, I refer you to Rainford and Gradwell (2006).

References

Concepts
Cardiac cycle, phases and functions
Heart valves, function
Heart, structure and function
Hemoglobin, structure and function
Transport vehicle of the cardiovascular system
Vocabulary
Arterial and venous
Arteries
Atria and ventricles
Blood pressure
Capillaries
Cardiac cycle
Cardiac output
Heart rate
Hemoglobin
mmHg
Plasma
Pulmonary circulation
Red blood cells, erythrocytes
Systemic circulation
Systolic and diastolic
Veins
1.2. Respiratory Physiology

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1.2.1. Function

The primary function of the respiratory system is the uptake of oxygen (O\textsubscript{2}) and the removal of carbon dioxide (CO\textsubscript{2}). Oxygen serves as the terminal electron acceptor at the end of the electron transport chain in the mitochondria and as such is essential to aerobic energy production, which accounts for more than 95\% of the body’s energy expenditure (Guyton and Hall, 2000).

Once oxygen picks up two electrons it combines with two H\textsuperscript{+} ions, forming water. This metabolic water can account for up to 10\% of the body’s daily water requirement. Carbon dioxide is produced during the breakdown of nutrients (carbohydrates, proteins, and lipids) in the citric acid or Krebs cycle. This CO\textsubscript{2}, produced in the mitochondria, diffuses out of the cells into the blood and is ultimately expired through the lungs (Guyton and Hall, 2000). The rate of O\textsubscript{2} consumption and CO\textsubscript{2} production are closely tied to, and regulated by, the energy requirements of the cells, tissues, organs, and ultimately the entire body.

The process of providing oxygen to the cells for energy production and removal of carbon dioxide can be divided into a series of functional events (also known as the phases of respiration):

1. **Pulmonary ventilation.** The movement of air into and out of the lungs.

2. **Diffusion.** Movement of gases between the lungs and the blood, sometimes called *external respiration*, down their respective pressure gradient.

3. **Transport.** Movement of the respiratory gases between the lungs and the tissues. A function of the cardiovascular system with blood as the transport medium.

4. **Diffusion.** Movement of gases between the blood, the interstitial fluid, and the cells, sometimes called *internal respiration*, down their respective pressure gradient.

5. **Utilization.** The consumption of O\textsubscript{2} and production of CO\textsubscript{2} in the cell, known as *cellular respiration* (Marieb and Hoehn, 2007).

Only the first two events are a special function of the respiratory system, but if the cells are to be provided with O\textsubscript{2} and have their CO\textsubscript{2} waste product removed, the respiratory and circulatory systems must work together and be closely regulated.
1.2.2. Anatomy of the Respiratory System

Air enters the conducting passages of the respiratory system via the mouth and nose. The nose does a particularly good job of conditioning (filter, warming, and humidifying) the inspired air. The hairs of the nose remove large particles, while conchae, vane-like projections within the nasal passages, are important to filtration of smaller particles through a process known as turbulent precipitation. As air flows through the convoluted passageways created by the conchae, it is forced to repeatedly change direction. The momentum of heavier particles, such as dust, pollen, and bacteria, causes them to impact the conchae and become entrapped in mucus coating the walls. Turbulent precipitation is so effective that virtually no particles larger than 6 µm that enter the nose make it to the lungs (Guyton and Hall, 2000). Ciliated cells of the mucosa lining the nasal passages sweep the contaminated mucus toward the throat where it is swallowed or coughed and expelled. In addition to cleansing the air, the large, highly vascularized surface area of the nasal passages also warms and humidifies the inspired air to within 1°F of body temperature and to 97% - 98% saturation with water vapor. The paranasal sinuses in the frontal, sphenoid, ethmoid, and maxillary bones lighten the skull and contribute to warming and humidifying inspired air (Marieb and Hoehn, 2007) (see also Figure 3.3.2.3-1). If the nasal mucosa becomes infected by viruses or bacteria, or is irritated by allergens, the mucosa can become inflamed with excessive production of mucus. Because the nasal mucosa is continuous with the rest of the respiratory tract, infection often progresses from nose to throat to chest. Spread of infection into the paranasal sinuses can result in sinusitis, with mucus blocking the passages connecting the sinuses to the nasal cavity, resulting in sinus headaches and difficulty in equalizing pressure between the sinuses and the ambient environment during altitude changes.

The nasal and oral cavities merge at the throat, or pharynx (Marieb and Hoehn, 2007). The pharynx serves as a passageway for both air and swallowed food. The eustachian tubes (also known as the pharyngotympanic or auditory tubes), which allow pressure to equalize between the middle ear and the atmosphere, open into the upper or nasopharynx. The tubal tonsil arches over the opening of the eustachian tube and helps prevent the spread of infection from the nasopharynx into the middle ear (see also Figure 3.3.2.2-2). The pharynx extends for about 13 cm to the larynx, or voice box, which is visible externally as the Adam’s apple. The larynx is responsible for routing air and food into the proper channels. When only air is flowing through the larynx, the inlet to the larynx is open and the epiglottis, a cartilage projection at the top of the larynx, is oriented upward. During swallowing, the larynx is pulled upward and the epiglottis tips to cover the laryngeal opening, directing food into the esophagus and preventing it from entering the lower respiratory passages. If anything other than air enters the larynx, a strong cough reflex is stimulated, expelling the substance. The larynx also contains the vocal chords and is involved in voice production. The true vocal chords, or vocal folds, and the opening between them through which air passes, are called the glottis. The glottis can act as a sphincter, preventing air passage. During the anti-G straining maneuver (AGSM), the glottis is closed to prevent exhalation while abdominal and thoracic muscles contract, increasing intrathoracic pressure.

The trachea, or windpipe, descends from the larynx until it divides into the two main bronchi at mid-chest level (Figure 1.2.2-1). The trachea is quite flexible but has C-shaped rings of cartilage that keep it from collapsing during the pressure changes
associated with breathing. The main bronchi extend to the lung on their respective sides. Once inside the lung the bronchi divide into lobar or secondary bronchi, two on the left and three on the right, each supplying one lung lobe. The lobar bronchi then undergo repeated branching for up to 23 generations. Branches less than 1 mm in diameter are called bronchioles, with the smallest terminal bronchioles being less than 0.5 mm in diameter. This repeated dichotomous branching pattern of the conducting respiratory pathways is called the bronchial or respiratory tree (Marieb and Hoehn, 2007). Ciliated and mucus producing cells are found in the lining of the larger bronchi but are absent in the bronchioles. Ciliated cells in the bronchi, trachea, and larynx sweep mucus upward toward the pharynx, where it is swallowed. Contaminants that reach the bronchioles are removed primarily by macrophages, a type of white blood cell. As successive branches become smaller, the amount of cartilage in the walls of the respiratory tree decreases and the amount of smooth muscle increases. The bronchioles completely lack cartilage support and have a complete layer of smooth muscle, which can alter their diameter.

**Figure 1.2.2-1. Respiratory System Anatomy**

All of the structures described above are referred to as conducting zone structures, as they function to conduct air to and from the site of gas exchange. Respiratory zone structures are characterized by the presence of gas-filled sacks called alveoli, the actual site of gas exchange within the lungs (Marieb and Hoehn, 2007). The respiratory zone structures include respiratory bronchioles, the smallest bronchioles that have scattered alveoli protruding from them; alveolar ducts, winding passages with out-pocketing alveoli that lead to terminal clusters of alveoli, which resemble a bunch of grapes; and the alveoli themselves (Figures 1.2.2-1 and 1.2.2-2). Alveoli, the functional unit of the lung, are composed of a single layer of epithelial cells surrounded by a thin basement membrane. Each is covered by a meshwork of pulmonary capillaries, creating a respiratory membrane with gas on one side and blood flowing past on the other. There are over 300 million alveoli in the lungs. They make
up most of the lung volume and provide a tremendous surface area for gas exchange. It is estimated that the total surface area of the respiratory membrane is 750 ft$^2$ (70 m$^2$), the area of a 25 ft x 30 ft room.

The lungs occupy almost the entire chest cavity, extending from the collar bones to the diaphragm, with the front, side, and back surfaces in close contact with the ribs. The lungs are slightly different sizes and shapes to accommodate the heart, with the left lung being the smaller. The pulmonary blood vessels enter and exit the lungs at the hilum, where the main bronchi also enter. Each lung is enclosed by a thin membrane called the visceral pleura (Figure 1.2.2-3). A second membrane, the parietal pleura, lines the inner surface of the chest wall and the top of the diaphragm and extends between the lungs and around the heart. The space between the membranes, the pleural cavity, is filled with pleural fluid, which is secreted by the membranes. The fluid provides lubrication so the lungs can glide easily over the chest wall and creates surface tension, which prevents the pleural membranes from separating, forcing the lungs to expand and contract with movements of the chest wall during breathing.
1.2.3.  Ventilation of the Lungs

When the glottis is open and there is no air moving into or out of the lungs, the pressure throughout the respiratory tree, including the alveoli, is equal to atmospheric pressure (Guyton and Hall, 2000). This is considered the zero reference pressure of the airways. During inspiration, the inspiratory muscles contract, expanding the volume of the thoracic cavity. The diaphragm contracts, pulling the lungs downward and lengthening the thoracic cavity (Figure 1.2.3-1).

![Figure 1.2.3-1. Breathing Movements](image)

The external intercostal muscles between the ribs contract, pulling the ribs upward and outward and increasing the diameter of the chest. In accordance with Boyle’s Law, as the volume of the chest increases the intra-thoracic pressure decreases, creating a slight negative pressure in the alveoli of approximately -1 cm H₂O. This pressure difference is sufficient to pull 500 mL of air into the lungs during the 2 seconds of normal quiet inspiration. During forced inspiration, in response to exercise, obstructive lung disease, or resistance from oxygen equipment, the diaphragm and external intercostal muscles contract more vigorously and accessory muscles that aid in expansion of the rib cage are recruited, resulting in greater expansion of the thoracic cavity and creation of a greater negative plural pressure, resulting in an increased rate of flow and volume of air entering the lungs.

Expiration during quiet breathing is a passive process due to the elastic recoil of the lungs and chest cage structures. As the inspiratory muscles relax, the rib cage descends and the diaphragm recoils upward, decreasing the volume of the thoracic cavity and increasing the plural pressure to approximately +1 cm H₂O. This increased pressure forces 500 mL of air out of the lungs during the 2 to 3 seconds of normal expiration. Under resting conditions, the respiratory muscles only perform work during inspiration and relax during expiration. On the other hand, forced expiration is achieved by contraction of the internal intercostal muscles, which depress the rib cage, and the oblique and transverse abdominal muscles, which compress the abdominal cavity, forcing the abdominal organs upward against the diaphragm, reducing thoracic volume and generating elevated intra-thoracic pressures. This allows rapid and forceful expulsion of air from the lungs during exercise, against the resistance of oxygen equipment, and during positive pressure breathing. During quiet breathing, 3% to 5% of the body’s total energy expenditure is dedicated to pulmonary ventilation, but during
heavy exercise or if breathing resistance is increased, the amount of energy required to ventilate the lungs can increase 50-fold and can become a limiting factor in exercise intensity (Guyton and Hall, 2000).

Replenishing the air in the alveoli where gas exchange can occur between the lungs and the blood is the ultimate purpose of pulmonary ventilation; however, because of the in and out reciprocal pattern of ventilation, not all of the tidal volume contributes to alveolar ventilation (Figure 1.2.3-2). The last air inhaled during inspiration is the first air exhaled during the subsequent exhalation. This approximately 150 mL of last air in/first air out fills the conducting passageways where no gas exchange occurs. This volume is known as the anatomical dead space ($V_D$).

![Figure 1.2.3-2. Pulmonary Ventilation](image)

Alveolar ventilation rate ($\dot{V}_A$) can be calculated as:

$$\dot{V}_A = f \times (V_T - V_D)$$

where:

- $f$ = ventilation frequency (breaths/min) (normal: 12 – 15 bpm)
- $V_T$ = tidal volume (mL/breath)
- $V_D$ = anatomical dead space volume (mL/breath)

For an average adult male breathing quietly:

$$\dot{V}_A = 12 \times (500 - 150) = 4,200 \text{ mL/min}$$

Because anatomical dead space is constant for any given individual (~ 1mL per pound ideal body weight), increasing $V_T$ by breathing more deeply is more effective in increasing $\dot{V}_A$ than increasing $f$; in fact, $\dot{V}_A$ drops rapidly with rapid shallow breathing because a greater percentage of $V_T$ remains in the dead space (Marieb and Hoehn, 2007). Oxygen masks increase functional dead space and reduce $\dot{V}_A$, forcing the aircrew member to increase the depth or frequency of breathing to maintain alveolar ventilation. As a result, minimizing dead space volume is an important consideration in oxygen equipment design (Rainford and Gradwell, 2006).
1.2.4. Alveolar Gas Tensions

The partial pressures of gases in the alveoli are significantly different from those of atmospheric air. Oxygen is continually absorbed from the alveoli into the blood, while at the same time it is constantly being replenished by alveolar ventilation (Guyton and Hall, 2000). Increased oxygen consumption, for example during exercise, reduces alveolar oxygen pressure ($P_{A\text{O}_2}$); conversely, increased alveolar ventilation elevates it. Moderate exercise increases oxygen absorption from the alveoli from 250 to 1,000 mL/min, requiring a four-fold increase in ventilation to maintain normal $P_{A\text{O}_2}$. On the other hand, carbon dioxide is continually being formed by the body and delivered to the lungs for elimination. Increased CO$_2$ production by the tissues will elevate alveolar CO$_2$ partial pressure ($P_{A\text{CO}_2}$) and unlike $P_{A\text{O}_2}$, $P_{A\text{CO}_2}$ is inversely proportional to alveolar ventilation rate. Under normal resting conditions, with a whole body CO$_2$ production rate of about 200 mL/min and alveolar ventilation of 4,200 mL/min, $P_{A\text{CO}_2}$ is 40 mmHg.

Alveolar $P_{A\text{O}_2}$ can be calculated using the alveolar gas equation (Rainford and Gradwell, 2006) (see also Section 2.2.8):

\[
P_{A\text{O}_2} = P_{I\text{O}_2} - \left[ P_{A\text{CO}_2} \times \left( F_{I\text{O}_2} + \left( \frac{1 - F_{I\text{O}_2}}{R} \right) \right) \right]
\]

where:

- $P_{I\text{O}_2}$ = oxygen partial pressure (PO$_2$) of inspired tracheal gas, i.e., gas saturated with water vapor at body temperature (37°C). Water vapor pressure at 37°C is 47 mmHg and is wholly dependent on temperature and, therefore, independent of altitude.

Therefore,

\[
P_{I\text{O}_2} = (P_B - 47) \times F_{I\text{O}_2}
\]

where:

- $P_B$ = barometric pressure
- $P_{A\text{CO}_2}$ = alveolar carbon dioxide partial pressure (40 mmHg under normal conditions)
- $F_{I\text{O}_2}$ = fractional concentration of oxygen in the inspired gas (0.21 for atmospheric air)
- $R$ = respiratory exchange ratio (also called the respiratory quotient, RQ), which is the ratio of CO$_2$ produced/O$_2$ consumed by the tissues (Rainford and Gradwell, 2006).

$R$ is dependent on diet because macronutrients enter the Krebs’ cycle at different points, resulting in different rates of CO$_2$ production. Oxidation of carbohydrates results in an $R = 1.0$; oxidation of fats or protein gives an $R = 0.7$. For a typical mixed diet, an $R = 0.85$ can be assumed. Figure 1.2.4-1 shows the $R$ values for varying oxidation combinations.
Gas exchange occurs between the alveolar gas and the blood by simple diffusion across the respiratory membrane.

The alveoli are covered by an almost solid network of interconnecting capillaries (Guyton and Hall, 2000). The respiratory membrane between the alveolar gas and the blood consists of:

- A layer of fluid lining the inside of the alveolus
- The alveolar epithelium, made up of a single layer of thin cells
• Epithelial basement membranes of the alveoli and the capillaries, which fuse in many places

• The capillary epithelium, again made up of a single layer of cells

Despite these many layers, the respiratory membrane is very thin, averaging only 0.6 µm. The distance gases must diffuse and, therefore, the functional thickness of the respiratory membrane is also reduced by the fact that the pulmonary capillaries average only 5 µm in diameter, forcing the red blood cells to deform and squeeze through the vessels, bringing the RBC membrane in contact with the capillary walls so that gases need not diffuse through a significant layer of plasma.

Fick’s Law of Diffusion explains the relationship between the factors that determine how the volume of a gas that passes through a membrane (see also Section 2.2.6):

\[
\dot{V}_{gas} = \frac{A \times D \times \Delta P}{T}
\]

where:
- \( \dot{V}_{gas} \) = the volume of gas per unit time
- \( A \) = the surface area of the membrane
- \( D \) = the diffusion coefficient of the gas within the membrane
- \( \Delta P \) = the partial pressure difference \( (P_1 - P_2) \) of the gas across the membrane
- \( T \) = the thickness of the membrane

The thickness of the respiratory membrane, although extremely thin, can be increased by accumulation of fluid in the space between the alveolar and capillary basement membranes or inside the alveoli themselves as a result of pneumonia or by diseases such as pulmonary fibrosis. Increasing the thickness of the membrane more than two to three times normal will significantly impair gas exchange. Surface area of the respiratory membrane can be severely decreased by diseases such as emphysema, cancer, or bronchial obstruction. The diffusion coefficient of a gas is a characteristic of the gas itself related to its solubility in the membrane, which is essentially the same as its solubility in water, and the square root of its molecular weight (see also Section 2.2.6). The rate of diffusion of CO\(_2\), for a given pressure difference, is 20 times greater than O\(_2\); therefore, CO\(_2\) moves across the respiratory membrane much faster than O\(_2\). The partial pressure difference across the respiratory membrane is the most important factor for aerospace physiology. The difference in partial pressure of a gas between the alveoli and the pulmonary capillary blood is a measure of the net tendency of the gas molecules to move through the membrane. The net movement of gas will be from an area of high partial pressure to an area of low partial pressure. At sea level, normal \( P_A O_2 \) = 104 mmHg (this value is often rounded to 100 mmHg for convenience) and \( P_A CO_2 \) = 40 mmHg. Oxygen pressure in mixed venous blood \( (PvO_2) \) is typically 40 mmHg, while mixed venous PCO\(_2\) \( (PvCO_2) = 45 \) mmHg. Therefore, the net movement of O\(_2\) across the respiratory membrane is from the alveolar gas to the blood, and the net movement of CO\(_2\) is from the blood into the alveoli.
The rate of gas exchange is very rapid. At rest it takes approximately 0.75 second for blood to transit the pulmonary capillary, yet oxygen tension in the blood is essentially equal to that of the alveolar gas within 0.2 – 0.25 second (Rainford and Gradwell, 2006). Even under heavy exercise when the capillary transit time for a red blood cell is reduced to one-third that at rest, there is still sufficient time for oxygen tensions to equilibrate across the respiratory membrane. At altitude under hypoxic conditions, when $P_{A}O_2$ is reduced, the driving force for the movement of oxygen across the respiratory membrane falls and the rate of transfer is slowed. If $P_{A}O_2$ drops to 40 mmHg, there is still time for equilibration between the alveolar gas and venous blood when at rest, but even moderate exercise will result in the oxygen tension of end capillary blood being below alveolar $PO_2$. Therefore, exercise can exacerbate hypoxia due to the slowed rate of oxygen exchange in the lung.

Another complication of gas exchange in the lung is the ventilation/perfusion ($\dot{V}_A/\dot{Q}$) ratio, that is, the balance between the ventilation of an alveolus and the blood flow through its alveolar capillaries (Figure 1.2.5-1). If the ventilation of a given alveolus is normal, and the flow of blood through the capillaries of that alveolus is also normal, $\dot{V}_A/\dot{Q}$ is said to be normal or equal to 1 (Guyton and Hall, 2000). If an alveolus is not ventilated but still has perfusion, then $\dot{V}_A/\dot{Q}$ equals zero. Under these conditions, the blood will absorb $O_2$ from the alveolar gas and deposit $CO_2$ until the alveolar gas pressures equal those of mixed venous blood: $P_{v}O_2 = 40$ mmHg, $P_{v}CO_2 = 45$ mmHg. This situation is known as a shunt because once gas pressures are equal between the alveolus and the blood, no gas exchange occurs and it is as if the blood did not flow through the lungs. On the other hand, if an alveolus is ventilated but not perfused, $\dot{V}_A/\dot{Q}$ equals infinity, no $O_2$ is removed from the alveolus, and no $CO_2$ is deposited, so the alveolar gas tensions will approach those of humidified air: $PO_2 = 149$ mmHg, $PCO_2 = 0$ mmHg. This situation effectively increases the physiologic dead space; that is, it increases the percentage of tidal volume going to parts of the lung where no gas exchange occurs. Various parts of the lung may experience degrees of $\dot{V}_A/\dot{Q}$ mismatch between these two extremes. One cause of $\dot{V}_A/\dot{Q}$ mismatch is gravity. For a healthy individual in an upright posture, gravity causes a stretching or distension of the upper part of the lung and a compression of the lower lung, resulting in greater ventilation of the lower alveoli compared to the upper alveoli, while at the same time blood flow through portions of the lung above the heart is reduced and flow below the heart is increased. Although both ventilation and perfusion increase from the upper to the lower lung, the difference in blood flow is greater than the difference in ventilation, so $\dot{V}_A/\dot{Q}$ may be as high as 2.5 at the top of the lung and as low as 0.6 at the bottom of the lung. The degree of mismatch is decreased during exercise due to increased blood flow to the upper part of the lung, and the effect is eliminated in the microgravity of space but accentuated in hyper-G environments (Rainford and Gradwell, 2006).
The body has control mechanisms to reduce the inefficiency of $V_A/\dot{Q}$ mismatch (Marieb and Hoehn, 2007). If $P_AO_2$ is low due to poor ventilation, the terminal arterioles feeding the capillaries of that alveolus constrict (hypoxic pulmonary vasoconstriction), redirecting blood flow to better ventilated parts of the lung. If $P_AO_2$ is high, the arterioles dilate. This response is opposite of that seen in the systemic circulation, where low tissue $PO_2$ results in vasodilatation. While this auto-regulatory mechanism is adaptive in reducing $V_A/\dot{Q}$ mismatch, under hypoxic conditions where the $PO_2$ in all alveoli falls it can result in generalized pulmonary vasoconstriction, which may contribute to the pulmonary hypertension and high altitude pulmonary edema observed during chronic hypoxia. In addition to the vascular response to $PO_2$, the bronchioles respond to alveolar $PCO_2$. Increased $P_ACO_2$ results in bronchiolar dilation, which increases ventilation and drives $PCO_2$ down; conversely, low $PCO_2$ causes bronchiolar constriction, limiting ventilation of regions with limited perfusion. These two mechanisms serve to synchronize ventilation and perfusion, increasing efficiency of gas exchange.

1.2.6. Transport of Oxygen in the Blood

Once oxygen has diffused from the alveolar gas to the blood, it is transported in two ways: 1) dissolved in the plasma and 2) bound to hemoglobin in the red blood cells. Because of the low solubility of $O_2$ in water, a normal arterial blood $PO_2$ of 95 – 100 mmHg results in only about 0.3 mL of oxygen being dissolved in every 100 mL of plasma. When blood $PO_2$ falls to 40 mmHg in the tissue capillaries, approximately 0.12 mL of $O_2$ remains dissolved in the plasma. Therefore, only 0.17 mL of $O_2$ is delivered in the dissolved state to the tissues by each 100 mL of blood (Guyton and Hall, 2000). This accounts for less than 3% of the oxygen delivered to the tissues.

*Over 97% of the oxygen transported by the blood is reversibly bound to the hemoglobin of the red blood cells as oxyhemoglobin. Each hemoglobin molecule has four subunits, each with an oxygen-binding heme group, so each hemoglobin molecule can bind four molecules of oxygen (Marieb and Hoehn, 2007).*
Oxygen binding to hemoglobin demonstrates the characteristic of cooperative binding, where once the first oxygen molecule binds a heme group, the hemoglobin molecule changes shape, facilitating the uptake of successive O₂ molecules until the hemoglobin is fully saturated with four O₂ molecules. Recall from Section 1.1.3 that each gram of hemoglobin is capable of carrying about 1.34 mL O₂ and that normal adult males have 14 – 16 grams hemoglobin in 100 mL of blood; adult females have 12 – 15 g hemoglobin/100 mL of blood (Des Jardins, 2013). Similarly, release of one O₂ molecule enhances the unloading of the next. Because the affinity of hemoglobin for O₂ changes with successive binding of each O₂ molecule, the relationship between hemoglobin saturation and PO₂ is not linear. This relationship is represented by the oxygen-hemoglobin dissociation curve, which has a characteristic sigmoid shape (Figure 1.2.6-1). The S-curve has a steep slope for PO₂s between 10 and 50 mmHg and then plateaus between 70 and 100 mmHg. Physiologically, the steep portion of the curve describes how large quantities of O₂ are released at PO₂ values found in tissue capillaries and allows large amounts of O₂ to be removed for only a small drop in capillary PO₂. The plateau portion of the curve allows arterial O₂ saturation (and content) to remain high and nearly constant despite fluctuations in PO₂. Under normal sea level conditions, the PO₂ of arterial blood is about 95 – 100 mmHg, resulting in 97% hemoglobin saturation (SₐO₂). The PO₂ of mixed venous blood is only 40 mmHg, just beyond the inflection point where the oxygen-hemoglobin dissociation curve becomes more steep, resulting in a hemoglobin saturation of 75%.

![Figure 1.2.6-1. Oxygen-Hemoglobin Dissociation Curve](image)

While hemoglobin saturation (SₐO₂) is often used as an indicator of O₂ transport, it’s important to keep in mind that the total amount of O₂ in the arterial blood, the arterial O₂ content (C_aO₂), is a more accurate assessment of the amount of O₂ available to the tissues. The total C_aO₂ consists of O₂ bound to hemoglobin and the relatively insignificant amount dissolved in plasma (see equations below). The difference between SₐO₂ and C_aO₂ can be critical in anemia and carbon monoxide poisoning, where an oximeter will give a normal hemoglobin saturation reading despite the fact that the C_aO₂ is reduced, resulting in hypoxia. During hyperbaric administration of O₂, the individual’s SₐO₂ may reach 100%, a relatively small change from normal, while the C_aO₂ is greatly increased due to more O₂ dissolved in the plasma (Figure 1.2.6-1).
The amount of $\text{O}_2$ bound to the hemoglobin in 100 mL of arterial blood at 97% saturation is about 19.5 mL (or 19.5 vol%), just below the maximum of 20 mL $\text{O}_2$/100 mL blood (or 20 vol%) present when hemoglobin is fully saturated (Guyton and Hall, 2000). The amount of $\text{O}_2$ bound to hemoglobin in 100 mL blood is calculated by:

\[
\text{Bound } \frac{\text{O}_2}{100 \text{ mL}} = \frac{\text{grams Hb}}{100 \text{ mL blood}} \times \frac{1.34 \text{ mL O}_2}{\text{gram Hb}} \times S_a \text{O}_2
\]

\[
\text{Bound } \frac{\text{O}_2}{100 \text{ mL}} = \frac{15 \text{ grams Hb}}{100 \text{ mL blood}} \times \frac{1.34 \text{ mL O}_2}{\text{gram Hb}} \times 0.97
\]

\[
\text{Bound } \frac{\text{O}_2}{100 \text{ mL}} = \frac{19.5 \text{ mL}}{100 \text{ mL blood}}
\]

where:

- 1.34 mL O$_2$ represents the carrying capacity of each gram of hemoglobin
- $S_a \text{O}_2$ = hemoglobin saturation (in decimal)

In addition to the $\text{O}_2$ bound to hemoglobin, a small amount is dissolved in the plasma.

\[
\text{Dissolved } \frac{\text{O}_2}{100 \text{ mL}} = P_a \text{O}_2 \times \frac{0.003 \text{ mL O}_2}{100 \text{ mL blood} \cdot \text{mmHg}}
\]

\[
\text{Dissolved } \frac{\text{O}_2}{100 \text{ mL}} = 104 \text{ mmHg} \times \frac{0.003 \text{ mL O}_2}{100 \text{ mL blood} \cdot \text{mmHg}}
\]

\[
\text{Dissolved } \frac{\text{O}_2}{100 \text{ mL}} = \frac{0.3 \text{ mL O}_2}{100 \text{ mL blood}}
\]

Therefore, the total amount of $\text{O}_2$ available to the tissues is:

\[
C_a \text{O}_2 = \text{Bound } \frac{\text{O}_2}{100 \text{ mL}} + \text{Dissolved } \frac{\text{O}_2}{100 \text{ mL}}
\]

\[
C_a \text{O}_2 = \left( \frac{\text{grams Hb}}{100 \text{ mL blood}} \times \frac{1.34 \text{ mL O}_2}{\text{gram Hb}} \times S_a \text{O}_2 \right) + \left( P_a \text{O}_2 \times \frac{0.003 \text{ mL O}_2}{100 \text{ mL blood} \cdot \text{mmHg}} \right)
\]

\[
C_a \text{O}_2 = (15 \times 1.34 \times 0.97) + (104 \times 0.003)
\]

\[
C_a \text{O}_2 = \frac{19.5 \text{ mL O}_2}{100 \text{ mL blood}} + \frac{0.3 \text{ mL O}_2}{100 \text{ mL blood}}
\]

\[
C_a \text{O}_2 = \frac{19.8 \text{ mL O}_2}{100 \text{ mL blood}}
\]

After the blood transits the tissue capillaries and blood $\text{PO}_2$ has fallen to 40 mmHg and hemoglobin saturation to 75%, the amount of $\text{O}_2$ bound to hemoglobin is about 15.1 mL. Under normal conditions, about 5 mL of $\text{O}_2$ is released to the tissues from each 100 mL of blood that transits the tissue capillaries, although this value varies...
between tissues. The percentage of \( \text{O}_2 \) released to the tissues is called the utilization coefficient and, in this case, would be 22%. During strenuous exercise the muscles consume oxygen at a rapid rate, lowering interstitial fluid \( \text{PO}_2 \) to as little as 15 mmHg. At a \( \text{P}_a\text{O}_2 \) of 15 mmHg, hemoglobin saturation falls to 22%, where only 4.4 mL of \( \text{O}_2 \) remain bound to hemoglobin in each 100 mL of blood, increasing the amount of \( \text{O}_2 \) delivered to the tissues from 5 to 15 mL, or three times the normal delivery rate. During heavy exercise the utilization coefficient for the entire body can increase to 75% - 85%. In some tissues where blood flow is restricted or metabolic rates are extremely high, coefficients approaching 100% have been recorded.

The S-shape of the oxygen-hemoglobin dissociation curve reveals two important characteristics of oxygen transport (Guyton and Hall, 2000). In the upper flat portion of the curve, large changes in \( \text{PO}_2 \) result in little change in hemoglobin saturation, buffering the effect of mild hypoxia. For example, at an altitude of 8,000 ft, \( \text{PAO}_2 \) falls to about 63 mmHg; however, hemoglobin saturation of blood leaving the pulmonary capillaries is still about 90%. In contrast, on the steep part of the curve at lower \( \text{PO}_2 \) values, small drops in \( \text{PO}_2 \) result in large changes in saturation and extraction coefficient, facilitating the rapid offloading of oxygen to the tissues. As a result, despite a significant drop in \( \text{PAO}_2 \) from 104 mmHg at sea level to 63 mmHg at 8,000 ft, delivery of a normal 5 mL \( \text{O}_2/100 \text{ mL} \) blood to the tissues only requires a drop in tissue \( \text{PO}_2 \) from 40 mmHg to 35 mmHg. In this way, hemoglobin buffers tissue \( \text{PO}_2 \) and stabilizes \( \text{O}_2 \) delivery.

### 1.2.7. Factors Affecting Hemoglobin Saturation

Several factors affect hemoglobin saturation including: 1) \( \text{CO}_2 \) concentration, 2) pH, 3) temperature, and 4) 2,3-diphosphoglycerate (DPG) concentration (Guyton and Hall, 2000). Each of these factors can shift the relative position of the oxygen-hemoglobin dissociation curve without changing its basic sigmoid shape. For example, in the lungs, \( \text{CO}_2 \) diffuses from the blood into the alveoli, lowering blood \( \text{PCO}_2 \) and raising pH due to a decrease in blood carbonic acid. This causes an increase in the hemoglobin’s affinity for \( \text{O}_2 \) and shifts the oxygen-hemoglobin dissociation curve to the left, resulting in greater hemoglobin saturation at lower \( \text{PO}_2 \)s, facilitating the unloading of oxygen in the lung (the Haldane Effect). In the tissues, where \( \text{CO}_2 \) diffuses from the cells into the blood, the opposite occurs: \( \text{PCO}_2 \) increases and pH falls, shifting the dissociation curve to the right. Under these conditions the affinity of hemoglobin for \( \text{O}_2 \) is reduced, so the percent saturation of hemoglobin will be lower at any given \( \text{PO}_2 \), facilitating the offloading of \( \text{O}_2 \) to the tissues (the Bohr Effect). An increase in temperature similarly shifts the dissociation curve to the right. During exercise these factors work to increase the oxygen delivery to the muscles. An active, exercising muscle fiber generates large quantities of \( \text{CO}_2 \), increasing tissue \( \text{PCO}_2 \) and lowering pH. The muscle fibers release several other acids (i.e. lactic acid), further driving down pH, and the metabolic heat of exercise can raise muscle temperature \( 2\text{°C} – 3\text{°C} \). These factors act together to shift the dissociation curve of hemoglobin in the muscle capillaries significantly to the right, allowing an increase in utilization coefficient from 22% to 75% - 85% with no decrease in tissue \( \text{PO}_2 \). 2,3-DPG is produced by the red blood cells as a product of their anaerobic metabolism. Under hypoxic conditions, 2,3-DPG production increases, shifting the dissociation curve to the right, increasing \( \text{O}_2 \) release to the tissues. Changes in 2,3-DPG concentration and its effect on hemoglobin saturation may be an important factor in adaptation to prolonged hypoxia, either due to
diminished blood flow through a tissue or due to residence at high altitude. Figure 1.2.7-1 summarizes the effects of the various factors on the oxygen-hemoglobin saturation curve.

![Diagram showing factors affecting oxygen-hemoglobin saturation curve]

**Figure 1.2.7-1. Factors Affecting the Oxygen-Hemoglobin Dissociation Curve**

1.2.8. Transport of Carbon Dioxide in the Blood

Although the solubility of CO₂ in water is 24 times greater than that of O₂, only about 5% - 10% of CO₂ is transported as a dissolved gas in the plasma (Des Jardins, 2013; Marieb and Hoehn, 2007). This is due to the small change in PCO₂ between venous and arterial blood. Mixed venous PCO₂ is normally 45 mmHg, where 2.7 mL/100 mL of CO₂ is found in solution. Arterial blood leaving the lung has a PCO₂ of 40 mmHg, with 2.4 mL/100 mL of dissolved CO₂. This small change in blood PCO₂ results in only 0.3 mL/100 mL of CO₂ being delivered to the lungs in the dissolved state.

The greatest proportion, 70%, of CO₂ is transported in the form of bicarbonate ions (HCO₃⁻). CO₂ combines with water to form carbonic acid (H₂CO₃), which then immediately dissociates into bicarbonate (HCO₃⁻) and a hydrogen ion (H⁺) via the following reaction:

\[
CO₂ + H₂O \overset{CA}{\leftrightarrow} H₂CO₃ \leftrightarrow H^+ + HCO₃^- 
\]

The formation of carbonic acid from CO₂ and water is reversibly catalyzed by the enzyme carbonic anhydrase (CA) in the red blood cells. When the carbonic acid formed in the red blood cells dissociates, most of the H⁺ combines with the globin portion of the hemoglobin molecules, which, like all proteins, is a powerful acid-base buffer and prevents a significant drop in blood pH (typically a pH drop of 7.4 to 7.34 is observed between arterial and venous blood). The HCO₃⁻ moves out of the RBC into the plasma in exchange for Cl⁻ via a bicarbonate-chloride carrier protein in the membrane of the red blood cell (chloride shift). As blood circulates through the lung, bicarbonate moves back into the red blood cell and Cl⁻ moves out. The HCO₃⁻ recombines with an H⁺ and carbonic anhydrase catalyzes the reformation of CO₂ and
H₂O, after which CO₂ diffuses down its partial pressure gradient into the alveoli. This is by far the most important means of CO₂ transport in the blood.

The remaining 20% of CO₂ transport occurs through the reversible reaction of CO₂ with amine radicals of the hemoglobin protein to form carbaminohemoglobin (HbCO₂):

\[ CO₂ + Hb \leftrightarrow HbCO₂ \]

CO₂ will combine via the same reaction with plasma proteins, but because hemoglobin is by far the most abundant protein in the blood, carbaminohemoglobin accounts for the majority of CO₂ transport in association with proteins.

Under normal conditions, these three methods result in the transport of about 4 mL of CO₂ per 100 mL of blood, or 4 vol%, from the tissues to the lungs. This is actually twice what would be predicted based on the change in PCO₂. The combination of oxygen with hemoglobin in the lungs displaces CO₂ from the blood via two mechanisms known as the Haldane Effect. First, oxygenated hemoglobin has a lower tendency to form carbaminohemoglobin than deoxygenated hemoglobin, displacing much of the CO₂ in the carbamino form from the blood. Second, oxygenated hemoglobin is a stronger acid than deoxygenated hemoglobin. The increased acidity causes it to release H⁺, which binds to bicarbonate ions to form carbonic acid, which dissociates into CO₂ and water. As hemoglobin is oxygenated in the lungs, the Haldane Effect facilitates the off-loading of CO₂; in the tissues, as hemoglobin gives up its oxygen to the tissues, the movement of CO₂ into the blood is enhanced, effectively doubling the amount of CO₂ transported by the blood.

1.2.9. Internal Respiration

Because the tissues are constantly consuming oxygen and producing carbon dioxide, favorable concentration gradients for the diffusion of O₂ from the blood into the interstitial fluid and CO₂ from the interstitial fluid to the blood are created. Both gases move down their partial pressure gradients, with the peripheral capillary blood quickly reaching equilibrium with the adjacent interstitial fluid (Guyton and Hall, 2000). The partial pressures of O₂ and CO₂ in the interstitial fluid of any given tissue are primarily determined by the blood flow through the local systemic capillaries, the metabolic rate of the tissue cells, and the distance from the nearest perfused capillary. If blood flow through the tissue is decreased, gas transport is decreased, O₂ levels will fall, and CO₂ levels will increase. Similarly, if cellular metabolic rate increases, the consumption of O₂ and the production of CO₂ both increase. Even if average blood flow and metabolic rate are in balance for a tissue so total gas exchange within the tissue is adequate, pockets of hypoxia can exist due to large diffusion distances. The further a cell is from the nearest perfused capillary, the lower the interstitial PO₂ (Rainford and Gradwell, 2006). Because CO₂ diffuses 20 times faster than O₂, it is rarely a limiting factor; however, for some cells the diffusion distances may become so great the diffusion of oxygen will be too slow to meet the metabolic demands of the cell. The oxygen supply to these cells can be increased either by increasing blood flow through the capillaries, reducing the fall in PO₂ across the tissue space, or by opening more capillaries, reducing the intercapillary distance and, therefore, the required diffusion distance. Normal interstitial PO₂ ranges from 5 – 40 mmHg, with an average of about 23 mmHg, depending on the above factors (Guyton and Hall, 2000). Because an intracellular PO₂ of only 1 – 3 mmHg is needed to meet the oxygen requirements of the cell, most cells
are more than adequately supplied, and only under conditions such as initiation of exercise before blood flow can be adjusted or extreme muscular exertion are cells forced to utilize anaerobic metabolism.

1.2.10. Control of Respiration

The above sections have taken us through the five phases of respiration from ventilation of the lungs to utilization of O₂ and generation of CO₂ by the cells. For the metabolic requirements of every cell within the body to be met, the respiratory system and its coordination with the circulatory system must be tightly regulated. Regulation of the circulatory system and the control of blood flow to the tissues are discussed in Section 1.1. Here we will discuss the control of whole body gas exchange through control of ventilation.

Rhythmic, subconscious ventilation is established and controlled by neural signals from the respiratory center of the brain in the medulla oblongata (Marieb and Hoehn, 2007). Inspiratory neurons in the medulla send motor signals to the diaphragm and external intercostal muscles, which contract and expand the thoracic cavity as described above in the section on ventilation of the lungs. Expiratory neurons of the medulla then fire, inhibiting the inspiratory neurons and stopping the motor signals. The inspiratory muscles relax and passive expiration occurs as the lungs and chest wall recoil. This on-off cycling of the inspiratory and expiratory neurons produces the normal ventilation rate of 12 – 16 breaths/min. This basic ventilation pattern is modified by another neural center located in the pons. The pons both smooths the breathing pattern and influences the duration of inspiration and, therefore, the frequency of ventilation (Guyton and Hall, 2000). A strong signal from the pons to the medulla shortens inspiration and, as a result, expiration, increasing the ventilation rate to as high as 30 – 40 breaths/min, while a weak signal extends inspiration and can slow ventilation to 3 – 5 breaths/min. Lesions to the pons can retard the switching off of the inspiratory signal, resulting in almost complete filling of the lungs with only short expiratory gasps. Under normal conditions, the regulatory influence of the pons is important in fine tuning the breathing rhythm during speech, sleep, and exercise (Marieb and Hoehn, 2007).

The rate and depth of breathing are modified in response to changes in the concentrations of CO₂, O₂, and H⁺ in arterial blood. Chemoreceptors in the medulla, the central chemoreceptors, and in the aortic arch and carotid bodies at the bifurcation of the common carotid arteries, the peripheral chemoreceptors, send both excitatory and inhibitory signals to the medulla, adjusting ventilation to the changing demands of the body. Of the three, CO₂ is the most important and the most closely regulated.

Arterial PCO₂ is maintained within 3 mmHg of its normal value of 40 mmHg (Marieb and Hoehn, 2007). CO₂ easily diffuses across the blood brain barrier from the blood into the cerebrospinal fluid, where it is hydrated, forming carbonic acid as described above. As the carbonic acid dissociates, H⁺ is released. The cerebrospinal fluid has much less buffering capacity than the blood because it has a much lower protein content, so changes in arterial PCO₂ result in changes in cerebrospinal fluid pH. If arterial PCO₂ increases, cerebrospinal fluid [H⁺] increases and pH drops, stimulating the central chemoreceptors in the medulla, which increase the rate and depth of
Increased alveolar ventilation flushes CO₂ out of the lungs and blood drawing arterial PCO₂ and cerebrospinal fluid pH back toward normal. A 5 mmHg increase in arterial PCO₂ doubles alveolar ventilation. Conversely, when arterial PCO₂ is abnormally low, cerebrospinal fluid pH rises and ventilation is inhibited, becoming slow and shallow. Breathing may even stop for a short period until arterial PCO₂ levels rise and stimulate ventilation. Although arterial PCO₂ is the proximate stimulus for ventilation, the central chemoreceptors actually respond to changes in cerebrospinal fluid [H⁺], so ultimately ventilation is primarily regulated by pH of the brain.

Changes in blood pH have a much smaller influence on ventilation than changes in PCO₂. Blood [H⁺] can increase and blood pH fall due to increased PCO₂ or accumulation of lactic acid during exercise or other metabolic acids in various disease states, but because H⁺ cannot cross the blood brain barrier, increased blood [H⁺] is not detected by the central chemoreceptors. Changes in blood pH are detected by the peripheral chemoreceptors, however, and a drop in blood pH results in an increase in ventilation rate and depth in an attempt to raise pH by eliminating CO₂ and carbonic acid from the blood. While this regulatory mechanism is much less sensitive than the central pH chemoreceptors, it is faster and may play a role in the initial respiratory response to exercise (Guyton and Hall, 2000).

The peripheral chemoreceptors, especially the carotid bodies, are also sensitive to arterial PO₂; however, arterial PO₂ must drop below 60 mmHg before it significantly influences ventilation rate (Marieb and Hoehn, 2007). This insensitivity to moderate falls in arterial PO₂ makes sense in light of the oxygen-hemoglobin dissociation curve. The percent saturation of hemoglobin and, therefore, the concentration of O₂ in the blood remain high until PO₂ falls below 50 – 60 mmHg. At a PO₂ less than 60 mmHg, the peripheral chemoreceptors stimulate the medulla to double the ventilation rate and depth and can increase ventilation as much as five-fold at very low PO₂S when PCO₂ and pH are at normal levels (Guyton and Hall, 2000). This is known as the hypoxic ventilatory response. When a person ascends to altitude and arterial PO₂ falls below 60 mmHg, the hypoxic ventilatory response is stimulated, which increases O₂ levels in the alveoli and transfer of oxygen to the blood, but it also decreases alveolar and blood CO₂ and raises pH, both of which inhibit ventilation. As a result, a balance of excitatory and inhibitory stimuli is reached with the increase in ventilation due to hypoxia being somewhat less than expected if CO₂ and pH were held at normal levels. With prolonged exposure to altitude, ventilation increases beyond this initial response due to reduced sensitivity of the central chemoreceptors to low PCO₂ and adjustment of body fluid pH by the kidneys. Over a period of 2 – 3 days of acclimatization to altitude, the inhibitory effect of low PCO₂ is diminished and the stimulatory effect of low PO₂ becomes more prominent, resulting in up to a four- to five-fold increase in alveolar ventilation. This is one reason mountain climbers ascend high peaks in gradual stages, to allow the body’s respiratory control functions to acclimatize to progressively greater altitudes.

In addition to the chemical regulation of respiration, higher brain centers can also influence ventilation. The hypothalamus modifies ventilation rate in response to strong emotions or pain (Marieb and Hoehn, 2007). Excitement and stress typically increase ventilation rate. We also have conscious voluntary control of our breathing via the cerebral cortex. Voluntary inputs bypass the medulla, stimulating the ventilatory muscles directly. Our ability to consciously hold our breath or hyperventilate is limited, however, because the medulla will reestablish control of ventilation if blood PCO₂ reaches critical levels. In an effort to maximize their time underwater, breath hold
divers will often voluntarily hyperventilate before holding their breath and submerging. This practice can be dangerous, resulting in what is known as shallow water blackout. Voluntarily hyperventilation reduces alveolar and blood PCO₂, diminishing the CO₂-driven urge to breathe. When divers descend, they consume oxygen from the lungs and blood but do not feel a need to return to the surface until CO₂ levels build back up (Figure 1.2.10-1). When divers do ascend after a prolonged breath hold, PₐO₂ in the lungs rapidly decreases to potentially critical levels due to the decreased water pressure on the body, resulting in a loss of consciousness and possible drowning.

Finally, the respiratory response to exercise is complicated and still not fully understood (Marieb and Hoehn, 2007). During exercise the muscles consume large amounts of oxygen and generate large quantities of CO₂, and ventilation can increase 10- to 20-fold to deal with these demands, but the respiratory response to exercise does not seem to be driven by changes in arterial PCO₂, PO₂, or pH. As exercise begins, ventilation increases abruptly then continues to increase gradually until it plateaus at a steady state. When exercise stops, there is a small abrupt drop in ventilation followed by a gradual return to a normal breathing rate. Throughout this pattern of respiratory adjustment, arterial PCO₂ and PO₂ are virtually unchanged, suggesting that neither PCO₂ nor PO₂ changes stimulate the respiratory response. The most widely accepted hypothesis suggests that the abrupt increase in ventilation at the beginning of exercise is due to the combined effects of: 1) a conscious anticipation of exercise, 2) simultaneous stimulation of the respiratory center by higher brain centers in conjunction with motor activation of skeletal muscles, and 3) excitation of the respiratory center by proprioceptors in the muscles, tendons, and joints. The combination of these neurogenic mechanisms produces the initial increase in ventilation, then the gradual increase and plateauing of breathing rate matches ventilation rate to the production of CO₂. Similarly, the abrupt drop in breathing rate at the end of exercise is believed to be due to a cessation of these neurologic inputs followed by a gradual decrease in ventilation as CO₂ flow decreases and O₂ debt is repaid. The generation of an oxygen debt, that is a buildup of lactic acid in the muscles and interstitial fluid due to an inability to deliver sufficient oxygen to working muscles to support aerobic metabolism, is not due to an inability of the lungs to provide enough
oxygen, as hemoglobin leaving the lungs is fully saturated even at maximal exercise. Rather, oxygen debt is due to cardiac output and muscle vascularization limiting the amount of blood that can be circulated through the muscle. This should make it obvious that breathing 100% oxygen at sea level during athletic events does not improve oxygen delivery to the muscles or speed the payback of oxygen debt because the blood is already carrying virtually all the oxygen it can. The problem is in the amount of blood that can be delivered to the muscles. At altitude, however, low $P_{A}O_{2}$, reduced hemoglobin saturation, and, therefore, reduced $O_{2}$ content of the blood can contribute to reduced exercise capacity, which can be helped by supplemental oxygen.

1.2.11. Summary

This chapter attempts to select and summarize key concepts of respiratory anatomy and physiology from respected texts that are of particular importance and interest to the aerospace physiologist. For further study of pulmonary anatomy, refer to Marieb and Hoehn. Guyton and Hall provide a detailed treatment of all of the respiratory physiology topics touched on in this chapter and many more. For a detailed examination of the respiratory consequences of the stressors associated with the aerospace environment, including hypoxia, anti-G straining maneuver, and positive pressure breathing, refer to Rainford and Gradwell.

References


Concepts

Alveolar gas equation
Gas exchange in the lung
Oxygen-hemoglobin dissociation curve
Primary function of the respiratory system
Rate and depth of breathing

Vocabulary

$P_{A}O_{2}$
Alveoli
Bohr Effect
Carbonic anhydrase
Cellular respiration
Diffusion
$F_{I}O_{2}$
Intercostal muscles
Oxyhemoglobin
Pulmonary ventilation
Trachea
1.3. Neural Physiology

Maj C. Brad Wilson, USAF, M.F.S., Ph.D.

1.3.1. Introduction

The nervous system is comprised of two main components: the central nervous system (CNS) and the peripheral nervous system (PNS). The CNS includes the brain and spinal cord. The PNS links sensory receptors in the body with the CNS. The PNS includes the somatic division, which regulates voluntary motor control, and the autonomic division, which regulates involuntary functions. The autonomic division is further broken down into the sympathetic and parasympathetic systems. The sympathetic system primarily speeds up actions and can be thought of as the “fight-or-flight” response. The parasympathetic system primarily slows down actions, and can be thought of as the “rest-and-digest” response. A third component of the autonomic division, called the enteric nervous system, is composed of neurons in the walls of the gastrointestinal system that are responsible for many functions of digestion and can actually operate autonomously of the CNS or PNS (Purves et al., 2007). Figure 1.3.1-1 summarizes the organization of the nervous system.

![Nervous System Organization Diagram]

Figure 1.3.1-1. Nervous System Organization

1.3.1.1. Central Nervous System Anatomy. The central nervous system (CNS) can be divided into seven primary parts. Starting at the anterior portion of the brain (rostral) and moving to the posterior (caudal), they are the: 1) cerebrum (left and right hemispheres), 2) diencephalon, 3) midbrain, 4) cerebellum, 5) pons, 6) medulla, and 7) spinal cord (Figure 1.3.1.1-1).
The outer layer of the brain, called the cerebral cortex, is comprised of grey matter and is found only in mammals. Grey matter consists of mostly neuronal cell bodies and is the site where higher functions such as memory, emotions, speech, and decision-making occur (Miller et al., 1980). In large mammals, including humans, the cerebral cortex is folded to create gyri (ridges) and sulci (furrows). These folds increase surface area and allow for increased functionality. Underlying the grey matter is the white matter, which contains mostly myelinated axons and appears white due to the lipid composition of myelin. The cerebral cortex is further divided into four lobes. From front to back they are the: 1) frontal lobe, 2) parietal lobe, 3) occipital lobe, and 4) temporal lobes, which sit on either side below the parietal lobe (Figure 1.3.1.1-2).

The diencephalon sits below the cerebrum and consists of the thalamus and hypothalamus. These regions have many sensory, thermoregulatory, and endocrine functions and will be discussed further in the section below covering CNS physiology, as well in Sections 1.5, 1.6, and 7.8.

The midbrain, pons, and medulla are collectively referred to as the brainstem. Although considered a more primitive part of the brain, the brainstem is the primary link for all information flow between the cerebrum and the rest of the body. To illustrate this, 10 of the 12 cranial nerves arise from the brainstem.
The cerebrum is the largest and best developed part of the human brain. It lies at the back of the brain, below the cerebral hemispheres, and has the appearance of a separate structure. The cerebellum primarily deals with motor control and contains several hundred independently functioning microzones. Although the cerebellum is approximately 10% of the total brain volume, it contains more than three times as many neurons as the cerebral cortex (Llinas et al., 2004).

Below the cerebellum and extending from the brainstem, the spinal cord travels caudally to the level of the 12th thoracic vertebra. Sensory information is carried to the spinal cord via the dorsal roots, and motor commands leave the spinal cord via the ventral roots (Purves et al., 2007). The interior of the spinal cord is composed of grey matter, surrounded by white matter on the outside. The grey matter is oriented in such a manner that it forms an X shape. The extensions of the X are referred to as horns (Figure 1.3.1.1-3). The dorsal horns receive sensory information and the ventral horns contain motor neurons that terminate on striated muscles and control movement.

Figure 1.3.1.1-3. Cross-Section Anatomy of the Spinal Cord (studyblue.com [public domain])

The blood-brain barrier is a unique feature of the CNS that protects it from toxins circulating in the bloodstream. It is a highly selective permeable membrane that surrounds the vasculature of the CNS and only allows the passage of water, some gases, lipid soluble molecules, and active transport of other small molecules. It prevents the passage of nearly all bacteria and large molecules, including antibodies (Purves et al., 2007). In certain instances, inflammation and cytotoxins released from some bacteria can increase permeability of the blood-brain barrier and cause serious infections. Such infections are extremely difficult to treat, as the blood-brain barrier also prevents the passage of many therapeutic agents (Purves et al., 2007). Cytokines themselves can even be transported across the blood-brain barrier, where they activate microglial cells and induce the production of more cytokines and perpetuate the inflammatory response (Banks, 2005). There are a few regions of the brain, however, where the blood-brain barrier is absent. These areas are the circumventricular organs (CVOs), and they exist as the connection between the CNS and peripheral blood flow.

1.3.1.2. Peripheral Nervous System Anatomy. As stated previously, the peripheral nervous system (PNS) refers to the parts of the nervous system that lie outside of the brain and spinal cord. This consists of: 1) the cranial nerves, 2) the spinal nerves, 3) peripheral nerves, and 4) neuromuscular junctions.
The 12 cranial nerves emerge directly from the brain and exchange information with various systems of the body. The cranial nerves are: I) Olfactory, II) Optic, III) Oculomotor, IV) Trochlear, V) Trigeminal, VI) Abducens, VII) Facial, VIII) Vestibulocochlear, IX) Glossopharyngeal, X) Vagus, XI) Accessory, and XII) Hypoglossal. The cranial nerves and their functions are summarized in Table1.3.1.2-1.

**Table 1.3.1.2-1. Cranial Nerves and Their Functions** (Purves et al., 2007)

<table>
<thead>
<tr>
<th>Nerve #</th>
<th>Name</th>
<th>Origin</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Olfactory</td>
<td>Forebrain</td>
<td>Smell</td>
</tr>
<tr>
<td>II</td>
<td>Optic</td>
<td>Forebrain</td>
<td>Vision</td>
</tr>
<tr>
<td>III</td>
<td>Oculomotor</td>
<td>Brainstem</td>
<td>Eye movement</td>
</tr>
<tr>
<td>IV</td>
<td>Trochlear</td>
<td>Brainstem</td>
<td>Eye movement</td>
</tr>
<tr>
<td>V</td>
<td>Trigeminal</td>
<td>Brainstem</td>
<td>Mastication, jaw movement, taste</td>
</tr>
<tr>
<td>VI</td>
<td>Abducens</td>
<td>Brainstem</td>
<td>Eye movement</td>
</tr>
<tr>
<td>VII</td>
<td>Facial</td>
<td>Brainstem</td>
<td>Facial expression, salivary glands, taste</td>
</tr>
<tr>
<td>VIII</td>
<td>Vestibulocochlear</td>
<td>Brainstem</td>
<td>Hearing, balance</td>
</tr>
<tr>
<td>IX</td>
<td>Glossopharyngeal</td>
<td>Brainstem</td>
<td>Carotid baroreceptors, taste</td>
</tr>
<tr>
<td>X</td>
<td>Vagus</td>
<td>Brainstem</td>
<td>Cardiac reflex, swallowing, vocal cords, gut</td>
</tr>
<tr>
<td>XI</td>
<td>Accessory</td>
<td>Brainstem</td>
<td>Shoulder and neck muscles</td>
</tr>
<tr>
<td>XII</td>
<td>Hypoglossal</td>
<td>Brainstem</td>
<td>Tongue movements</td>
</tr>
</tbody>
</table>

There are 31 pairs of spinal nerves that run the length of the spinal cord. There are 8 **cervical** nerves, 12 thoracic nerves, 5 lumbar nerves, 5 sacral nerves, and a single coccygeal nerve. The nerves are situated on each side of the midline of the spinal cord and connect to the spinal cord in two separate branches: 1) the dorsal root, which connects to the dorsal horn and, 2) the ventral root which connects to the ventral horn. The dorsal root contains afferent sensory neurons that send information to the spinal cord, while the ventral root sends efferent signals to the muscles. The dorsal root also contains the dorsal root ganglion, which is a collection of neuronal cell bodies and appears as a slight bulge in the nerve. The two branches join after they exit the spinal cord and both afferent and efferent nerves travel together (Figure 1.3.1.2-1).

Figure 1.3.1.2-1. Spinal Nerve
1.3.1.3. **Cellular Components.** The nervous system is comprised of neurons of many different shapes and sizes, and it is estimated that there are over 100 billion in the brain alone. The work of Santiago Ramon y Cajal in the early 1900s elucidated the structure of neurons and neural networks, and eventually led to the acceptance of neuron doctrine in the 1950s (Lopez-Munoz et al., 2006). Neuron doctrine states that neurons are individual cells composed of a cell body, an axon, and dendrites, and they may differ in size, shape, and function based on their location (Figure 1.3.1.3-1). Dendrites receive synaptic input from other neurons, relay the information to the cell body, and a response is generated down the axon to the next cell.

![Figure 1.3.1.3-1. Neuron Structure](image)

In addition to neurons, the nervous system contains many other cells that serve supportive roles and maintain the signaling capabilities of neurons (Figure 1.3.1.3-2). These cells are referred to as glial cells, and they outnumber neurons by about three to one. The most abundant of these cells are astrocytes, which are restricted to the CNS. They regulate the chemical environment of neurons, and their projections surround the CNS vasculature and make up the blood-brain barrier. Oligodendrocytes, also restricted to the CNS, produce the myelin sheath that wraps around some (but not all) axons and increases the speed of electrical transmission. In the PNS, cells that produce myelin are referred to as Schwann cells. The third type of glial cells are called microglia. These cells are considered resident macrophages, and they function to remove cellular debris and secret cytokines that modulate inflammatory responses during infection or injury. Lastly, ependymal cells are involved in the creation and secretion of cerebrospinal fluid (CSF), and they have cilia on their surface to circulate the CSF.
1.3.2. Neural Signaling

1.3.2.1. Electrical Potentials. Neurons transmit information by utilizing a variety of electrical signals. These signals are generated by the transfer of ions across the plasma membrane. When a neuron is at rest, the intracellular charge is always negative. The negative internal environment is usually around -70mV, but it can vary slightly. This negative state is established by concentrating certain ions on either side of the membrane, creating an unbalanced distribution and a concentration gradient. When a neuron is in this state, the unequal distribution of ions creates the resting membrane potential. Inside the cell, the primary ions are potassium (K+) and negatively charged proteins referred to as anions (A-). Outside the cell, the primary ions are sodium (Na+), chloride (Cl-), and calcium (Ca2+) (Figure 1.3.2.1-1).

While the phospholipid bilayer prevents the free exchange of these ions across the membrane, there are two forces that are constantly acting on the ions. These two forces are the concentration force and the electric force. The concentration force refers to the direction in which the ions are being pulled down their gradient. The electric force refers to the direction ions are being pulled based on their charge. If one force is
pulling the ions in one direction while the other force is pulling in the opposite direction, that ion is said to be at equilibrium. If both forces are pulling in the same direction, the ion is not at equilibrium. For example, $K^+$ is pulled outward by the concentration force, but inward by the electric force. Therefore, $K^+$ is at equilibrium. By contrast, $Na^+$ is pulled inward by both the concentration and electric force, so $Na^+$ is not at equilibrium. The $Na^+$ gradient is maintained via two factors that oppose $Na^+$ influx. The first is the low membrane permeability of $Na^+$, and the second is the $Na^+/K^+$-ATPase, which ejects three $Na^+$ for every two $K^+$ brought back into the cell.

Establishing equilibrium is accomplished by an electrical gradient that expands until it balances the chemical gradient. The electrical charge that must be present to balance an ion with its chemical gradient is determined by the Nernst equation. This equation is expressed as:

$$E_x = \frac{RT}{zF} \ln \left( \frac{X_2}{X_1} \right)$$

where:

- $E_x$ = the equilibrium potential for any ion x
- $R$ = universal gas constant = 8.3145 J/mol·K
- $T$ = temperature (in Kelvin; $K = °C + 273.15$)
- $z$ = the valence of the ion (+1 for $Na^+$, -1 for $Cl^-$, etc.)
- $F$ = Faraday’s constant (96,485 C/mol [Coulombs per mole])
- $X_1$ = the concentration of ions inside the cell
- $X_2$ = the concentration of ions outside the cell

The equation, simplified for standard body temperature and base 10 logarithm, is expressed as:

$$E_x = 62 \log \left( \frac{X_2}{X_1} \right)$$

As an example, $K^+$ has an approximate concentration of 140 mM inside the cell and 5 mM outside the cell. The equilibrium potential for $K^+$ is expressed as:

$$E_x = 62 \log \left( \frac{5}{140} \right) = -89 \text{ mV}$$

Therefore, it takes -89 mV of electrical energy to maintain equilibrium with the chemical gradient.

### 1.3.2.2. Action Potentials

In neurons, action potentials are the primary mechanism for cell-to-cell communication. An action potential is a moving exchange of ions that travels from the axon hillock (where the axon connects to the cell body) down the length of the axon. At the terminal end of the axon, synaptic vesicles respond by releasing their neurotransmitters into the synaptic cleft and the signal passes to the next neuron.

Action potentials begin via messages received by the dendrites or cell body of the neuron. These messages can be neurotransmitters from previous neurons or from
sensory transduction (a physical stimulus that initiates an action potential) (Purves et al., 2007). When a message is received, ion channels on the cell membrane open. The ion channels are very selective for the specific ions they will allow across the membrane. The first channels that open are the Na\(^{+}\) channels. As Na\(^{+}\) enters, the inside of the cell becomes less negative as it approaches the threshold potential (approximately -55 mV). If the threshold is not reached, the neuron returns to the resting potential. If the threshold is reached, more Na\(^{+}\) channels open and the polarity across the membrane is temporarily reversed (positive inside and negative outside). This is the initiation of the action potential. As the spike progresses, the membrane potential rapidly rises to approximately +40 mV in a process called depolarization. At the apex of the spike, Na\(^{+}\) channels close and K\(^{+}\) channels open. The rapid efflux of K\(^{+}\) reestablishes the polarity across the membrane (negative inside and positive outside). Na\(^{+}\)/K\(^{+}\)-ATPase then pumps out three Na\(^{+}\) ions and pumps in two K\(^{+}\) ions, which sets the membrane back to its starting point (Figure 1.3.2.2-1). This entire process, however, happens only in one segment of the axon. The propagation down the axon occurs in sections, similar to dominos falling.

![Figure 1.3.2.2-1. Ion Transfer during an Action Potential](image)

Certain factors influence the speed and efficiency of the propagation of the action potential. These factors include the membrane resistance \(r_m\), the membrane capacitance \(c_m\), and the longitudinal resistance \(r_l\) (Purves et al., 2007). The \(r_m\) and \(r_l\) comprise the length constant \(\lambda\), which is expressed as:

\[
\lambda = \frac{\sqrt{r_m}}{r_l}
\]

The length constant determines how far a current will influence the voltage. The higher the value of \(\lambda\), the further the current will flow. The time constant \(T\) determines the speed of the propagation and is expressed as:

\[
T = r_m * c_m
\]

Therefore, to increase the action potential velocity, either \(r_m\) or \(c_m\) must be increased or \(r_l\) must be decreased. An example of decreased \(r_l\) is the giant squid axon. Although unmyelinated, the large diameter of the giant squid axon sufficiently reduces \(r_l\) to increase action potential velocity. This was described in the Nobel Prize winning work of Alan Hodgkin and Andrew Huxley (Hodgkin and Huxley, 1952). Since most
axons in humans have a considerably smaller diameter than the giant squid axon, action potential velocity is increased by adding a myelin sheath, which increases $r_m$ and $c_m$. Myelin is an electrically insulting material composed of cholesterol and other lipids. Myelin allows the action potential to “hop” down the axon in a process called saltatory conduction (Figure 1.3.1.3-1). Since myelin is an insulating material, it has no voltage-gated ion channels. It covers the axon and exposes it to the extracellular environment only at the Nodes of Ranvier. These nodes are gaps in the myelin sheath and contain large amounts of ion channels (Figure 1.3.1.3-1).

1.3.2.3. Synaptic Transmission. Neurons communicate their signals via a process called synaptic transmission. Neurons are not connected to other neurons or cells they influence, but they receive and send information across very narrow gaps called synapses. These synapses, or synaptic clefts, are small extracellular spaces between the cells through which neurotransmitters pass and send information (Figure 1.3.2.3-1). The cell initiating the information is referred to as the presynaptic terminal, and the cell receiving the information is referred to as the postsynaptic terminal. As the action potential propagates down the axon, the electrical changes act upon neurotransmitters at the presynaptic terminal.

Small vesicles, or sacs, at the presynaptic terminal contain various neurotransmitters. When the action potential reaches the end of the axon, it causes $Ca^{2+}$ to rapidly enter the cell. When $Ca^{2+}$ enters, it causes the vesicles to adhere to the presynaptic terminal cell membrane and release their neurotransmitters into the synaptic cleft. The neurotransmitters then attach to receptors on the postsynaptic terminal and either continue or terminate the message (Figure 1.3.2.3-1). Any remaining or unused neurotransmitters in the synaptic cleft are either broken down by enzymes on the postsynaptic terminal membrane or are taken back up (re-uptake) by channels on the presynaptic terminal membrane.

![Figure 1.3.2.3-1. Synaptic Transmission of Neurotransmitters (antranik.org [public domain])](image)

The messages sent by the presynaptic neuron can be classified as either excitatory or inhibitory. An excitatory transmission causes the postsynaptic cell to either continue or act upon the message. An inhibitory transmission causes the
postsynaptic cell to either stop the message or cease action. Synaptic transmission, however, is often not a single cell to single cell message. Neurons have multiple afferent (incoming) and efferent (outgoing) connections (Figure 1.3.1.3-1). The neuron is thus influenced by the combined effects of the afferent connections. The multiple connections are “added up” by a process called summation, and the net product of the signals determines whether an excitatory or inhibitory signal is sent. Spatial summation refers to impulses received by multiple afferent connections in various places of the cell, and temporal summation refers to combined impulses by a single presynaptic cell (Purves et al., 2007).

1.3.2.4. **Neurotransmitters.** Neurotransmitters are the substances used by neurons to transmit signals to other cells. Neurotransmitters are often incorrectly referred to as either excitatory or inhibitory, but the excitatory or inhibitory message is determined by the action of the postsynaptic terminal receptors. Neurotransmitters are comprised of many different substances, but for classification purposes they are often divided into four primary categories: 1) amino acids, 2) monoamines, 3) peptides, and 4) gases. Examples of amino acid neurotransmitters include glutamate and gamma-aminobutyric acid (GABA). Glutamate is considered the only neurotransmitter that always generates an excitatory response, while GABA is considered the only neurotransmitter that always generates an inhibitory response. Monoamines are derived from amino acids and include substances such as epinephrine, norepinephrine, dopamine, and serotonin. Epinephrine, norepinephrine, and dopamine are derived from the amino acid tyrosine, while serotonin is derived from the amino acid tryptophan. Peptide neurotransmitters include substances such as somatostatin and neuropeptide-Y (NPY). Lastly, the gaseous neurotransmitters include nitric oxide (NO) and carbon monoxide (CO) (Purves et al., 2007). A very common neurotransmitter that does not readily fall into one of the previous categories is acetylcholine (ACh). ACh is an ester of acetic acid and choline and is the only neurotransmitter used at the neuromuscular synaptic cleft to initiate muscular contraction. In cardiac tissue, however, ACh has an inhibitory effect.

Neurotransmitters act on postsynaptic receptors, and these receptors can either be categorized as ionotropic, metabotropic, or genotropic. Ionotropic receptors open or close membrane ion channels (ligand-gated). Metabotropic receptors activate second-messengers following activation of G-protein coupled receptors (GPCRs). Genotropic receptors turn certain genes on or off.

1.3.3. **Nervous System Physiology**

1.3.3.1. **Synaptic Plasticity.** Prior to the work of Cajal, it was believed that the nervous system “wiring” was a continuous, unbroken system similar to the vascular system. Cajal demonstrated that the nervous system was in fact billions of individual neurons. As mentioned previously, neurons connect or “talk” to each other via synaptic connections. Unlike a fixed circuit board, however, these synaptic connections are dynamic and constantly changing. These changes either strengthen or weaken synaptic transmission, and they are usually classified as either short-term or long-term. Short-term plasticity refers to changes lasting minutes or less. Synaptic facilitation is a process whereby two or more action potentials reach the presynaptic terminal within milliseconds of each other. The end result of this is that more neurotransmitters are released. The mechanism behind this action is that the
increased number of action potentials increases $\text{Ca}^{2+}$ influx, creating persistent binding of neurotransmitter vesicles at the presynaptic membrane (Purves et al., 2007). The opposite action is called synaptic depression.

Since short-term synaptic plasticity denotes rapid changes in transmission strength, it does not provide the basis for long-term changes that persist for weeks, months, or even years. Long-term synaptic plasticity refers to persistent strengthening or weakening of signal transmission between neurons. Persistent increases are called long-term potentiation (LTP), and persistent decreases are called long-term depression (LTD). Of the two mechanisms, LTP has been more extensively studied. It is believed that the increased synaptic strength produced by LTP is one of the major cellular mechanisms responsible for memory and learning. In 1949, Donald Hebb proposed a theory now known as Hebb’s postulate. In its simplest terms, Hebb stated that persistent firing between cells increased synaptic efficiency and created a symbiotic relationship. This relationship may explain how neurons connect themselves and become engrams, or neural networks.

1.3.3.2. Endocrine Control. Situated just beneath the thalamus and above the brainstem is the hypothalamus. The hypothalamus has many functions, but one of its primary functions is to link the nervous system to the endocrine system via the pituitary gland. The hypothalamus contains magnocellular and parvocellular neurons that send messages to the pituitary gland and control its endocrine functions. Magnocellular neurons are concentrated in the paraventricular and supraoptic nuclei and project directly to the posterior pituitary. These neurons release vasopressin and oxytocin. Vasopressin, also called anti-diuretic hormone (ADH), is responsible for water retention and vasoconstriction. Oxytocin has various roles in females during childbirth and in both sexes regarding feelings of intimacy. The parvocellular neurons secrete various releasing hormones into the hypophyseal portal system, where they diffuse to the anterior pituitary and cause the release of other hormones. The link between the hypothalamus and the hypophyseal portal system occurs at the median eminence, which is a circumventricular organ and therefore devoid of the blood-brain barrier. The hypothalamus thus controls body temperature, sleep, fatigue, hunger, and many other actions.

The hypothalamic-pituitary-adrenal (HPA) axis is a critical component of the CNS stress response. During a stressful event, afferents from the thalamus and sensory cortex signal the amygdala of possible danger. The amygdala has efferents that project to the hypothalamus, where the parvocellular neurons of the paraventricular nucleus (PVN) release corticotropin-releasing factor (CRF) at the median eminence and it is carried to the anterior pituitary gland via the hypophyseal portal system. In response, the anterior pituitary releases adrenocorticotropic hormone (ACTH) into the systemic circulation. ACTH causes the release of cortisol from the adrenal cortex, which serves a variety of purposes in the stress response (Figure 1.3.3.2-1).
Cortisol's primary functions are to increase blood sugar, depress immune function, and provide negative feedback to the pituitary and PVN. Current research has shown that the interaction between cortisol and the immune system may not be as simplistic as previously believed. Cytokines are upregulated in the brain and systemic circulation during the stress response (Wilson, McLaughlin et al. 2013), which contradicts the belief that cortisol is always immunosuppressive. Researchers have also demonstrated that CRH and glucocorticoids can influence the immune system in both directions, indicating a stress response could increase pro-inflammatory cytokines (PICs) and inflammation and alter normal HPA axis function (Chrousos and Gold 1992, Chrousos 1995).

1.3.3.3. Stress and Memory Consolidation. The hippocampus lies deep in the temporal lobe and is part of the limbic system (Figure 1.3.3.3-1). It exerts important influences on the endocrine and autonomic systems, as well as affecting motivation and mood. It plays an important role in the consolidation of both long-term and short-term memories, and it is chiefly responsible for mediation of stress responses via the HPA axis. The hippocampus has been implicated in fear conditioning (Fendt et al., 2005), spatial memory (Kessels et al., 2001), depression (Malberg, 2004), epilepsy and seizure susceptibility (McEwen and Magarinos, 2001), CNS-mediated glucoregulation through cholinergic epinephrine secretion (Uemura et al., 1989), and cognitive disorders such as Alzheimer’s Disease (Garzon et al., 2002) and post-traumatic stress disorder (PTSD) (Wilson et al., 2013; Wilson et al., 2014).
The hippocampal formation is comprised of six distinct regions, linked by primarily unidirectional projections (Hasselmo, 1995). These regions include: 1) the dentate gyrus, 2) the hippocampus proper (Cornu Ammonis [CA] 1, 2, 3), 3) the subicular cortex (comprised of the subiculum, the presubiculum, the parasubiculum), and 4) the entorhinal cortex (composed of two or more subdivisions) (Hasselmo, 1995).

Although the neurophysiological roles for each of these areas are now being elucidated and reported, distinct functions remain incompletely understood, as they are based mainly on computational models derived from hippocampal connectivity studies (Strange and Dolan 1999; Guzowski et al., 2004). What is well known and widely reported, however, is that the hippocampus exhibits a large degree of functional and structural plasticity and even throughout adulthood the hippocampus generates large numbers of neurons (van Praag et al., 1999; van Praag et al., 2002; Kozorovitskiy and Gould, 2004).

The sympathoadrenal medullary system (SAM) system works in concert with the HPA axis during the stress response. The system is very complex and most pathways between nuclei are bi-directional with multiple connections. As mentioned previously, the amygdala receives inputs from sensory areas of the brain and transmits signals to the appropriate regions for response. The amygdala and the PVN have efferents to the rostral ventrolateral medulla (RVLM), which projects to the intermediolateral nucleus (IML) (Fisher and Paton, 2012). The amygdala and PVN also project to the locus coeruleus (LC), the brainstem nucleus responsible for norepinephrine synthesis in the CNS and contains sympathetic projections to the IML. The IML sends preganglionic efferents to the adrenal medulla from the thoracic spinal cord, and the adrenal medulla acts as a specialized sympathetic ganglion (Sapru and Allan, 2007). In response to stimulation, chromaffin cells in the adrenal medulla synthesize and release catecholamines into the systemic circulation. Catecholamine release during stress influences neuroplasticity, memory, emotions, behavior, and other actions in the CNS (Benarroch, 2009). In the systemic circulation, epinephrine and norepinephrine increase heart rate, trigger glucose release, and prepare the body for a “fight-or-flight” response.

1.3.3.4. Thermoregulation. Careful regulation of internal temperature is a critical homeostatic component of both endotherm (warm-blooded) and ectotherm (cold-blooded) organisms. Endotherms create heat by metabolic processes, whereas
ectotherms use the ambient environment to regulate temperature. In both, however, thermoregulation is controlled by the hypothalamus (Romanovsky, 2007). In the anterior portion of the hypothalamus, there lies a small concentration of cell bodies referred to as the preoptic nucleus. The preoptic nuclei are considered thermosensitive and they act as a thermoregulatory coordinating center for effector areas in the brain stem and spinal cord. The preoptic nucleus also receives input from thermoreceptors in the skin, and thus serves as both a central and peripheral regulator (Boulant, 2000). These thermosensitive neurons are also affected by pyrogens, which are any substances that have fever-producing qualities. Pyrogens cause fever by changing the activity of warm-sensitive and cold-sensitive neurons, thus producing elevated set-point temperatures (Boulant, 2000).

In the case of infectious conditions, the presence of a fever usually indicates activity by the immune system. Cytokines produced by leukocytes (white blood cells) elevate temperature in an attempt to create an unfavorable environment for pathogens. Many infectious agents are temperature sensitive, and higher temperatures may make it more difficult for replication. For years it was commonly accepted that exogenous pyrogens (e.g. bacterial toxins, viruses, etc.) induced fever via production of a host-derived molecule called endogenous pyrogen (EP). It was later revealed that one of the main EPs was interleukin-1 (IL-1) (Dinarello, 2004). Other EPs, such as IL-6 and tumor necrosis factor-α (TNF-α), were also discovered and they are now collectively referred to as pyrogenic cytokines. This fit with the EP model, but it was later challenged when experiments blocking IL-1 or TNF-α did not attenuate the febrile response to lipopolysaccharide (LPS). LPS is a potent endotoxin released by Gram-negative bacteria, and is a well-known pyrogen. It is now believed that the Toll-like receptors (TLRs), which act as sentinels on the surface of cells and induce cytokine production, act in concert with pyrogenic cytokines in the vascular network of the anterior hypothalamus to induce fever.

1.3.3.5. Fine Motor Control and Balance. As previously mentioned, basic motor control is conducted via synapses of motor neurons with skeletal muscles. These movements, however, would be very disjointed without coordination and fine tuning by the CNS. The brain region responsible for this fine tuning is the cerebellum. Most of what is known about control of movement by the cerebellum has been derived through studies of humans and animals with cerebellum damage. Such damage can produce notable malfunctions in equilibrium, fine movement, motor learning, and posture (Fine et al., 2002). Thus, the cerebellum is responsible for calibrating detailed forms of movement, but is not responsible for initiation of such movements (Ghez, 1985). One of the unique features of the cerebellum is that, unlike most other regions of the brain, signals move almost exclusively in a feedforward manner. To illustrate, higher brain regions like the cerebral cortex conduct extensive internal “cross-talk.” The cerebellum, however, operates almost exclusively on an “information in – information out” system. According to Eccles et al., this type of operation gives the cerebellum a distinct advantage in information processing and allows it to act like a computer. The brain regions that require input from the cerebellum do not need a complex set of reverberatory information, but rather quick and concise inputs based on received information (Eccles et al., 1967).

Although control of fine motor movement is the most well established function of the cerebellum, recent studies suggest it is involved in many more functions. Imaging studies have demonstrated activation of the cerebellum in relation to attention,
language, and mental imagery (Doya, 2000; Rapp, 2001). A condition known as cerebellar cognitive affective syndrome is characterized by deficits in executive functions, language, behavior, and spatial recognition, and it results from damage to the cerebellum (Schmahmann and Sherman, 1998). This condition has been reported in adults as well as children (Levisohn et al., 2000).

1.3.4. Summary

The purpose of this chapter was to provide a basic overview of nervous system anatomy and physiology. This is by no means a comprehensive guide and many systems and processes were excluded in the interest of scope and depth. Presented first was an overview of the major nervous systems, followed by an introduction to CNS and PNS anatomy. This was followed by the basic cellular components, neural signaling, action potentials, synaptic transmission, and neurotransmitters. Introduced next was basic neural physiology to include synaptic plasticity, endocrine control, and finally stress and memory consolidation. Further research on any of the nervous system components is highly encouraged.

References


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Concepts
- Neural signaling
- Electrical potential
- Synaptic plasticity
- Memory consolidation

Vocabulary
- Autonomic nervous system (ANS)
- Cranial nerves
- Central nervous system (CNS)
- Parasympathetic
- Peripheral nervous system (PNS)
- Sympathetic
1.4. Vision Physiology

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1.4.1. Introduction

Early aerospace medicine pioneers Col W.H. Wilmer and Maj Conrad Berens (1920) wrote: “Of all the physical qualifications necessary for the military aviator there is not one that is more important than good sight.” This aeromedical insight has been proven true many times over. It is commonly accepted that more than 80% of the sensory information necessary to accomplish the flying mission is gathered by the visual system, even in modern technically enhanced aircrafts. Accordingly, USAF aviators are selected, in part, on excellent visual ability and are periodically evaluated to ensure continued optimal visual capability.

However, not all aircrew begin and end their career with so-called “perfect” vision. Perfect vision assumes visual acuity is as sharp as humanly capable at distance and near. It also assumes both eyes are equal in performance and a given person will correctly perceive a visual scene. Some aviators do, in fact, have virtually ideal visual capability as dictated by the selection standards. Most, however, have minor visual imperfections that are typically easy to compensate. As a result of relaxed aeromedical vision standards to allow more aircrew candidates and as trained aircrew simply get older, approximately 40% of all aircrew including pilots require some form of vision correction for flight duties.

Aviator visual performance is further challenged by utilization of devices designed for vision enhancement and/or protection that may, in fact, alter the normal visual perception. For example, visors/spectacles designed to protect eyes from laser threats can mask or alter critical color cues. Even clear chemical/biological or eye hazard protection lenses can create spatial orientation anomalies caused by prismatic effects, effectively altering an aircrew’s situational awareness.

The flying environment itself presents challenges to normal visual performance, ranging from physical dynamics created in flight by gravitational forces (G-forces), fatigue, and hypoxia to visual dynamics such as compressed visual processing time due to aircraft speeds, absence or misleading visual cues, and multiple nonredundant or conflicting sources of spatial information.

This chapter provides a basic understanding of the vision principles that contribute to the efficiency and effectiveness of USAF flying personnel. Psychophysiological and physical aspects related to aircrew vision are applied. By design, information presented is not an exhaustive reference of the human vision but rather selected details presented to assist in understanding. Complete information about the function, anatomy, and physiology of the eye and its neurological interactions can be found in appropriate references.

1.4.2. Form and Function of the Eye

One core concept to understand aviation vision is that all vision occurs peripherally at the eye and centrally in the brain. The eye is, in essence, a device to capture information, while the brain is the information decoder. If the eye doesn’t capture critical visual information, then the brain cannot make judgment and action
based on that missing visual stimuli (i.e., not looking where necessary). Conversely, the eye may capture the “right” stimuli, but the brain may not attend to that information or may make judgment based on other sensory input.

It is also important to understand that conversion of visual stimuli and subsequent action based on visual inputs occur in steps: presence of electromagnetic energy, capturing and conversion of that energy to a neurological signal, transfer of the signal to the brain for interpretation and integration with other sensory input, and finally action taken based on the information.

The eye is a system of elements that focus light energy onto a systematic pattern of cells, which photochemically convert energy into neurological signals transferred to the occipital area of the brain for processing and interpretation as vision. The interpretation of the visual input is then integrated with other sensory sources and then acted upon as necessary.

Each eye is contained in a bony orbit, which provides both protection and support of the globe (Figure 1.4.2-1). The bony orbit is configured like a quadrilateral pyramid, which allows the anterior portion of the eye to be exposed with a wide aperture. The posterior portion of the bony orbit tapers down to an opening at the apex. The posterior opening allows the cranial nerves and blood supply to communicate with the eye. The eye is aligned and moved by six extraocular muscles, which, except for one (the inferior oblique), take their origin from a fibrous ring, the annulus of Zinn, located at the bony orbit’s apex. The eye itself sets in Tenon’s capsule, which acts as an articulate socket. The remainder of the space in the bony orbit is completely filled with fat pads.

![Figure 1.4.2-1. Bony Structure of the Eye](image-url)
The eye globe is approximately 25 mm (1 inch) in diameter and consists of three layers (Figure 1.4.2-2). The outer two layers support, protect, and provide nutrition. The outermost layer, the sclera, is a tough, fibrous coat of collagen bundles laced together in an irregular pattern, which makes the sclera appear white. The anterior portion has a bulge about 12 mm in diameter called the cornea. The cornea is composed of collagen fibers similar to the sclera but formed into a regular pattern of parallel fibers, creating the transparent surface. This is the “window” that allows electromagnetic energy to enter. The shape of the cornea, in combination with the indices of refraction (refractive density), cause the initial focusing of electromagnetic energy (within a limited range of energies) on the retina. In fact, the cornea and its overlying tear film are the most powerful components in the ocular refracting system.

The interior to the scleral layer, the uvea, is the pigmented vascular layer of the globe. The anterior portion of the uvea forms the iris or colored part of the eye. The iris color is a function of the degree of pigmentation. For example, brown eyes are heavily pigmented, while blue or green eyes are relatively lightly pigmented. The iris forms a circular opening, the pupil, observed as the black center of the anterior eye. Part of the iris’s function is to control the amount of electromagnetic energy entering the eye by constricting or dilating the size of the pupil. The posterior portion of the uvea nourishes the outer sclera and the innermost layer, the retina.

![Diagram of the Eye](image)

**Figure 1.4.2-2. Anatomy of the Eye**

Immediately behind the iris is the crystalline lens. The purpose of the crystalline lens is to increase the optical power of the eye’s focusing system, also called accommodation. Physically it’s about 9 mm in diameter and 4 mm thick. In a person with “perfect vision,” distance vision is focused when the crystalline lens is in its natural relaxed state, i.e., no accommodation. When that person’s attention is drawn to a close object, constriction of the ciliary muscle causes the crystalline lens to thicken, increasing the refractive power (accommodates) to maintain a clear focused image. This action of accommodation applies to those whose distance vision is corrected by spectacles, contact lenses, or refractive surgery. In youth, the crystalline lens is quite flexible. Over time accommodative flexibility will gradually decrease. About age 40, the flexibility has decreased sufficiently to affect its ability to accommodate to normal
reading distance. This condition is called presbyopia and continues a progression of reduced accommodative power until about age 65.

The retina contains the elements (rods and cones) that convert electromagnetic energy into neurological signals. This retina actually consists of two layers. The outer layer where the rods and cones are positioned is nourished primarily by the vascular uvea. The innermost nerve fiber layer of the retina is nourished by retinal blood vessels. This difference in metabolic support plays a role in the visual response to high-G exposures that will be discussed later. Visual light energy must pass through the nerve fibers, scattering the energy, before they are captured by the photosensitive cells. It would seem that the retinal layers are opposite to optimal engineering design. This certainly impacts the resolution potential of the eye. However, there are two factors that reveal the advantages of the retinal design. First, the photoreceptors have a high metabolism demand, higher than the neural cells. This high nutritional demand is met by the close proximity of the vascular uvea. Second, the optics of the eye focus light energy to a central area of the retina called the fovea or macula. In the foveal area, nerve fibers are dramatically thin, enabling this area to provide the eye’s sharpest vision.

The retina consists of two classes of photosensitive cells, rods and cones, and various interconnecting nerves. There are three cones types: 1) those sensitive to short (blue), 2) medium (green), and 3) long (red) wavelengths. There is only one rod type, which is sensitive to a range of wavelengths in the middle of the cone sensitivities. The two classes combine to give the human visual system a tremendous operating illumination range from very bright (photopic) to very dim (scotopic) conditions. Additionally, the two classes of photosensitive cells perform different functions and are distributed in different density patterns. This will be discussed later.

The retinal photosensitive cells convert electromagnetic energy into neurological signals. These signals are passed along nerve cells, which merge into a bundle of nerves called the optic nerve, exiting through an opening in the posterior globe. The optic nerve bundle of one eye continues back, intertwining with the optical nerve bundle from the other eye to the occipital area of the brain. The occipital area of the brain decodes the neurological signal and begins the interpretation and action of the visual input.

As a final consideration of form and function, normal human vision includes two “redundant” eyes for capturing and converting electromagnetic energy. In part, two eyes provide us with a survivability aspect should one eye be injured or lost. However, if both eyes are functioning equally, the visual information from each eye is combined and processed, giving rise to higher visual performance than possible from a single eye system. In other words, combined two-eye interpretation (binocular) is better than each individually (monocular). Each eye views the environment with a slightly different perspective caused by the physical horizontal separation of the eyes. When an object is selected as the point of attention, that specific point of attention is placed on corresponding locations on each retina, a common reference point. Other points of interest located at equal corresponding locations are judged to be in the same plane in space. Points of interest that result in disparate placement on the two retinas are judged to be further or closer (Figure 1.4.2-3). The advantage and limitations of binocular and monocular vision will be discussed later.
1.4.3. Photosensitive Cells (Rods and Cones)

Electromagnetic energy spans a wide range of wavelengths. Very short wavelengths (ultraviolet (UV), gamma, and x-rays) and very long wavelengths (infrared, microwave, radio) do not stimulate human photosensitive cells and are therefore invisible to humans. The narrow range of mid-wavelengths that do stimulate these cells is referred to as visible light (peak sensitivity for color vision is at 555 nm).

Normal human color vision results from the stimulation of cone cells (one or more of the three cone cell types). Cone cells sensitive to short visible wavelengths perceived as “blue” light (peak at 440 nm) are called “blue” cone cells. Cone cells sensitive to medium visible wavelengths perceived as “green” light (peak at 535 nm) are called “green” cone cells. Photosensitive cells that give perception of “red” light (peak at 570 nm) are called “red” or long-wavelength cone cells (Figure 1.4.3-1).

Figure 1.4.3-1. Peak Sensitivities of Photoreceptors
Other color perceptions result from stimulating more than one cone cell type (stimulating both red and green cone cells is perceived as a “yellow” light) or from the relative lack of a given wavelength (a filter that removes blue wavelengths from a full spectrum light will make the light appear yellow). In humans, there are twice as many “red” cones as there are “green” cones. There are 10 times as many “red” and “green” cones as there are “blue” cones.

The rod system of photosensitive cells is stimulated by a narrow range of wavelengths between the blue and green cones (peak at 510 nm). However, there is only one rod cell type. As a result of the single-rod cell design, the rod cells are either stimulated and thereby give a “vision” response or they are not stimulated and give a “no vision” output. There is no wavelength discrimination as found in the cone system and consequently no rod color perception.

Cone cells are closely packed together in an area called the macula and become more widely spaced peripherally. It is this dense array of cone cells in the macula that gives rise to the high resolution portion of our vision. Cones are linked to the occipital portion of the brain in a one-cone-to-one-nerve pathway. The combination of one cell to one nerve pathway along with the densely packed central retina cone pattern (macula) enables the eye to have very high resolution. Stimulation of a single cone enables very accurate location of the stimulating source, but at the expense of sensitivity. Cones, compared to rod cells, are relatively insensitive and correspondingly work best in daytime or simulated daytime light (high illumination). In general, the cone system is used to detect color and for fine image resolutions, (i.e., I am looking at what?). Rod cells, in contrast, are more sensitive to appropriate visible light and are interconnected by common nerve paths. A one-rod-cell-to-many-nerve-paths configuration along with high sensitivity make the rod system more responsive to detecting a dim light source (just stimulating one cell sends a signal along many nerve paths). Rod cells are absent in the center of vision (the fovea), are most dense around the macula (parafoveal), and become less dense in the peripheral retina. The rod system sacrifices accuracy in spatial location of the source for sensitivity. In general, the rod system is used to detect motion or the “something happened” (i.e., I am looking where?). The distribution of the rods and cones (Figure 1.4.3-2) is important in aviation vision and will be further discussed later.

![Figure 1.4.3-2. Retinal Distribution of Photoreceptors in the Human Eye](image-url)
1.4.4. Visual Acuity

The human eye is constantly sampling the world around us. An image reaching our retina has spatial, temporal and color properties, with different views from each eye, essential for binocular depth perception. The following discussion will concentrate on the spatial properties of visual perception. First, the various types of visual acuity are presented as a foundation to introduce the concept of contrast. This is then followed by a discussion characterizing spatial vision in terms of the contrast sensitivity function. This will provide an understanding of the omnipresent term “contrast sensitivity” and low contrast vision. Finally, pictorial examples are used to demonstrate the clinical and operational importance of contrast sensitivity and low contrast visual performance. Ultimately, understanding the relationship between visual acuity and contrast allows us to better comprehend visual perception.

1.4.4.1. Measuring Acuity. There are several ways to measure visual acuity based on the type of the task utilized in the measurement. The first type of acuity is Minimum Detectable Acuity, which is defined as the smallest detectable object (e.g., a “dot” in the blue sky). In humans, it can be measured as fine as 1 second of arc (1/60th minute; 1/3,600th degree), which is 30 times smaller than the angle subtended by a single retinal cone cell. Minimum Detectable Acuity has limited practical application and it is rarely measured clinically, though it remains operationally important and a basis for future research.

The second type of acuity is Minimum Misalignment (position or alignment) Acuity, also known as “Vernier Acuity.” It involves the ability to align adjacent spots or contours. Our ability to do this is 10-times more precise than standard clinically measured acuity (3 seconds of arc) and depends on processing in the visual cortex. Vernier acuity, stereopsis, orientation alignment, and minimum motion displacement are considered “hyper-acuities” since they exceed standard acuity and are 10-times finer than the separation between retinal cone cells and depend on visual cortex processing. Vernier acuity is illustrated in Figure 1.4.4.1-1.

![Figure 1.4.4.1-1. Minimum Misalignment Acuity](image)

It should be emphasized that Minimum Detectable Acuity and Minimum Misalignment Acuity are measured in terms of seconds of arc. The acuities that will be introduced next (Minimum Resolvable and Recognition Acuity) are measured in minutes of arc, or arc minute. Minimum Detectable and Minimum Misalignment Acuities are an order of magnitude more sensitive than the acuities that are typically used clinically.

The third type of acuity is Minimum Resolvable Acuity, defined as the smallest resolvable separation (e.g., is it one or two lines?). It is typically determined as the finest pattern (i.e. highest spatial frequency) of black and white stripes or gratings that...
can be discerned. The limit for the human eye equates to an acuity of approximately 20/10. Minimum Resolvable Acuity is typically not used for clinical purposes; however, it is used by the U.S. Armed Forces for focusing and verifying calibration of optical devices. Figure 1.4.4.1-2 depicts a 3 x 3 square wave grating that is used when adjusting the focus of night vision goggles.

![3 x 3 Square Wave Grating used for Focusing Night Vision Goggles](image)

The final type of acuity is Recognition Acuity and is defined as the smallest recognizable letter or object (e.g., it is an F-16 and it is “friend not foe”). Recognition Acuity requires resolving object or letter details. Based on theoretical calculations of retinal cone density, the human eye may be capable of achieving 20/8 acuity using this method. Recognition Acuity is the most common clinical measure of acuity and is the basis for the traditional eyechart developed by Herman Snellen in 1862. Snellen defined 20/20 acuity as a letter that subtends 5 arc minute on the retina and whose details subtend 1 arc minute, Figure 1.4.4.1-3.

![Snellen 20/20 Letter Depicting Angular Sub-tense of Detail](image)

1.4.4.2. Snellen Visual Acuity Test. As mentioned above, in 1862 Snellen devised a method of measuring visual acuity using black letters on a white background. He chose black on white simply because this form could be reliably obtained in a standard and consistent form from a printer. Snellen used the concept that all focused light entering the eye crosses an optical nodal point. Light rays from the extreme opposite edges of a target will cross the nodal point and form a visual angle (Figure 1.4.4.2-1).
As the target approaches the eye, the rays at extreme target edges will subtend a larger visual angle, forming a larger image on the retina. Snellen found that the smallest letter that could be seen by most visually normal patients at 200 feet was about 9 cm tall and composed of lines about 1.8 cm wide. Similarly, the smallest letter seen by normal patients at 20 feet was 0.9 cm tall with 0.18 cm lines. Both targets subtend a visual angle of about 5 arc minutes. The lines had a minimum separation of 1 arc minute (1/60th of a degree). This was consistent with the understanding at that time as determined by ancient astronomers that two stars had to have about 1 minute of angular separation to be distinguished as two separate bodies. Snellen concluded that the minimum visual angle was therefore 1 arc minute. Since that time, standard chart design has used letter targets with line and space widths of 1 arc minute, for example the letter “E” with three horizontal lines that are 1 arc minute wide and separated from each other by 1 arc minute of space. The total letter height is 5 arc minutes (Figures 1.4.4.1-3 and 1.4.4.2-1).

Snellen expressed visual acuity as a fraction of normal as follows:

\[
\text{Visual acuity} = \frac{\text{Distance at which a person can read a letter}}{\text{Distance at which a person with normal acuity should be able to read a letter}}
\]

In other words, the bottom number is the distance from which the chart is designed to be viewed. The top number is the distance to which the patient must move relative to the chart to recognize the same letter that a normal person should see. Normal visual acuity is therefore

\[
\text{Normal visual acuity} = \frac{20 \text{ ft}}{20 \text{ ft}} \text{ or } \frac{6 \text{ m}}{6 \text{ m}}
\]

This equates to a minimum visual angle of 1 arc minute. If an individual is unable to read the letter and must move closer, such as when nearsighted, visual acuity is then

\[
\text{Nearsighted visual acuity: } \frac{10 \text{ ft}}{20 \text{ ft}}
\]
One method to use the Snellen letters is to create a chart of letters all equal in size. To measure visual acuity, the person would simply walk forward until he/she could read the letters. The distance is measured and visual acuity is determined. The U.S. Navy (USN) used this method for many years.

Currently, all U.S. military services (and civilian examiners) use a chart with letters of various sizes placed at 20 feet or in an optical device that simulates 20 feet (Figure 1.4.4.2-2). The numerator is always 20 under these conditions. A 20/20 letter on this chart subtends a visual angle of 5 arc minutes; a 20/200 letter subtends a visual angle of 50 arc minutes. The latter visual acuity indicates that a person viewing from 20 feet can identify letters that a normal person could see at 200 feet.

![Snellen Visual Acuity Test](image)

**Figure 1.4.4.2-2. Snellen Visual Acuity Test Used by the Military**

### 1.4.5. Contrast Sensitivity

Visual acuity is an important metric of visual function, but it represents only a single point on the contrast sensitivity curve. Contrast sensitivity provides a more complete picture of visual status. Contrast sensitivity evaluates our perception of low, mid and high spatial frequencies, while visual acuity relates only to the high spatial frequencies. Studies demonstrate that contrast sensitivity is more relevant than visual acuity in correlating function on real-life visual tasks. Various ophthalmic and systemic conditions may negatively impact contrast sensitivity without significant change to visual acuity. This has the potential to interfere with daily activities such as driving in a more profound fashion than would be predicted based on visual acuity alone. This should be a consideration when a patient’s subjective complaints outweigh the normal clinical findings.

#### 1.4.5.1. Spatial Frequency

The Snellen visual acuity test was designed with high contrast black letters on a white background, ideal conditions rarely found in the natural world and in particular in the aviation environment. As the contrast of a target against its background decreases, visual performance will degrade. Currently, there is no standard for contrast measurement or performance level. With the assumption that vision performance will degrade under certain conditions, aircrew are required to meet strict standards and should perform their duties with optimally corrected vision.
To relate visual acuity to contrast sensitivity, we must introduce the concept of a cycle, which is an increase and decrease in luminance (bright and dark bar). As shown in Figure 1.4.5.1-1, a cycle is the distance required to go from the beginning of one peak (or trough) to the beginning of a second peak (or trough).

![Figure 1.4.5.1-1. Square Wave Function Depicting Complete Cycle](image)

If we relate this concept back to the Snellen letter, we see that a single cycle is comprised of one black stroke of detail and one white stroke of detail (Figure 1.4.5.1-2).

![Figure 1.4.5.1-2. Complete Cycle of Details in Snellen Letter “E”](image)

Thus, the entire letter represents 2.5 cycles (Figure 1.4.5.1-3).

![Figure 1.4.5.1-3. Each Snellen “E” Contains 2.5 Cycles](image)

Now if we consider that there are 60 arc minutes per degree, and each 20/20 “E” is five arc minutes, it is evident that there are twelve 20/20 “Es” per degree (20 x 5 = 60). Since each letter is comprised of 2.5 cycles, a 20/20 letter has a spatial frequency of 30 cycles per degree (cpd): 60/5 = 12 x 2.5 = 30 cpd. Expanding on this concept, we are able to convert a target of any acuity into a spatial frequency (Figure 1.4.5.1-4).
A useful equation to convert Snellen to spatial frequency, and vice versa, is:

\[
\text{Snellen denominator} = \frac{600}{\text{Spatial frequency}}
\]

where:

\[
\text{Spatial frequency} = \text{cycles per degree (cpd)}
\]

Hence, \(\frac{600}{30} \text{ cpd} = 20/20\) visual acuity; for \(20/50\) visual acuity, \(\frac{600}{50} = 12 \text{ cpd}\) (Rabin, 1982).

### 1.4.5.2. Stimulus Contrast Relative to Background.

Up to this point, our discussion has concentrated on black and white stimuli of varying spatial frequencies. This provides one axis of the total contrast sensitivity function. The second axis is defined by the contrast of the stimulus relative to the background. The two most common definitions of contrast are Weber and Michelson, whose formulas appear below. Understanding the mathematics is not essential to this discussion; simply understand that contrast refers to the difference in brightness (luminance) between the target stimulus and its surround. Weber Contrast is the luminance difference relative to the background, while Michelson Contrast is the luminance difference relative to the mean level. The equations for each are:

\[
\text{Weber Contrast} = \frac{L_s - L_b}{L_b}
\]

\[
\text{Michelson Contrast} = \frac{L_s - L_b}{L_s + L_b}
\]

where:

\(L_s = \text{luminance of the stimulus}\)

\(L_b = \text{luminance of the background}\)

If we consider spatial frequency to be the horizontal axis and contrast to be the vertical axis, we arrive at overall contrast sensitivity function (Figure 1.4.5.2-1).
Figure 1.4.5.2-1. Contrast Sensitivity Function

Figure 1.4.5.2-2 shows the typical contrast sensitivity curve for the human eye. The peak of the human sensitivity curve is around 3 – 5 cpd, referred to as mid spatial frequencies. Stated simply, the human eye requires the least amount of contrast to identify objects at mid spatial frequencies, as depicted by the letters in Figure 1.4.5.2-2. Visual acuity would be determined by travelling along the horizontal axis at the bottom of the function where there is 100% (nominal) contrast. It is evident that this represents a very small portion of the overall visual function, despite the degree to which it is used to define visual status.
There are several ways that the human contrast sensitivity (CS) curve may be measured. Eyecharts, such as the Pelli-Robson Chart (Pelli et al., 1988) and those shown in Figure 1.4.5.2-3, have letters of fixed size that reduce in contrast as the test progresses. The size of the letters will determine the position on the horizontal axis and the subject's threshold will determine the point on the curve on the vertical axis.

![Figure 1.4.5.2-3. Charts used to Measure the Contrast Sensitivity Function. 20/25 Letter Size on Left; 20/50 on Right. CS Curve Depicts Approximate Portion of Curve Measured By Each Chart.](image)

Similarly, eye charts may have letters of fixed contrast (Figure 1.4.5.2-4), but varying in size. The contrast level fixes the position on the vertical axis of the contrast function and the subject's threshold will determine the point on the curve on the horizontal axis. The complete contrast curve can also be measured using computer generated images that control both the contrast and spatial frequency of sine wave gratings.

![Figure 1.4.5.2-4. Charts Used to Measure the Contrast Sensitivity Function. 25% Contrast on Left; 5% Contrast on Right. CS Curve Depicts Approximate Portion of Curve Measured by Each Chart.](image)

Studies have shown that contrast sensitivity plays an important role in daily life. Specifically:
- Contrast sensitivity, specifically mid spatial frequency sensitivity, is the strongest predictor for someone’s ability to decipher real world targets such as signs, faces, etc. (Owsley and Sloane, 1978).

- High contrast visual acuity is not highly predictive of performance on everyday tasks (Wood and Owens, 2005).

- Ocular pathologies (e.g. cataracts) may cause a significant reduction in contrast sensitivity with minimal changes to high contrast visual acuity (Adamsons et al., 1992).

- Measuring visual acuity alone would be analogous to a hearing test using only a single high frequency.

This is not to imply that visual acuity has no value, but rather that low and mid spatial frequencies offer additional information that is not embedded in the high spatial frequency information. Figure 1.4.5.2-5 demonstrates a picture that has been filtered into the corresponding low (coarse), middle, and high (fine) frequency images. Notice the degree to which shading and depth cues are lost when the image is limited to high spatial frequencies. High-spatial frequencies give us information on edges and borders; however, low and mid frequencies add additional and important information.

The fact that low, mid, and high spatial frequencies contain unique information is further demonstrated in Figure 1.4.5.2-6. When viewed from a distance (or defocused), the eye is only able to perceive the low to mid frequencies and the image on the left appears as a skull when defocused and a woman in a mirror when in focus. When brought into better focus, the higher spatial frequencies are visible and the picture takes on a completely new meaning. The image on the right appears as a portrait of Abraham Lincoln when defocused and a collage of random images when viewed in focus.
1.4.5.3. Operational Examples. Contrast sensitivity can be reduced in the human eye for a number of reasons. Opacifications within the optical pathway (e.g. cataract, corneal scar), loss of retinal or optic nerve function (optic neuritis, glaucoma), systemic medical conditions (multiple sclerosis, diabetes), refractive surgery, and even normal aging effects can result in reduced contrast sensitivity without significant change to high contrast visual acuity. Reduced contrast sensitivity has the effect of “washing out” images and makes target detection more difficult (Figure 1.4.5.3-1).
1.4.6. Lag in Perception

Visual perception is also affected by cell physiology. Conversion of electromagnetic energy to a neurological signal and its subsequent interpretation don't happen instantaneously. While the process is amazingly quick, there is a time lag. Review the figure below (Figure 1.4.6-1). Note the approximate times for each response to occur. The time to detect an event can be very short. The highly sensitive rod system detects a change in the field of view. Due to the relatively poor resolution of the rods, there may be no information about what changed. To determine "what" changed, the high resolution cone system is directed toward the event, and the subsequent perception is processed. The longest period of time is the decision-making process needed to make a decision on the appropriate action to take relative to the perceived event. This occurs in the brain and then some input is given to the aircraft controls followed by the aircraft response.

To illustrate this time lag, imagine you are flying. Something catches your attention in your peripheral vision (rods detection motion). You look toward the area of the motion to determine what is happening. About 1 second later you determine there is another aircraft approaching. You take another 2.4 seconds to make a decision and initiate a physical reaction to the threat, e.g. to bank right and up. The aircraft reacts to your control input and responds as directed, albeit with an inherent mechanical delay (another 0.2 seconds or so). If you are flying at 500 knots, you will have traveled about 0.5 nautical miles (nmi) (~3,000 ft/920 m) between detecting some motion and the aircraft’s initial response. If you are traveling at 700 knots, you have just covered about 0.7 nmi (4,250 ft/1,300 m). And that is on a good day! Now consider the other aircraft might be flying at a similar rate. If you assume the other pilot does not react at all, within a little more than 3 seconds you both have traveled a net distance of 1.4 nautical miles before your “quick” action and agile aircraft begin to respond to each other. Add to the situation fatigue, hypoxia, or inattention and the zone of safety must become much greater to avoid a mishap.

![Figure 1.4.6-1. Distance Traveled vs. Aircraft Speed and Visual Processing](image)

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1.4.7. Environmental Influences on Vision

1.4.7.1. Effects of Hypoxia on Vision. Hypoxia can cause several changes in vision. Visual disturbances may occur at any altitude above sea level. The
indifferent stage of hypoxic hypoxia occurs in a range between sea level and 10,000 ft. Within this range, daytime vision (photopic vision) is not significantly affected as most
targets of information are generally high contrast and with high illumination. For USAF
aircrew, supplemental oxygen is not required when flying below 10,000 ft. There is,
however, a slight impairment of night vision (scotopic vision) when targets of
information are lower in contrast. For this reason, it is imperative for night combat
flyers to use supplemental oxygen from the ground up.

The stage of adaptation ranges between 10,000 and 16,000 ft of altitude. At
these altitudes, unpressurized flight with no supplemental oxygen will impair visual
function. The following ocular changes occur, becoming more marked with increasing altitude:

a. Retinal vessels appear dark due to the cyanotic condition.

b. The diameter of the arterioles increases 10% - 20%.

c. Retinal blood volume increases up to four times normal.

d. Retinal and systemic arteriolar blood pressure increase.

e. Intraocular pressure increases with the increase in blood volume.

f. Pupils constrict.

g. At 16,000 ft, 40% of night vision ability is lost.

h. Ocular accommodation and binocular convergence decrease.

i. Ability to maintain binocular alignment diminishes.

j. Visual reaction time is reduced.

All these changes return to normal by either use of supplemental oxygen or with
return to ground level.

The stage of inadequate compensation is the region between 16,000 ft and
25,000 ft. In this stage, one or more of the above changes become severe enough
when exposed to unpressurized flight and no supplemental oxygen to significantly
interfere with visual performance. Visual reaction time is significantly slowed and motor
response to visual stimuli is sluggish, as all mental processes are affected. Double
vision may result from binocular eye misalignment for which compensation would
normally adjust. Loss of accommodation and convergence will result in the inability to
clearly interpret cockpit instrumentation. All changes are reversible by use of
supplemental oxygen or returning to ground level.
Finally, the stage of decompensation or zone of lethal altitude occurs above 25,000 ft. In this zone circulatory collapse occurs and there is loss of both vision and consciousness. As a result of neuron death from severe hypoxia and lack of circulation, the aviator may suffer permanent damage to the retina and brain.

1.4.7.2. Brightness of the Field of View. The amount of light reflected back to the eye determines the overall brightness of the individual’s field of view. Snow, for example, reflects back 85% to 90% of the light falling on it. White sand, coral, and white clouds may reflect as much as 75% - 80%. Grass and forests reflect as little as 10% of the light. The apparent “coolness” of green fields probably depends as much upon low light reflectance as it does upon any specific psychological effect of the color. The feeling of sunlight brightness balances on two factors: the amount of light falling on a surface and the amount of light reflected by that surface.

1.4.7.3. Glare at High Altitude. Glare is caused by brightness difference between various parts of a visual field. For example, an eye can be dazzled by a brighter object when adapted for a darker field of view, or view of an object is difficult due to a bright off-axis light source.

At altitudes above 40,000 ft, glare can occur from the cloud layer below the aircraft. The human facial contour is not shaped to protect from glare below the eyes as the brow does for glare above the eyes. A similar effect occurs when standing on snowy or sandy surfaces. It has been theorized that subjective haze is a persistent positive after-image of the bright cloud floor. Other causes suggest fluorescence of the crystalline lens caused by ultraviolet energy or intraocular scattering of light. While the actual cause has not clearly been established, some solutions use filters to eliminate or minimize transmitted glare light. Visor or spectacle techniques considered for below the eye reflected glare include:

a. Maximum absorption in the central portion with increasing transmission superiorly and inferiorly (gradient lenses dark on top and bottom, lighter in center).

b. Maximum absorption superiorly with increasing transmission below (gradient lenses dark on top, lightest at bottom).

c. Self-attenuating variable density filter (photosensitive lenses – increase UV, increase density).

Technique “c” would seem ideal, but in practice the dark-to-light cycling and reverse is too long to be useful in the aviation environment. Further, photosensitive lenses respond to the level of UV energy reaching the lens material. However, UV energy is largely attended by windscreens and canopies. Therefore, these lenses cannot react as intended. Techniques “a” and “b” are possible solutions. However, there is not a standardized method to produce gradient lenses that would be optimal for all airframes and aircrew positioning. These options remain available on a special order basis.

Side glare and glare from below combined with lack of light scatter at high altitude may cause a relative shadow on instrumentation. This is not a physiological condition, and the human has no adaptation for glare. Since the external environment
is bright and relatively little light diffuses from outside of the cockpit, the instrument panel may appear dark as the aircrew’s attention is drawn from outside the aircraft to the panel. One solution is to use while instrument light to brighten and equalize the panel illumination with environment lighting.

It is frequently stated that lens filters reduce glare. This statement is incorrect. Glare is the result of a bright area imposing on a darker area. Typical filters reduce the overall brightness of objects by blocking portions of the energy transmission. But they don’t change the ratio of brightest and dimmest areas. Therefore, glare remains. Only polarizing filters are capable of decreasing glare. Polarizing lenses transmit wavelengths that vibrate in a particular orientation. Wavelength vibrations of other orientations are blocked, thereby reducing glare caused by off-axis light. There are limitations to use of polarizing filters in the aviation environment, however. Stress patterns in windscreens and canopies due to tempering or tension effects create polarization in those transparencies. If the orientation of the polarizing filters is perpendicular to the stress patterns, then dark bands occur and will potentially block vision.

1.4.7.4. Night Vision. There are two types of sensory receptors in the retina: rods and cones. The rods are responsible for vision under very dim illuminations (scotopic vision) and the cones for higher illuminations (photopic vision). As stated previously, cones enable color perception. This is commonly known as the “duplicity theory of vision,” in that the presence of rods and cones enables the human eye to function over an impressively wide range of lighting conditions. A common misconception is that the rods function only at night (dark conditions) and the cones function only during the day (light conditions). Actually, both rods and cones function over a wide range of light intensities, and at intermediate illumination levels they function simultaneously (Figure 1.4.7.4-1).

![Figure 1.4.7.4-1. Iluminance and Visual Function (Sacek, 2006)](image-url)

The transition zone between scotopic and photopic vision is called mesopic vision. Here both rods and cones are active, although not at peak efficiency. Mesopic vision may be of great importance to aviators, because a low level of light is present at dawn and dusk, as well as during night operations. Under dark conditions (below
moonlight intensity), cones cease to function and rods alone are responsible for vision (scotopic vision). Scotopic vision is characterized by poor visual acuity and a lack of color discrimination, but it greatly enhances sensitivity to light. Rods can function in dim light equivalent to an overcast night with no moonlight. The dimmest light cones can function is roughly equivalent to a night with 50% of a full moonlight. Another way to think of this: a white light barely seen by the rod system under scotopic conditions must increase in brightness 1,000 times to be visible to the cone system.

As discussed earlier, rods and cones are uniformly distributed through the retina. Cones are concentrated primarily in the center of the retina, while rods are primarily parafoveal and absent in the foveal (macula). Normal high resolution visual acuity is a result of the densely packed and centrally located cones located in the retina area of central vision. Therefore, under photopic conditions, the cones are functioning as intended and visual acuity is optimized in the fovea. As lighting condition reduces to mesopic, visual acuity is reduced in relation to the reduced cone function. Under scotopic conditions, cones cease to function, and, correspondingly, visual acuity is poorest. Further, under scotopic lighting, the central area of vision (cone vision) becomes a central area of no-vision (the physiologic blind spot). By eccentrically fixating (looking 17 – 20 deg to one side, above, or below an object), along with scanning techniques, placing the object of interest on the densest rod distribution enables rod vision to overcome the physiological blind spot. The "diamond scanning" technique involves looking above, below, and to each side of an object in a diamond-shaped scanning pattern to keep the dimly lit object from bleaching out and disappearing.

Both rods and cones undergo a chemical change when exposed to light that initiates visual impulses in the retina. The retinal photopigments regenerate to continue light detection. Under dark adaptation, the retinal photopigment cells become fully regenerated, and retinal sensitivity is at its maximum. Rods and cones differ in their rate of dark adaptation. Rods require 20 – 30 minutes (or longer) in absolute darkness to attain maximal sensitivity. Cones reach maximum sensitivity in about 5 – 7 minutes (Figure 1.4.7.4-2).

![Figure 1.4.7.4-2. Rod and Cone Dark Adaptation](image-url)
Rods are not sensitive to wavelengths above 650 nm (i.e., red light), while some of the cone cells are. This is the basis for use of red goggles when working in photopic illumination or red light illumination to aid in cone-based mobility. Illumination can then be high enough to enable cone function and yet still preserve dark-adapted vision.

In general, peak sensitivity of rods is 507 nm. The peak sensitivity of cones is 555 nm. If a dark-adapted person views an object, for example, a red car, under photopic conditions, the cones are fully functioning and the car will appear red. As illumination is reduced, there will be a brightness difference between the red car and its background. The red (longer wavelengths) car will appear less bright as the cones become less sensitive and rod vision becomes predominant. With further reduction of illumination the cone cells cease function and the red car will appear to lose its relative brightness and will appear as a gray car. This shift from cone vision to rod vision is called the “Purkinje Shift” (Figure 1.4.7.4-3).

![Figure 1.4.7.4-3. Purkinje Shift](image)

1.4.7.5. Effect of Empty Visual Field. At altitude, aircrew may develop a physiological myopia secondary to ciliary muscle tone when the eye is at rest. At high altitude, one may not have a distant object on which to fixate. In such an empty visual field, reflex accommodation may occur, creating 0.50 to 2.00 diopters (focusing power) of nearsightedness (myopia). Under this condition, normal-sighted (emmatropic) or distance-vision-corrected aircrew would have reduced visual acuity at distance.

1.4.7.6. Ophthalmic Optics. Ophthalmic optics pertains to the science of refracting (bending, redirecting) light wavelengths. While the topic is primarily targeted for eye care providers and optical scientists, it has application to aviation vision. Aircrew often perform target acquisition, refueling, landing, and various other tasks through multiple optical materials: spectacles/contact lenses, visors, windscreens, ballistic/laser eye protection, etc. Distortions induced by these materials are minimized by design. But some distortions cannot be designed out or are simply an artifact of normal optical systems.
1.4.7.7. **Light Characteristics.** All electromagnetic energy moves or propagates, spreading light energy along a wavefront. Vergence is a description of the propagation of light. Light propagating as a parallel wavefront is called a plane or zero wave. Divergent light is a wavefront spreading out as it propagates. Conversely, a wavefront that comes together is called convergent light (Figure 1.4.7.7-1).

![Divergent Plane Convergent](image)

**Figure 1.4.7.7-1. Electromagnetic Propagation (Vergence)**

1.4.7.8. **Sign Convention.** Whether a given wavefront is converging or diverging would seem to depend on the point of view. In the figure above, for example, the left image (labeled divergent) is diverging left to right. But it could also be described as converging right to left. To avoid ambiguity, there are certain rules, known as sign convention. This is based on Cartesian coordinates. In general, figures are drawn with light traveling left to right. All distances are drawn from a certain reference surface, such as a wavefront or a refracting surface, located graphically at (0,0) of an x-y graph. The x-axis extends from - to +. Distances measured to the left of the reference surface, in a direction opposite to light propagation, are considered negative. Distances to the right are considered positive. Accordingly, the radii of curvature of the above figures are measured from the wavelength toward the center of curvature. In the figure representing divergent wavefront, the radii of curvature extend to the left or negative. The convergent wavefront radii extend to the right or in a positive direction. The measure of vergence is the inverse of the distance between the reference surface and the point the radii meet. For example, what is the vergence (V) 2 meters from a point source? \[ V = \frac{1}{2M} = -0.50 \text{ M}^{-1}. \] M^{-1} is also called diopter. Dioptries are commonly used to describe the refractive power of spectacles or contact lenses.

1.4.7.9. **Refraction of Light.** As light wavelengths propagate, they encounter media in their path. If the media are clear, like glass or water, the light will pass through without much loss. Clear media are also called transparent. If the light passes through a material but the medium is not transparent, it is called translucent. Examples of translucent media are frosted or milky glass. If light is unable to pass through a medium, then it is called opaque. Aircrew spectacles, windscreen, visors, sunglasses, etc. are fabricated in transparent materials.

Fermat’s principle of light states that “Light takes the path that requires the least time.” All laws of reflection and refraction are derived from this theorem. For example, a light wave coming from a point “A” is reflected from a mirrored surface to a point “B.” The angle created by point “A” and the mirrored surface is called the angle of incidence. The angle created by point “B” and the mirrored surface is called the angle of reflection. In the case of reflection, both angles are equal. In reflection, light moves in the same media; in this example, light originates and is reflected in air (Figure 1.4.7.9-1).
The principle of refraction differs in that the light does not move in the same media. If there is no difference between the media, light would travel in a straight line. Refraction occurs when light passes from one medium that has a given density (index of refraction) to a different density. For example, originating in air and passing through glass, the path of a light wave is no longer straight. It is refracted. Snell’s law of refraction states that light passing from a lower index (density) to a higher more dense index is bent toward the surface normal (Figure 1.4.7.9-2). If the light travels in the more to less dense medium, it is bent away from the surface normal. One example of this effect is when viewing fish swimming in water. The actual location of the fish is displaced from the visually perceived location if the observer is viewing out of the water.
1.4.8. **Operational Aspects of Night Vision**

1.4.8.1. **Effects of Altitude.** Exposure to reduced oxygen at high altitude causes an increase in the adaptation time and a reduced visual performance. At 12,000 ft without supplemental oxygen (as required by current AF instructions), night vision is functioning at about 25% of its capability at sea level. For this reason, supplemental oxygen is required from ground level and up on all tactical or combat operations. One hundred percent oxygen is not required, since the object is to maintain the blood oxygen content at an equivalent of 5,000 ft mean sea level (MSL) or lower. Therefore, the diluter on an oxygen regulator should be set in the “Normal Oxygen” setting. Note: carbon monoxide hypoxia affects vision in the same manner as high altitude. For example, 5% carbon monoxide saturation (5% carboxyhemoglobin) has an effect equal to hypoxia at 8,000 to 10,000 ft MSL. Smoking three cigarettes can result in 4% carboxyhemoglobin.

1.4.8.2. **Contrast Discrimination.** Visual acuity is reduced at night under low illumination conditions; 20/20 vision cannot be sustained at a low photopic or mesopic range. Accordingly, objects are seen at night because they are either lighter or darker than their background, in other words, contrast differences. These contrast differences may be reduced by reflected light (windshields, visors, spectacles especially with scratched or dirty surfaces, fog, or haze). Because visual acuity and contrast detection are a function of small differences in luminance between objects and their background, any transparent medium through which the flyer must look should be spotlessly clean for night operations. Knowledge of contrast at night may be used by aircrew to detect or avoid detection by enemy observers. Pilots should fly below the enemy, if possible, when over dark areas such as land. They should fly above the enemy when over white clouds, desert, moonlight water, or snow.

1.4.8.3. **Enhancing and Maintaining Dark Adaptation.** For maximum utilization of scotopic vision, 20 – 30 minutes in total darkness is required for adaptation. A more practical alternative is to wear red-tinted goggles. Properly designed goggles (not spectacles) block all light transmission except red wavelengths. Since rod vision is not activated by the red wavelengths, the rods are able to fully regenerate, and scotopic adaptation occurs. The downsides to use of red goggles include the following: red letters on a white background may appear invisible (color confusion) and near vision may blur for pre-presbyopic or presbyopic aircrew (inadequate accommodative stimulus).

Dark adaptation develops rather slowly but can be lost in a second or two upon exposure to bright lights, either external lights (environmental, flares, afterburners, gun flashes, etc.) or bright instrument lighting. If light must be used, it should be as dim as possible and used for the shortest time possible. Dark adaptation is an independent process in each eye. Even though a bright light may shine in one eye, the other will retain its dark adaptation if protected from the light.

1.4.8.4. **Cockpit Illumination.** A question often arises as to what illumination should be used in the cockpit. Red light for cockpit illumination has been used since World War II because, like red goggles, it has less of an impact on dark adaptation. However, as stated previously, it may cause reduced near vision clarity for older aircrew and there may be some color confusion. It should be also noted that
night vision devices operate in a difference spectrum of wavelengths than the human eye. For example, night vision goggles (NVGs) are designed to detect infrared wavelengths, which overlap with visible red light. The result is that even very dim red light as perceived by human vision is highly visible to NVGs. White light provides a more natural visual environment without degrading the color of objects, which would enable the best visual efficiency for aircrew. But dark adaptation is sacrificed. Blue-green lighting, commonly used in modern aircraft, has several advantages. The blue-green light is near the peak wavelength sensitivity for cones, and it’s easiest for accommodative focus. It is readily seen by the rod system, and it lies outside of NVG sensitivity. However, blue-green light is also more visible to enemy unaided observers than the traditional dim-red illumination. The decision on appropriate illumination is, therefore, dependent on the visual requirements and environment in which it will be used.

1.4.9. Visual Illusions and Problems

Spatial disorientation may arise from labyrinthine, proprioceptive, or visual mechanisms. This section will only address those illusions associated primarily with visual perception. There are two forms of visual processing: foveal (central, focal) and peripheral (ambient). Foveal vision is mainly concerned with object recognition, whereas peripheral vision deals mainly with spatial orientation.

The visual perception of something existing objectively but misinterpreted is a visual illusion. In a study by Bell and Chunn (1964), spatial disorientation and visual restrictions were the psychological factors responsible for approximately 23% of the aircraft accidents they investigated. Visual illusions and reduced vision may cause judgment errors that cannot be compensated for by corrective actions. For example, landing at night under inclement weather presents reduced visual situations for a pilot. The poor visual cues complicate distance judgments, making time for corrective actions limited.

The visual system has to make sense of the world in which everyday objects are distorted by perspective. Visual interpretation of two distant illuminated objects can lead to the perception that one is closer than the other when they may be equally distant but differ in brightness. Obviously a large portion of our perceptual experience is based on real world observations. For the aviator, these learned perceptions can lead to visual illusions. One important phenomenon is the apparent unchanged size of a perceived image of an object as its distance from the observer changes. The retinal image reduces in size as the observer moves further from the object; the mental image does not change proportionally. This is the perceptual phenomenon of size constancy. Related to this is the perception of depth. Retinal displacement (disparity) of images on the observer’s two eyes gives rise to stereoscopic (binocular) depth perception.

Aircraft windscreens, visors, and other transparencies can cause refraction, magnification, and distortion of images. Debris, heavy rain, spectacle tints, and such can reduce vision sufficient to make objects like power lines, flag poles, trees, and other aircraft invisible. Further, distance judgment may be reduced as ground lighting may appear less intense, thereby giving the illusion of greater distance. Depth perception arises from monocular and binocular cues.
Monocular cues (single-eye perception) include:

- **Size of the retinal image (size constancy).** Judgment is based on known and/or comparative object sizes.

- **Motion parallax.** The relative motion of images across the retina. Objects nearer than a fixation point move against observer’s motion. Further objects move in the same direction.

- **Interposition.** One object is obscured by another.

- **Texture or gradient.** Detail is lost with increasing distance.

- **Linear perspective.** Parallel lines converge at a distance.

- **Apparent foreshortening.** A circle appears as an ellipse at an angle.

- **Illumination perspective.** Light sources are assumed to come from above.

- **Aerial perspective.** Distance objects appear more bluish and hazy.

Binocular cues (two-eye perception) are vergence and accommodation, useful for near distances and stereopsis (up to 200 yards). Binocular cues occur due to disparate retinal images sufficient to give rise to stereopsis but not to the level to cause diplopia (double vision).

Common aviation visual illusions are runway aerial perspective, visual autokinesis, linear and angular vection, black hole approach, sloping cloud decks, and lean-on-the-sun illusion. A more in-depth discussion on illusions and spatial disorientation can be found in Section 7.3.

1.4.9.1. **Runway Aerial Perspective.** Visual orientation during runway landing is based in part on learn perception. Repetition of aircraft landing on a given runway develops perception of the relative size (length, width) of the runway at various time intervals of successful attempts. The skill developed is essential to a pilot’s performance. However, variation from a highly practiced runway landing requires adaptation to relatively subtle changes. A runway that is sloped upwards 3 degrees will give the illusion that the runway is narrower than expected and, therefore, the aircraft is too high on the approach. If the pilot corrects the aircraft altitude to match the visual perception, the glide slope will be shallower than planned, resulting in a short or hard landing. The opposite will occur on a downward-sloping runway. A runway narrower or longer than expected will likewise give the visual perception of a too high approach.

At night, the pilot attends to the runway lights for visual confirmation of his/her aircraft’s approach. If the runway lights are displaced laterally, a similar misperception will occur as described above. The terrain near the runway may also give false impression of height as well. If the terrain at the approach end is descending, the pilot may fly too shallow. If the terrain is sloping up to the runway, the pilot may approach steeper than required.
Other hazardous runway illusions that may give false height perception may be produced by the presence of snow, fog, smooth water nearby, size of buildings, size of vegetation, and differences in runway light brightness.

1.4.9.2. Visual Autokinesis. The apparent wandering of an object or a light when viewed against a visually unstructured background is called autokinesis. A bright star may be seen as moving in a circle or moving linearly. For example, during a night formation flight, only one running light of the lead aircraft is seen; other pilots may have difficulty in distinguishing real movements of the aircraft. The exact cause is unknown, but it may be related to normal subconscious eye movements. To counter this illusion, avoid staring (fixating) at solitary lights for more than a few seconds and establish a reliable reference in the aircraft such as the canopy border. Look at Figure 1.4.9.2-1. Note that the image at the point of your attention has no perception of motion, while other portions appear to have motion.

![Figure 1.4.9.2-1. Autokinesis](image)

1.4.9.3. Linear and Angular Vection. If a large structure nearby moves forward, there is an illusion that one is slipping backward. The most familiar situation occurs when one is stopped at a traffic light and a nearby car rolls forward. The perception is created that your vehicle is rolling backward.

1.4.9.4. Black Hole Approach. The black hole illusion is produced during night landing, when there are no visual references except runway lights. The situation may worsen when the lights of a city near the end of the runway make the approach appear high (no distinct horizon). To make the perception match expectation, the pilot lowers the aircraft and lands short. A similar situation is produced by blowing snow or sand and also when the runway and nearby terrain are covered by fresh snow or sand.

1.4.9.5. Sloping Cloud Decks. A sloping cloud deck may cause the pilot to adjust the aircraft attitude to what is perceived as the real horizon. This is particularly hazardous when flying near mountainous terrain. There is a strong tendency to accept the level appearance of the clouds as the true horizon, especially if it is indistinct. An unperceived angle of bank will lead to altitude loss, if not corrected.
1.4.9.6. Lean-on-the-Sun Illusion. Terrestrial-based observers are accustomed to seeing the brighter part of the horizon above and the darker ground below. When flying in weather, an attempt to position the brighter cloud layer above may result in an unexpected aircraft attitude. When flying in and out of cloud layers, the pilot generally will remember the relative bearing of the sun before the weather was encountered. This causes the pilot to unconsciously seek the “correct” visual image, resulting in imprecise flight. Formation flight under these conditions can be hazardous.

References

Concepts
Dark adaptation
Diamond scanning
Visual acuity
Contrast sensitivity

Vocabulary
Cone cells
Crystalline lens
Foveal vision
Night vision goggles (NVGS)
Peripheral vision
Photopic vision
Presbyopia
Retina
Rod cells
Scotopic vision
1.5. Vestibular Physiology

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1.5.1. Introduction

Spatial orientation is the interpretation of one’s linear and angular position and motion relative to the plane of the Earth’s surface. Orientational input is necessary in order to know: 1) the body’s position in relation to self and 2) the body’s position in relation to the Earth’s surface. Both have to occur simultaneously and require accurate and reliable input from all senses. Proper input regarding the attitude, heading, and velocity of the body is necessary in order to maintain head stabilization, postural control, orientation, and locomotion (Cheung, 2004a). Incorrect or conflicting inputs lead to disorientation.

In order to maintain our sense of balance, input from multiple receptors located throughout the body must continuously be received and integrated. Postural maintenance and self-motion perception is based on simultaneous stimulation of the visual, vestibular, and somatosensory systems (Cheung, 2004a). Out of all of these inputs, the vestibular component displays the least conscious awareness, yet its subconscious control and coordination of eye movements, postural inputs and corrections, and motor responses to changes in head and body position are critical for maintaining a relatively stable center of gravity relative to the ground while in a dynamic environment, such as walking, running, and even sitting in a chair.

As terrestrial animals, our vestibular system evolved to operate in a 1 G environment with a constant fixed reference point (i.e., the ground) and moving at a relatively slow speed. Under these conditions, vestibular input normally works in close coordination with the other senses, especially vision, to provide accurate orientational inputs. As advancements in technology have allowed us to move at higher speeds and eventually take to the air, conflicting inputs from the vestibular, visual, and somatosensory systems have led to problems of motion sickness and spatial disorientation, both of which are discussed in more detail in separate chapters.

1.5.2. Functions of the Vestibular System

The vestibular system provides three major functions for maintaining balance and orientation (Parmet and Ercoline, 2008). The first major function is to help maximize the effectiveness of the visual system by providing a structural and functional basis for reflexes that stabilize vision. It does so by providing information regarding linear and angular accelerations of the head and body. Without this input, the image on the retina would blur with any movement of the head. Second, orientational information necessary for performing both skilled and reflexive motor activities is provided to various brain centers. Stabilizing reflexes are organized via eye and skeletal motor systems that receive information from vestibular receptors. And third, in the absence of vision, the vestibular system provides a reasonably accurate perception of motion and position (Parmet and Ercoline, 2008). By sensing angular and linear accelerations, the vestibular system provides input for the brain to determine three dimensional head movements in space. By sensing gravitational acceleration, the otolith organs provide a spatial frame of reference that allows the brain to maintain vertical posture with respect to the ground (Lopez and Blanke, 2011).
Another area of orientation that the vestibular system appears to play an important role in is that of positional orientation (i.e., navigation) within our environment. There is increasing evidence that the vestibular system helps track our movement as we navigate through our environment by maintaining and organizing our navigational maps (Lopez and Blanke, 2011; Jamon, 2014). Head direction signaling appears to originate primarily in the otolith organs (although input from the semicircular canals also plays a role) and provides the perception of directional heading with respect to the environment. Input from the vestibular nuclei to the cortex helps develop the internal representation of the head and body movements to aid the perception of self-motion (Jamon, 2014). The input of head rotations associated with path turns, balance perturbations, and other somatosensory inputs helps the brain build internal maps of our external environment that we can then use to navigate and guide our movements. An example is walking around a familiar environment in the dark (Fitzpatrick et al., 2006). While visual inputs are also important, they appear to be intermittent to this process and function more to update the maps than build them. Disruption of vestibular inputs leads to navigation errors while walking in the absence of vision (Fitzpatrick et al., 2006). Vestibular inputs also aid the brain in determining the relative motion of an object: is it moving relative to the head, or is its motion due to the head moving relative to the object (Shinder and Taube, 2010). This information is used to update self-motion, as well as to build the internal maps mentioned above.

In addition to its roles in stabilizing vision and balance and orientation, the vestibular system provides input to the cardiovascular system and the regulation of arterial blood pressure (Cheung, 2004a). Changes in body orientation stimulate the vestibular system, which provides input for the subconscious anticipation of demands placed on maintaining blood pressure to the brain. Stimulation of the otolith organs due to head tilt increases muscle sympathetic nerve activity and helps to defend against orthostatic challenges (Ray and Monahan, 2002). Feedback via the vestibulosympathetic reflex may act immediately to maintain blood pressure before cardiovascular baroreceptors detect a drop in blood pressure. The combination of vestibulosympathetic reflex and baroreceptor inputs has an additive effect on increasing muscle sympathetic nerve activity, and therefore maintenance of blood pressure and blood flow (Ray and Monahan, 2002). It has been proposed that the vestibulosympathetic reflex also plays a role in cardiovascular adaptation to high $+G_z$ (Newman, 1999).

### 1.5.3. Peripheral Vestibular System

#### 1.5.3.1. Location and General Characteristics

The structures of the peripheral vestibular system (PVS) are located within the temporal bone of the skull, some of the densest bone of the body. Its location within the temporal bone provides for a large degree of protection and stability (Figure 1.5.3.1-1). The PVS consists of a membranous labyrinth housed within a bony labyrinth, as well as the mechanoreceptors, or hair cells, responsible for the detection of linear and angular accelerations of the head. The PVS, along with the cochlea, is referred to as the inner ear.
On both the left and right sides of the head, the bony labyrinth of the inner ear is made up of three semicircular canals (SCCs), the vestibule containing the utricle and saccule, and the cochlea of the auditory system. These structures are arranged in a mirror image fashion relative to each other on opposite sides of the head. A cross-section of the bony labyrinth reveals an inner membranous tube, the membranous labyrinth, which is continuous between the three SCCs, the vestibule, and the cochlea (Figure 1.5.3.1-2). This continuity partially explains why loud noise can stimulate a vestibular response.

The space between the bony and membranous labyrinths is filled with perilymph fluid that is similar in composition to cerebral spinal fluid (high sodium:potassium ratio, Na⁺:K⁺) (Hain and Helminski, 2007). The membranous labyrinth is suspended within the bony labyrinth by the perilymph fluid and connective tissues and is filled with endolymph fluid. Unlike cerebrospinal fluid, endolymph fluid more closely resembles the composition of intracellular fluid with a high potassium:sodium (K⁺:Na⁺) ratio (Hain and Helminski, 2007). This composition plays an important role in the generation of action potentials when the hair cells of the vestibular system are deflected.
Near the junction of the three semicircular canals with the vestibule, the membranous labyrinth is enlarged to form an ampulla that houses the mechanoreceptors, or hair cells, responsible for the detection of angular accelerations of the head. These structures are covered in more detail below.

1.5.3.2. Functional Unit of the Vestibular Sensory System.
Rotational head movements that elicit angular accelerations are detected by the semicircular canals, while linear acceleration generated either from linear motion of the head or static tilting of the head is detected by the otolith organs. Neither the otolith organs nor the semicircular canals respond to constant velocity, but rather respond to acceleration (i.e., changes in velocity). This allows the system to detect changes in head position with regards to changes in angular or linear motion. Due to their specific orientations, each semicircular canal and otolith organ can selectively respond to head motion in a particular direction and the brain is therefore able to determine head position.

Within the enlarge membranous labyrinth of each semicircular canal and the vestibule, specialized hair cells serve as mechanoreceptors that convert the spatial and temporal patterns of mechanical energy into neural signals that are then transmitted to the brainstem and higher brain centers. This functional unit of the vestibular system consists of bundles of approximately 60 – 100 cilia, or stereocilia, and one larger kinocilium (Figure 1.5.3.2-1). These sensory cells are often referred to as vestibular hair cells. The graduated arrangement of stereocilia relative to the kinocilium leads to functional polarity or directionality—deflection towards the kinocilium results in a depolarization and generation of an action potential; deflection way, hyperpolarization. Deflection in a vector off the axis of polarity does not result in signal transduction (Colclasure and Holt, 2003).

Figure 1.5.3.2-1. Vestibular Sensory Hair Bundles (Holt and Cory, 2000)

The tips of each stereocilia are linked together such that when the stereocilia are deflected, the connection opens mechanically gated channels and allows K⁺ in the endolymph fluid bathing the hair cells to enter the stereocilia within 10 microseconds of deflection, faster than sensory transduction occurs in the visual or olfactory systems (Boyer et al., 1998; Holt and Cory, 2000; Colclasure and Holt, 2003). This influx of K⁺ depolarizes the cell and opens voltage-gated calcium channels allowing an influx of calcium (Ca²⁺). Increased intracellular Ca²⁺ causes the release of excitatory neurotransmitter (i.e., glutamate) and the generation of a neural signal that is carried to the brainstem and higher brain centers (Figure 1.5.3.2-2).
Under resting conditions, vestibular sensory cells have a continual, steady neural firing rate. For a frame of reference, the resting discharge rate of a semicircular canal is about 100 action potentials per second. By comparison, a retinal ganglion cell has a rate of about one action potential per second in the dark (Wolfe et al., 2012).

Continual stimulation and deflection of the sensory receptor cells results in vestibular adaptation to the stimulus. Adaptation occurs as a result of depletion of neurotransmitter in the pre-synaptic vesicles, post-synaptic desensitization of receptors, or other post-synaptic processes that result in decreased afferent neuronal firing (Goldberg et al., 2012). A similar phenomenon occurs with repetitive exposure that leads to vestibular habituation to a stimulus. With training and repeated exposure, an individual can learn to suppress the response to vestibular inputs to the brain (Parmet and Ercoline, 2008). This vestibular suppression is possible due to the dominance of the visual system overriding the vestibular cues. Ballet dancers use a technique called spotting where they utilize a fixed visual reference point that allows the head to lag behind the rotation of the body. By rapidly snapping the head forward and fixating on a new fixed reference point, the sequence of head motions mimics those of normal head movements and does not result in prolonged deflection of the semicircular canal cupula. Figure skaters, on the other hand, utilize vestibular suppression and habituation to avoid disorientation following a spin (Goldberg et al., 2012). Without visual inputs, vestibular suppression is not possible (Parmet and Ercoline, 2008).

As mentioned earlier, deflection of the stereocilia towards the kinocilium results in an opening of the $K^+$ channels in the tips of the stereocilia and an influx of $K^+$. The resulting depolarization increases the neural firing rate of the vestibular nerve to specific areas of the brainstem and cerebellum. Deflection of the stereocilia away from the kinocilium results in hyperpolarization and a decreased neural firing rate (Figure 1.5.3.2-3). This change in potential is proportional to the degree of bending of the sensory cells and determines the amount of neurotransmitter released to bind with the afferent neuron. The greater the deflection, the more neurotransmitter released, and
the greater the firing rate. The brain uses this information to determine both changes in head position (i.e., direction of motion) and also the rate of head movement.

![Figure 1.5.3.2-3. Neural Discharge of Vestibular Nerve](image)

Unlike some vertebrates, mammals lack an ability to regenerate hair cells in the peripheral vestibular organs (Goldberg et al., 2012). Damage to the sensory hair cells, or to the neurons that innervate them, can result in a permanent loss of function within the vestibular system. Even partial loss of sensory hair cells results in deterioration of function. Sensory hair cells can be lost due to exposure to ototoxic agents, overstimulation, or simply the aging process (Goldberg et al., 2012).

### 1.5.3.3. Otolith Organs

The otolith organs, or otoliths, refer collectively to the utricle and saccule structures that are contained within membranous labyrinth inside the vestibule of the bony labyrinth (Figure 1.5.3.1-1). They respond to changes in the magnitude and direction of transient linear acceleration and static head tilt relative to gravity. Unlike the semicircular canals, the function of the otolith organs is much simpler—no hydrodynamic forces are necessary in order to displace and activate the hair cells (Hain and Helmsinki, 2007).

While there are three semicircular canals on each side of the head to detect motion in the pitch, roll, and yaw axes, there are only two otolith organs in each vestibule. The utricle is oriented to detect changes primarily in the horizontal plane and the anterior portion is tilted approximately 30° from the transverse plane, roughly matching the orientation of the horizontal semicircular (Figure 1.5.3.4-1). The hair cells of the utricle are oriented vertically relative to the ground. The orientation of the hair bundles allows it to detect lateral accelerations (in the y axis) as well as accelerations in the anterior-posterior plane (in the x axis) (Hain and Helmsinki, 2007). The saccule is oriented to detect changes primarily in the vertical plane (Figure 1.5.3.3-1), with the saccular hair cells oriented horizontally relative to the ground. It is capable of detecting linear acceleration in the occipitocaudal axis (z axis), as well as along the anterior-posterior axis (Hain and Helmsinki, 2007). The combined shape and orientation of the utricle and saccule allows them to detect linear motion or head tilt in all three axes, thereby determining pitch, roll, and yaw motion of the head.
Structurally, the utricle and saccule consist of a macula, or base, that contains the sensory hair bundles, supporting cells, and afferent neurons (Figure 1.5.3.3-2). Each macula is roughly 3 mm long by 2 mm wide and contains approximately 16,000 hair cells in the saccule and 30,000 hair cells in the utricle (Wolfe et al., 2012). This is the site where sensory transduction occurs. The sensory hair bundles protrude from the macula into a gelatinous layer called the otolithic membrane. This layer is roughly three times as dense as the endolymph fluid that bathes the entire otolith organ. The otolithic membrane completely encompasses the sensory hair bundles and ensures they move as a single unit. Embedded in the otolithic membrane are millions of calcium carbonate crystals, or otoconia. The otolithic membrane and otoconia combine to add mass to the otolith organ and are therefore responsive to inertial forces and gravity. Their movement relative to the macula causes the sensory hair cells to bend, altering their receptor potential as described earlier. The macula of both the utricle and saccule contain a central curved band called the striola (Figure 1.5.3.3-1). The sensory hair bundles on either side of the striola are oriented in a mirror fashion to each other. In the utricle, the kinocilia are oriented facing the striola, while in the saccule the kinocilia are oriented away from the striola (Cheung, 2004a). With any change in linear motion or head tilt, some hair cells will be depolarized and others hyperpolarized in both otolith organs. During angular acceleration, such as during yaw rotation, an equal number of hair cells on both sides of the striola in each otolith organ are stimulated. This equal and opposite stimulation results in a cancelling out of input to the brain. As a result, it has been shown that the otolith organs are unaffected by angular accelerations (Cheung, 2004a).
While the otolith organs accurately detect head position with respect to gravity in a static environment, their function can be compromised by becoming habituated to an altered head position or sustained linear acceleration. With sustained linear acceleration and a lack of visual cues, the brain can misinterpret the input from the deflected sensory hair cells. During sustained forward acceleration, the rearward deflection of the utricular hair cells can be interpreted as an upward, or backward, tilt of the head leading to an illusion called the Pitch-Up Illusion (Figure 1.5.3.3-3). This and other somatogravic illusions will be discussed in more detail in Section 7.3.

![Figure 1.5.3.3-3. Misinterpretation of Linear Acceleration](image)

The limitations of the otolith organs in the aerospace environment are summarized as follows (Cheung, 2004a):

1. No transduction of linear velocity, but only linear acceleration.
2. An inherent inability to distinguish between gravity and sustained linear acceleration of the body.
3. Accuracy for determining the direction of the vertical only with the head upright pitched forward 25 – 30 deg, because the utricular macula and [horizontal] semicircular canals are pitched up by 25 – 30 deg with respect to the Horsley-Clarke plane.
4. A limited threshold ($Y$ and $X = 0.005 – 0.01 \text{ G}; Z = 0.01 – 0.1 \text{ G}$).
5. A higher threshold and greater error in detecting vertical motion.

### 1.5.3.4. Semicircular Canals.

The semicircular canals (SCCs) are the primary receptors for head acceleration. At velocities and durations of normal head rotations, they act as integrating accelerometers (Cheung, 2004a). Output from the semicircular canals provides information about movement within the environment and aids in coordination of posture, body movement, and eye gaze during locomotion (Cox and Jeffery, 2010).

There are three SCCs within each labyrinth on each side of the head: an anterior (or superior) SCC, a posterior SCC, and a horizontal (or lateral) SCC. Each of the three SCCs on the same side of the head lies perpendicular to each other and the arrangement across the head is a mirror image. The semicircular canals are not aligned perfectly with the anatomical axes. The anterior and posterior SCCs are
oriented roughly 45° from the sagittal plane. While the posterior SCC is nearly vertical, the anterior SCC is tilted approximately 15° from the vertical. The horizontal SCC is tilted up anteriorly approximately 30° from the transverse plane (Figure 1.5.3.4-1).

![Figure 1.5.3.4-1. Orientation of the Semicircular Canals](image)

Each semicircular canal has an axis of maximal sensitivity that results in it generally being associated with rotation in a particular plane. In reality, due to the actual orientation of the SCCs, there is no plane of rotation that stimulates a single pair of canals (Cheung, 2004a). This can partially be explained by the fact that the semicircular canal anatomy is such that each canal has some torsion, or twisting of the canal out of its principal plane. This torsion allows for detection of out-of-plane accelerations (Cox and Jeffery, 2010). In general, the horizontal SCC is associated with rotation around the z axis (yaw), the anterior SCC with rotation around the y axis (pitch), and the posterior SCC with rotation around the x axis (roll). The horizontal SCC also appears to be more highly correlated with agility and navigation in mammals, and appears to distinguish between animals that move largely in a two dimensional environment, such as on land, versus those that move in a three dimensional environment such as aerial, arboreal, or aquatic environments, with the horizontal SCC being more important in the three dimensional environment (Cox and Jeffery, 2010). The vertical anterior and posterior SCCs appear to play a larger role in reflex adjustments in response to movement and maintenance of balance, with the posterior SCC being the least correlated with agility (Cox and Jeffery, 2010; Fitzpatrick et al., 2006).

When comparing the planes of the paired SCCs in both labyrinths, the six SCCs form three coplanar pairs: 1) right and left horizontal, 2) left anterior and right posterior, and 3) left posterior and right anterior (Hain and Helminski, 2007) (Figure 1.5.3.4-1). This functional pairing of canals is called a push-pull arrangement. When one canal in the pair is excited, the other canal is inhibited. This differential coding of neural input is used by the brain to determine head rotation. Another advantage is that this arrangement of the SCCs aligns them very closely with the extraocular muscles and helps in vestibular stabilization of the eye. This connection will be discussed in more detail later.
In addition to helping to stabilize vision, the coplanar arrangement of the SCCs has three advantages. The first is that it provides for sensory redundancy. Second, it allows the brain to ignore changes in neural firing that occur on both sides of the head. Third, it assists in compensation for sensory overload (Hain and Helminski, 2007).

Anatomically, the three semicircular canals occupy a space roughly the size of a pea. The bony labyrinth of the semicircular canals is approximately 1.5 mm in diameter and 15 mm in length and is elliptical in shape (Wolfe et al., 2012, Cheung 2004). Each canal forms about ¾ of a toroid, with the remaining portion passing through the vestibule (Figures 1.5.3.1-1 and 1.5.3.1-2). As discussed above, the membranous labyrinth is suspended within the bony labyrinth and surrounded by perilymph fluid. At 0.3 mm, the cross-sectional diameter of the membranous labyrinth is slightly larger than a human hair and is filled with endolymph fluid (Wolfe et al., 2012).

The elliptical shape and size of the canals play a role in their sensitivity and vary greatly among mammals. There is wide intra-individual, as well as inter-individual, variation in shape and size (Cox and Jeffery, 2010). These differences have been shown to play a role in functional differences in locomotion, with a significant positive correlation between semicircular canal radius and agility across a wide sample of mammals. This correlation is likely due to the increased sensitivity to angular acceleration conferred by a larger radius of curvature (Cox and Jeffery, 2010). In general, across mammals the anterior canal is larger than the posterior and horizontal canals. This suggests greater sensitivity to changes in pitch, which could serve an evolutionary benefit while walking upright.

At the junction of the canals with the vestibule, the membranous labyrinth enlarges to form the ampulla (Figure 1.5.3.4-2). Contained within the ampulla are the sensory epithelial cells called the crista ampullaris. The crista ampullaris is similar to the otolithic macula in that it contains the sensory hair cells, the stereocilia and kinocilia, and neurons responsible for signal transduction. Each ampulla contains approximately 7,000 hair cells (Wolfe et al., 2012). Unlike the orientation of the sensory hair bundles around the striola of the otolith organs, all of the stereocilia and kinocilia within an ampulla are oriented in the same direction. The stereocilia and kinocilia project up into the lumen of the membranous labyrinth into the cupula, a flexible jelly-like substance that extends across the entire width of the ampulla (Figure 1.5.3.4-2). The cupula serves as a rudder that is deflected with the motion of the endolymph fluid in response to angular accelerations of the head. Because the cupula has the same density as the endolymph fluid, it is not sensitive to gravity.

![Figure 1.5.3.4-2. Ampulla of a Semicircular Canal](image)

During rotation of the head, the bony and membranous labyrinths move with the rest of the head; however, due to inertia, the endolymph fluid within the membranous labyrinth initially lags behind. This results in a relative flow of endolymph fluid, and
therefore deflection of the cupula, in the opposite direction of the head movement (Figure 1.5.3.4-3). Deflection of the cupula deflects the stereocilia and results in either depolarization or hyperpolarization as previously discussed. In each SCC, all of the hair cells deflect in the same direction.

![Figure 1.5.3.4-3. Deflection of the Cupula during Head Rotation](image)

When rotation of the head decelerates or stops, the inertia of the endolymph fluid can cause it to continue moving in the direction of the initial rotation, despite the lack of movement of the bony and membranous labyrinths. This results in a change in the deflection of the cupula to the opposite direction. This reversal of stimulation results in a perception of the head rotating in the opposite direction of the initial rotation, even in the absence of actual rotation.

For the semicircular canals to function correctly as a hydromechanical sensor of angular acceleration, the specific gravities (i.e., the densities) of the cupula and the endolymph fluid must be equal (Kondrachuk and Boyle, 2011). Due to the sensitivity of the semicircular canals, even small density differences between the two compartments can significantly alter their response. Differences of approximately $10^{-4} \text{ g/cm}^3$ have been shown to be enough to make the SCCs sensitive to gravity and centrifugal forces that are comparable to gravity (i.e., during simulation of 1 G during microgravity) (Kondrachuk and Boyle, 2011). The following conditions can potentially lead to density differences between the cupula and the endolymph fluid in the membranous labyrinth of the semicircular canals (Kondrachuk and Boyle, 2011):

1. Different compositions of the cupula and endolymph.
2. Change of intra-labyrinthine pressure (discussed in more detail below).
3. Ingestion of some substances, such as alcohol, glycerol, or heavy water (discussed in more detail below).
4. Pathology or local inflammatory processes.

While the hydrodynamic properties of the semicircular canals typically provide reliable inputs for head acceleration and position, there are a couple of hydrodynamic characteristics that can lead to an inaccurate perception of head rotation.

With constant acceleration, the endolymph fluid eventually catches up and moves at the same speed as the membranous labyrinth. With no more mechanical pressure on the cupula, it returns to the resting, upright position relative to the crista ampullaris. In this state, there is no deflection of the hair cells, no altered neural...
discharge, and therefore no acceleration signal sent to the brain. The lack of neural input is interpreted as a lack of head rotation, even if the head is still rotating. This condition is referred to as adaptation and, depending on the initial acceleration, can occur within approximately 15 seconds (Wolfe et al., 2012).

The second hydrodynamic characteristic necessary for accurate perception is than any rotation of the head must be above a minimum threshold to result in deflection of the sensory hair cells in the SCC and alter the neural firing rate of the afferent neurons. Stimulation is dependent on both the magnitude of the angular acceleration and the duration (Cheung, 2004a). Any rotation of the head that does not meet the minimum threshold will not be detected, and will result in an incorrect awareness of head position. Each semicircular canal has a different threshold for stimulation, and it appears that for transient (1 – 5 seconds) angular movements, the threshold for movement around the z axis (yaw plane) is 1.5 deg/sec, movement around the y axis (pitch plane) is 2.1 deg/sec, and movement around the x axis (roll plan) is 2.0 deg/sec (Benson et al., 1989; Cheung, 2004a). Any rotation that does not meet threshold, and is therefore not perceived by the brain, is referred to as sub-threshold rotation. As land animals, sustained angular acceleration (either the magnitude or time component) is rarely encountered, and sub-threshold rotation is rarely a problem. However, this changes in the aerospace environment.

Semicircular canal adaptation and sub-threshold stimulation are the basis for several aviation relevant forms of spatial disorientation, and will be discussed in more detail in Section 7.3.

The limitations of the semicircular canals in the aerospace environment are summarized as follows (Cheung, 2004a):

1. A limited threshold of vestibular perception:
   a. Yaw (sustained): 0.14 deg/sec$^2$
   b. Yaw (transient, < 5 sec): 1.5 deg/sec
   c. Roll (sustained): 0.5 deg/sec$^2$
   d. Roll (transient, < 5 sec): 2.1 deg/sec
   e. Pitch (sustained): 0.5 deg/sec$^2$
   f. Pitch (transient, < 5 sec): 2.0 deg/sec

2. A perceived angular velocity that is progressively less than the actual angular velocity during prolonged rotation.

3. An absence of sensation of rotation during constant velocity rotation.

4. An apparent sensation of rotation in the opposite direction during deceleration.

5. A persistent apparent sensation of rotation in the opposite direction, after physical rotation has actually stopped.

6. A greater chance in developing error in roll than in pitch, which in turn is greater than in yaw.

7. A rotation-induced involuntary oculomotor response that destabilizes the retinal image.
1.5.3.5. Innervation of Peripheral Vestibular System. The signals from the peripheral vestibular structures in the inner ear are integrated in the brainstem, the cerebellum, and the cortex. The cerebellum and cortex provide control over the sensory integration and motor control process that occurs predominantly in the brainstem. Input from the vestibular system lacks the conscious prominence of the other orientation systems (vision, hearing, touch).

Inputs from the peripheral vestibular system are transmitted to the higher processing centers via the eighth cranial nerve, also known as the vestibulocochlear nerve. The afferent nerves from the utricle, saccule, and each semicircular canal are bundled together to form the vestibular nerve portion the vestibulocochlear nerve (Figure 1.5.3.5-1). The initial grouping of afferent neurons is based on their origin within the vestibular apparatus. The superior division of the vestibular nerve carries afferent fibers from the hair cells of the anterior and horizontal semicircular canals and utricle, while the inferior division carries afferent fibers from the posterior semicircular canal and saccule (Figure 1.5.3.5-1). The connections within the brain stem and associated vestibular nuclei are discussed in more detail below.

Figure 1.5.3.5-1. Divisions of the Vestibulocochlear Nerve

A slightly more detailed map of the vestibular neural pathways is shown in Figure 1.5.3.5-2.

Figure 1.5.3.5-2. Major Projections of the Vestibular Neural Pathway
1.5.4. Central Vestibular System (Brain Centers)

1.5.4.1. Cerebellum. The cerebellum lies at the base of the brain under the occipital lobe and just above the upper portion of the brain stem (Figure 1.5.3.5-2). It is comprised of at least three distinct areas (anterior lobe, posterior lobe, and the flocculonodular lobe) that are collectively concerned with subconscious control of movement. The ability of the cerebellum to influence motor inputs to maintain balance is due to its organization and connections to other parts of the central nervous system. While the cerebellum makes up roughly 10% of the total brain volume, it contains more than half of all its neurons. In addition, there are nearly 40 times more axons projecting into the cerebellum than leave it (Ghez and Thach, 2000). This provides the cerebellum with information about the goals, commands, and feedback regarding motion of the body and allows it to evaluate disparities between intention and action and provide input back to the motor centers in the cortex and brainstem, both during movement and in anticipation of movement (Ghez and Thach, 2000).

Motor commands are not initiated in the cerebellum, but rather it plays a major role in the timing of motor activities and helps to make adjustments to the body's motor activities while they are being executed (i.e., it provides real-time coordination so that the actual movements conform to the motor signals directed by the cerebral motor cortex and other parts of the brain) (Hall, 2011). If the directed and actual movements are in conflict, the cerebellum provides instantaneous corrections to the specific muscles via subconscious signaling. It also aids in planning sequential movements a fraction of a second before they are needed (Hall, 2011). This allows for smooth progression between movements, especially when rapidly changing from one direction to another. The cerebellum also plays an important role in motor learning by adapting and fine-tuning neuromuscular communication to improve such activities as hand-eye coordination.

A few of the vestibular afferents from the organs in the inner ear directly enter the cerebellum, while most vestibular inputs arrive in the cerebellum from the vestibular nuclei located in the brainstem (discussed in more detail below).

1.5.4.2. Vestibular Nuclei. The cell bodies of the first-order afferents from the individual semicircular canals, the utricle, and the saccule converge in the internal auditory meatus of the skull to form the vestibular, or Scarpa's, ganglion. Their axons form the vestibular nerve component of the vestibulocochlear nerve, which then enters the brain stem at the junction between the pons and the medulla. The majority of the vestibular afferents project to one of the four (superior, inferior, lateral, and medial) vestibular nuclei located in the rostral medulla and caudal pons (Figure 1.5.4.2-1).
The vestibular nuclei of the brainstem act as part of vestibular reflex arcs to provide input to the extraocular muscles to help stabilize vision, as well as postural muscles to maintain balance. Vestibular nuclei neurons use signals from the peripheral vestibular structures to differentiate between head translation and head tilt with respect to gravity (Lopez and Blanke, 2011). Information moves via the descending lateral or medial vestibulospinal tracts of the spinal cord to innervate the necessary musculature to maintain posture. Projections of the lateral vestibulospinal tract extend the length of the spinal cord and project to cervical, thoracic, and lumbar regions to provide excitatory tone to extensor muscles. This pathway is important for helping to maintain an upright posture while walking. The medial vestibulospinal tract projects predominately to the cervical segments and facilitates the vestibulocollic reflex discussed below.

There are numerous connections between the vestibular nuclei and higher brain centers, particularly the cerebellum. Projections from the vestibular nuclei extend to the thalamus and cortex, where they can give rise to conscious awareness of body orientation.

1.5.4.3. Cortex and Thalamus. The primary cortical receiving areas for the vestibular system are more diffuse than for other sensory systems, and there is some dispute about their locations. In general, vestibular inputs ultimately reach the primary vestibular projection area of the cerebral cortex that is located in the parietal and parietotemporal cortex (Parmet and Ercoline, 2008). This area of the cortex receives inputs from other sensory systems and provides for integration with higher-order motor activity and conscious awareness of orientation.

The thalamus plays an important role in relaying and modulating the flow of sensory information to the cortex (Lopez and Blanke, 2011). Second order afferents from the vestibular nuclei, as well as neurons from the cerebellar cortex, ascend to the thalamic nuclei. Input from the vestibular nuclei is integrated with information from proprioceptive and cutaneous receptors before being relayed to higher order areas of the cortex (Lopez and Blanke, 2011). While it has been demonstrated that the thalamic nuclei play an important role in integrating sensory input and relaying it to the cortex, it is unknown whether conscious attention and signals from the cortex to the thalamus can influence this thalamic processing (Lopez and Blanke, 2011).
1.5.5. Vestibular Reflexes

As previously stated, most vestibular inputs are processed with very little conscious awareness. Coordination of eye movements, postural inputs and corrections, and motor responses to changes in head and body position are all carried out via subconscious control through a number of reflexes. It takes up to 40 milliseconds for the eye to process the visual image and up to 100 milliseconds for the nerves to transmit this image to the cerebral cortex. The vestibular system, on the other hand, transmits its information in about 12 milliseconds (Previc, 2004; Cheung, 2004a).

While the focus when presenting vestibular system function is generally done in regards to its role as a balance system and for stabilizing gaze and posture, it contributes to a multitude of functions that range from automatic reflexes and more complicated motor strategies to spatial perception and path finding (Goldberg and Cullen, 2011). Only a brief overview of a few of the vestibular reflexes is provided here. For a more detailed description, the reader is directed to the Parmet AJ and Ercoline WR reference, as well as the Cheung, 2004a reference.

1.5.5.1. Vestibulo-ocular Reflex. As we move in our environment, it is necessary to compensate for head oscillations that occur during walking, running, or shaking your head. Without this compensation, we would see a blurry world due to the fact that natural head movements are of high frequency, while retinal processing is relatively slow. As a result, the visual system is unable to produce eye movements that are capable of stabilizing the retinal image (Cheung, 2004a). In order to compensate, the vestibulo-ocular reflex (VOR) provides input to the extraocular muscles that allow it to lag slightly behind the motion of the head, essentially slowing the movement of the eye to allow sufficient time for retinal processing of the image on the retina (Figure 1.5.5.1-1). The result is a stable retinal image. While semicircular canal inputs to the VOR are best understood (angular VOR), a similar reflexive response is associated with the otolith organs (translational VOR). Only angular VOR is presented here.

![Figure 1.5.5.1-1. Extraocular Muscles (Superior View, Right Eye)](image-url)
The vestibulo-ocular reflex is a gaze-stabilizing reflex. It relies on a three-neuron reflex arc connecting the vestibular organs, secondary vestibular neurons in the vestibular nuclei of the brainstem, and ocular motor neurons (Goldberg and Cullen, 2011). The VOR works by generating an eye movement that matches the velocity of the head movement (Hain and Helminski, 2012). Head rotation is detected by the semicircular canals and neural inputs are sent to the appropriate extraocular muscles to rotate the eyes in the opposite direction. For example, if you move your head to the left, you will excite the left horizontal canal. To keep your eyes fixed on a stationary point, you need to fire the right lateral rectus & the left medial rectus muscles. On the other side of the head, the right horizontal canal is wired to the complementary set of muscles. Since it is inhibited, it will not excite its target muscles (right medial rectus & left lateral rectus). The net result is to pull the eyes to the right and keep the image stable on the retina long enough for it to be processed (Figure 1.5.5.1-2). Without the VOR, it would be difficult to read signs or recognize individual features during movement.

Figure 1.5.5.1-2. The Vestibulo-Ocular Reflex

An easy demonstration of the VOR is to hold your finger up in front of your face. If you shake your finger rapidly back and forth, it appears blurry as the image of your finger rapidly moves across the retina. However, if you rapidly shake your head left and right while holding your finger still and focusing on it, the image of your finger appears stable, or fixed, due to the compensatory eye movements that rotate the eyeball independently of the head movements in order to keep the image stable on the retina.

The vestibulo-ocular reflex is adaptive in nature, meaning that the reflex is constantly updated as demands change during dynamic motion of the head in order to stabilize the retinal image (Jones, 2000). It also displays plasticity, in that it retains its ability to constantly adapt. Once adaptation has occurred, it will be maintained until a new condition occurs and readaptation takes place. During adolescence, when the head is still growing, the VOR is constantly adjusted to compensate for changes in head circumference (Goldberg et al., 2012). Later in life, it may undergo further adjustments to compensate for changes in vision and the wear of glasses or contact lenses. This VOR adaptation period may be noticeable until an individual becomes used to a new prescription.
1.5.5.2. Vestibulocollic Reflex. In order to have an accurate perception of our orientation relative to the earth, and to maintain balance with respect to it, it is necessary to keep the head still in space and on a level plane while walking or running. This is done through the vestibulocollic reflex (VCR), which functions to stabilize the head during unexpected movements by generation of inputs to move the head in the opposite direction of the initial movement (Goldberg and Cullen, 2011). It is elicited by the position of the head in different orientations with respect to gravity and works to ensure that the head and neck stay centered, steady, and upright on the shoulders. The VCR is activated by input from both the otolith organs and the semicircular canals and has a strong response to pitch stimuli (Wilson, 1991).

Information from the vestibular system, primarily by input from the otolith organs, is sent to the vestibular nuclei in the brainstem, then to the spinal cord centers that control the muscles of the neck. Similar to the VOR, the VCR relies on a three-neuron reflex arc connecting the vestibular organs, secondary vestibular neurons in the vestibular nuclei of the brainstem, and neck motor neurons. In addition to the reflex arc, there is strong evidence that more complex neural pathways also make dominant contributions to mediate the VCR. In contrast to the VOR, the VCR must control a complex musculature of the neck, with over 30 muscles controlling pitch, roll, and yaw rotations of the head (Goldberg and Cullen, 2011). Without the vestibulocollic reflex, the head would not automatically stay balanced and level.

1.5.5.3. Vestibulospinal Reflex. The vestibulospinal reflexes (VSR) are important for postural maintenance, equilibrium, and resting muscular tone. They help to keep the body upright relative to the earth by providing input to maintain a stable center of gravity during movement. As bipedal animals with a high center of gravity, humans are inherently unstable and require constant inputs to the postural muscles to maintain our vertical orientation. The VSR is initiated by inputs primarily from the otolith organs, although the semicircular canals are also involved (Gleason, 2008). Stimulation of the VSR results in motor inputs to a number of muscle pairs throughout the body in order to stabilize body position. Some of the muscles responsible for postural control include the abdominal muscles, paraspinal and back muscles, hip flexors, the hamstrings and quadriceps in the thigh, and the gastrocnemius and tibialis anterior in the calf.

1.5.5.4. Vestibulosympathetic Reflex. As discussed earlier, stimulation of the otolith organs due to head tilt increases muscle sympathetic nerve activity and helps to defend against orthostatic challenges (Ray and Monahan, 2002). Feedback via the vestibulosympathetic, or vestibulo-autonomic, reflex may act immediately to maintain blood pressure before cardiovascular baroreceptors detect a drop in blood pressure. The combination of vestibulosympathetic reflex and baroreceptor inputs has an additive effect on increasing smooth muscle sympathetic nerve activity, and therefore maintenance of blood pressure and blood flow (Ray and Monahan, 2002). It has been proposed that the vestibulosympathetic reflex also plays a role in cardiovascular adaptation to high +Gz (Newman, 1999).

The vestibulosympathetic reflex also plays a role in the on-set of motion sickness (Wolfe et al., 2012). It is thought that motion sickness arises partly due to inappropriate activation of the vestibulosympathetic pathways that attempt to return the body to a state of homeostasis following conflicting inputs between the visual, vestibular, and somatosensory systems (Goldberg et al., 2012).
1.5.6. Environmental Influences on Vestibular Function

1.5.6.1. Hypoxia. Hypoxia has well documented impacts on central nervous function and is discussed in more detail in Section 3.1. As it applies here, hypoxia’s direct influence on the peripheral vestibular system is not well established. Changes in balance that occur during hypoxia may be due to altered cerebral processing of vestibular inputs within the brainstem, within the cerebellum, and/or within the higher brain centers. In addition, balance issues could be the result of impaired neuromuscular communication between the central nervous system and the postural muscles responsible for maintaining balance (Wagner et al., 2011).

While debate still exists as to whether there are any physiological differences between normobaric hypoxia and hypobaric hypoxia, there is some evidence that a difference does exist in regards to postural stability. It has been well documented that hypobaric hypoxia alters vestibular inputs through multiple mechanisms; however, recent research indicates that postural instability may be more influenced by altered barometric pressure than by changes in the fraction of inspired oxygen (Degache et al., 2012).

1.5.6.2. Temperature Differences. Due to its location within the temporal bone of the skull, the vestibular system is relatively well protected from temperature changes relative to body temperature or differential temperatures between the two sets of peripheral vestibular structures. Generally, any change in temperature that affects one side of the head affects the other side in the same way. As a result, both sets of vestibular organs register the same conditions and there are no conflicting inputs to the brain. If a temperature differential does occur, the result is an unbalanced neural response (i.e., not equal and opposite) between the two systems.

Based on the anatomy and orientation of the vestibular system, the structure most commonly affected by temperature differentials is the horizontal semicircular canal. The ampulla of the horizontal SCC lies closest to the middle ear, and can therefore be influenced by changes in the air temperature of the middle ear cavity. This can occur as the temperature in the external auditory canal is transmitted across the tympanic membrane and then through both the air space and surrounding bone to the horizontal SCC. The altered temperature causes a change to the temperature of the endolymph fluid inside the membranous labyrinth, resulting in the formation of convection currents within the canal (Goldberg et al., 2012; Perrella and Caovilla, 2012). The warmer, less dense endolymph fluid rises within the membranous labyrinth while the colder, denser fluid sinks. This causes a deflection of the cupula and a neural discharge (Goldberg et al., 2012). The result of this stimulation of the horizontal SCC due to a caloric difference across the head is horizontal nystagmus. The effect is maximized when the head is lowered by 30° to bring the horizontal SCC parallel to the ground or when the head is raised 60° so that the horizontal SCC is vertical. When the horizontal SCC is vertical, the movement of the endolymph fluid and stimulation of the canal is perceived as rotation in the horizontal plane.

Caloric horizontal nystagmus can be induced by the introduction of either air or water down the external auditory canal to the tympanic membrane. Water temperatures of ±7°C from body temperature (30°C or 44°C) or air temperatures of ±13°C (24°C or 50°C) are typically used in clinical tests of vestibular function to stimulate a response (Perrella and Caovilla, 2012). When cold water (or air) is used to irrigate the external auditory canal, the fast phase of the nystagmus is opposite the
filled ear; when warm water (or air) is used, the fast phase of the nystagmus is towards the filled ear. The mnemonic COWS (cold, opposite; warm, same) can be used to predict the direction of the fast phase.

Examples of situations that may result in caloric nystagmus include exposure to cold air without proper head gear (such as skiing or running in cold weather) or during swimming or diving in cold water.

1.5.6.3. **Trapped Gases.** In a manner similar to caloric nystagmus, the vestibular system can be stimulated due to pressure differences between the middle ear cavities on the left and right side of the head. This can occur due to the expansion of trapped gases in the middle ear cavity or with the addition of additional pressure when performing the Valsalva maneuver (Wicks, 1989). The resulting condition is referred to as alternobaric vertigo, and may include sensations of spinning, rolling, pitching, or nystagmus. While the exact mechanism is not fully understood, it is most likely due to increased pressure in the middle ear cavity that is transmitted to the membranous labyrinth of the semicircular canals through the round window of the cochlea (Cheung, 2004b). This may occur due to the connections between the cochlea, the vestibule, and the semicircular canals that allow the endolymph fluid to flow between the various compartments (Figure 1.5.3.1-2). Normally, there is no pressure difference between the perilymph fluid and the endolymph fluid of the inner ear structures. As a result, the intra-labyrinthine pressure is sensitive to any change in intra-cranial pressure and transfers that pressure change to the receptors of the vestibular system (Kondrachuk and Boyle, 2011).

Typically, alternobaric vertigo is of short duration, lasting less than approximately 20 seconds; however, it has been reported to last for minutes (Cheung, 2004b). An aircrew member may experience alternobaric vertigo when they are unable to clear one ear when experiencing a change in pressure.

1.5.6.4. **Acceleration.** The structures most susceptible to changes in gravity are the otolith organs, as they respond to changes in the magnitude and direction of transient linear accelerations and static head tilt relative to gravity.

1.5.6.4.1. **Microgravity.** For the purposes of our discussion here, microgravity is considered anything less than normal 1 G exposure.

Exposure to microgravity leads to an alteration in the transduction and integration of signals from the systems responsible for normal orientation and body awareness. Until an individual adapts to the altered sensory inputs, the following conditions are common among astronauts (Legner, 2003):

1. Illusory self and/or surround motions.
2. Space motion sickness (SMS).
3. Eye-head coordination impairment.
4. Equilibrium control disturbance.
After return to normal gravity, many of these same problems exist during the re-adaptation period. The severity and duration of recovery depends on the duration of the microgravity exposure (Legner, 2003).

In microgravity, the peripheral vestibular structures are affected differently. Because the semicircular canals respond to angular acceleration, they are relatively unaffected by exposure to microgravity. The otolith organs, on the other hand, are significantly affected. In microgravity, without adequate gravitational force to act on the otoliths, signals regarding head tilt become meaningless (Legner, 2003). As a result, they are no longer capable of indicating static head position. The postural reflexes that rely on otolithic inputs are degraded while in microgravity and perception of static limb position becomes impaired. The vestibulo-ocular reflex, which plays an important role in helping to stabilize the retinal image, is negatively impacted during weightlessness due to the lack of otolithic inputs (Reschke et al., 1996).

1.5.6.4.2. Hypergravity. For the purposes of our discussion here, hypergravity is considered anything greater than normal 1 G exposure. In addition, sustained hypergravity refers to the type of exposures that may be experienced during aerial maneuvers. Chronic, or prolonged, hypergravity refers to exposures that occur under research conditions, can last for days or weeks, and deal primarily with animal models.

While humans have evolved in response to a 1 G terrestrial environment, our physiological systems and homeostatic mechanisms have the capability to rapidly respond to and accommodate increases in G (Burton and Smith, 2011). Sustained hypergravity exposure alters the stimulation of, and input from, the otolith organs. Under normal 1 G loading, linear acceleration or head tilt is determined by the magnitude of otolith movement relative to the macula. When exposed to hypergravity conditions, the increased magnitude of the force acting on the otoliths increases their shearing action and results in a much larger deflection of the hair cells. (Figure 1.5.3.3-3). The result is a sensation that the resultant vector has moved farther away from the vertical centerline and leads to a misperception of attitude. Linear acceleration, bank angle, and rate of climb can all add an additional acceleration/G component that further increases the magnitude of otolith shift and misperception. The end result leads to a somatogravic illusion, such as the G-Excess effect, which is discussed in more detail in Section 7.3.

Chronic exposure to hypergravity studies have typically looked at the effects on non-vestibular systems, such as the musculoskeletal and cardiovascular systems. Relatively little research has been conducted on long-term exposure to hypergravity and the effects on vestibular structure or function. From the few animal studies that have been done, it appears that the functioning of mature peripheral vestibular structures is not negatively affected after long-term exposure to hypergravity, with fully mature vestibular sensory epithelium qualitatively unaffected after a prolonged exposure to 2.5 G (Wubbels et al., 2002). Structurally, hair cell structure remains intact with no changes in the cytoskeletal elements. In the vestibular nuclei, exposure to hypergravity during development appears to advance the maturation; microgravity delays it (Jamon, 2014). Hypergravity also appears to lead to changes in cerebellar development.

Depending on the timing of exposure to hypergravity during development, the effects can vary from no effects to decreases in otoconia size to delayed synaptic stabilization of hair cells (Jamon, 2014). It appears that any long-term effects from
hypergravity exposure during development primarily impact the otolith organs and related otolith reflexes.

The vestibular system’s large dynamic range and adaptation mechanisms allow it to compensate for the hypergravity stimulus (Wubbels et al., 2002). Any altered postural responses after return to normal G are transient, with normal function typically returning within a few hours, although some centrifuged birds took up to a few weeks to return to normal (Burton and Smith, 2011).

1.5.6.4.3. Centrifuge Exposure. One other potential issue that can occur due to exposure to linear accelerations or following exposure in a centrifuge is a condition called cupulolithiasis. This condition occurs when otoconia become dislodged and are introduced into the posterior semicircular canal, where they settle on the cupula and alter its density relative to the endolymph fluid (Brandt, 1990). Spontaneous degeneration of the otolithic membrane, such as can occur with age or head trauma, may also result in debris being introduced into the semicircular canals (Brandt, 1990). Inorganic particles that don’t attach to the cupula, but remain loose within the endolymph fluid of the membranous labyrinth, lead to a condition referred to as canalithiasis. Collectively, cupulolithiasis and canalithiasis are associated with benign paroxysmal positional vertigo (Lee and Kim, 2010). Although any of the semicircular canals can be affected, the posterior semicircular canal is the most common location for the dislodged otoconia to settle due to its location relative to the utricle. Symptoms are typically a spinning sensation, or vertigo, induced by head movements, especially when getting out of bed, rolling over in bed, tilting the head back, or when bending forward (Lee and Kim, 2010). Light-headedness, postural instability, nausea, non-specific dizziness, and nystagmus may also occur (Lee and Kim, 2010; Brandt, 1990).

1.5.6.5. Alcohol. Alcohol, as well as some medications, affects the neural centers located in the brainstem and cerebellum that control eye movements, motor, sensory, and cognitive integration areas in the brain (Citek et al., 2003). The actions of alcohol on these central functions are the result of pharmacological interactions with ion channels and neurotransmitter receptors, while also having a direct physical effect on the peripheral vestibular structures (Goldberg et al., 2012). The vestibular effects from alcohol include postural instability and positional alcohol nystagmus (PAN).

The specialized structures of the semicircular canals, such as the crista ampullaris, are more highly vascularized than the rest of the membranous labyrinth. After being consumed and entering the blood, alcohol diffuses from the blood to the crista ampullaris and is ultimately absorbed into the cupula. The alcohol lowers the density of the cupula relative to the surrounding endolymph fluid and as a result, the cupula becomes buoyant relative to the crista ampullaris (Goldberg et al., 2012; Kondrachuk et al., 2008). Typically, the cupula of the SCC is not sensitive to linear accelerative forces on the head. However, due to its alcohol-altered density, the cupula can now be displaced when the head is tilted relative to gravity. The resulting stimulation is perceived as rotation, with the illusionary rotation leading to a feeling of postural instability. This condition is further complicated by the expectation of the brain, arising from signals from the otolith organs, somatosensory receptors, and visual system, that the body is rotating. When the inputs from these systems do not confirm the rotation, the conflicting signals can lead to motion sickness (Goldberg et al., 2012).
Over time, alcohol diffuses into the endolymph fluid surrounding the cupula and the density of both components equalizes. When this occurs, the sensation of motion will lessen or disappear. As the body continues to clear the alcohol, the alcohol in the cupula is cleared faster than from the endolymph fluid. This results in a density imbalance between the cupula and surrounding endolymph fluid, and as a result, the associated symptoms may return. However, during this period the sensation of rotation will be in the opposite direction relative to the initial symptoms (Goldberg et al., 2012). Positional alcohol nystagmus can be present in both density phases. It has been demonstrated that at a blood alcohol concentration (BAC) as low as 0.027%, well below most legal limits for driving or flying, the ability to visually suppress inappropriate nystagmus is impaired (Cheung, 2004b). The result is degraded visual function regardless of other effects on postural stability.

Of particular interest to the aviation community is the length of time that the effects of alcohol on the vestibular system, both the central centers and the peripheral vestibular structures, can persist. Even after the blood alcohol concentration has returned to zero, there continues to be measurable effects on the brain and vestibular system. The effects of alcohol may last up to 34 hours or more after drinking and increase the risk of disorientation and motion sickness (Cheung, 2004b). This is well beyond the 12 hour “bottle-to-throttle” rule commonly employed by aircrew.

1.5.7. Summary

This chapter summarized the structures and functions of the vestibular system and provided a basic overview its role in maintaining orientation. The functional unit of the vestibular system is the specialized hair cells that act as mechanoreceptors to convert the physical deflection of the stereocilia into neural signals that are transmitted to various locations in the brainstem and brain. The otolith organs (utricle and saccule) respond to changes in linear acceleration and static head tilt relative to gravity, while the semicircular canals respond to changes in angular acceleration. Inputs from the vestibular system are processed primarily at the subconscious level and provide input for vestibular reflexes that connect the vestibular system with the postural muscles for maintaining balance and orientation and eye movements for stabilizing the retinal image, as well as inputs to the cardiovascular system to help regulate blood flow and pressure. While its location within the temporal bone of the skull helps to stabilize and protect the vestibular structures, environmental factors can alter the normal function of the vestibular system.

References


**Concepts**
- Depolarization / Hyperpolarization of vestibular sensory cells
- Environmental effects on vestibular function
- Innervation of peripheral vestibular system
- Vestibular reflexes
- Vestibulo-ocular reflex (VOR)

**Vocabulary**
- Ampulla
- Bony labyrinth
- Cupula
- Kinocilium
- Macula
- Membranous labyrinth
- Otolith organ
- Otolithic membrane
- Saccule
- Semicircular canal
- Stereocilia
- Utricle
1.6. Somatosensory Physiology

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1.6.1. Introduction

As has been previously presented, input from multiple receptors located throughout the body must continuously be received and integrated in order to maintain our sense of balance and orientation. Postural maintenance and self-motion perception are based on simultaneous stimulation of the visual, vestibular, and somatosensory systems (Cheung, 2004). So far, the visual and vestibular systems have been presented and their contributions to balance and orientation discussed. The final orientation system, the somatosensory system, is composed of the non-visual and non-vestibular receptors located throughout the body that provide the brain sensory inputs for pressure and stretch, primarily in response to gravity, but also information about temperature, pain, and limb location. In some ways, the sense of touch, or somatosensation, is the most complex spatial sense due to the number of different receptors and the sensations that they detect (Lindeman et al., 2006). Additionally, the sense of touch is the only one that conducts both sensing and actuation. Figure 1.6.1-1 outlines the relationship of the somatic senses.

![Figure 1.6.1-1. Taxonomy Related to Touch (Myles and Binseel, 2009)](image)

This chapter is intended to provide a basic overview of somatosensory receptors and their functions to ensure the reader has a general understanding of the inputs that aid the central nervous system in knowing the relative motion and position of the limbs. This background will hopefully aid in understanding some of the illusions and other problems associated with the aviation environment discussed in later chapters.
1.6.2. Terminology

Kinesthesis refers to the perception of the position and movement of the limbs in three-dimensional space, with internal sensations arising from joints, tendons, and muscles. Inclusion of vestibular inputs with kinesthetic inputs from the internal environment is known as proprioception. It involves the processes, both voluntary and involuntary, by which the body varies muscle contractions to immediately respond to changing body or limb position (Sargant, 2000). Technically, the term somatosensation, or somatosensory, refers, collectively, to all the sensory signals from the body (Wolfe et al., 2012). The term somatosensory refers to inputs that are not a special sense (sight, hearing, taste, smell, vestibular).

1.6.3. Types of Receptors

The receptors located throughout the body respond to changes in both limb position and acceleration (Figure 1.6.3-1). Evidence suggests that previous exposure or experience helps to pre-program muscle activity patterns by allowing for a state of muscle tension to exist before actually activated. This allows for rapid response and compensation that is critical for maintaining dynamic joint stability (Sargant, 2000).

Figure 1.6.3-1. Proprioceptive Receptors

The somatosensory system consists of several different and distinct types of receptors corresponding to their specific function. These can be grouped into the exteroceptors that sense and respond to stimuli from outside the body, and the enteroceptors that detect changes that occur inside the body. We are primarily concerned with the orientation inputs that are provided from the exteroceptors.

Somatosensory input can be categorized based on the information provided to the spinal cord and higher brain centers. This information is then processed at both the subconscious and conscious levels. In general, there are four major somatosensory modalities (i.e., perceptual characteristics) (Gardner et al., 2000):
1. **Discriminative touch.** This allows for recognition of size, shape, and texture of objects and their movement across the skin.

2. **Proprioception.** Provides for the sense of static position of the body and the movement of limbs and body. Consists of two sub-modalities:
   a. Stationary position of the limbs (limb-position sense).
   b. Sense of limb motion (kinesthesia).

3. **Nociception.** Signals damage to tissues or chemical irritation, typically perceived as pain or itch.

4. **Temperature sense.** Provides for the sensation of warm and cold.

1.6.3.1. **Cutaneous and Subcutaneous Mechanoreceptors.** The skin contains a variety of sensory receptors that are specialized to relay information to the brain. Many of these cutaneous receptors respond to mechanical pressure (mechanoreceptors), while others respond to vibration, pain (nociceptors), and temperature (thermoreceptors). While the cutaneous mechanoreceptors are typically not associated with proprioceptive inputs, they are important for discriminative touch (tactile input) and helping to provide inputs on limb location and movement and therefore aid in kinesthetic awareness. As we are concerned with the somatosensory inputs as they pertain to orientation, we will focus our discussion here on the primary mechanoreceptors (Figure 1.6.3.1-1).

![Figure 1.6.3.1-1. Cutaneous and Subcutaneous Mechanoreceptors](image)

1.6.3.1.1. **Meissner’s Corpuscle.** Meissner’s corpuscles are found at the junction of the epidermis and dermis and have a small receptive field (Wolfe et al., 2012). The Meissner’s corpuscle is a rapidly adapting receptor that is located in the superficial layers of the dermis and responses to deformation of the skin. The receptor portion is a fluid-filled, globular structure than encloses a stack of flattened epithelial cells with the sensory nerve ending entwined between the layers (Gardner et al., 2000).
Rapidly adapting mechanoreceptors have lower stimulation thresholds and are therefore able to detect small vibrations or deformations of the skin. They are also more sensitive to the motion of objects across the skin. They respond by increasing their neural firing rate in proportion to the speed of motion and the duration of neural firing indicates the duration of motion.

1.6.3.1.2. Merkel Disk Receptor. Merkel disk receptors are found at the junction of the epidermis and dermis and have a small receptive field (Wolfe et al., 2012). In contrast to the Meissner's corpuscle, Merkel disk receptors are slow adapting receptors composed of a small epithelial cell wrapped around a nerve ending. It conveys compression strain from the skin by evoking sustained, slowly adapting responses (Gardner et al., 2000).

1.6.3.1.3. Pacinian Corpuscle. Pacinian corpuscles are located deep within the dermis layer of the skin. In relation to the other types of mechanoreceptors, the Pacinian corpuscle is relatively large with a large receptive field. It consists of a nerve ending covered in layers of connective tissue that give it an appearance similar to an onion. The Pacinian corpuscle is activated by rapid indentation of the connective tissue lamellae when the skin is compressed (Gardner et al., 2000). Similar to the Meissner's corpuscle, the Pacinian corpuscle detects compression of the papillary ridges of the skin, but do so over a larger area. These mechanoreceptors also respond to vibrations, especially vibrations in the 200 – 300 Hz range, and are able to detect vibrations that occur several centimeters away (Gardner et al., 2000).

In addition to their location in the skin, Pacinian corpuscles are also located between layers of muscle and on the interosseous membranes (Gardner et al., 2000).

1.6.3.1.4. Ruffini Ending. Like the Pacinian corpuscles, the Ruffini endings are located deep within the subcutaneous layer of the dermis. Ruffini endings are slowly adapting mechanoreceptors that link the subcutaneous tissue located in the folds of skin found in joints, as well as in the palms or the fingernails (Gardner et al., 2000). They are stimulated by compression of the nerve endings that occurs during skin stretch or bending of the fingernails. They play an important role in providing information regarding the shape of an object when grasped.

1.6.3.2. Muscle Mechanoreceptors. Muscle mechanoreceptors provide input to the central nervous system as to limb location, movement, and joint angle. As a result, they are collectively referred to as kinesthetic receptors (Wolfe et al., 2012). Below is a brief discussion of the primary muscle mechanoreceptors.

1.6.3.2.1. Muscle Spindle Receptors. Muscle spindles play a crucial role in the ability to maintain precise control of muscle activity. They function as stretch receptors to provide information about the length of the muscle and the velocity at which it is being stretched (Sargant, 2000).

Muscle spindle receptors lie within the belly of the long axis of the muscle. They consist of a collagenous capsule that is continuous with the fibrous tissues that separates individual muscle fibers, or extrafusal fibers (Figure 1.6.3.2.1-1).
Figure 1.6.3.2.1-1. Muscle Spindle Receptor

Contained within the capsule are specialized muscle fibers, known as intrafusal fibers. The central portion of the intrafusal fibers is non-contractile and is wrapped with the ends of sensory afferent neurons (Sherwood, 2001). These primary receptors are responsible for detecting changes in the length of the fibers when stretched, as well as the rate of stretching. A second component of the muscle spindle, known as a secondary receptor, only responds to changes in length and is located closer to the ends of the intrafusal fibers (Sherwood, 2001). When stretched, a neural signal is sent via the afferent sensory neurons, and along with the motor neurons that innervate the extrafusal muscle fibers, forms part of a negative feedback loop that works to resist muscle stretch (Figure 1.6.3.2.1-2).

Figure 1.6.3.2.1-2. Muscle Spindle Feedback Loop

Muscle spindle receptors are located in all skeletal muscles, with increased density in those muscles that are responsible for maintaining posture. The density of muscle spindle receptors in a muscle is dependent on the type of movement the muscle is involved in. Muscles that are used to perform delicate, precise movements contain a higher density of muscle spindles than those muscles involved in more gross motor movements (Sargant, 2000). In addition, for the brain to accurately determine the location of a particular joint or limb, input from more than one muscle spindle is
necessary. This multiple input requirement allows for the determination of joint angles, acceleration, and position (Sargant, 2000).

1.6.3.2.2. **Golgi Tendon Organ.** Unlike the muscle spindle receptors, Golgi tendon organs are located in the tendons that attach the muscle to the bone. From here, they are able to provide input regarding muscle tension. Structurally, Golgi tendon organs are much simpler than muscle spindles (Sargant, 2000). The Golgi tendon organ consists of afferent nerve endings woven into bundles of connective tissue that form the tendon (Sherwood, 2001) (Figure 1.6.3.2.2-1). The intertwined orientation of the nerve endings and collagen fibers makes the Golgi tendon organ very sensitive to small changes in muscle function (Sargant, 2000). Functionally, the Golgi tendon organ is very similar to the muscle spindles discussed above, but rather than detecting muscle stretch, they respond to changes in muscle tension. The two structures have opposing functions that work together to prevent muscle damage.

Unlike most other sensory neurons, the Golgi tendon organs do not have a resting discharge (Sargant, 2000). Neuronal firing only occurs when the tension in the muscle is very high, such as when the entire muscle undergoes a maximal contraction. When stimulated, a neural signal is sent via the afferent sensory neurons to the spinal cord. A signal is then transmitted via an efferent motor neuron that innervates the extrafusal muscle fibers to cause muscle relaxation and reduce the tension on the muscle and tendon. This forms a negative feedback loop that works to prevent muscle damage by limiting muscle tension, in much the same way that the muscle spindles work to prevent muscle damage by limiting muscle stretch.

![Golgi Tendon Organ](image)

**Figure 1.6.3.2.2-1. Golgi Tendon Organ**

1.6.3.2.3. **Joint Capsule Mechanoreceptors.** In addition to the receptors located within the muscles and tendons that provide information about muscle performance, receptors are also located within the joints and joint capsules. Joint capsule receptors are located within the connective tissues of the capsule, the ligaments, and fat pads that comprise most joints. These receptors provide information about joint angle, location, movement, and stress in regards to the components of individual joints (Sargant, 2000). While the threshold for stimulation of joint receptors is similar in all joints, distal joints (such as the fingers) are more readily perceived than more proximal joints such as the shoulder.
There are four types of joint capsule receptors, with Types I, II, and III described as true joint receptors, while Type IV is viewed more as a type of pain receptor (nociceptor) (Sargant, 2000). The morphology differs between the different types of joint mechanoreceptors, with many similarities to Pacinian corpuscles, Ruffini endings, free nerve endings, and Golgi tendon organs. Figure 1.6.3.2.3-1 shows a general representation of joint receptors. The differences in morphology allow for specialization, with some receptors providing input regarding position, while others provide input on movement or joint angle.

Figure 1.6.3.2.3-1. Joint Receptors

1.6.3.3. Thermal Receptors. As our focus in this chapter is on those receptors involved with providing proprioceptive input, only a brief description of thermoreceptors will be presented here. A complete discussion on thermoregulation can be found in Section 7.8.

There are two types of peripheral receptors found in the skin that respond to thermal inputs: cold receptors and warm receptors. With this set of receptors, humans are able to detect four distinct thermal sensations: 1) cold, 2) cool, 3) warm, and 4) hot (Gardner et al., 2000). Both cold and warm receptors display a tonic neural discharge consisting of a slow rate of action potentials when the skin is at a normal temperature of about 34°C. When stimulated, the afferent nerve fibers from the thermal receptors transmit the sensory information to the temperature regulation center in the hypothalamus. Here, skin temperature input is integrated with information regarding the core body temperature and compares it to the body’s normal set point (approximately 37°C or 98.6°F) (Sherwood, 2001). The hypothalamus is able to detect and respond to changes in body temperature (i.e., blood temperature) as low as 0.01°C.

Cold receptors are stimulated at temperatures starting around 30°C (86°F). When stimulated, cold receptors initiate an increase in neuronal firing rate, reaching a maximum discharge around 25°C (77°F) (Gardner et al., 2000; Wolfe et al., 2012). As the temperature drops, the sensation of cold is replaced by the sensation of pain (Figure 1.6.3.3-1). Some cold receptors can also be stimulated by compounds such as menthol and result in a sensation of cold (Belmonte and Viana, 2008).
Warm receptors are stimulated at temperatures starting around 34°C (93°F) and fire maximally at 45°C (113°F) (Gardner et al., 2000). Above 50°C (122°F), the sensation is no longer felt at heat, but rather pain (Figure 1.6.3.3-1).

1.6.3.4. Nociceptors. As our focus in this chapter is on those receptors involved with providing proprioceptive input, only a brief description of nociceptors will be presented here.

Nociceptors are receptors that respond to stimuli that are capable of damaging tissues. Nociceptors are able to respond directly to some stimuli, while most respond indirectly to tissue damage by detecting chemicals released from traumatized cells (Gardner et al., 2000). There are three classes of nociceptors that are determined by the type of stimuli they respond to: 1) mechanical, 2) thermal, and 3) polymodal nociceptors.

Mechanical nociceptors consist of bare nerve endings with a myelinated axon. They are fast conducting receptors that require strong, tactile stimuli to be activated (Gardner et al., 2000). They are activated by stimuli that penetrate, squeeze, or pinch the skin by increasing their firing rate.

Thermal nociceptors are stimulated primarily by extreme temperatures, but can also be stimulated by strong mechanical stimuli (Gardner et al., 2000). As mentioned above, one group is activated by cold temperatures starting at approximately 15°C (59°F), while another group is activated by warm temperatures starting around 40°C (104°F) (Figure 1.6.3.3-1) (Belmonte and Viana, 2008).

Polymodal nociceptors can respond to mechanical, thermal, and chemical stimuli that results in tissue damage (Gardner et al., 2000). When activated, they provide a sensation of slow, burning pain. They are insensitive to light pressure or inputs.

1.6.4. Innervation

While each of the somatosensory modalities listed above has a separate and distinct pathway to the brain, they all utilize dorsal root ganglion neurons. The peripheral terminals of the individual dorsal root ganglion neurons respond to different stimuli due to their morphological and molecular specialization (Gardner et al., 2000).
These differences are presented above in the discussion of the individual receptors. In general, the cell body is located in the ganglion of the dorsal root of a spinal nerve (Figure1.6.4-1). Its axon has two branches (pseudounipolar), with one projecting out to the peripheral tissues and the other projecting into the spinal cord or brainstem of the central nervous system (Gardner et al., 2000). The peripheral terminal end of the neuron consists of either a bare nerve ending or is encapsulated in non-neural tissue. The mechanoceptors responsible for proprioception and touch discrimination contain myelinated axons that allow them to transmit sensory signals rapidly to the central nervous system. The length and diameter of the myelinated axons vary depending on the location and type of receptor.

![Figure 1.6.4-1. Receptor Neural Pathway](image)

When stimulated, the receptor initiates an action potential along the afferent neuron to the spinal cord. If an immediate response is necessary, a signal is sent via an efferent neuron to the effector as part of a spinal reflex arc. In some cases, coordination with higher brain centers occurs so that there is conscious awareness of receptor activation. An example is when a hot plate is touched. Other inputs from receptors are processed subconsciously, such as inputs necessary for maintaining balance.

### 1.6.5. Somatosensory Input in Aviation

In aviation, the term “seat-of-the-pants” is often used when describing inputs from the somatosensory system. It has its origins in the early days of flying when instruments were either not readily available or utilized and pilots would rely on the sensations of muscle stretch and pressure due to gravity to help orient them with the vertical. Unfortunately, the inputs from the mechanoceptors are only capable of providing accurate orientation cues when the individual is in direct contact with the ground. As a result, it is generally felt that the somatosensory system is the least reliable system for providing accurate orientation inputs in the aviation environment.

As with the vestibular system, our somatosensory inputs evolved to operate in a 1 G environment with a constant fixed reference point. On the ground, the gravitoinertial force (GIF), or the resultant force that represents the sum of the gravity vector and acceleration of the body, provides fairly accurate information due to the relatively slow linear accelerations that humans are exposed to. However, the somatosensory system was not designed to work in the three dimensional environment.
found in aviation (Cheung, 2004). The greater linear and angular accelerations that act on the somatosensory receptors are able to inappropriately stimulate them, leading to inaccurate inputs to the central nervous system. When inverted, accelerative forces can push a pilot into the seat. The pressure on the back of the thighs and the gluteal surface can be misinterpreted as the downward pull of gravity, leading to a sensation of being upright if other orientational inputs are not available or utilized. Input from other gravity-dependent receptors (i.e. the otolith organs) often reinforces somatosensory inputs, even if the information is incorrect, and can lead to spatial disorientation (Cheung, 2004). Other somatosensory illusions are discussed in more detail in Section 7.3.

While somatosensory inputs are unreliable in flight, they can provide information about aircraft performance. The main advantage that tactile inputs provide over other senses is the large area of possible stimulation, a relatively large dynamic range, and natural directional capabilities (Myles and Binseel, 2009). Aircraft or engine vibrations are detected through stimulation of mechanoreceptors located throughout the body, while feedback from the control surfaces is relayed through the fingertips to the central nervous system. One example of where somatosensory input is used to alert a pilot is the “stick shaker” warning (Cheung, 2004).

Another example of where somatosensory input has been used to increase pilot awareness is the Tactile Situation Awareness System (TSAS) initially developed by NASA and the US Navy. The military requirements driving its development stemmed from the loss of situational awareness and spatial disorientation problems arising primarily in the aerospace (both flying and microgravity) and diving environments, but that can also occur in ground operations (Rupert et al., 2002).

The Tactile Situation Awareness System consists of an array of stimulators, or tactors, that provide vibratory stimulation around the torso to provide aircraft attitude cues (Figure 1.6.5-1) (Rupert, 2000). The full TSAS configuration consists of an upper-body vest, shoulder straps and a seat that all respond to hardware and software in the aircraft (Curry et al., 2008). There are two types of tactors, pneumatic and electromagnetic, that are utilized in different areas of the body to provide differential input to the pilot (Rupert et al., 2002). The pneumatic tactors are contained within a cooling vest, while the electromagnetic tactors are located under the thighs and on top of the shoulders. A TSAS-Lite version has also been developed, consisting of eight tactors located around the waist to reduce the bulk, cost, and maintenance requirements of the TSAS vest (Figure 1.6.5-2). It has been shown to effectively provide helicopter crews directional cues while hovering over dynamic targets, while diminishing the perceived pilot workload and allowing the aircrew to safely maintain a closer position to the target than without the TSAS-Lite (Kelley et al., 2013).
By providing tactile information via the under-utilized sense of touch, a pilot can maintain orientation while looking away from the flight instruments (Kelley et al., 2013). While the intent was to utilize the system in all types of aircraft to augment visual and auditory displays, it has shown the most promise in rotary-wing aircraft, especially during hover or rescue operations. In addition to attitude cues, TSAS is capable of providing drift direction, body position, velocity, navigation, acceleration, threat location, and target location (Kelley et al., 2013; Rupert and Lawson, 2010). Information is provided to the pilot in an intuitive manner by alternating the pattern of tactor vibrations. Simulator studies using the TSAS while flying and hovering with night vision goggles (NVGs) demonstrated a 22% reduction in horizontal position error and a 41% reduction in vertical position error (van Erp et al., 2002). The study also showed that the TSAS reduced horizontal position error by 32% and vertical position error by 63% under full vision conditions. The TSAS was successfully shown to increase pilot situation awareness and reduce pilot workload, especially during complex flight conditions in poor visibility, as well as enhancing control of hover maneuvers in the Joint Strike Fighter (JSF) V/STOL aircraft as part of the JSF TSAS flight demonstration (McGrath et al., 2004). The TSAS has shown the same advantages during land-based deck landings, indicating it would be beneficial in high sea state environments (Jennings et al., 2004).

In addition to the potential aviation advantages, it has been proposed that such tactile input may be beneficial in special operations such as urban warfare and underwater operations, space flight operations such as extravehicular activity and orientation in microgravity, clinical applications for the elderly and those with vestibular disorders or traumatic brain injuries, and the entertainment industry (Rupert, 2000; Alahakone and Senanayake, 2009). TSAS configurations have also demonstrated effectiveness in unmanned aerial vehicle (UAV) operations and high-altitude high-
opening (HAHO) parachuting operations (Rupert et al., 2002). In the HAHO and underwater environments, the TSAS has shown that navigation can be performed faster with tactile cues than with visual cues, and with a greater degree of accuracy and less mental fatigue (Chiasson et al., 2002). It also has application for dismounted soldiers by providing directional cueing and target/threat location information while doing so with less competition for cognitive-perceptual resources and virtually silent operation (Brill et al., 2004). These are significant advantages when operating at night and when noise discipline is essential.

While the TSAS has demonstrated that it can help improve pilot performance and reduce spatial disorientation, it does have some downsides. The complete TSAS array is bulky, hot, expensive, and difficult to maintain, especially in harsh field conditions (Curry et al., 2008). As a result, it has not been fully integrated into real-world operational missions. In 2014, the DoD Coalition Warfare Program awarded the U.S. Army Aeromedical Research Laboratory funding to continue TSAS development and prepare it for delivery to the aviation rotary-wing community (Lawson and Rupert, 2014).

1.6.6. Auditory Inputs to Orientation

Before leaving our discussion on the systems involved with providing orientation inputs, the contribution of the auditory system should be briefly mentioned. While auditory inputs play an important role in helping to build situational awareness, they are extremely limited in helping to orient an individual with regards to the ground. Auditory cues regarding aircraft orientation and location typically provide geographic orientation (Cheung, 2004). Despite limited contributions to orientation, auditory cues can lead to illusions of self-motion, such as the audiogravic illusion, although this is much more likely to occur in the absence of visual cues than with them.

Research involving the use of three-dimensional auditory cues has shown limited success, with improvements in target acquisition, speech intelligibility, situational awareness, and decreased workload relative to a no-visual and no-sound environment (Cheung, 2004). These improvements, however, are not significant when used in conjunction with visual displays. Further research in the field of three-dimensional auditory cues for indicating attitude and orientation is necessary before it is at the same level as visual displays.

1.6.7. Summary

This chapter provided a basic overview of the somatosensory receptors located throughout the body. The various receptors provide sensory input regarding objects that come in contact with the skin (touch), body position and movement (proprioception), and the internal environment (visceral sense). Most sensory information is processed and responded to subconsciously via spinal reflex arcs, with additional integration/awareness provided by higher brain centers.

While it is generally accepted that the somatosensory system is the least reliable sense in the aerospace environment for providing accurate orientational cues, it can be utilized to provide targeting/threat information, navigational cues, and increased situational awareness regarding aircraft performance.
References
Concepts
Negative feedback loop
Seat-of-the-pants
Spinal reflex arc
Tactile Situation Awareness System

Vocabulary
Golgi tendon organ
Kinesthesia
Mechanoreceptors
Meissner's corpuscles
Merkel disk receptor
Muscle spindle
Nociceptors
Pacinian corpuscle
Proprioception
Ruffini ending
Somatosensory
1.7. Space Physiology

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1.7.1. Introduction

Over the past 50+ years, the National Aeronautics and Space Administration (NASA), its international partners, and the aerospace community at large have learned much about the effects of spaceflight and weightlessness on humans through increasingly longer duration missions and exposures. Crewmembers now routinely spend about six months at a time exposed to the effects of weightlessness while on board the International Space Station (ISS), orbiting about 250 miles above Earth. The first of what will likely be multiple one-year missions on board ISS has begun. Each crewmember serves as a test subject to improve the body of knowledge regarding the consequences of the space environment on human physiology (Figure 1.7.1-1). To eventually explore Mars, these effects must be understood so that risks can be recognized and mitigated either through engineering or human health countermeasures.

![Figure 1.7.1-1. General Physiological Consequences of Weightlessness on the Human Body](http://www.zerog2002.de/bodyreactions.html)

Volumes of books and literature on these topics have been published. This chapter will serve as a synopsis of current knowledge of the effects of spaceflight on all aspects of human physiology, as well as a discussion of the ongoing research that is being done in order to plan for safe human exploration further than low earth orbit.

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1.7.2. Bone Physiology and Countermeasures in Spaceflight
Robert Mulcahy, MD, MPH

Since the commencement of manned spaceflight, bone and calcium loss during flight have commanded attention. NASA plans for forthcoming exploration missions to Mars (in the 2030s) and to an asteroid (in 2025), which will entail long periods in reduced gravity, so concern for skeletal changes will persist for the foreseeable future. For instance, the major current skeletal risks identified by NASA include: fracture (both traumatic and fragility due to osteoporosis), renal stone formation due to hypercalciuria, and intervertebral disc injury. A trip to Mars will take about 6 – 9 months (based on current technology), so if bone loss is not mitigated, astronauts would be at high risk for fracture and injury when gravity is reintroduced and work commences at the destination as well as at increased lifetime risk after return to Earth.

Variations to bone composition and strength develop during exposure to reduced gravity. These changes become more pronounced on long-duration missions such as a 6-month or longer stay on board the ISS or a much longer exploration-class mission. Several factors may influence these alterations: low light with reduced vitamin D levels, high ambient CO\textsubscript{2} causing acidosis, reduced skeletal loading, and high dietary sodium intake (Buckey, 2006; Heer, 2002). Soon after arrival on orbit, bone resorption increases, as evidenced by elevated urinary calcium and N-telopeptide (bone breakdown marker); these changes persist until after return to Earth (Sibonga, 2013). Studies suggest that it takes much longer to regain bone loss than to lose it; it may take up to 3 years to regain 50% of the loss (Sibonga, 2013). Furthermore, there is no commiserate increase in bone formation (bone-specific alkaline phosphatase is reduced or unchanged during flight), leading to net loss of bone mass (Buckey, 2006; Orwoll et al., 2013). Studies have demonstrated a 0.3% loss of total body calcium per month (Buckey, 2006). However, this loss is not distributed equally; there is preferential bone mineral density (BMD) loss from weight-bearing bones (i.e. lower extremities and lumbar spine) with some measures demonstrating up to 1.7% decrease in BMD per month at the hip (average 1.0 to 1.5% per month) (Figure 1.7.2-1) (Buckey, 2006; Sibonga, 2013). Typical age-related bone loss occurs at 0.5 to 1.0% per year on Earth. This accelerated hip demineralization may increase risk of fractures, which is especially troubling given increased mechanical loading upon landing after an extended transit time to Mars or on return to Earth.
Figure 1.7.2-1. Declines in DXA aBMD in Long-Duration Astronauts on Mir and ISS Spaceflights. Percentage change of preflight areal BMD per month was calculated by subtracting the first post flight DXA BMD measurement from the preflight measurement and normalizing by the mission duration (typically 4 – 6 months). The Advanced Resistive Exercise Device (ARED) is exercise hardware capable of providing up to 600 pounds of force (lbf). Data are plotted for groups of crewmembers who served on the Mir (n = 28 cosmonauts and 7 U.S. astronauts), on the International Space Station (ISS) pre-ARED (n = 24 U.S. astronauts exercising on an interim Resistive Exercise Device [iRED]), and on ISS after access to ARED hardware was available (n = 11 U.S. astronauts). BMD changes are reported for lumbar spine, femoral neck, trochanter, total hip, and wrist (wrist is 1/3 radius + ulna, and is for ISS crewmembers only). All group mean aBMD changes from pre-flight to post-flight were significant (P < 0.05), except at the wrist, as determined by 1-tailed, unpaired t-tests using absolute data. Likewise, except for the effect on the wrist, the comparison of exercise effects between ARED and iRED suggests an improved “step function” with ARED exercise hardware—attenuating the expected declines in aBMD with spaceflight. The impact on actual bone strength and on fracture risk remains unknown. (Sibonga, 2013)

With regards to bone surveillance, since 1998, dual-energy X-ray absorptiometry (DXA) has been a medical requirement for NASA astronauts. DXA monitoring is utilized at astronaut candidate selection, then preflight and post flight. Selection and preflight T-scores must be above -1, with a post flight permissible outcome of -2 (Orwoll et al., 2013). However, the utility of DXA in evaluating bone health among astronauts has been questioned as it does not necessarily correlate with bone strength or fracture risk after spaceflight. There is concern that DXA may underestimate the skeletal fracture risk in astronauts returning from microgravity (Orwoll et al., 2013). Bone density/quantity and bone strength/quality are separate entities. DXA is a surrogate for bone quantity but not quality, and may underestimate fracture risk after spaceflight because abundant bone does not necessarily correlate with strong bone. For this reason, new imaging modalities are under investigation. The most promising of these alternatives is quantitative computed tomography (QCT). QCT allows for individual characterization of the cortical and trabecular components of bone. Discordance has been noted between areal BMD (aBMD) by DXA and volumetric BMD (vBMD) by QCT when applied to returning astronauts. In the lumbar spine, aBMD improved after return, while vBMD declined during the same time period. Furthermore, femoral neck aBMD increases after return in most individuals, but one astronaut whose femoral neck aBMD exceeded preflight measurements showed a reduction in trabecular vBMD (Orwoll et
These incongruences between DXA and the more detailed QCT raise concern regarding the ability of DXA to accurately characterize BMD changes and resultant fracture risk. The addition of finite element modeling (FEM) to QCT (QTC-FEM) may further improve its predictive power (Orwoll et al., 2013). FEM is a computational tool that estimates hip strength for specific loading orientations (e.g., axial loading in one-legged stance). When employed on astronauts, FEM detected significant declines in hip strength post-flight. Further comparison yielded poor correlation between DXA and QCT-FEM (Orwoll et al., 2013). While these data cannot indicate whether DXA or QCT-FEM provides superior predictive power for fracture risk, they do suggest that QCT-FEM may detect changes in skeletal structure that DXA cannot (Orwoll et al., 2013). Studies involving these surveillance techniques are underway.

Current countermeasures for bone loss include: 1) vitamin D supplementation, 2) an exercise regimen while on orbit, and 3) intensive physical therapy upon return to Earth. During a typical mission, astronauts are scheduled to exercise for at least 2 hours daily, 6 days per week (Smith, 2012). Exercise on orbit consists of resistive exercise on the Advanced Resistance Exercise Device (ARED) and aerobic exercise on a treadmill and cycle ergometer. Resistive exercise is thought to improve bone health by providing mechanical loading to areas that would otherwise receive none in reduced gravity. ARED is an improvement over the previous Interim Resistance Exercise Device (IRED), which raises the maximum resistance load from 300 pounds to 600 pounds. This equipment change in addition to nutritional changes including increased caloric intake and vitamin D supplementation coincided with a decrease in average hip BMD loss from approximately 1.1 to 0.5% per month. (Smith, 2012) The improvement is encouraging, but further investigation is ongoing to confirm efficacy and rule out confounding factors.

Aggressive post-flight rehabilitation will be unavailable to astronauts arriving on Mars after a long-duration transit. Furthermore, volume constraints on an exploration-class mission may limit the feasibility of a comprehensive resistive exercise device. As such, additional countermeasures are under investigation. These include selection standards using QCT-FEM, bisphosphonate therapy, dietary modification to reduce sodium content and increase calcium and vitamin D intake, and artificial gravity via continuous or intermittent centrifugation (LeBlanc et al., 2013; Heer, 2002; Smith, 2012). Selection standards that screen out candidates with baseline low bone mass may improve post-transit bone mass and reduce the risk of fracture during re-exposure to mechanical loading. Bisphosphonate treatment using alendronate has shown promise in mitigation of bone mass reduction (LeBlanc et al., 2013). In a recent NASA study, the combination of alendronate and ARED diminished the expected bone demineralization in almost all markers: DXA in the spine, hip, and pelvis, QCT in trabecular and cortical compartments of the hip, FEM in the hip, levels of bone resorption markers, and urinary calcium excretion (LeBlanc et al., 2013). These results are encouraging, but additional evaluation will need to confirm the efficacy of bisphosphonate therapy. Artificial gravity would be the ultimate countermeasure for many space-induced adaptations. However, it faces significant engineering roadblocks. If artificial gravity is to be pursued, many questions remain regarding the strength and frequency of exposure sufficient to mitigate bone demineralization.

Much is known of the altered physiology of bone in reduced gravity environments, but uncertainties persist. Further investigations should better assess the utility of QCT-FEM or other modalities for fracture risk prediction and characterization of
bone microarchitecture. Moreover, optimization of countermeasures such as astronaut candidate screening, dietary manipulation, exercise regimens, pharmacologic countermeasures, and even artificial gravity is necessary prior to undertaking any exploration-class mission (Buckey, 2006; Orwoll et al., 2013).

1.7.3. Skeletal Muscle Alterations in Space

Tony Chao, M.S.

Maintaining skeletal muscle health is critical to maintaining overall health. Muscle strength and function can prevent fall injury, fracture, and improve daily quality of life. The effects of unloading from microgravity have been shown to have deleterious effects on muscle morphology and function. In space, astronauts experience increased rates of muscle atrophy that can be attributed to a decrease in muscle fiber cross-sectional area (Adams et al., 2003). Some indirect effects may be due to altered levels of anabolic hormones or glucocorticoids (Vandenburgh et al., 1999). This was demonstrated when tissue-cultured skeletal muscle cells were engineered in bioreactors and flown into space (Vandenburgh et al., 1999). Significant muscle atrophy was observed due to a decrease in muscle protein synthesis without any change in protein breakdown. Once the muscle tissue was returned to earth, protein synthesis was again stimulated (Vandenburgh et al., 1999). Contrary to this finding, rat studies showed increased muscle protein breakdown by simulation of spaceflight. Hind limb suspension of rats for an excess of 10 days caused ubiquitination and degradation of myosin heavy chain (MHC) in the gastrocnemius (Ikemoto et al., 2001). This suggests an increase of protein degradation through ubiquitin-dependent proteolytic pathway in spaceflight.

Heterogeneity in muscle tissue fiber types in humans is affected differently during prolonged spaceflight. Investigations on nine ISS crewmembers showed a greater loss in type I muscle fiber versus type II (Fitts et al., 2010). The loss of muscle cross-sectional area and strength is seen and contributes to a reduction in peak force and power (Adams et al., 2003; Fitts et al., 2010). Muscles in the lower extremities as well as postural muscles are more affected by prolonged unloading than muscles of the upper extremities (Fitts et al., 2010).

Exercise has been used as a countermeasure to maintain muscle size and strength. However, even with treadmill running, cycling, and resistance exercise, astronauts still experience significant loss in muscle size, strength, and power (Trappe et al., 2009). Advancement in technology and engineering in exercise equipment recently developed has improved the results of preventing muscle atrophy. New data from use of the Advanced Resistive Exercise Device (ARED), which has been on board ISS since 2008 and allows absolute loads up to 600 pounds, has been more effective in maintaining or increasing lean body mass than the previous model Interim Resistive Exercise Device (iRED) (Smith et al., 2012). The difficulty is engineering exercise equipment simulating the load during the eccentric phase of muscle contraction that we experience on earth, yet be small enough to fit in confined places on board a spacecraft. This will be even more of an issue when we transition from the large volume of the ISS to the likely much smaller volume of a spacecraft in transit to Mars. Ongoing work is underway to determine if ARED definitively maintains muscle mass, strength, and function over long periods.

The optimal exercise prescription has yet to be determined. The optimal frequency and duration of exercise during spaceflight is still not known; however,
several investigations are ongoing to determine ideal prescriptions. These will determine how many bouts per day and to what intensity exercise should be performed to mitigate the effect of weightlessness while minimizing energy expenditure. The goal is to optimize astronaut time, energy, and conserve resources for exploration class missions. Variations in intensity, type, and time have been investigated in spaceflight and in bed rest analog environments (Matsuo et al., 2012). One such study has been the first to report that exercise is “completely effective for the prevention of cardiovascular and skeletal muscle deconditioning during strict bed rest using exercise equipment similar to that on the ISS” (Ploutz-Snyder et al., 2014). Future and ongoing studies of astronauts on ISS will help determine the best approach to an ideal exercise prescription.

Long duration space flight can have detrimental effects on skeletal muscles; however, advances in technology and engineering have attenuated some of these effects on muscle atrophy, strength, and endurance. Today, with the use of countermeasures, astronauts are able to maintain muscle health and function better than in previous decades. Further study to integrate optimal exercise prescriptions, nutrition and other components are warranted to improve muscle health and function during prolonged spaceflight.

1.7.4. Cardiovascular Effects of Spaceflight

Eric Blacher, MD, MPH

Considerations for spaceflight include the consequences of expected cardiovascular physiological changes induced by spaceflight, as well as consequences of preexisting subclinical cardiovascular abnormalities that may be exacerbated by spaceflight (Barratt and Pool, 2008).

There are known cardiovascular physiological changes induced by spaceflight. These changes are fairly well understood from bed rest and microgravity research on ISS, but much is still not fully understood about orthostatic intolerance after flight, reduced exercise capacity, the effect of vascular-smooth muscle loss on other physiologic systems, and fluid shift mechanisms and their countermeasures (Hargens and Richardson, 2009).

Physiologic cardiovascular changes induced in weightlessness include: fluid shift from gravity dependent muscles of the lower body to the upper body, a decrease in blood volume and red cell mass, an increase in cardiac ectopy, reduced baroreceptor gain response, reduced thermoregulation, and myocardial atrophy (Buckey, 2006). As cephalad fluid shifts occur, stretch receptors in the central circulation sense an increase in central blood volume, which then stimulates adaptive mechanisms (decreased renal sympathetic nerve activity, increased plasma renin activity, aldosterone secretion, release of atrial natriuretic peptide) to reduce plasma volume (Buckey, 2006).

The reduced plasma volume results in an increase in hematocrit, likely leading to decreased erythropoietin secretion and reduced red cell mass (Buckey, 2006; Gunga et al., 1996). Evidence does not show that urine output increases during space flight, but suggests that albumin-containing fluids move from the intravascular to extravascular space, which accounts for the volume loss. Combined plasma volume and red cell mass results in approximately 11% total blood volume reduction, which gives a central blood volume which is similar to what the body sees in an upright position on Earth (Buckey, 2006). Blood pressure, on average, may be slightly reduced and cardiac muscle mass may decrease up to 8 – 10% during spaceflight (Charles et al., 1999).
Figure 1.7.4-1 illustrates the fluid shifts that occur during exposure to microgravity and upon return to Earth.

Cardiovascular changes manifest as orthostatic intolerance and impaired exercise capacity. Orthostatic intolerance can make landing and vehicle egress very challenging (Buckey, 2006). A crewmember might not show a propensity to orthostatic intolerance pre-flight, but can be affected after spaceflight. Crews should be in the best possible condition pre-flight as well as work throughout flight to maintain musculoskeletal and aerobic conditioning to tolerate the cardiovascular stress of the transition between microgravity and Earth (and/or Martian) gravity.

In preparation for potential cardiovascular stresses during landing after a long duration mission, several countermeasures are used. Carefully designed programs for maintaining aerobic and muscular fitness throughout the duration of the mission are vital (Ploutz-Snyder et al., 2014). These programs on the International Space Station (ISS) currently include scheduling 2.5 hours per day for physical exercise 6 days per week. Exercise includes running on a treadmill using a harness from the shoulders and hips to create loads of about 80% body weight, aerobic exercise with the cycle ergometer, and weight lifting with the Advanced Resistive Exercise Device (ARED).

Studies are underway to determine the optimal exercise program to maintain aerobic capacity, muscular strength, and bone integrity; preliminary data indicate that with consistent use of an optimal mix of high intensity aerobic and resistive exercises, crews

Figure 1.7.4-1. Cephalad Fluid Shifts Due to Microgravity Exposure
https://asgsr.org/index.php/education/slide-sets
can maintain aerobic capacity as well as muscle strength, which was previously not thought possible (Ploutz-Snyder et al., 2014).

Countermeasures to orthostatic intolerance can be employed just before landing; they include fluid loading with electrolyte solutions and/or NaCl tablets with water, G-suits, compression dressings or garments. Lower body negative pressure (LBNP) in-flight is another measure which can be used to gauge an astronaut’s orthostatic intolerance as well as a countermeasure to orthostatic intolerance, if used throughout the mission and/or in the weeks prior to landing (Buckey, 2006). Promising results have been seen with elastic graded compression garments (GCG) that apply graduated pressures from the feet to the abdomen. The GCG can be easily donned and are relatively comfortable to wear, but have not been validated after long-duration spaceflight (Stenger et al., 2013). Lastly, pharmacologic vasoconstrictors such as midodrine could play a role in mitigating orthostatic hypotension secondary to cardiac deconditioning, although it is not currently in use for long duration spaceflight crewmembers (Buckey, 2006).

Most large-vessel coronary vascular diseases are not symptomatic until stenosis of the vessel reaches about 80%. For missions with durations longer than one year, preexisting subclinical cardiovascular abnormalities must be ruled out to the extent possible with cardiovascular selection criteria (Gillis and Hamilton, 2012). This becomes even more relevant in deep space missions where emergency de-orbit and return to Earth is not possible (i.e. Mars) as it is now from the ISS in low Earth orbit. Crew screening may include comprehensive physical examination as well as tests including 24-hour Holter monitoring, echocardiography, EKG, and exercise stress testing. Other modalities such as stress echocardiography, coronary CT, carotid ultrasound can assist in screening for coronary artery disease (CAD). Basic serologies and cardiac biomarkers such as C-reactive protein (CRP), lipids, and coronary calcium scores could be obtained on selection and prior to spaceflight. Prevention of cardiovascular illness should be the primary goal in selecting a crew for a long-duration mission and maintaining the health of that crew (Barratt and Pool, 2008).

In summary, careful astronaut selection standards should be used to screen out pre-existing subclinical cardiovascular disease that could potentially be exacerbated by cardiac deconditioning of spaceflight and jeopardize the mission. Once a carefully screened crew is chosen, the crew will maintain cardiovascular aerobic and muscular fitness before, during, and after spaceflight with regular high intensity aerobic and resistive exercise (Ploutz-Snyder et al., 2014; Matsuo et al., 2012). Getting the crew safely through the mission is only half of the battle. Making the microgravity-to-Earth or microgravity-to-Mars transition safe can be challenging if proper countermeasures are not in place. Fluid loading, G-suits, GCG, and LBNP can be utilized to mitigate the microgravity-to-gravity transition of re-entry, landing, and egress.
1.7.5. Effects of Spaceflight on the Neurovestibular System
Ben Johansen, MD, MPH

The vestibular system integrates input from visual cues, proprioceptive sensations, and the inner ear to allow us to maintain posture and perform coordinated movements. In a weightless environment each of these input sources are affected, forcing the body to adapt to new sensations of movement.

In spaceflight, gravity is no longer present to supply a vector for linear acceleration sensed by the otolith organs, the body’s main gravity receptors. Instead the stimulus is static despite the orientation of the body. The organs are sensitive only to translational movement from inertia of the otoliths on the macula. Sensations from the semicircular canals, which normally sense body and head angular acceleration, are disturbed. This forces the astronaut to rely more on proprioception and visual cues to provide a reference of position and can have effects on the balance system (Buckey, 2006).

On spacecraft such as the ISS, there is no orientation in the traditional sense of up or down. All sides are used to maximize space and take advantage of weightlessness. Although useful, this can be disorienting to crewmembers as they try to adapt. It is as though walking into a room to suddenly find one standing on his head. Astronauts also rely more on their upper body to propel themselves through the station. Using only their lower body to secure themselves to foot holds for stability providing a new source of proprioceptive input to which the brain must adapt.

Just as the human takes some adaptation on arrival to weightlessness, on return to a 1 G environment the body must readapt. Again the astronaut must rely more on their visual and proprioceptive input, as vestibular senses may be unreliable. Post flight astronauts adopt a wide based gait and attempt to minimize any sudden head movements as to not provoke an illusion of excessive tilt (Buckey, 2006). False sensations are especially important to consider when performing tasks immediately on re-entry such as landing the shuttle or an emergency egress from a spacecraft (Small et al., 2012).

In many astronauts, it can be difficult to adapt back to a 1 G environment and the time to return to pre-flight abilities is dependent on the duration of the mission. With shuttle missions, the recovery time was relatively fast, a few days. However, recovery from a six month ISS expedition may take many days to weeks (Small et al., 2012). This is important to consider with longer duration missions to a planetary destination. A period of vestibular adaptation will have to occur to the new gravitational environment. This may impact specific tasks that will be required upon landing. Wearing and carrying bulky equipment or having to perform tasks with less than ideal lighting may be added challenges to an already compromised vestibular system (Peters et al., 2013).

Various countermeasures have been employed in attempts to shorten the adaptation time and prepare the astronauts for the sensations they may experience. Examples include spatial orientation exercises while walking on unstable surfaces, centrifuge runs, and parabolic flights (Peters et al., 2013; Shelhammer and Beaton, 2012). A novel analogue that has been studied in subjects in an effort to train to perform tasks while experiencing conflicting sensorimotor input is galvanic vestibular stimulation. This process can disrupt vestibular input in normal subjects by passing small electrical currents between mastoidal surface electrodes (Moore et al., 2011).

Space motion sickness (SMS) has been a focus of study since the beginning of human spaceflight experience. There have been conflicting theories for the cause and
mitigation strategies for SMS. Motion sickness has been experienced in many environments, including in planes, cars, boats, and other environments where humans experience motion. However, it can be experienced when there is no actual motion; conflicts between visual and vestibular stimuli may cause similar symptoms, such as in simulators and 3D or IMAX movies. Also, motion sickness can be a conditioned response such that a person can develop symptoms while walking to an aircraft, or smelling the cockpit. The current prevailing theory for the cause of SMS is the sensory conflict or sensory rearrangement theory, in which a conflict between senses or between a sense and past experience leads to motion sickness (Yates et al., 1998).

On Earth, our visual, vestibular, and proprioceptive senses agree. In a weightless environment, visual orientation cues are changed due to the floating crewmember, yet the visual cues will match what the semicircular canals sense with head movements; however, otoliths no longer sense directional motion or translation. Therefore, there is a mismatch within the vestibular system (between otoliths and semicircular canals) and between the vestibular and visual systems that result in SMS symptoms (Reason and Brand, 1975; Yates et al., 1998; Thornton and Bonato, 2013). Humans will adapt over a period of a few days, and these inputs will normalize. These normalizations from space then require re-adaptation on return to Earth when gravity and visual cues are reintroduced, which may result in similar symptoms to SMS which may include pallor, sweating, nausea, vomiting, dizziness and headache. Other signs may include drowsiness, malaise, reduced appetite, salivation, yawning, belching, or flatulence (Buckey, 2006; Thornton and Bonato, 2013).

Incidence of SMS has been reported as high as ~50 – 80% in the first 2 – 3 days of flight in varied sources, with up to 50% characterized as moderate to severe, requiring treatment (Buckey, 2006; Jennings, 1998; Thornton and Bonato, 2013). Treatment in flight has included elimination of provocative activities such as head movement and critical procedures or providing pharmacological treatment (Jennings, 1998). The most effective medication strategy to date is intramuscular promethazine, although it can cause sedative side effects requiring counteracting amphetamine use for critical tasks (Jennings, 1998; Weerts et al., 2014). Symptoms typically resolved in 24 – 72 hours. Other treatments currently undergoing study include meclizine and scopolamine, both of which have undesired side effects and varied results (Jennings, 1998; Weerts et al., 2014). Further study is needed on the best mitigation strategies for SMS as well as for the re-adaptation sickness which occurs on return to Earth as the transition from weightlessness to a full or partial gravity environment will be important to understand going forward to exploration class missions.

Future exploration missions to other planetary bodies will require astronauts to perform landing or docking maneuvers that may be compromised by unreliable vestibular sensations (Moore et al., 2011) and SMS. Training exercises to help crew to understand that sensations they experience may not be reliable will be important so they can perform mission tasks, and medication may be important to mitigate symptoms of motion sickness. It will be necessary to estimate the length of time required for individual crew members to adapt and to factor this into mission schedules; particularly knowing that the longer the crew is exposed to weightlessness the longer it may take to adapt back to a gravitational environment. A combination of countermeasures will need to be employed to mitigate the effects of weightlessness and improve performance once crews reach their destination.
1.7.6. Radiation Effects of Spaceflight
Natacha Chough, MD, MPH

While radiation is a known unavoidable risk in human spaceflight beyond Low Earth Orbit (LEO), current countermeasures remain a significant challenge.

1.7.6.1. Present Knowledge. Currently, the physical nature of radiation is relatively well-understood, existing in the form of either photons (gamma- and x-rays), which impart low Linear Energy Transfer (LET) through cells; or particles (protons, neutrons, atomic nuclei), which impart high LET (Buckey, 2006). The end result of this energy transfer is ionization, which is most relevant to human spaceflight because it creates free radicals that can damage DNA and other cellular processes necessary for life. Of most concern in the LET category is Galactic Cosmic Radiation (GCR), which is constant background radiation, and Solar Particle Events (SPEs).

Based on the above known characteristics of various types of ionizing radiation (e.g., energy, flux, dose rates), occupational dosimetry is currently implemented for U.S. crewmembers, with both mission and career threshold doses. The Gray (Gy) is the unit used to describe the absorbed dose available to do damage to tissues, or 1 J/kg. The Sievert (Sv), while a 1:1 conversion factor with the Gy, describes the measure of radiation exposure and exposure limits. Currently the career limit for a U.S. astronaut (gender and age-dependent) is 1.0 – 4.0 Gy (Barratt and Pool, 2008). This monitoring serves as the current primary mitigation strategy for crewmembers in the form of limiting overall exposure. Future interplanetary missions will require better propulsion systems to also minimize total exposure time. Figure 1.7.6.1-1 shows the comparative doses of ionizing radiation for reference.

![Chest X-Ray](image)

Sub-Orbital Flight, Large SPE
Terrestrial Background (1 year)
Orbital Flight (10 days)
Defibrillator Malfunction
CT Scan (typical)
Pacemaker Malfunction
Pacemaker Failure
Defibrillator Failure

Radiation Dose, mGy

**Figure 1.7.6.1-1. Comparative Doses of Ionizing Radiation (Reyes et al., 2014)**

The biological effects of radiation in humans are less-well known, mostly due to ethical issues with designing randomized control trials using radiation exposure. Therefore, most of the knowledge on biological effects comes from Hiroshima survivors and cancer patients. What is known is that rapidly dividing cells are more sensitive to radiation, such as that of the gastrointestinal (GI) tract. This is presumably due to DNA strands being unwound for replication a higher percentage of the time, rendering these
tissues more susceptible to breaks and other damage. This also explains phenomena such as radiation GI syndrome, which can occur at ~3 Gy, and why CNS effects are not seen until near-lethal doses, i.e. 10+ Gy (Buckey, 2006; Barratt and Pool, 2008).

Although the Earth’s magnetic field offers some radiation protection from solar particle events (SPEs), GCR and other ionizing radiation, this cannot be relied upon for long-duration and interplanetary spaceflight.

1.7.6.2. Knowledge Gaps and Posited Countermeasures. Current gaps in space radiation include the ability to predict SPEs and adequate mitigation strategies. However, even with the best astrophysical devices, prediction of SPEs may not be any better than attempting to predict seismic events on Earth. In the end, SPEs will still occur, and are a risk, necessitating shielding and other mitigation methods regardless of any prediction capabilities. Preparing for the worst and hoping for the best remains the keystone of preventive health and space medicine. Therefore, the remainder of this summary will focus on mitigation.

Both active and passive shielding methods have merit as countermeasures. While dense materials such as lead are undesirable passive shielding due to mass constraints and secondary radiation, they may be of use as an internal "storm shelter" in the event of a disastrous SPE (Buckey, 2006). Water and hydrogen-containing compounds are still discussed as options for a lighter form of passive shielding (Bagshaw, 2010). Ideally, a combination of both dense and light materials will be implemented to minimize launch mass while still addressing crew safety.

Active shielding in the form of a superconducting coil to create the spacecraft's own magnetic field, and indeed, technology developments have the potential to make this a reality (Buckey, 2006). NASA has been working to develop high-temperature superconductors, and current discussions involve the investigation of solenoids, double-helix toroids, inflatable and other coil forms potentially ideal for long-duration spaceflight (Westover et al., 2012). Based on the inevitable risk of radiation, this area of research and development should receive funding priority for Mars and other long-duration missions.

A number of sources cite the benefit of antioxidants as a tool to help combat reactive oxygen species (ROS) (Giardi et al., 2013; Wambi et al., 2009). While no data supports excessive dosing, there is likely no harm in dietary supplementation with these compounds, both for radiation reasons as well as cardiovascular, immunologic and other benefits while on a long-duration mission.

While extremely difficult from a feasibility standpoint, it may also be beneficial to further study extremeophiles such as Deinococcus radiodurans, which has been found to resist radiation exposures of up to 12,000Gy (Daly, 2012). D. radiodurans possesses metals such as manganese, which help with combating ROS that can be a result of ionizing radiation, free radicals, and can damage DNA. If the technology for gene therapy or some sort of radiation vaccine development were harnessed for use in crewmembers, this may provide an effective biological shield that may be the most efficient countermeasure in terms of resources and mass mitigation for launch manifests.

In short, radiation remains one of our most important risks to address for long-duration spaceflight due to its ubiquitous presence. Based on current knowledge, a combination of countermeasures in mission duration, spacecraft design, crew nutrition, and biotechnology will need to be employed to minimize mission risk and maximize crew safety.
1.7.7. **Nutritional Aspects of Space Flight**

James Pattarini, MD, MPH

Though spaceflight appears to present a similar environment to long-duration submarine or remote polar deployment, the obstacles to providing adequate nutrition in space extend far beyond the consumables mass and preservation constraints of these analogs. The microgravity environment induces physiologic changes in astronauts including sarcopenia, bone resorption due to longitudinal unloading, and hematologic changes with down-regulation of marrow red and white cell production that all influence how oral nutrition may affect the crew (Smith et al., 2012; Sonnenfeld and Shearer, 2002; Willoughby et al., 2007). Since Space Lab and Mir, weight loss in space has been a concern, with the inciting factors difficult to control due to their multifactorial nature; space motion sickness and some of the medications used to treat it have an early effect on blunting appetite, and the lack of regular lower extremity use drives large muscle group mass decline. The unique stress of navigating the microgravity environment, circadian disruption and activities such as EVA also combine to influence appetite at designated mealtimes. Elevated CO$_2$ levels may also play a role in appetite suppression, though this is unclear.

The necessities of providing adequate nutrition that will remain unspoiled and free of contamination for the prolonged periods needed for human spaceflight has also resulted in markedly high sodium content of food, with the unfortunate side effect of possibly exacerbating already accelerated bone loss. Conversely, cephalad fluid shift in microgravity commonly results in sinus congestion early in flight and decreased taste sensitivity, further reducing the palatability of foodstuffs. While energy requirements on orbit appear close to terrestrial measurements, astronauts consistently consume less food in space, resulting in a caloric deficit that can ironically be exacerbated by the required aerobic and resistance countermeasures implemented to prevent sarcopenia and bone loss.

The spacecraft environment, being enclosed and weightless, is an ideal environment to speed bone resorption – without sunlight, astronauts are at risk for vitamin D deficiency, and calcium resorption from long bones is high enough to suppresses parathyroid hormone (PTH). Supplementation of Ca$^{2+}$ and vitamin D is weighed against the unknown risks of aggravating hypercalciuria and increasing the risk of nephrolithiasis on orbit, however terrestrial data shows no increase in renal stone formation with aggressive supplementation. A similar balancing act must be accomplished when protein intake is considered – given the active countermeasures against sarcopenia and the average adult male requiring a dietary protein load of ~20 grams to stimulate protein synthesis, adequate intake is critical. A high-protein diet unfortunately increases plasma acidity that may in turn exacerbate bone loss (Smith et al., 2012; Willoughby et al., 2007).

Dietary antioxidant supplementation as a component of a cohesive radiation mitigation strategy is an area of ongoing study; during the ISS and the Space Shuttle era, astronauts’ primary source of radiation exposure was derived from regular transits of the South Atlantic Anomaly, with the associated high energy protons therein. As we look to longer duration missions that will extend beyond the Earth’s magnetosphere, the source and types of radiation will change to include greater magnitude solar particle events, and galactic cosmic radiation with heavy nuclide components. While these sources come from without, the production of free radicals from metabolic processes may be increased during times of stress as well, compounding the amount of oxidizing...
species available to produce cellular damage (Fang et al., 2002). While a number of antioxidants have shown promise in terrestrial trials for cancer prevention or reducing oxidative stress from radiation exposure including Alpha-lipoic acid, N-acetyl cysteine, and glutathione, other data has been conflicting, with supplementation with beta-carotene showing an increase in lung cancer risk, contrary to expectations (Kennedy and Todd, 2003; Omenn, 1998). Furthermore, many antioxidants are toxic in moderate to high doses, making dosing recommendations difficult when attempting to balance risks.

While U.S. astronauts are selected to have lower than average risk factors for cardiovascular disease (CVD) at the time of selection, a preventive health strategy must include a diet tailored to reduce CVD risk during any long duration mission where definitive intervention will be unavailable. While a diet low in saturated fat and hydrogenated oils is favorable for cardiovascular health, the need for long-term food stability on missions where resupply may not be possible again calls for a balance between optimal nutritional content and partially hydrogenated oil content, which may raise LDL cholesterol but prolong shelf life. Due to the aforementioned difficulties in maintaining crew weight and caloric intake targets, a likely strategy for long-duration missions to other planetary bodies will include selecting crew members at low risk for cardiovascular events over the mission duration (1 – 2 years), and allow a certain amount of hydrogenated and saturated fat intake in favor of ensuring unspoiled consumables will last the duration of the mission.

The psychological impact of palatable food is also a consideration that should not be overlooked; when a mission to our nearest planetary neighbor will include a >40 minute round-trip communication delay at times, the mental wellbeing of the crew is equally important to their physical health. The ability to provide recognizable and desirable food items for a crew who may not see Earth for several years should be a goal of our nutritional research programs.

The combination of microgravity, radiation exposure, vestibular adaptation and circadian rhythm alteration makes terrestrial studies of nutrition questionable analogs, limiting our ability to close gaps in our knowledge except in direct ‘learn-by-doing’ studies involving active astronauts during spaceflight activities. While we have made great strides in addressing sarcopenia and bone loss through resistive exercise countermeasures, more study is needed to find the optimal nutritional protein and carbohydrate load to combine with these countermeasures to maximize muscle and bone gains while minimizing losses where caloric shortfalls occur. Additionally, while antioxidant supplementation may yield questionable gains in radiation protection, active research on the use of carbon nanotubes for pharmacological pre- or post-exposure prophylaxis may prove a valuable component of future radiation mitigation strategy (Wu et al., 2008). Advances in prolonging shelf life while preserving nutritional value (i.e. minimizing hydrogenated oils and sodium load while maximizing plant phenols and protein/fiber content) will need to be aggressively pursued for a 2-year mission to be viable; currently, ISS resupply shipments are a staple of ongoing 6-month and 1-year duration missions. Such periodic consumable restocking will be unavailable for missions outside of the Earth-Luna system, and research into hydroponic and aeroponics as renewable supplements to stored consumables should be seriously pursued (Wu et al., 2008). While further study in these areas is needed, advances in understanding of human metabolism and altered nutritional needs during spaceflight will have profound implications for our ability to support missions beyond our moon in the decades to come.
1.7.8. Visual Impairment Intracranial Pressure in Spaceflight
Rahul Suresh, MD, M.S.

Astronauts have observed decrements in near visual acuity during spaceflight for several decades. A recent survey of 300 NASA astronauts confirmed these anecdotal reports, finding that 48% of long duration and 23% of short duration astronauts complained of near vision difficulties (Mader, 2011). Prescription glasses, or “space anticipation glasses,” are simple but effective counter measures regularly flown with astronauts (Mader, 2011). However, the clinical and operational significance of these visual deficits gained increased attention after identification of ophthalmic changes including posterior globe flattening (PGF), optic disc edema (ODE), cotton wool spots (CWS), choroidal folds, and retinal nerve fiber layer (RNFL) thickening associated with decreased near visual acuity and hyperopia in seven astronauts (Mader, 2011). No clinical signs or symptoms were associated with decrements in visual performance, but in some astronauts these findings persisted for greater than a year post flight, raising further concern for the long-term clinical and operational impact (Mader, 2011). Since this initial report, NASA has instituted a more comprehensive ocular surveillance program to carefully monitor all astronauts pre-, in-, and post-flight using 2D ultrasound, head and orbit magnetic resonance imaging (MRI), vision exams, tonometry, fundoscopy and ocular coherence tomography. A retrospective review of post-flight MRIs in 27 astronauts confirmed posterior globe flattening in some and in addition showed orbital and intracranial abnormalities including optic nerve distension (OND) and optic nerve sheath distension (ONSD), optic nerve kinking and partially empty sella turcica suggestive of elevated of intracranial pressure (Kramer et al., 2012). The above-mentioned constellation of findings defines the visual impairment and intracranial pressure (VIIP) syndrome (Alexander et al., 2012).

A number of anatomic, physiologic, and environmental factors have been linked to development of VIIP but a unifying etiology remains elusive. Based on the proposed mechanisms, the etiology of VIIP is likely a primary ocular/intraorbital problem and/or an intracranial problem related to elevated intracranial pressure manifesting as ophthalmic changes. Known cardiovascular adaptations to spaceflight and environmental factors may help initiate or exacerbate these problems in those who are predisposed, since not all exposed to weightlessness develop the syndrome, and the exact incidence is not published to date. The variable penetrance and severity so far observed suggest a multifactorial etiology. The following discussion will provide a brief overview of the important physiological mechanisms currently under consideration.

Development of optic disc edema (also known as papilledema when intracranial pressure (ICP) is elevated) is considered the most severe manifestation of VIIP and can lead to loss of vision if uncorrected. Optic disc edema has now been noted in some astronauts raising concern for permanent visual deficits. Typically papilledema is seen with elevated ICP. Other predisposing factors related to ocular anatomy and architecture have also been implicated in development of optic disc edema. The optic disc, or the optic nerve head, is the anatomical location where the retinal ganglion cell axons converge to leave the eye. As the axons (and the central retinal artery and vein) traverse the optic canal, they must pass through the lamina cribrosa, a lattice like structure of successive cribiform plates that stabilizes intraocular pressure (IOP) by providing a barrier between the vitreous humor and the extraocular space. In the normal eye, the pressure gradient across the lamina cribrosa (called the translaminar
pressure gradient, TLPG) favors intraocular pressure over cerebral spinal fluid (CSF) pressure. However, as TLPG is reversed, either due to decreased intraocular pressure, increased intracranial pressure, or failure of the lamina cribrosa, optic disc edema ensues.

Limited measurements of intraocular pressure are available from spaceflight and suggest an initial increase in IOP followed by stabilization at pre-flight levels and then decrease below pre-flight levels upon landing (Draeger et al., 1987). This has been confirmed in terrestrial analogs including parabolic flight and head-down tilt (Mader et al., 1990; Mader, 1991; Mader et al., 1993). The initial spike in IOP is postulated to be secondary to rapid venous congestion of the choroidal vessels due to spaceflight induced fluid shifts (discussed below). Measured increases in inflight IOP are inconsistent with development of optic disc edema. However, it is possible that decreased IOP on landing may contribute to optic disc edema seen post-flight. The thickness and structural integrity of the lamina cribrosa are also an important determinant of the effects of abnormal translaminar pressure gradient. With age, the load bearing connective tissues within the lamina cribrosa become damaged, leading to its weakening and elevated shear forces on the traversing axons and vessels. A weakened lamina cribrosa increases risk for retinal nerve fiber layer thickening and optic disc edema. Optical coherence tomography (OCT) is an emerging technology for performing high-resolution cross-sectional imaging of the retina. In OCT studies of astronauts, the structurally weaker areas of the lamina cribrosa correspond to the appearance of retinal nerve fiber layer thickening most prominent in the superior and inferior portions of the optic disc. Glaucoma and myopia are also associated with thinner lamina cribrosas, highlighting additional risk factors for optic disc edema.

The perineural subarachnoid space of the optic nerve is anatomically contiguous with the intracranial subarachnoid space and ends in a blind “cul-de-sac” immediately posterior to the globe, a region referred to as the bulbar optic nerve. In circumstances of increased ICP, excess pressures in the intracranial space are transmitted to the bulbar optic nerve and lead to pathological remodeling and persistent bulbar optic nerve sheath distension. Severe bulbar optic nerve sheath distension may cause compression of nearby structures such as the sclera anteriorly causing posterior globe flattening. In idiopathic intracranial hypertension patients for example, posterior globe flattening is nearly 100% specific for chronically elevated ICP when compared to healthy controls and those with acute ICP elevation. Idiopathic intracranial hypertension, also known as pseudotumor cerebri, is a disorder most commonly seen in obese young women and is characterized by headache, blurred vision, and visual changes resulting from increased intracranial hypertension. On clinical examination, papilledema is detected but on neuroimaging studies there is no evidence of an intracranial mass lesion and the ventricles are either of normal size or small. If untreated, idiopathic intracranial hypertension occasionally results in permanent visual loss and is of an unknown cause. Increasing optic nerve sheath distension can also compress structures within the optic nerve sheath including the nerve and retinal artery and vein leading to axonal swelling and optic disc edema. Prolonged compression of these structures may lead to axonal plasma stasis, interstitial edema and local ischemia, which further exacerbate optic disc edema and lead to retinal nerve fiber layer thickening and cotton wool spots. In terrestrial MR studies of patients with elevated ICP, bulbar optic nerve sheath distension exceeding 5.0 – 6.0 mm as seen in VIIP is predictive of intracranial hypertension (Geeraerts et al., 2008). Increased venous congestion may also play a role, leading to increased post-capillary pressures
and venous stasis. It has been hypothesized that choroidal folds represent engorgement of the choroidal vessels secondary to venous congestion or may be secondary to deformation of the globe and choroid as a result of optic nerve sheath distension.

Although intracranial hypertension is implicated in the ocular findings so far described, direct evidence of elevated ICP in-flight remains lacking. However, lumbar punctures performed between 12 and 60 days after landing in four of the seven astronauts studied by Mader et al. have demonstrated mild to moderately elevated opening pressures of 22, 21, 28 and 28.5 cmH₂O (normal < 20.4 cmH₂O), suggesting altered regulation of ICP post flight (Mader et al., 2011). Findings in VIIP such as posterior globe flattening, optic nerve sheath distension, empty sella turcica, and optic nerve tortuosity have also been noted in patients with idiopathic intracranial hypertension (Alexander et al., 2012). Idiopathic intracranial hypertension, like VIIP, is characterized by increased ICP without clinical, laboratory, or radiologic evidence of space-occupying lesion, meningeal inflammation or venous outflow obstruction.

Cephalad fluid shift upon exposure to microgravity is currently considered the primary initiating factor for elevated ICP in orbit. The phenomenon manifests as facial edema, sensation of sinus fullness and loss of leg mass and has been termed the “puffy face, bird leg” syndrome. Loss of the hydrostatic column and gravity assisted drainage of venous vessels above the heart leads to a redistribution of vascular and interstitial fluids towards the head and causes venous congestion. Animal models have shown elevated jugular vein bulb pressures and increased ICP immediately after exposure to free fall (Gotoh et al., 2003; Gotoh et al., 2004). Ultrasound evaluation in orbit of the jugular vein has confirmed increased jugular venous cross-sectional area that persists throughout exposure to microgravity (Arbeille et al., 2001). Moreover, jugular venous changes can be reversed with application of a mild compression tourniquet, called a braslet, to the bilateral lower extremities, strengthening the case for fluid shifts from the lower body towards the head (Herault et al., 2000).

Alternatively, some have postulated a proximal venous outflow obstruction as a mechanism for venous congestion. Indeed, spaceflight induced visual deficits have even been referred to as space obstructive syndrome (Weiner, 2012). Frank vascular occlusions from thrombosis or external compression have not yet been identified during flight. However, in the absence of gravity, even small changes in systemic venous compliance may impair venous drainage from the head. Unlike the lower body venous system, head and neck veins are otherwise unassisted by muscular forces or one-way valves. Non-occlusive venous outflow obstruction in the form of decreased systemic venous compliance secondary to increased sympathetic tone as seen in hypertensives has therefore been suggested (Safar and London, 1985). In general, increased vascular stiffness may explain the higher frequency and severity of VIIP in males as compared to females.

Due to limited cranial compliance, small changes in intracranial mass reflect dramatic changes in pressure as described by the Monro-Kellie doctrine. In the absence of pathology such as a space-occupying lesion, the primary determinants of intracranial pressure are neatly described by an adapted version of Davson’s equation (Chapman et al., 1990):
ICP = CSF Formation \times CSF Outflow Resistance + Superior Sagittal Sinus Pressure

where:

ICP = intracranial pressure
CSF = cerebral spinal fluid

In addition to increased venous pressures, CSF hydrodynamics is a potential determinant of elevated ICP. A rise in cerebral venous pressures blunts the hydrostatic diffusion gradient between the subarachnoid space and the dural venous sinuses diminishing CSF resorption in the arachnoid granulations. In addition, recent data has suggested that between 13 – 80% of CSF resorption may occur through craniospinal lymphatics (Koh et al., 2005). Data from animal models suggests that lymphatic function is impaired in spaceflight (Gashev et al., 2006). Magnetic resonance derived CSF hydrodynamics have recently been measured in astronauts post-flight (Kramer et al., 2015). Decreased CSF production rate and diminished CSF systolic velocity were associated with posterior globe flattening and optic disc edema. In the setting of increased CSF outflow resistance, CSF secretion appears to decrease in flight and increase post flight in animal models (Gabrion et al., 1995). Over correction in CSF secretion upon returning to 1G may therefore explain increased ICP post-flight in the 1G environment. Finally, increased venous pressures may alter hydrostatic gradients at the capillary beds, increasing transcapillary pressure with subsequent net permeation of fluid into brain parenchyma further increasing ICP.

Environmental factors, diet, and physical activity have been hypothesized to increase ICP. CO\(_2\) levels aboard the ISS are typically an order of magnitude higher than on earth (0.3% vs 0.03%, respectively). CO\(_2\) is a known cerebral vasodilator exacerbating vascular congestion and impairing the cerebral auto-regulatory response. Moreover, elevated CO\(_2\) levels may also trigger increased CSF production at the arachnoid granulations (Ainslie and Duffin, 2009). Arguably the acute effects of exposure to CO\(_2\) differ from chronic exposure secondary to physiologic adaptation. It remains unclear whether over the course of a 6-month mission elevated CO\(_2\) levels on ISS alter baseline acid-base balance and impact cerebral vascular function. Altered sodium handling in IIH has been postulated to exacerbate ICP, although the mechanism remains unclear (Smith and Zwart, 2008). Nutritional study of astronauts has also revealed baseline differences in one carbon metabolism between VIIP cases and non-cases that persisted during and after flight (Zwart et al., 2012). The authors of the study postulate that these differences highlight polymorphisms in enzymes in the 1-carbon pathway that interact with microgravity to cause pathophysiological changes associated with VIIP. The clinical significance is yet to be determined. Finally, resistive exercise with Valsalva maneuvers has been shown to increase ICP transiently (Edwards et al., 2002). Astronauts currently perform 1 – 2 hours of such exercises aboard the ISS daily to maintain bone and muscle health. It remains unclear whether transient ICP elevations have similar effects to chronic ICP elevation, and ongoing study of these effects is needed.

There remains significant controversy as to the prevalence of elevated ICP during flight and its role in the development of the ophthalmic and intracranial findings noted (Scott-Connor et al., 2013). Unlike in patients with IIH, astronauts do not have associated clinical symptoms such as chronic headaches, diplopia, transient visual obscurations, or pulse synchronous tinnitus. ICP in patients with IIH that exhibit ophthalmic findings noted in astronauts are usually significantly higher in the range of

1-140
40 – 50 cmH₂O. Post flight lumbar punctures have documented only mildly elevated opening pressures. Moreover, these are static values often obtained long after landing that may not accurately reflect inflight ICP. Both invasive and non-invasive means of measuring ICP in flight are currently under investigation that will provide definitive evidence for ICP dysregulation in microgravity.

Visual disturbances secondary to spaceflight have been well known for many years. However recent studies have revealed insidious ocular and intracranial changes such as optic disc edema, globe flattening, optic nerve sheath distension and empty sella turcica associated with near vision deficits that in some astronauts persist for some time after flight. These changes define the visual impairment intracranial pressure syndrome but it remains unclear what their clinical and operational impacts are. Elevated ICP and intracranial and ocular vascular congestion secondary to headward fluid shifts are thought to be the primary mechanism responsible for these changes. However, significant controversy surrounds the exact etiology of VIIP and many areas of investigation are actively underway to resolve the exact nature of this interesting and important clinical syndrome.

1.7.9. Summary

The reduced gravity environment and remote nature of spaceflight result in significant changes in human physiology. Humans adapt quite readily to weightlessness. Nevertheless, many adaptations can result in possible illness or injury when gravity is reintroduced or if left unhindered for long periods. Significant musculoskeletal, neurovestibular, ocular, and cardiovascular changes occur which, if left unchecked, may limit further human exploration of space. Longer lasting impacts can also result, as in the case of increased cumulative radiation exposure experienced by astronauts. This chapter has attempted to summarize many of the effects of spaceflight on humans, and is by no means exhaustive; immunologic, psychologic, and gender differences have not been addressed here.

Countermeasures such as proper nutrition, physical exercise, and medications are proving our ability to reduce some of the effects of weightlessness, but many investigations are ongoing to improve human tolerance to longer duration spaceflight, which will be required for exploration to destinations such as Mars. We are explorers. To ensure the success of future exploration, we must understand and overcome the medical and physiologic challenges associated with human spaceflight.

References


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Recommended Reading


1.8. Exercise Physiology

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The dim glow of red cabin lights reflects off each of their faces... at the 2-minute warning the team is on their feet, shuffling toward the open ramp, each saddled with over 80 pounds of gear, jumpmaster making the final checks. The light goes green and the mass of bodies moves quickly to the door. For the last 2 hours, each one has been propped against the hull of the fuselage balanced on the web seating and trying to stay limber. Up until now, it's been mental preparation: insertion to the landing zone, deployment to the objective, preparation for recovery...check, double-checks. Now the moment of truth: heart pounding, muscles straining against the mass of equipment harnessed head to foot; like pack mules each one steps to the ramp and out into the darkness. Now begins the complex ballet of muscles and tendons holding body position, maintaining control during freefall...no sudden moves, subtle adjustments only. Prepare to land: new muscle activity and mental preparation to gather togther equipment and get ready to move to the objective. The event relies upon the principles of total body fitness: muscle movement, stamina, and endurance. Developing proactive and purposeful activities is the focus of this chapter and the core of success in every dynamic arena in the Air Force.

1.8.1. Physical Fitness Training Principles

1.8.1.1. Introduction. It is inherent that aircrew establish and maintain a consistent state of physical fitness for both health and performance. A comprehensive aircrew functional physical fitness program must follow fundamental exercise principles and exercise program design and planning. The program should include: (1) primary physical fitness components of cardiorespiratory endurance, body composition, muscular strength, muscular endurance, flexibility-stability-mobility, and as applicable (2) skill-based physical fitness components of agility, balance, coordination, power, reaction time, and speed. A program of deliberately designed multiyear to weekly training cycles should encompass progressively applied physical training. Within these cycles a daily general exercise pattern should address primary workout phases of movement preparation, cardiorespiratory endurance, muscle fitness across the prioritized physical movement patterns, combination fitness, skills training, and movement transition-cessation. Finally, the training should include lessons on body composition, injury prevention and treatment, and fitness testing and standards. Accomplished in the above manner, this functional fitness program will ensure aircrew are prepared for the physical challenges of combat operations as well as stress the vital importance of maintaining physical fitness throughout one's career.

1.8.1.2. Reasons to Exercise. Exercise is a powerful medicine quite unlike any pill available, and it's a health-saving nostrum that brings intoxicating benefits with few side effects. It can lengthen the span and quality of life, decrease the risk of several diseases, and alleviate mental anxiety and depression as well as enhance human performance in athletic and occupational arenas. However, time and effort to perform exercise are involved, and these are barriers for many that make regular physical activity a bitter pill to swallow. For years philosophers, leaders, and
physicians have extolled the virtues of regular physical activity, but only in the recent decades have scientific data emerged from research by exercise physiologists, preventive medicine physicians, and other professionals to prove the benefits. Greatest benefits are incurred when very sedentary people begin and maintain a regular program of physical activity. Therefore, from a public health viewpoint, getting the most physically inactive portion of the population to become moderately active will lead to the strongest health gains. To avoid the pitfalls of sedentary lifestyle and address the first or health-related “tier” of fitness, the American College of Sports Medicine (ACSM), the American Heart Association (AHA), and the U.S. Centers for Disease Control and Prevention (CDC) recommend that every adult perform cardiorespiratory endurance exercise at moderate intensity 30 minutes a day 5 days a week or at vigorous intensity 20 minutes a day 3 days a week and accomplish 8 to 10 muscular strength training exercises, 8 to 12 repetitions of each exercise, twice a week. This first “tier” is gender specific and occupationally independent, whereas the second or performance/occupational-related “tier” of fitness is specific to one’s occupation or athletic activity and independent of gender. To address this second “tier” of fitness, one must, for most occupations and sports, physically train with greater exercise volume and specificity, usually transcending the basic first “tier” physical activity recommendations.

1.8.1.3. **Components of Physical Fitness.** Operational definitions of physical fitness vary with the interest and need of investigators and instructors (CDC). Dividing the construct of physical fitness into components allows for measurement.

1.8.1.3.1. **Health-Related Physical Fitness.** Health-related components of fitness are cardiorespiratory endurance, body composition, muscular strength, muscular endurance, and flexibility—mobility—stability. These health-related physical fitness components are least affected by genetics, hence more modifiable via behavior change, and are more important to both health and performance than are the skill-related components. Each component is a movement-related trait or capacity that is generally independent of the others. An underlying concept here is—better status in each of the constituent components is associated with both lower risk for development of disease or functional disability and higher performance capacity. Again, this multifactorial construct of several components is most important for both health and performance.

- **Cardiorespiratory Endurance.** Ability to perform large muscle, dynamic, moderate-to-high intensity exercise for prolonged periods. Performance of such exercise depends on the functional state of the respiratory, cardiovascular, and skeletal muscle systems. It is more simply defined as the ability to produce energy; your level of aerobic fitness determines how long and how hard you can exercise.

- **Body Composition.** Relative percentage of body mass that is fat and fat-free tissue.
- **Muscular Strength.** The maximal force that can be generated by a specific muscle or muscle group (properly expressed in Newtons, although kg is commonly used). It is more simply defined as the ability of your muscles to move your body and the objects around you.

- **Muscular Endurance.** The ability of a muscle group to execute repeated contractions over a period of sufficient time duration to cause muscular fatigue, or to statically maintain a specific percentage of maximum voluntary contraction for a prolonged period of time. It is more simply defined as how long your muscles can perform a task, move objects, or successfully hold items or a position.

- **Flexibility.** The maximum ability to move a joint through a range of motion. Many believe flexibility is lost with age; while this may be true, it is primarily due to the decrease in activity associated with age.

  **Note:** *Mobility* and *stability* are terms recently combined with flexibility in this final health-related component to designate a broader term that encompasses the role of stability and mobility in posture and daily functional living. Stability deals with maintaining nonmovement functional positions, including postural stability. Stability ranges from shoulder to ankle with shoulder, core, and hip stability as primary. Mobility, similar to stability, is stable, controlled, functional movement through an active range of motion in the various planes of motion.

**1.8.1.3.2. Skill-Related Physical Fitness.** Skill-related components of fitness are *agility, balance, coordination, power, reaction time,* and *speed.* These components are more genetically dependent than the health-related components and play a role in some AF specialties (occupation specific).

**1.8.1.3.3. Performance-Related Physical Fitness/Total Fitness.** Includes both health-related components plus skill-related components of physical fitness and may be termed total fitness. Note that “Total Fitness” (the complete ability to perform physical activity) = Health-Related Fitness + Skill-Related Fitness. Although the skill-related components are particularly important to athletes and some AF occupations, the five health-related components are most important for daily functional living and weight gain prevention. Interestingly, research shows that fit athletes who become inactive are less fit than nonathletes who remain consistently active. So, to perform your daily tasks with vigor and alertness, without undue fatigue, and with ample energy to enjoy leisure time pursuits and meet unforeseen emergencies, you should focus your lifelong physical activity on the five health-related fitness components above.

**1.8.1.4. Exercise Prescription/Professional Guidelines.**

**1.8.1.4.1. Exercise Program Goal and Objectives.** The fundamental goal of a physical fitness program is to bring about a change in personal fitness behavior to enhance health and performance, which includes, at a minimum, habitual physical activity. This regular physical activity should result in long-term exercise compliance and attainment of individual fitness goals and objectives. As addressed above, these individual fitness goals and objectives are either at “tier one”—gain health
benefits, prevent hypokinetic (inactivity) disorders, and maintain functional capacity, or at “tier two”—seek to attain greater health benefits and higher levels of fitness/human performance by engaging in physical activity of more vigorous intensity and greater volume (longer duration and greater frequency). To meet exercise goals, one must carry out an exercise prescription.

1.8.1.4.2. Exercise Prescription: Purpose/Art. The art of exercise prescription (Ex Rx) is the successful integration of exercise science with behavioral techniques that result in long-term program compliance and attainment of an individual's goals; i.e., techniques presented should be applied with flexibility and careful attention to the needs of the individual. Various purposes of Ex Rx include promoting health, enhancing physical fitness, and ensuring safety during exercise. Based on individual interests, health needs, clinical state, and performance status, these purposes of Ex Rx do not carry equal or consistent weight. Specific individual outcomes are the ultimate target for the Ex Rx.

Recommended exercise guidelines from leading professional organizations address the first tier and are listed just below. This document addresses both tiers; to meet tier one and tier two goals and objectives, one must execute a balanced exercise program that trains the human energy systems and follows primary exercise principles, addressed in the sections following these exercise guidelines.


1.8.1.4.3.1. 2007 Physical Activity and Public Health Guidelines of the ACSM and the AHA. Guidelines for healthy adults under age 65 – Basic Recommendations from the American College of Sports Medicine (ACSM) and the American Heart Association (AHA) include:

- Do moderately intense cardio 30 minutes a day, 5 days a week

  or

- Do vigorously intense cardio 20 minutes a day, 3 days a week

  and

- Do 8 to 10 strength training exercises, 8 to 12 repetitions of each exercise, twice a week.

Moderate-intensity physical activity means working hard enough to raise your heart rate and break a sweat yet still being able to carry on a conversation. It should be noted that to lose weight or maintain weight loss, 60 to 90 minutes of physical activity may be necessary. The 30-minute recommendation is for the average healthy adult to maintain health and reduce the risk for chronic disease. For further info: [http://www.acsm.org//AM/Template.cfm?Section=Home_Page](http://www.acsm.org//AM/Template.cfm?Section=Home_Page)
1.8.1.4.3.2. 2008 Physical Activity Guidelines of the HHS. Key Guidelines for Adults include:

- All adults should avoid inactivity. Some physical activity is better than none, and adults who participate in any amount of physical activity gain some health benefits.

- For substantial health benefits, adults should do at least 150 minutes (2 hours and 30 minutes) a week of moderate-intensity aerobic physical activity, or 75 minutes (1 hour and 15 minutes) a week of vigorous-intensity aerobic physical activity, or an equivalent combination of moderate- and vigorous-intensity aerobic activity. Aerobic activity should be performed in episodes of at least 10 minutes, and, preferably, it should be spread throughout the week.

- For additional and more extensive health benefits, adults should increase their aerobic physical activity to 300 minutes (5 hours) a week of moderate-intensity aerobic physical activity, or 150 minutes a week of vigorous-intensity aerobic physical activity, or an equivalent combination of moderate- and vigorous-intensity activity. Additional health benefits are gained by engaging in physical activity beyond this amount.

- Adults should also do muscle-strengthening activities that are moderate or high intensity and involve all major muscle groups on two or more days a week, as these activities provide additional health benefits.


1.8.1.4.3.3. Centers for Disease Control and Prevention (CDC). Physical activity is anything that gets your body moving. According to the HHS 2008 Physical Activity Guidelines for Americans, you need to do two types of physical activity each week to improve your health—aerobic and muscle strengthening. For important health benefits adults need at least:

- 2 hours and 30 minutes (150 minutes) of moderate-intensity aerobic activity (i.e., brisk walking) every week

  and

- Muscle-strengthening activities on two or more days a week that work all major muscle groups (legs, hips, back, abdomen, chest, shoulders, and arms).

  or

- 1 hour and 15 minutes (75 minutes) of vigorous-intensity aerobic activity (i.e., jogging or running) every week

  and
• Muscle-strengthening activities on two or more days a week that work all major muscle groups (legs, hips, back, abdomen, chest, shoulders, and arms).

or

• An equivalent mix of moderate- and vigorous-intensity aerobic activity

and

• Muscle-strengthening Activities on two or more days a week that work all major muscle groups (legs, hips, back, abdomen, chest, shoulders, and arms).

For further info:  http://www.cdc.gov/

1.8.1.5. Muscle for Movement – Fiber Types. Skeletal muscle contains two major fiber types, slow twitch (ST) and fast twitch (FT), classified by speed of action. Myosin ATPase, the enzyme that catalyzes adenosine triphosphate (ATP) to adenosine diphosphate (ADP) + inorganic phosphate (Pi) + energy for muscle filament action (myosin filament binding to actin filament), is in a slow form for ST and a fast form for FT. Further classification of muscle fibers is based on the characteristics of the fibers. ST or Type I fibers have high oxidative capacity but slow contractile speed, hence the label SO for slow oxidative. ST fibers also have low glycolytic capacity and low motor unit strength but high fatigue resistance. FT or Type II fibers have low oxidative capacity but fast contractile speed, hence the label FG for fast glycolytic (for glycolysis for energy production). FT fibers also have high motor unit strength but low fatigue resistance. FT fibers are further divided into Type IIa and Type IIb or FOG (fast oxidative glycolytic) and FG. Type IIa or FOG has characteristics intermediate to ST and FG; endurance training can transition FT Type IIb towards FOG Type IIa.

1.8.1.6. Exercise Principles. Within any exercise program, certain principles apply. Understanding these principles will not only provide a solid foundation for a lifestyle of physical activity and human performance but will also help one recognize false claims about exercise and fat gain prevention. Ten primary principles of exercise follow:

1. Overload. To obtain health and fitness improvements, i.e., an overall change in the body, physical activity must place a demand on the body at a level beyond its current ability. The appropriate overload for each person can be achieved by varying the combinations of frequency, duration, and intensity of the physical activity. Regular physical activity is, in essence, a series of small challenges that gradually bring about improvement of body systems. As adaptation occurs, more load is necessary for continued improvement. This concept of individual and progressive overload applies to all, from the very sedentary unfit person to the elite athlete.

2. Progression. To avoid injury, illness, and discouragement with program progress, we must be patient with the exercise prescription and therefore ensure that increases in duration, frequency, and especially intensity of activity occur in a gradual fashion. This is especially important in the first 2
3 weeks of starting a new program or activity regime. Major physiological improvements take at least a few weeks, so without a certain measure of patience some people who fail to see immediate improvement will unfortunately resume an inactive lifestyle—don’t fall prey to this. Stick with the Ex Rx and positive changes will occur over time.

3. **Regularity.** We cannot store fitness or health benefits, so we must remain active; a physical activity/exercise stimulus must occur at minimal levels of duration and frequency over time and over the life span. There is an element of truth to the adage “Use It or Lose It.”

4. **Balance.** A balanced activity/exercise program is necessary to address the components of fitness and, in turn, prevent fat gain and obtain health benefits and performance improvements. Additionally, moderation goes hand in hand with balance. Temper zealous dedication with judgment and moderation and beware of “overtraining”—too much or too sudden can be harmful.

5. **Specificity.** Health benefits and body fat gain prevention are not highly specific to a certain type or mode of activity as long as the activity involves some whole body movement that meets the basic minimal exercise prescription requirements. However, specificity does apply to a certain degree in fitness in that gains or adaptations in fitness are fairly specific to the mode or type of activity/exercise applied. For example, swimming and running are activities that will improve aerobic fitness, but a swimmer will tend to perform at higher levels in swimming than in running and vice versa.

6. **Variety.** To keep a fresh outlook on physical activity and to prevent boredom, staleness, and the consequences of a one-dimensional program, it’s a good idea to select a variety of activities. Also, we recommend a hard-easy format: a hard session followed by an easy one, long with short, fast with slow. Avoid the same routine each day but remain consistent, and try not to sacrifice the other exercise principles addressed here.

7. **Recovery.** Another adage that is important is “Rest is Part of Adaptation.” Including easy or off days in the activity schedule, varying the intensity of activity sessions, and obtaining adequate sleep all permit physical adaptations to take place. Proper recovery also helps prevent injury and illness while helping to keep a positive view of regular physical activity.

8. **Individual Response/Differences.** Man was created with varying capacities to adapt to exercise. Many factors can potentially contribute to this—heredity, age, nutrition, rest and sleep habits/patterns, fitness level, attitude/motivation, environmental influences, disease, or injury. Therefore, be aware of specific attributes, limitations, and needs. Also, one must remember to compare individual gains to one’s own baseline fitness level and fixed standards, not necessarily to others.

9. **Reversibility/Regression/Use and Disuse.** Unfortunately, the gains achieved through regular physical activity/exercise are lost with inactivity.
Physical inactivity results in flabby muscles, weak heart, poor circulation, shortness of breath, excess body fat, weakening of bones and connective tissue, and other disuse problems. As stated above, a fit athlete who becomes inactive will be less fit than a nonathlete who has remained physically active via a proper exercise prescription. Again, inactivity is the culprit. The body does not necessarily wear out; rather, it thrives on activity. Therefore, combat disuse problems with a maintenance program of physical activity, full of enjoyable exercises and activities.

10. Potential. Everyone has a ceiling of maximal physical performance, and few achieve it or necessarily need to do so. However, through regular moderate physical activity, one can reach a reasonable percentage of potential, improve quality of life and longevity, better face life’s trials and challenges, and avoid the consequences of inactivity mentioned above.

Few programs have been validated for specific performance outcomes in the aviation and operational environment. For example, it is well accepted that higher anaerobic strength is only one component of G-performance in a high sustained G aircraft. The endurance component of a sustained duration mission must be coupled with good core strengthening and muscle tone. Likewise, balanced aerobic and anaerobic conditioning is critical for the Battlefield Airman to perform the myriad of activities in a typical mission.

1.8.2. Designing and Planning

1.8.2.1. Designing an Exercise Training Program. It is a good approach in exercise program design to work from general to specific and keep program goals at the forefront. Elite athlete training programs often start with multiyear layouts and goals with more specific annual and seasonal objectives. Similarly, the human performance training program for the military member should be designed with training periods or cycles over time in mind. To optimally reach goals and objectives, reduce injury/illness rates, and best prescribe rest periods, program designers can lay out physical training stimuli in a progressive step-wise fashion over the following breakouts:

1. **Annual Cycle.** Training cycle of 1 year that consists of three or four macro training cycles.

2. **Macro Cycle.** Training cycle of 3 to 4 months duration that consists of three or four meso training cycles. The macro cycle should be the longest duration of continuous training prior to an active rest period.

3. **Active Rest.** A rest period, usually 7 – 10 days in duration, from the just completed macro cycle that must not include the same physical and mental stimuli of the recent macro cycle. However, the active rest period should include physical activity that is less structured, mentally refreshing, and different in modality, e.g., a swimmer does not swim during the rest period but plays tennis and cycles.
4. **Meso Cycle.** Training cycle of 1 month duration that consists of weekly micro cycles. Within a macro cycle, each meso cycle builds upon the previous meso cycle in a progressive pattern of training stimuli/exercise prescription application. Recommend scheduling a slight downturn in training stimuli at the end of each meso cycle to aid in overall physical and mental progress, e.g., weekly training volume (swim mileage, resistance training volume) increases each week for 3 weeks and then returns to baseline for week four before repeating at slightly higher levels for the next meso cycle.

5. **Micro Cycle.** Training cycle of 1 week duration that consists of daily general workout sessions. Within a micro cycle the training stimuli can follow a hard-easy-hard format or a hard-hard-easy format. Hard and easy are defined by the combination of frequency, duration, and intensity.

1.8.2.2. **General Workout Session.** The salient phases of a recommended general workout session that ensure a comprehensive approach are described below in the following order: Movement Preparatory Phase, Cardiorespiratory Endurance (Aerobic) Phase, Muscle Fitness Phase, Combined Activity Phase, Skill Phase, and Movement Transition/Cessation Phase.

1.8.3. **Movement Preparation**

1.8.3.1. **Warm-Up.** Unlike static stretching (more on this in Movement Transition/Cessation Phase), activity-specific warm-up prior to exercise does enhance performance and reduce injury risk. Warm-up should always precede physical activity to increase body temperature and blood flow and to guard against muscle, tendon, and ligament strains and tears. Recommend warm-up of 10 – 20 minutes depending on the environmental conditions.

1.8.3.2. **Dynamic Drills.** In addition to warm-up, dynamic drills are helpful to complete the movement preparation phase by providing slight core temperature increase, enhanced blood flow/perfusion, and joint capsule lubrication. These are:

- Leg swing, fore-aft
- Leg swing, side-side
- Hi knee, hi toe
- Leg over (supine)
- Knee to chest (supine)
- Bent leg over-Scorpion (prone)
- Walking knee lift
- Carioca
- Carioca with quick step
- Backward run
- Lunge step + hip up
1.8.4. Cardiorespiratory Endurance

Again, cardiorespiratory endurance is the ability to perform large muscle, dynamic, moderate-to-high intensity exercise for prolonged periods. Cardiorespiratory endurance or cardiorespiratory fitness is the sum physiological capability of the pulmonary system, cardiovascular system, and relevant musculature at rest, during submaximal exercise, maximal exercise, and prolonged work. Many terms have essentially the same meaning as cardiorespiratory endurance: cardiorespiratory fitness; maximal oxygen consumption or uptake; aerobic fitness; functional capacity; physical work capacity; cardiovascular endurance, fitness, or capacity; and cardiopulmonary endurance, fitness, or capacity. It is important to measure cardiorespiratory endurance for exercise prescription, progress, feedback, and motivation in an exercise program; prediction of medical conditions and further diagnoses of health problems; and performance status.

1.8.4.1. Exercise Prescription for Cardiorespiratory Endurance Training. As stated above, the basic goals and objectives of an exercise program focus on health and performance. In seeking to achieve these objectives, an exercise program must place primary emphasis on cardiorespiratory endurance or aerobic fitness. In turn, the key components in an exercise prescription for cardiorespiratory endurance training are modality, frequency, duration, and intensity of exercise.

1.8.4.1.1. Modality or Type of Activity. This is a major factor in an exercise prescription (Ex Rx). Generally, if one meets the minimum thresholds for frequency, duration, and intensity of exercise, then the overall cardiorespiratory fitness improvement is fairly independent of the mode of activity. For an individual program, select activities based on interests, abilities, time availability, equipment, facilities, and personal goals. Studies show that maximal improvements in aerobic fitness occur when the activity:

- Involves a large proportion of total muscle mass
- Maximizes use of large muscles, e.g., muscles around the thigh and hip
- Involves dynamic, rhythmic muscle contractions
- Minimizes static contractions and use of small muscles

Many modes of activity meet the above requirements. These include cross-country (Nordic) skiing, running, cycling, swimming, skating, rowing, walking, aerobic dance, indoor aerobic exercise machines (e.g., cycle ergometer, elliptical, rower, versa climber, stair), and some sports if they are continuous in nature (soccer, basketball, court sports). In battlefield-oriented military training programs, the primary cardiorespiratory endurance training modalities are running, swimming, and ruck marching with nonambulatory alternate cardiorespiratory endurance training activities included in the program to aid in injury prevention and add variety. In aviation-related training programs, the primary training modalities are generally less specific but can include neck strengthening exercises (fast jet) and abdominal strengthening exercises (reduction in lower back pain and better preparation for AGSM).
1.8.4.1.2. Frequency, Duration, Intensity. The final three steps in a proper Ex Rx are frequency or “How Often,” duration or “How Long,” and intensity or “How Hard.” Frequency, duration, and intensity (FDI) are interrelated, and one must exceed minimum thresholds in each to achieve gains in aerobic fitness. Furthermore, this threshold will increase as aerobic fitness improves. Table 1.8.4.1.2-1 below shows the minimum, optimal, and do-not-exceed (safety) levels for cardiorespiratory endurance Ex Rx.

Table 1.8.4.1.2-1. Exercise Prescription – Frequency, Duration, Intensity

<table>
<thead>
<tr>
<th>FDI</th>
<th>Frequency (days/wk)</th>
<th>Duration (min)</th>
<th>Intensity (beats/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Minimum</td>
<td>3</td>
<td>20</td>
<td>Minimum Heart Rate</td>
</tr>
<tr>
<td>Optimal</td>
<td>4 – 6</td>
<td>30 – 45</td>
<td>Target Heart Rate</td>
</tr>
<tr>
<td>Do-Not-Exceed</td>
<td>6</td>
<td>60</td>
<td>DNE Heart Rate</td>
</tr>
</tbody>
</table>

Initial military training should prescribe minimum levels for FDI and gradually move towards optimal levels as training progresses. Program design must include combinations of moderately intense and vigorously intense aerobic activity.

1.8.4.1.3. Intensity Determination. Moderately intense aerobic activity equates to continuous exercise that raises heart and respiratory rates, initiates sweating (varies with climate), and permits conversation; vigorously intense aerobic activity elicits higher physiological responses and permits light or broken conversation. One can objectively assess intensity of exercise via heart rate (HR) formulas:

1.8.4.1.3.1. Maximal HR Formula. Aerobic activity corresponding to HRs in the range of 60% - 90% of age-specific estimated maximal HR (220 – age in years).

\[ \text{Max Heart Rate} = (220 - \text{age}) \]

1.8.4.1.3.2. Karvonen Formula, the HR Range or HR Reserve Formula. Preferred method as percent HR range directly correlates with percent VO\(_2\) max. Steps are:

1. Calculate maximal HR by subtracting age in years from 220.

\[ \text{Males: Max Heart Rate} = (220 - \text{age}) \]

\[ \text{Females: Max Heart Rate} = (230 - \text{age}) \]

\[ \text{or} \]

\[ \text{Max Heart Rate} = (205 - (0.5 \times \text{age})) \]
2. Measure resting HR for 3 to 4 days shortly after waking for a 60-second period while in the same body position each day. Take an average of the measures.

3. Calculate HR range.

\[
\text{Heart Rate Range} = \text{Max Heart Rate} - \text{Resting Heart Rate}
\]

4. Calculate minimum, optimal (target), and do-not-exceed (safety) exercise HRs:

\[
\text{Minimum Exercise Heart Rate} = (50\% \text{ Heart Rate Range}) + \text{Resting HR}
\]

\[
\text{Optimal Exercise Heart Rate} = (75\% \text{ Heart Rate Range}) + \text{Resting HR}
\]

\[
\text{Do Not Exceed Exercise Heart Rate} = (85\% \text{ Heart Rate Range}) + \text{Resting HR}
\]

For example, a 30-yr-old AF member with a resting HR of 70 beats/min (bpm) calculates maximal HR as \(220 - 30 = 190\) bpm and HR range as \(190 - 70 = 120\). Applying the equations:

\[
\text{Minimum Exercise Heart Rate} = (50\% 120) + 70 = 60 + 70 = 130 \text{ bpm}
\]

\[
\text{Optimal Exercise Heart Rate} = (75\% 120) + 70 = 90 + 70 = 160 \text{ bpm}
\]

\[
\text{Do Not Exceed Exercise Heart Rate} = (85\% 120) + 70 = 102 + 70 = 172 \text{ bpm}
\]

Therefore, this individual should keep exercise HR above 130 bpm, but below 172 bpm, targeting 160 bpm for at least 20 to 25 minutes 3 days/week. Unfit individuals should start at the lower end of this exercise HR range. As fitness level increases, the resting HR will decrease; therefore, increase the intensity percentage from low (50%) towards optimal (75%).

1.8.4.1.3.3. Borg Scale. Measuring HRs is not always practical and sometimes problematic. An alternative is to measure exercise intensity subjectively using the Rating of Perceived Exertion (RPE) scale (Table 1.8.4.1.3.3-1), also known as the Borg Scale. While exercising, individuals subjectively rate how hard they are working and select a corresponding numerical rating on the RPE scale that ranges from 6 to 20. The range between 13 and 17 is needed to change aerobic fitness; however, some lifestyle health benefits can be obtained at a lower range of 12. Note that adding one order of magnitude (placing a 0) on the scale number will roughly equate to HR at the corresponding intensity, e.g., a 15 on the scale is roughly equivalent to a 150 BPM HR. Finally, ability to select individual level-of-effort will improve with consistent aerobic exercise over a prolonged period; studies show the scale has high reliability.
1.8.4.2. **Rate of Progression.** A physiological conditioning or training effect will occur at the onset of an exercise program, especially for individuals with low initial fitness levels. Adjustments in modality, frequency, duration, and intensity are necessary to reach higher levels of health and fitness. Patience and perseverance are critical to maintain an active lifestyle and effective exercise program because many will start a physical activity program but quit within the first 2 or 3 weeks of starting and return to an inactive lifestyle. One *must* maintain regular activity for at least 3 or 4 weeks before tangible and lasting health and performance improvements, including body fat loss, will occur. To help ensure that increases in frequency, duration, and especially intensity of exercise occur in a *gradual* fashion, the following stages of progression are helpful to avoid injury, illness, and potential discouragement (see Table 1.8.4.2-1).

**Table 1.8.4.1.3.3-1. Borg Scale**

<table>
<thead>
<tr>
<th>RPE Scale</th>
<th>Subjective Rating</th>
<th>Corresponding HR Equivalence (bpm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>6</td>
<td>60</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Very, very light</td>
<td>70</td>
</tr>
<tr>
<td>8</td>
<td>Very light</td>
<td>80</td>
</tr>
<tr>
<td>9</td>
<td>Fairly light</td>
<td>90</td>
</tr>
<tr>
<td>10</td>
<td>100</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>Somewhat hard</td>
<td>110</td>
</tr>
<tr>
<td>12</td>
<td>120</td>
<td></td>
</tr>
<tr>
<td>13</td>
<td></td>
<td>130</td>
</tr>
<tr>
<td>14</td>
<td></td>
<td>140</td>
</tr>
<tr>
<td>15</td>
<td>Hard</td>
<td>150</td>
</tr>
<tr>
<td>16</td>
<td></td>
<td>160</td>
</tr>
<tr>
<td>17</td>
<td>Very hard</td>
<td>170</td>
</tr>
<tr>
<td>18</td>
<td></td>
<td>180</td>
</tr>
<tr>
<td>19</td>
<td>Very, very hard</td>
<td>190</td>
</tr>
<tr>
<td>20</td>
<td></td>
<td>200</td>
</tr>
</tbody>
</table>

**Table 1.8.4.2-1. Stages of Progression Table for Healthy Individuals, General Guidance**

<table>
<thead>
<tr>
<th>Program Phase</th>
<th>Week</th>
<th>Frequency (sessions/wk)</th>
<th>Duration (min)</th>
<th>Intensity (% HR range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial Stage</td>
<td>1</td>
<td>3</td>
<td>12</td>
<td>40-50</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>3</td>
<td>14</td>
<td>50</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>3</td>
<td>16</td>
<td>60</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>3</td>
<td>18</td>
<td>60–70</td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>3</td>
<td>20</td>
<td>60–70</td>
</tr>
<tr>
<td>Improvement Stage</td>
<td>6 – 9</td>
<td>3 – 4</td>
<td>21</td>
<td>70–80</td>
</tr>
<tr>
<td></td>
<td>10 – 13</td>
<td>3 – 4</td>
<td>24</td>
<td>70–80</td>
</tr>
<tr>
<td></td>
<td>14 – 16</td>
<td>3 – 4</td>
<td>24</td>
<td>70–80</td>
</tr>
<tr>
<td></td>
<td>17 – 19</td>
<td>4 – 5</td>
<td>28</td>
<td>70–80</td>
</tr>
<tr>
<td></td>
<td>20 – 23</td>
<td>4 – 5</td>
<td>30</td>
<td>70–80</td>
</tr>
<tr>
<td></td>
<td>24 – 27</td>
<td>4 – 5</td>
<td>30</td>
<td>70–85</td>
</tr>
<tr>
<td>Maintenance Stage</td>
<td>28+</td>
<td>3</td>
<td>30 – 45</td>
<td>70–85</td>
</tr>
</tbody>
</table>
1.8.4.2.1. **Initial Stage.** Include low level aerobic activities and light muscular endurance exercises for minimal muscle soreness or discomfort. Do not be aggressive in this stage. Set individual goals that are achievable and realistic; include a system of personal rewards. The majority of failures occurs in this stage—persevere to experience benefits.

1.8.4.2.2. **Improvement Stage.** Progress more rapidly here at a higher intensity; steadily increase duration to 30 minutes of continuous exercise. Increase frequency as adaptation to exercise permits.

1.8.4.2.3. **Maintenance Stage.** After 6 months of regular activity, focus on maintenance. Review goals, ensuring that long-term focus is on a lifestyle exercise behavior of consistency.

1.8.4.3. **Acute Responses and Chronic Adaptations to Exercise.** Extensive coverage of 1) the physiological responses to an acute bout of exercise and 2) the physiological adaptations to regular exercise training can be found in reputable exercise physiology texts and American College of Sports Medicine publications and are addressed in the USAF Exercise Principles and Methods Course.

1.8.5. **Run Training**

Running is likely the most common cardiorespiratory endurance exercise modality in military training. This section focuses on running, specifically distance running, but one must note that other modalities such as cycling and swimming are also quite adequate to elicit physiological training effects.

1.8.5.1. **Running Performance.** Three primary determinants that explain greater than 70% of the intersubject variance in distance running performance are the functional abilities:

1. Maximal Oxygen Consumption/Uptake (VO$_{2\text{max}}$)
2. Lactate Threshold/Onset of Blood Lactate Accumulation (OBLA)
3. Running Economy

These three functional abilities have both genetic and environmental components. The genetic component is lesser and fixed, where as physical training exerts profound effects on the three determinants and, in turn, performance.

1.8.5.1.1. **Maximal Oxygen Consumption/Uptake (VO$_{2\text{max}}$).** VO$_{2\text{max}}$ is expressed as the maximal volume of oxygen taken in, transported, and used by the pulmonary, cardiovascular, and skeletal muscular systems. The true or gold standard laboratory measurement of VO$_{2\text{max}}$ is the collection and analysis of expired air samples during exercise of progressing intensity to maximal levels. The final measure is expressed as: 1) volume of oxygen consumed per minute (liters O$_2$·min$^{-1}$) as absolute VO$_{2\text{max}}$ for nonambulatory exercise (e.g., cycling), or 2) more typically as volume of oxygen consumed relative to body weight per minute (mL·O$_2$·kg$^{-1}$·min$^{-1}$) for ambulatory exercise (e.g., running).
The interaction between cardiac output and arterial-venous oxygen \( (O_2) \) difference defines \( VO_{2\text{max}} \) or cardiorespiratory endurance. Cardiac output, the product of heart rate (HR) and stroke volume (SV), is the amount of oxygenated blood pumped by the heart per minute. Arterial-venous oxygen difference is the difference between the oxygen content of the arterial blood and the oxygen content of the venous blood, which equals the amount of oxygen extracted by the tissues – primarily the working muscle. The product of cardiac output and arterial-venous oxygen difference is the rate at which the body tissues are consuming oxygen.

\[
VO_{2\text{max}} = (HR \times SV) \times (a - v O_2 \text{ difference})
\]

where:
- \( HR \) = heart rate (beats per minute)
- \( SV \) = stroke volume (mL per beat)
- \( a - v O_2 \text{ difference} \) = arterial-venous oxygen difference (mL O\(_2\) per mL)

\( VO_{2\text{max}} \) or cardiorespiratory endurance is measured as above in the laboratory via a progressive maximal test to volitional exhaustion. \( VO_{2\text{max}} \) may also be predicted via field tests or submaximal exertion tests. Field tests include walk, walk-run, run, cycle, or swim tests for a set time or for timed completion of a specified distance. For optimal prediction of maximal \( O_2 \) consumption, these are maximal efforts with strong motivation and some sense of pacing. Submaximal tests predict \( VO_{2\text{max}} \) from submaximal measures of efficiency of measured variables (usually HR response). Step, treadmill, cycle ergometer, or other exercise modes are used with single- or multiple-stage protocols.

1.8.5.1.2. Lactate Threshold/Onset of Blood Lactate Accumulation. Lactate Threshold (LT) is the point at which blood lactate accumulates above resting levels during exercise of increasing intensity; turn point on the curve. Onset of Blood Lactate Accumulation (OBLA) is the point at which blood lactate accumulation begins, approximately 4 mmol·L\(^{-1}\).

LT determines the fraction of \( VO_{2\text{max}} \) that can be sustained, usually expressed in terms of percent \( VO_{2\text{max}} \). For two individuals with the same \( VO_{2\text{max}} \), the one with the higher LT or OBLA will elicit the better performance.

1.8.5.1.3. Running Economy. Oxygen cost per rate of work; better economy equates to a lower \( VO_2 \) value for the same rate of work. How efficient the runner is at converting available energy into running velocity. Dependent on percent slow twitch fibers, mechanical efficiency, musculotendonous stiffness, and ventilatory cost.

1.8.5.2. Chronic Adaptations to Exercise Run Training/Training to Improve. Current run training methods have largely developed from a trial-and-error approach of runners and coaches, while contributions from scientists have been relatively small due to a variety of reasons, including reluctance of coaches to acknowledge potential merit of research for improving training methods and scientific research has been limited by methodological problems. Therefore, available training methods often include elements of sound principles mixed with questionable or nonefficacious methods. Program designers must view traditional-historical methods
with caution and always ask, “Why should I include this in my program?” Finally, scientists can still provide valuable training recommendations by integrating information from the limited quality training studies with related scientific knowledge. Run training elicits changes in morphological/physiological components across the three determinants of performance.

1.8.5.2.1. $\text{VO}_{2\text{max}}$ – Two General Means:

1. **Morphological/Physiological Factors – Central**
   - Increase left ventricular chamber size and wall thickness – increased SV
   - Increase plasma volume – increased blood volume (BV) and SV
   - Increase erythrocyte mass – increased BV, SV, arterial $\text{O}_2$ content
   - Training to improve central factors: intensity, at or near $\text{VO}_{2\text{max}}$ training. Time spent at $\text{VO}_{2\text{max}}$ – control the recovery intervals

2. **Morphological/Physiological Factors – Peripheral**
   - Increased skeletal muscle mitochondrial density and oxidative enzyme content (and proximity) – increased widening of arterial-venous $\text{O}_2$ difference
   - Increased skeletal muscle capillarity – increased $\text{O}_2$ diffusion and uptake
   - Increased myoglobin concentration – increased $\text{O}_2$ diffusion from sarcolemma to mitochondria
   - Training to improve peripheral factors – volume, submaximal training; base and maintenance work
     - Higher the $\text{VO}_{2\text{max}}$ the less dependence on anaerobic capacity (which is limited); lower the RPE at same work rate
     - Goal is not necessarily to tolerate lactic acid ($\text{H}^+$) buildup but to reduce lactic acid buildup at race pace – do so by increasing aerobic contribution, which is one means of increasing LT pace

1.8.5.2.2. Lactate Threshold/Onset of Blood Lactate Accumulation

1. **Morphological/Physiological Factors**
   - Changes in anaerobic enzymes (decreased phosphofructokinase (PFK), changes in lactate dehydrogenase (LDH) and monocarboxylate transporter (MCT) isofrom) – decreased lactate production
   - Increased skeletal muscle mitochondrial density and oxidative enzyme content – increased pyruvate to Krebs cycle versus lactate production
   - Increased beta oxidation enzymes – increased lipid oxidation, decreased demand for carbohydrate (CHO) metabolism and lactate production at given work rate
   - Increased muscle strength – reduced recruitment of Type II fibers and reduced blood flow occlusion
2. Training to Improve

- Threshold runs, interval runs (same as with VO$_{2\text{max}}$)
- Also repetition runs
- Some muscle fitness training, especially “aerobic rotations”

1.8.5.2.3. Running Economy

- Typically improves with increased training time (chronic) and concomitant improvement in running skill
- Cumulative distance over years of training, not the training volume per se

1. Morphological/Physiological Factors

- Change in fast twitch towards slow twitch (FOG) muscle fiber - reduced energy cost per given force production
- Increased mechanical efficiency – reduced whole body energy demand
- Increased musculotendinous stiffness – increased storage and return of elastic energy and muscle stabilization
- Decreased minute ventilation for a specific run velocity – reduced respiratory energy demand

2. Training to improve

- Long runs
- Repetition runs
- Muscle fitness work – resistance training, plyometrics, stability-mobility (core)/elasticity, stiffness

1.8.5.2.4. Brief Summary of Training to Elicit Adaptations

- Quantity to quality spectrum includes: long, steady state, threshold, hills, fartlek, interval, and repetition runs/other training

- Program designer must determine and balance the training load – a product of intensity, duration, and frequency. Leaders may prescribe training load based on training volume (duration x frequency), i.e., miles per week. However, one must not forget intensity, considered the most important variable in training. Intensity may be prescribed as percentage of maximum velocity or race pace or percentages of physiological variables – percent VO$_{2\text{max}}$ or percent HR max.

- A balance of quantity and quality must be included to elicit improvements in the three primary determinants above. Generally, quantity runs affect VO$_{2\text{max}}$ peripheral factors and running economy while quality runs affect all three determinants.
1.8.5.3. Run Ability Groups. When training in groups, e.g., students in a course, it is efficacious to employ ability groups to provide optimal application of the physical training stimulus. Group individuals according to their estimated VO\textsubscript{2max} determined from a baseline run test, i.e., the 1.5 mile run (sample grouping below in Table 1.8.5.3-1). Conduct training runs (steady state, interval, fartlek) by ability group.

<table>
<thead>
<tr>
<th>Ability Group</th>
<th>1.5 Mile Test (min:s)</th>
<th>Estimated VO\textsubscript{2max} (mL O\textsubscript{2}/kg/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A+5</td>
<td>≤ 9:12</td>
<td>≥ 56</td>
</tr>
<tr>
<td>+++</td>
<td>9:13 – 9:45</td>
<td>53 – 55</td>
</tr>
<tr>
<td>+++</td>
<td>9:46 – 10:10</td>
<td>51 – 52</td>
</tr>
<tr>
<td>++</td>
<td>10:11 – 10:37</td>
<td>49 – 50</td>
</tr>
<tr>
<td>+</td>
<td>10:38 – 11:06</td>
<td>47 – 48</td>
</tr>
<tr>
<td>A</td>
<td>11:07 – 11:38</td>
<td>45 – 46</td>
</tr>
<tr>
<td>B</td>
<td>11:39 – 12:14</td>
<td>43 – 44</td>
</tr>
<tr>
<td>C</td>
<td>12:15 – 12:53</td>
<td>41 – 42</td>
</tr>
<tr>
<td>D</td>
<td>12:54 – 13:36</td>
<td>39 – 40</td>
</tr>
<tr>
<td>F</td>
<td>≥ 13:37</td>
<td>≤ 38</td>
</tr>
</tbody>
</table>

1.8.5.4. Run Training Methods. Several modes of run training from the quantity to quality spectrum include long, steady state, fartlek, hill, threshold, interval, and repetition runs along with other training.

1.8.5.4.1. Assessment Runs. Runs that include baseline tests, course evaluations, or races that are typically run on flat measured courses such as 400-m tracks or roads. In putting forth a maximal best effort, runners should employ a race plan to achieve the best time for distance and apply learned pacing technique with the use of watches.

1.8.5.4.2. Long and Steady State. Runs that are longer in duration (up to an hour or more) and lower in intensity than other running modes employed. Duration is of import here; run these at a non-straining intensity (Table 1.8.5.4.2-1). Long and steady state runs primarily affect the portion of aerobic metabolism that occurs in the muscle cell, improving the working muscles' ability to take up and use oxygen for energy production. In addition to building cardiorespiratory endurance, these runs also enhance the musculoskeletal system; therefore, these runs build the base for more intense workouts. Intensity for these runs is 65% - 79% of HR max, 59% - 74% of VO\textsubscript{2max} or Karvonen HR reserve (as stated above, the percent Karvonen HR range is identical to the HR equivalent of the same percentage of VO\textsubscript{2max}), or an RPE of 12 – 13. These runs form a strong foundation for the weekly mileage base. The periodic (every 7 to 10 days) long run should amount to no more than 25% of total weekly training mileage. These may be run on grass and dirt surfaces across fields, over hills, through woods, or on roadways. Running on asphalt should be reduced as much as possible and running on concrete should be avoided.
Table 1.8.5.4.2-1. Run Training Pace (Intensity) Guide

<table>
<thead>
<tr>
<th>Ability Group</th>
<th>1.5-Mi Test (min:s)</th>
<th>Long/ Easy Pace (min/mi)</th>
<th>Fartlek Base Pace (min/mi)</th>
<th>Threshold Pace (min/mi)</th>
<th>Interval Pace (min/400 m)</th>
<th>Repetition Pace (min/400 m)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A+5</td>
<td>≤ 9:12</td>
<td>≤ 8:36</td>
<td>≤ 7:20</td>
<td>≤ :54</td>
<td>≤ 1:35</td>
<td>≤ 1:28</td>
</tr>
<tr>
<td>A++++</td>
<td>9:13 - 9:45</td>
<td>9:04</td>
<td>7:45</td>
<td>7:17</td>
<td>1:40</td>
<td>1:33</td>
</tr>
<tr>
<td>A+++</td>
<td>9:46 - 10:10</td>
<td>9:25</td>
<td>8:04</td>
<td>7:34</td>
<td>1:44</td>
<td>1:37</td>
</tr>
<tr>
<td>A++</td>
<td>10:11 - 10:37</td>
<td>9:47</td>
<td>8:24</td>
<td>7:52</td>
<td>1:48</td>
<td>1:41</td>
</tr>
<tr>
<td>A+</td>
<td>10:38 - 11:06</td>
<td>10:10</td>
<td>8:46</td>
<td>8:12</td>
<td>1:53</td>
<td>1:46</td>
</tr>
<tr>
<td>C</td>
<td>12:15 - 12:53</td>
<td>11:37</td>
<td>10:04</td>
<td>9:24</td>
<td>2:10</td>
<td>2:03</td>
</tr>
</tbody>
</table>

1.8.5.4.3. Fartlek. The Swedish word for “speed play”; a type of running that improves mental and physical abilities to adapt to varied intensity, duration, and terrain. In the beginning (first 5 – 8 minutes) of a fartlek run, leaders should establish a base pace (Table 1.8.5.4.2-1), which is slightly faster than long-steady state pace but slower than threshold. Thereafter, instructors should inject surges of varied intensity and duration/distance (50 – 1200 meters) into the base pace. Fartlek run leaders should ensure runners are not “in formation” on these runs; rather runners should run in a loose cluster or “beehive” grouping, maintaining visual awareness and not watching legs and feet of other runners. Runners strive to stay within 10 meters of the leader but can fall back as much as 200 – 400 meters if visual contact is maintained. Leaders can employ teardrop turns to accommodate slower runners.

1.8.5.4.4. Hill. Included in steady state and fartlek runs to improve running fitness and form.

1.8.5.4.5. Threshold. Run as a steady state run but at slightly higher intensity that pushes the limit of aerobic metabolism. The intensity of threshold runs, 88% - 92% of HR max, 83% - 88% of VO_{2max} or Karvonen HR reserve, or an RPE of 14 – 16, approaches the anaerobic threshold or level where anaerobic metabolism nearly supplies a portion of the necessary energy. The “comfortably hard” intensity is roughly between 10 km and half marathon race pace (Table 1.8.5.4.2-1). Threshold runs should be limited to approximately 10% of total weekly training mileage.

1.8.5.4.6. Interval. Repeated bouts of shorter run distances, typically 100 to 1,600 meters in work distance, interspersed with rest periods designed to improve: 1) cardiovascular system’s ability to deliver oxygen to your working musculature, 2) pacing ability, and 3) running form. Concerning the cardiovascular system, interval runs primarily affect the central factors, stimulating an increase in left ventricular chamber size and wall thickness and expansion of blood volume, leading to an increase in stroke volume and concomitant reduction in heart rate for the same work rate. The intensity of interval runs is 98% - 100% of HR max, 94% - 100% of VO_{2max} or Karvonen HR reserve, or an RPE of ≥ 17 (see Table 1.8.5.4.2-1 for interval pace guidance for 400 meter work distance). This intensity is at or near VO_{2max} and the total time spent at VO_{2max} is essential for optimal results; therefore, one must control the recovery intervals. Leaders should monitor these closely to ensure runners start the next repetition on time (see sample guidance in Tables 1.8.5.4.7-1 and 1.8.5.4.7-2).
The goal for the runner is controlled pacing across the work repetitions: maintain at or slightly faster than ability group goal pace across the entire workout. Starting out the initial repetitions at a faster than goal pace and slowing across the workout is worse than remaining consistent across the workout a few seconds slower than pace. Traditional intervals cross the anaerobic threshold (AT) and have both aerobic and anaerobic components for energy supply.

1.8.5.4.7. Repetition. Repeated bouts of shorter run distances, typically 100 to 400 meters in work distance, interspersed with rest periods designed to primarily improve running economy, running mechanics, and speed for goal race pace. Secondly, these runs will also help improve anaerobic or lactate threshold. See Table 1.8.5.4.2-1 for repetition pace guidance for 400-meter work distance. Repetition pace is employed for some of the shorter work distances in the sample guides found below in Tables 1.8.5.4.7-1 and 1.8.5.4.7-2.

<table>
<thead>
<tr>
<th>Wk</th>
<th>Sets x Reps x Work Distance (m)</th>
<th>Work:Rest Ratio</th>
<th>Set Rest (min)</th>
<th>Ability Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2 x 3 x 300</td>
<td>1:0.5</td>
<td>2:00</td>
<td>A 1:26, B 1:29, C 1:35, D 1:39, F 1:40</td>
</tr>
<tr>
<td>2</td>
<td>2 x 4 x 400</td>
<td>1:0.5</td>
<td>3:00</td>
<td>A 1:58, B 2:03, C 2:10, D 2:16, F 2:17</td>
</tr>
<tr>
<td>3</td>
<td>1 x 3 x 1,200</td>
<td>1:0.5</td>
<td>N/A</td>
<td>A 5:53, B 6:10, C 6:29, D 6:49, F 6:50</td>
</tr>
<tr>
<td>4</td>
<td>1 x 3 x 600</td>
<td>1:1</td>
<td>3:00</td>
<td>A 2:46, B 2:55, C 3:04, D 3:15, F 3:16</td>
</tr>
<tr>
<td></td>
<td>2 x 4 x 200</td>
<td>1:1</td>
<td>3:00</td>
<td>A 0:55, B 0:58, C 1:01, D 1:04, F 1:05</td>
</tr>
</tbody>
</table>

Retest 1.5 mile run, then adjust ability group

<table>
<thead>
<tr>
<th>Wk</th>
<th>Sets x Reps x Work Distance (m)</th>
<th>Work:Rest Ratio</th>
<th>Set Rest (min)</th>
<th>Ability Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>5</td>
<td>1 x 3 x 800</td>
<td>1:1</td>
<td>3:00</td>
<td>A 3:56, B 4:07, C 4:19, D 4:33, F 4:34</td>
</tr>
<tr>
<td></td>
<td>1 x 3 x 600</td>
<td>1:1</td>
<td>3:00</td>
<td>A 2:46, B 2:55, C 3:04, D 3:15, F 3:16</td>
</tr>
<tr>
<td></td>
<td>1 x 4 x 300</td>
<td>1:1</td>
<td>3:00</td>
<td>A 1:22, B 1:27, C 1:31, D 1:36, F 1:37</td>
</tr>
<tr>
<td>6</td>
<td>4 x 5 x 200</td>
<td>45/35/25/15 cutdown</td>
<td>2:00</td>
<td>A 0:55, B 0:58, C 1:01, D 1:05, F 1:06</td>
</tr>
<tr>
<td>7</td>
<td>2 x 4 x 400</td>
<td>1:0.5</td>
<td>N/A</td>
<td>A 1:58, B 2:03, C 2:10, D 2:16, F 2:17</td>
</tr>
</tbody>
</table>

*Cutdown rest: 200-m work intervals with progressively shorter rep rest; 45/35/25/15-seconds rep rest for four sets, respectively, i.e., 45 seconds between reps in first set, 35 seconds between reps in second set, etc. Two minutes rest between sets.
### Table 1.8.5.4.7-2. Sample 8-Week Interval Run Guide, Ability Groups A+5 – A+

<table>
<thead>
<tr>
<th>Wk</th>
<th>Sets x Reps x Work Distance (m)</th>
<th>Work:Rest Ratio</th>
<th>Set Rest (min)</th>
<th>A+5</th>
<th>A+4</th>
<th>A+3</th>
<th>A+2</th>
<th>A+</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2 x 3 x 300</td>
<td>1:0.5</td>
<td>2:00</td>
<td>1:09</td>
<td>1:13</td>
<td>1:16</td>
<td>1:18</td>
<td>1:22</td>
</tr>
<tr>
<td>2</td>
<td>2 x 4 x 400</td>
<td>1:0.5</td>
<td>3:00</td>
<td>1:35</td>
<td>1:40</td>
<td>1:44</td>
<td>1:48</td>
<td>1:53</td>
</tr>
<tr>
<td>3</td>
<td>1 x 3 x 1,200</td>
<td>1:0.5</td>
<td>N/A</td>
<td>4:45</td>
<td>5:00</td>
<td>5:12</td>
<td>5:25</td>
<td>5:38</td>
</tr>
<tr>
<td>4</td>
<td>1 x 3 x 600</td>
<td>1:1</td>
<td>3:00</td>
<td>2:12</td>
<td>2:19</td>
<td>2:25</td>
<td>2:31</td>
<td>2:39</td>
</tr>
<tr>
<td></td>
<td>2 x 4 x 200</td>
<td>1:1</td>
<td>3:00</td>
<td>0:43</td>
<td>0:46</td>
<td>0:48</td>
<td>0:50</td>
<td>0:52</td>
</tr>
</tbody>
</table>

Retest 1.5-mi run, then adjust ability group:

<table>
<thead>
<tr>
<th>Wk</th>
<th>Sets x Reps x Work Distance (m)</th>
<th>Work:Rest Ratio</th>
<th>Set Rest (min)</th>
<th>A+5</th>
<th>A+4</th>
<th>A+3</th>
<th>A+2</th>
<th>A+</th>
</tr>
</thead>
<tbody>
<tr>
<td>5</td>
<td>1 x 3 x 800</td>
<td>1:1</td>
<td>3:00</td>
<td>3:10</td>
<td>3:20</td>
<td>3:28</td>
<td>3:36</td>
<td>3:46</td>
</tr>
<tr>
<td>6</td>
<td>1 x 3 x 600</td>
<td>1:1</td>
<td>3:00</td>
<td>2:12</td>
<td>2:19</td>
<td>2:25</td>
<td>2:31</td>
<td>2:39</td>
</tr>
<tr>
<td></td>
<td>1 x 4 x 300</td>
<td>1:1</td>
<td>3:00</td>
<td>1:04</td>
<td>1:09</td>
<td>1:12</td>
<td>1:15</td>
<td>1:18</td>
</tr>
<tr>
<td>7</td>
<td>4 x 5 x 200</td>
<td>45/35/25/15 cutdown&lt;sup&gt;a&lt;/sup&gt;</td>
<td>2:00</td>
<td>0:43</td>
<td>0:46</td>
<td>0:48</td>
<td>0:50</td>
<td>0:52</td>
</tr>
<tr>
<td>8</td>
<td>2 x 4 x 400</td>
<td>1:0.5</td>
<td>N/A</td>
<td>1:35</td>
<td>1:40</td>
<td>1:44</td>
<td>1:48</td>
<td>1:53</td>
</tr>
</tbody>
</table>

<sup>a</sup>Cutdown rest: 200-m work intervals with progressively shorter rep rest; 45/35/25/15-sec rep rest for four sets, respectively, i.e., 45 sec between reps in first set, 35 sec between reps in second set, etc. Two minutes rest between sets.

### 1.8.5.5. Run Training Plan.

Each micro cycle in the run plan may contain all types or a mixture of the run types above. Alternate cardiorespiratory endurance training, indoor aerobic exercise machines (see above), should be included in the overall plan to attenuate injury risk and add variety and may be prescribed by physicians or physical therapists for partially limiting injuries. The run plan should progressively build in volume across meso cycles (= months) with minor volume reductions between meso cycles that, in turn, build to a complete macro cycle. A no-running active rest period should follow each macro cycle.

### 1.8.5.6. Run Shoes and Running Form.

Quality running (versus court, cross training, or other athletic shoes) shoes are necessary for training. Shoes are classified into three basic types:

1. **Cushioned.** For neutral, high arched feet with no overpronation (excess inward motion of the ankle and foot upon foot strike)

2. **Stability.** For moderate to high arches with slight overpronation

3. **Motion Control.** For low arches with overpronation
With regards to running form, one should conduct a mental checklist from head to foot to avoid biomechanical deficits in running form:

- Run with eyes forward, looking ahead, not at the feet of the person running in front of you.
- Hold head straight and steady, don’t rock.
- Keep upper body perpendicular to running direction with shoulders relaxed.
- Arms pivot at the shoulder, not the elbow joint, holding approximately an 80-deg to 90-deg angle at the elbow.
- Increase speed by increasing stride rate/turnover, not by abnormally lengthening stride.

1.8.6. Muscle Fitness

A key to a fit force is reduction of injury and improved recovery following activities. Muscle fitness is in the center of the target ring for any task that relies upon individual strength, flexibility, and movement.

1.8.6.1. Physical Endurance. Physical endurance is composed of two separate but related concepts—cardiorespiratory endurance (covered above) and muscular endurance; the former refers to the whole body whereas the latter refers to the capacity of individual or groups of musculature.

1.8.6.2. Muscle Fitness Principles. Muscular fitness is a linked term that combines muscular strength and muscular endurance and infers the interdependence between these two physical fitness (PF) components. Muscular strength is the maximum force generated by a specific muscle or muscle group, and muscular endurance is the ability of a muscle group to execute repeated contractions over a period of sufficient time duration to cause muscular fatigue. A balanced physical activity program should address the five health-related components of physical fitness, with primary emphasis on aerobic fitness, but muscular fitness is also important as inclusion of muscular strength and muscular endurance exercise provides several benefits:

- Develops muscular strength and endurance to enhance the ability to live a physically independent lifestyle, i.e., improves daily functional living.
- Increases and maintains fat-free (lean) mass, helping to maintain resting metabolic rate, which is beneficial for preventing fat gain.
- Increases the strength and integrity of connective tissue.
- Increases bone mineral density, preventing age-related bone deterioration.
- Combats chronic low back problems.
• Improves the ability of the muscles to recover from physical activity.

• Provides injury protection during deployment, daily work, and sports and recreational activities.

• Alleviates some common musculoskeletal complaints that result in lost duty time and medical treatment costs.

• May provide modest gains in cardiorespiratory fitness.

• May improve mood and self-image.

Muscular fitness exercise is essential to gain the above benefits and to develop muscular fitness to optimal human performance levels.

1.8.6.2.1. Overload, Specificity, and Progression. The three main principles of exercise training must be included in a muscle fitness training program. First, training must provide an overload, or a stimulus, for the muscle to adapt—the means for development of muscular strength and muscular endurance. Through adaptation muscles become stronger and better able to sustain muscular activity. Physiological adaptations to properly executed muscular fitness training include both neuromuscular adaptations and muscle cell adaptations, with the former accounting for nearly all of the overall performance gains in the first weeks of a muscle fitness training program and the latter, muscle cell hypertrophy, accounting for the majority of gains later in a program (more detail on physiological responses and adaptations to muscle fitness training can be found in reputable exercise physiology texts, ACSM publications). Secondly, training should be individualized, i.e., the principle of specificity – designed to meet individual needs. Finally, training should be progressive by including periodic increases in workload as muscular fitness improves.

1.8.6.2.2. Mode and Pattern. The above process of overloading the muscular system is referred to as resistance training, which includes, but is not limited to, calisthenics, weight training, plyometrics, and field exercises. This training should focus on major muscle groups in the core and lower and upper regions of the body and should address the primary movement patterns (in priority order) of run, bend, twist, squat, pull, and push.

1.8.6.2.3. Volume and Intensity. For general fitness and health, ACSM, AHA, and CDC recommend accomplishing 8 to 10 resistance training exercises involving all major muscle groups at least twice per week. They recommend one set of 8 to 12 repetitions of each exercise performed at moderate or high intensity to muscular fatigue. However, for higher levels of fitness with a human performance focus, one should perform multiple-set resistance training regimens. Generally, for muscular strength development two to three sets of 10 to 12 repetitions of an exercise should be conducted at a 1:1 to 1:3 work:rest ratio. Resistance load should be set so the last few repetitions of an exercise are challenging but still feasible with controlled form. For muscular endurance development, two to three sets of 15 to 20 repetitions of an exercise should be conducted at a 1:1 work:rest ratio. Finally, for muscular power
development, three or more sets of fewer repetitions at longer work:rest ratios are needed. For muscular strength and endurance workouts, a maximum of 1 minute to transfer between exercise stations should be allowed. Other set-repetition-work:rest combinations may be used, e.g., 1-minute cycle (below in calisthenics section).

1.8.6.2.4. Procedures and Safety. The following resistance training procedures work in conjunction with the above recommendations:

1.8.6.2.4.1. Frequency. One can conduct resistance training nearly every day of the week (recommend no more than 6 days/week) as long as sufficient variety and balance are designed into the program. Exercises specific to location or pattern typically are spaced across the week, i.e., ≈ every 48 hours. Different apparatus, free weights, machines, medicine balls, etc. can be used here.

1.8.6.2.4.2. Order of Execution. Try to not work the same muscle group with consecutive exercises. Rather, work the major areas of the body over the priority movement patterns at one time, i.e., core, lower, then upper with occasional mixing of exercises within major areas to maintain variety and prevent staleness in the workout routine. Also, start with exercises of greatest priority and follow with exercises of lesser importance.

1.8.6.2.4.3. Control. Training activities should be rhythmic, performed at a controlled (not necessarily slow) speed, and not interfere with normal breathing.

1.8.6.2.4.4. Range of Motion (ROM). Conduct exercises over the joint’s full ROM in a controlled manner; however, limit the ROM on open and closed chain exercises involving the knee (e.g., squats, avoid dropping past 90 degrees of flexion; leg extensions, limit to ≈ 20 deg to ≈ 60 deg of motion to protect the knee joint).

1.8.6.2.4.5. Rest Time. Keep rest time between exercises (and between sets if multiple sets are used) controlled as above. Avoiding long, time-wasting breaks results in better fitness and a more efficient workout and increases the likelihood for retaining resistance training as a lifestyle behavior.

1.8.6.2.4.6. Safety. Perform a proper warm-up, work antagonistic (opposite) muscle groups, and use a spotter if using free weights.

1.8.7. Muscle Fitness Testing

Muscular fitness testing varies in assessment tools and standardization. Prior to any testing, leaders must consider member familiarity with equipment and procedures, safety, breathing, and rest between assessments.

1.8.7.1. Muscular Strength Testing. Static or isometric muscular strength can be measured by cable tensiometers and handgrip dynamometers. Measures are specific to both the muscle group and joint angle involved in testing; therefore, their utility in describing overall muscular strength is limited. Dynamic or isotonic strength can be measured by various 1-repetition maximum (1-RM) (heaviest
weight that can be lifted only once) tests. Also, isokinetic testing involves use of expensive machines that assess muscle tension generated throughout a range of joint motion at a constant angular velocity, a measurement of peak rotational force or torque.

1.8.7.2. **Muscular Endurance Testing.** Test by lifting a submaximal level of resistance and measuring the number of repetitions or duration of static contraction to fatigue. Measurements are conducted via machine or calisthenic field tests (push-ups, pull-ups, sit-ups).

1.8.8. **Stability and Mobility**

Stability and mobility are terms recently combined with flexibility in the final health-related physical fitness component to designate a broader term that encompasses the role of stability and mobility in posture, occupational functional movement, and daily functional living. Stability deals with maintaining nonmovement functional positions, including postural stability. Stability ranges from shoulder to ankle with shoulder, core, and hip stability as primary. Mobility, similar to stability, is stable, controlled, functional movement through an active range of motion in the various planes of motion. No published tests or standards are yet available for assessing mobility or stability. However, exercises that are known to enhance stability and mobility, especially in the core region, for functional fitness should be added to the muscle fitness training regimen. The core region encompasses the musculature that anchors and acts on the pelvis, providing the stable framework for motion. These muscles include the abdominals, obliques, quadratus lumborum, hip abductors and adductors, anterior hip musculature (iliopsoas group), posterior hip musculature (gluteals and tensor fasciae latae), hip rotators, and lower back muscles (erector spinae-lumbar and semispinalis).

1.8.9. **Resistance Training Modes**

1.8.9.1. **Calisthenics.** Calisthenics are body resistance exercises to improve muscular fitness, e.g., sit-ups, push-ups, and pull-ups. A calisthenics-based approach to exercise and, more specifically, exercise testing has advantages and disadvantages:

**Advantages:**

- Minimal space required
- Minimal cost for mats and incidentals
- May be used in the field (outdoor)
- Historical – traditional basis: historically, military services have incorporated calisthenic-type measures as metrics (does not necessarily mean the best or optimal means for testing)
- Sister Service normative scores exist; however, not anchored in criterion standards
Disadvantages:

- Relatively high degree of interindividual test variability (confirmed by AF feasibility testing)
- Relatively high degree of subjectivity in test administration (confirmed by AF feasibility testing)
- Not optimal scientifically; Sister Service science experts confirm significant negatives
- Traditional pattern of sit-up, push-up, and pull-up does not assess (or very limited) lower body
- Does not assess muscular strength component of physical fitness, i.e., measures muscular endurance
- Difficult, at best, to set either health- or performance-related criterion standards for calisthenic tests

1.8.9.1.1. Calisthenic Methods. One of several means of physical training with calisthenic exercises is a basic set procedure that provides a fairly objective means for controlling an individual exercise prescription. The goal of this procedure is to accomplish 33% of a one-test (one-set) maximum in a three-set pattern on a controlled 1-minute cycle. First, determine the current maximum number of repetitions of the exercise in a 1-minute period i.e., use the most current evaluation result. Second, take 33% of that total number for the amount of repetitions to perform for each of three sets. Rest between sets is on the minute cycle, i.e., perform the first set of repetitions, rest for the remainder of 1 minute, start the next set at the 60-second cumulative time mark, and start the third set at the cumulative 120-second mark. Perform these three sets of exercise once per day starting at 2 to 3 days per week, building to a maximum of 6 days per week. For example, an individual with a sit-up test maximum of 44 repetitions will perform three sets of 15 repetitions per set on the minute cycle: 15 repetitions – rest remainder of the first minute, 15 repetitions – rest remainder of second minute, 15 repetitions. Over time add repetitions to each set as the last few repetitions of the third set become easier.

1.8.9.1.2. Calisthenic Exercises (Limited Listing).

- Core (Bend and Twist). Crunch, cross-knee crunch (feet on wall), windshield wiper, hanging knee to elbow, sit-up, flutter kick, V-raise, plank series (bridges) with leg raises

- Lower and Whole Body (Run and Squat). Squat, lateral squat, single leg squat, lunge, walking lunge, Iron Mike, squat thrust, mountain climber, multi-count body builder, Burpee

- Upper (Pull, Push). Pull-up; pull-up in L-position; pull-up with partner; arm rotations; push-up series; push-up, raised (box, wall)

1.8.9.1.3. Weight Training. Major series of resistance exercises to improve muscular fitness that employ an external object, e.g., machine-based weights (weight stacks, cables, etc.), free weights (bars, plates, etc.), medicine balls, sand bags, weighted vests, and many more.
1.8.9.1.3.1. **Machine and Free Weights.** Some commonly recommended resistance training exercises that are in accordance with ACSM’s major muscle group recommendations are:

- **Core.** Abdominal “crunch,” incline ab, barbell balance with toe touch, trunk rotator, back extension, Captain’s chair
- **Lower.** Leg press, leg (knee) extension, leg (knee curl) flexion, hip flexion and extension, hip abduction and adduction, calf (heel) raise
- **Whole.** Squat, dead lift, thrusters, clean, clean and jerk, snatch
- **Upper.** (lat) pull down, seated or erect row; shoulder or push (overhead) press; bi-directional bent-arm flys (shoulder horizontal flexion and extension); chest (bench) press; arm (bicep) curl; tricep extensions or dips; running dumb bell; lateral raise

1.8.9.1.3.2. **Weight Equipment.** For the beginner, machines are often more optimal than free weights, as machines require less skill, one can start at lower resistance and increase in smaller increments, and it’s easier to control ROM. However, for intermediate and advanced weight training, free weights and other apparatus should be used.

1.8.9.1.3.3. **Medicine Balls.** Another means of weight training with high utility. A partial list of medicine ball (MB) exercises:

- **Core.** Basic rotation close, basic rotation distance with cross-over, reaching wall tap, prone leg hold with arm pass, sit-up and throw, throw from knees to pop-up, two-arm trunk push
- **Lower.** Squat MB thruster with jump/toss/turns, one-legged squat with MB rotation, mule kick
- **Whole.** Three distance rapid wall throw, log throw, wood chopper, wall ball squat thrust, overhead toss, front and rear with partner/with run chase
- **Upper.** Two-arm over, ball slam, two-arm putt, one-arm under, one-arm putt, one-arm over

1.8.9.1.3.4. **Stability/Physio Balls, Bosu Balls, and other Unstable Platforms.** Numerous exercises can be performed with these devices to enhance neuromuscular recruitment patterns and elevate the degree of difficulty.

1.8.9.1.3.5. **Other Weighted Objects.** Other objects for weight training include sand bags, weighted vests, bars, bands, etc.
1.8.9.1.4. **Plyometrics.** Activities that train a muscle to reach maximal force in the shortest possible time. Training exercise drills employed as a supplement to resistance training to develop quick, explosive, and powerful muscles. Plyometric movements are generally classified into four exercise modalities:

1. **Jump.** Two-leg take-off and landing

2. **Hop.** Single-leg take-off, landing on the same foot

3. **Bound.** Single-leg take-off, landing on the opposite foot

4. **Skip.** Single-leg take-off, landing with two feet contact

Plyometric exercises include box, depth, squat, double, lateral jumps, and bounding.

1.8.9.1.5. **Field Activities.** These include carries and drags (buddy, rescue), weighted pulls (sleds, tires), rope pulls and climbs, crawls (low, high, bear), partner relays, grass and guerilla drills.

1.8.9.2. **Functional Fitness.** As in the above paragraph on stability and mobility, functional fitness training requires further development than basic fitness activities. It is physical training that addresses whole body fitness via multi-joint movement, multi-task activities that have increased functionality and relevance to AF operational/occupational actions.

1.8.10. **Combination Training**

1.8.10.1. **Combined Activity Phase.** Physical training that combines aerobic and muscular fitness actions can be quite effective for overall fitness development that, depending upon design, can contain varied degrees of functional fitness training with high occupational task relation.

1.8.10.2. **Rotations.** Multi-station rotations are designed courses that include cardiorespiratory, muscle fitness, and skill components. Designs vary, but in a typical rotation, members run between stations where they must accomplish muscle fitness exercises or skill requirements prior to advancing to the next station. Rotations can be designed to require a set number of requirements with time as the dependent variable or a number of requirements accomplished in a fixed time. Rotations should be designed and applied in a progressive manner, i.e., beginner, intermediate, and advanced levels with increased degree of difficulty (physical, mental, skill), task complexity, or occupational specificity.

1.8.11. **Skill**

Occupational- or athletic-specific skill training should be accomplished at this point of an overall general exercise session.
1.8.12. Movement Transition/Cessation and Flexibility

1.8.12.1. Cool-Down. Although frequently ignored, a brief cool-down period after an exercise session is important. Cool-down, a gradual reduction in activity, permits reduction in the elevated cardiovascular (prevents blood pooling) and metabolic systems and may hasten recovery and avoid injury.

1.8.12.2. Flexibility/Static Stretching. Flexibility is the maximum ability to move a joint freely, without pain, through a range of motion. Flexibility tends to decrease with age, primarily due to the decrease in activity associated with age. Although no single test can be generalized to evaluate total body flexibility, it is important to health and functional living and should be part of a well-balanced exercise routine.

1.8.12.2.1. Timing and Guidelines. Despite the popular perception that stretching prior to exercise enhances performance and prevents injury, little scientific evidence exists to support such long-held beliefs. Recent research actually shows that static stretching prior to exercise causes a temporary weakening of muscle and does not decrease the risk of injury. Therefore, static stretching prior to exercise or an athletic event is contraindicated. Rather, engage in a gradual, activity-specific warm-up that includes the movement patterns of planned activity, e.g., if running for the workout then warm-up with brisk walking, jogging, and dynamic movements or drills such as leg swings and knee raises (above). To help maintain flexibility one should stretch after a workout when muscles, tendons, ligaments, and connective tissue are warmer (above normal body temperature). Static stretch according to the following ACSM guidelines:

- **Type.** Static stretch, with a major emphasis on the major muscle groups to include the low back, hips, iliotibial band, quadriceps and hamstrings, calf, and shin. Do not ballistic (bounce) stretch.

- **Frequency.** 2 to 3 days/week

- **Duration.** 10 to 30 seconds for each stretch

- **Intensity.** To a position of mild discomfort, not to point of pain

- **Repetitions.** Two to three for each stretch

Again, first increase body temperature; don’t “cold” stretch. Finally, avoid comparing one’s level of flexibility to others, as it varies widely across individuals due to several factors that include gender, age, activity level, temperature, and extensibility of the muscles and tendons surrounding the joints.

1.8.13. Body Composition

1.8.13.1. Body Composition Hierarchy. Body composition deals with the relative portion of the body that is composed of fat and fat-free tissue. Body weight and body fat are related to health status, but misconceptions exist regarding body measurements and application of results. In the assessment of fat gain and
associated disease risk and performance degradation, the focus must go beyond body weight measures to relative body fat and body fat distribution.

1.8.13.2. Weight and Height. Measurements of weight and weight relative to height—scale readings, height-weight tables, body mass index (BMI)—do not differentiate between fat and fat-free tissue and do not account for fat distribution pattern. Unfortunately, the societal norm for “measuring” body composition is the scale. Routine use of the home, gym, or doctor’s scale enforces the misconception that body weight is more important than body fatness. BMI, an index of weight and height, is a health screen with recognized, evidence-based guidelines. However, it also cannot fully describe body composition and related health status, as it is not a direct measure of body fat, does not pick up individual changes in body fatness, and is difficult to project changes with exercise and diet via BMI alone. Therefore, scale and BMI readings can mislead one into thinking a change in body weight is a gain or loss of fat alone. Weight changes occur in fat-free components (bone, muscle, organs) as well as in fat. Many are concerned about losing weight to be thin, without realizing that thin does not necessarily equal lean. A lean person with low body fat may weigh more, due to greater muscle mass, than a thin person. Also, inactive thin individuals may not be overweight but often are overfat, whereas lean individuals with fit muscle tissue are typically physically active. Therefore, activity, specifically aerobic and resistance exercise, is very important for preventing loss of fat-free mass and gain in fat mass.

1.8.13.3. Relative Body Fat and Body Fat Distribution. The amount of total body tissue that is fat and where fat is deposited or carried on the body are necessary to complete a body composition assessment. This is done via “non-scale” measurements.

1.8.13.4. Percent Body Fat. Total body fat relative to body mass is known as percent body fat. Average and at-risk levels are 15% and 25% for males, 23% and 32% for females, respectively.

1.8.13.5. Abdominal Circumference. Increased health risks associated with overfat are not only related to total body fat but also and more closely to fat distribution. Upper body or trunk fat, specifically abdominal fat, presents the greatest health risk; it is highly linked to cardiovascular diseases and metabolic disorders such as type II diabetes. Reducing abdominal girth or circumference is more important than normalizing body weight, since exercise-induced increases in muscle mass can mask reductions in girth, i.e., with proper exercise body weight may stay the same or even increase, but “belt size” will reduce. Therefore, as abdominal fat is an independent risk factor for disease, the evaluation of abdominal circumference (AC) is used. A high risk of current and future disease exists for males with an AC > 39 inches regardless of age or height. The health risk is moderate for males with an AC of 35 to 39 inches and low for an AC < 35 inches.

1.8.13.6. Aerobic Fitness and Visceral Adipose Tissue. With a very strong scientific connection between aerobic fitness and abdominal adiposity, unhealthy visceral fat exists. Recent and compelling research data show:
• Moderate to high levels of aerobic fitness provide health risk protection independent of body weight or total adiposity.

• Aerobic fitness provides protection, not by reducing body mass per se, but by reducing fat from the region in which it is most dangerous – reduces visceral fat.

• Aerobic fitness mitigates elevated health risk associated with increases in abdominal fat.

1.8.13.7. **Spot Reducing.** Finally, remember spot reducing is a fallacy. “Ab” machines may aid in strengthening abdominal muscles, but these exercises alone will not reduce abdominal fat or girth. Physical activity and caloric restriction are the best means for reducing total and regional body fat.

References
2007 Physical Activity and Public Health Guidelines of the American College of Sports Medicine (ACSM) and the American Heart Association (AHA).

Concepts
Health

Vocabulary
Exercise and Exercise physiology
Exercise training
Muscle contraction
Physical activity and Physical fitness
1.9. Nutrition

Mark White, M.S.

Mission One: Consider the influence of preflight meals on the aircrew that is alerted to a med-evac mission at the 8th hour of a 12-hour alert period. The crew scrambled from a forward-operating base on a night combat search and rescue mission. As part of the mission, they were to transport two casualties to a forward medical station. Now 14 hours into their day, the crew could have chosen to stay in place as they were off alert status or return to their forward-operating base. The Joint Search and Rescue Center directed the crew to RTB. Following a minimal preflight briefing, the pilot performed a modified marginal power climb profile, the aircraft entered a brown-out condition, and the pilot failed to maintain forward motion, allowing the tail to strike the ground and causing the aircraft to roll inverted.

Mission Two: What influences does daily well-being have on nutritional in-take? A double turn sortie was briefed at 0615 with a planned hot-pit refueling between sorties. Take-off was at 0730 with hot-pit at 0930 – 0950, and second take-off was scheduled for 1015 hours. Approximately 90 minutes into the second sortie (6.5 hours into the pilot’s day without adequate breakfast), the mission pilot began feeling sick to his stomach (nausea) with headache and dry mouth. Symptoms of nausea persisted, so he terminated the mission and RTB. Only upon examination did the pilot admit to mild gastritis exasperated by dehydration, poor nutrition, and daily aspirin therapy.

Mission Three: How does circadian rhythm influence nutrition? Mission crew is scheduled as a replacement crew over a 4-day period. Initial take-off is 0800L for an 8.2-hour sortie, with subsequent mission launches at 1230L, 1745L, and 0230L spanning a 5-day period. Each mission is scheduled to be 6.4 to 10.2 hour in length, and there is ample food available in the galley of the aircraft. Problem is shifting the body’s sleep-rest schedule and eating schedule over a short period of time leaves several of the mission crew forgoing meals in place of rest.

The human body’s need for nourishment is as fundamental as its requirement for oxygen. To sustain life, the body must balance whole-body energy metabolism, that is, the ingestion, degradation, and absorption of nutritional food sources for the development, growth, and movement of individuals as it pertains to their lifestyle. When considering the lifestyle of military personnel, i.e., occupational athletes, their requirements for fit-for-duty are at any given moment and may mean the difference between successful mission-completion and failure. Therefore, military warfighters must take into account the importance of nutrition as it relates to their physical and cognitive performances, both at work and home. The purpose of the following section is to provide basic nutritional facts and recommendations as they are applied to the military warfighter.
1.9.1. Whole Body Metabolism

To provide comprehensive nutritional recommendations, the topic’s foundation begins with the fundamental explanation of whole-body energy metabolism. The body maintains energy balance by the application of Newton’s First Law of Thermodynamics, i.e., energy can neither be created nor destroyed. Essentially, the body acquires energy input from nutritional sources, and then utilizes metabolic processes to convert the chemical energy to mechanical and thermal energy, as depicted in Figure 1.9.1-1. The body receives energy input from three major nutritional constituents – carbohydrates (CHO), proteins (PRO) and lipids (FAT) – and the energy stored is measured (i.e., kilocalories (kcal) or joules) by their complete metabolic combustion (Table 1.9.1-1).

Figure 1.9.1-1. Overview of Whole Body Energy Metabolism
As these constituents (CHO, PRO and FAT) are metabolized through their respective metabolic pathways (i.e., glycolysis, gluconeogenesis, CHO and FAT oxidation), energy is given off (i.e., energy output) in several different forms: resting metabolic rate, diet-induced thermogenesis, nonshivering thermogenesis, and physical activity. An individual’s resting metabolic rate (RMR), aka basal metabolic rate (BMR), represents the metabolic requirement of that particular body to maintain minimal aspects of life (e.g., generate/maintain ion gradients, signal transduction, respiratory and circulatory work, heat regulation, etc.) for a 24-hour period of time. Energy expenditure for RMR is measured in units of kcals or liters of O\textsubscript{2} and is approximately 20 – 25 kcal X kg of body weight\textsuperscript{-1} or 0.2 – 0.25 L of O\textsubscript{2} X min\textsuperscript{-1} for the general adult human, respectively. The regression equations listed in Table 1.9.1-2 are accepted methods for estimating RMR in male and female adults.

### Table 1.9.1-1. Summary of Nutritional Source Metabolic Combustion

<table>
<thead>
<tr>
<th>Nutritional Constituent</th>
<th>kcal gram\textsuperscript{-1} (Produced)</th>
<th>liter O\textsubscript{2} gram\textsuperscript{-1} (Used)</th>
<th>kcal liter O\textsubscript{2} \textsuperscript{-1} (Produced)</th>
<th>Respiratory Quotient \textsuperscript{(RQ)}</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carbohydrates (CHO)</td>
<td>4.2</td>
<td>0.84</td>
<td>5.0</td>
<td>1.00</td>
</tr>
<tr>
<td>Proteins (PRO)</td>
<td>4.3</td>
<td>0.96</td>
<td>4.5</td>
<td>0.80</td>
</tr>
<tr>
<td>Lipids (FAT)</td>
<td>9.4</td>
<td>2.00</td>
<td>4.7</td>
<td>0.70</td>
</tr>
<tr>
<td>Mixed Fuel</td>
<td>---</td>
<td>----</td>
<td>4.8</td>
<td>0.85</td>
</tr>
</tbody>
</table>

### Table 1.9.1-2. Validation of Harris-Benedict and Mifflin Equations for Resting Metabolic Rate in Obese and Nonobese People (from Frankenfield et al., 2003)

<table>
<thead>
<tr>
<th>Gender</th>
<th>w = wt in kg</th>
<th>h = ht in cm</th>
<th>a = age in yr</th>
<th>MGF\textsuperscript{a}</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(13.75 x w) +</td>
<td>(5 x h) -</td>
<td>(6.76 x a) +</td>
<td>66</td>
</tr>
<tr>
<td>Men</td>
<td>(9.56 x w) +</td>
<td>(1.85 x h) -</td>
<td>(4.68 x a) +</td>
<td>655</td>
</tr>
<tr>
<td>Women</td>
<td>(10 x w) +</td>
<td>(6.25 x h) -</td>
<td>(5 x a) +</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>(10 x w) +</td>
<td>(6.25 h) -</td>
<td>(5 x a) -</td>
<td>161</td>
</tr>
</tbody>
</table>

\textsuperscript{a}Metabolic gender factor.

Generally, the central nervous and musculoskeletal systems account for 40% and 20% to 30% of RMR, respectively. Approximately 80% of inter-individual differences are accounted for by lean muscle mass, fat mass, age, and gender, leading to generalizations such as younger individuals have higher RMR than the elderly and women have a lower RMR than men. For average sedentary adults, RMR is about 60% - 70% of their total daily energy expenditure (Figure 1.9.2.1-2). Additional energy expenditures one might incur due to changing environmental conditions pertain to the energy cost of digesting food (i.e., diet-induced thermogenesis), metabolic production of heat (i.e., nonshivering thermogenesis), and any energy used for minor to major physical activity (i.e., fidgeting, manual labor, athletic performance, etc.) (Figure 1.9.1-1). While diet-induced and nonshivering thermogenesis equal 5% to 15% of daily energy expenditure (DEE), the physical activity of daily life can be as great as 20% to 30%. Tables 1.9.1-3 and 1.9.1-4 are based upon general and specific activity exertion levels, respectively.
Table 1.9.1-3. Classification of Physical Activity Based upon Gender  
(from McArdle et al., 2001)

<table>
<thead>
<tr>
<th>Activity Level</th>
<th>kcal X min(^{-1})</th>
<th>L O(^2) X min(^{-1})</th>
<th>mL O(^2) X kg(^{-1}) X min(^{-1})</th>
<th>METs(^a)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Energy Expenditure, Women</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Light</td>
<td>1.5 - 3.4</td>
<td>0.30 - 0.69</td>
<td>5.4 - 12.5</td>
<td>1.2 - 2.7</td>
</tr>
<tr>
<td>Moderate</td>
<td>3.5 - 5.4</td>
<td>0.70 - 1.09</td>
<td>12.6 - 19.8</td>
<td>2.8 - 4.3</td>
</tr>
<tr>
<td>Heavy</td>
<td>5.5 - 7.4</td>
<td>1.10 - 1.49</td>
<td>19.9 - 27.1</td>
<td>4.4 - 5.9</td>
</tr>
<tr>
<td>Very Heavy</td>
<td>7.5 - 9.4</td>
<td>1.50 - 1.89</td>
<td>27.2 - 34.4</td>
<td>6.0 - 7.5</td>
</tr>
<tr>
<td><strong>Energy Expenditure, Men</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Light</td>
<td>2.0 - 4.9</td>
<td>0.40 - 0.99</td>
<td>6.1 - 15.2</td>
<td>1.6 - 3.9</td>
</tr>
<tr>
<td>Moderate</td>
<td>5.0 - 7.4</td>
<td>1.00 - 1.49</td>
<td>15.3 - 22.9</td>
<td>4.0 - 5.9</td>
</tr>
<tr>
<td>Heavy</td>
<td>7.5 - 9.9</td>
<td>1.50 - 1.99</td>
<td>23.0 - 30.6</td>
<td>6.0 - 7.9</td>
</tr>
<tr>
<td>Very Heavy</td>
<td>10.0 - 12.4</td>
<td>2.00 - 2.49</td>
<td>30.7 - 38.3</td>
<td>8.9 - 9.9</td>
</tr>
</tbody>
</table>

\(^a\)One MET = average resting oxygen consumption; L X min\(^{-1}\) based on 5 kcal per L of oxygen; mL X kg\(^{-1}\) based on 55-kg woman and 65-kg man.
Table 1.9.1-4 Energy Expenditures in Men (Based on 70-kg Male)

<table>
<thead>
<tr>
<th>Activity Level</th>
<th>Activity</th>
<th>kcal X min(^{-1})</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. Rest</td>
<td>Basal, aircraft pilots(^a)</td>
<td>1.0</td>
</tr>
<tr>
<td></td>
<td>Lying down fully relaxed(^b)</td>
<td>1.2</td>
</tr>
<tr>
<td></td>
<td>Basal, helicopter pilots(^a)</td>
<td>1.2</td>
</tr>
<tr>
<td></td>
<td>Rest KC-135 flight (experienced pilot)(^c)</td>
<td>1.3</td>
</tr>
<tr>
<td></td>
<td>Routine KC-135 flight (experienced pilot)(^c)</td>
<td>1.5</td>
</tr>
<tr>
<td></td>
<td>Piloting helicopters during aerobatics(^a)</td>
<td>1.6</td>
</tr>
<tr>
<td></td>
<td>Sitting in helicopter prior to engine start(^a)</td>
<td>1.6</td>
</tr>
<tr>
<td></td>
<td>Descent/landing helicopter(^a)</td>
<td>1.7</td>
</tr>
<tr>
<td></td>
<td>Sitting at rest(^b)</td>
<td>1.7</td>
</tr>
<tr>
<td></td>
<td>Seated in aircraft prior to engine start(^a)</td>
<td>1.7</td>
</tr>
<tr>
<td></td>
<td>Aerobatics by aircraft pilots(^a)</td>
<td>1.8</td>
</tr>
<tr>
<td></td>
<td>Emergency KC-135 flight (experienced pilot)(^c)</td>
<td>1.9</td>
</tr>
<tr>
<td>B. Light Activity</td>
<td>4-G turns(^a)</td>
<td>2.3</td>
</tr>
<tr>
<td></td>
<td>Landing(^d)</td>
<td>2.5</td>
</tr>
<tr>
<td></td>
<td>Taxi for takeoff(^a)</td>
<td>2.8</td>
</tr>
<tr>
<td></td>
<td>Rolls(^d)</td>
<td>3.0</td>
</tr>
<tr>
<td></td>
<td>Takeoff(^d)</td>
<td>3.2</td>
</tr>
<tr>
<td></td>
<td>Takeoff by aircraft pilots(^d)</td>
<td>3.2</td>
</tr>
<tr>
<td></td>
<td>Barrell rolls(^a)</td>
<td>3.6</td>
</tr>
<tr>
<td></td>
<td>Slow walking(^b)</td>
<td>3.8</td>
</tr>
<tr>
<td></td>
<td>Walking (3.5 mph)(^f)</td>
<td>4.7</td>
</tr>
<tr>
<td></td>
<td>Aerial combat maneuvering(^d)</td>
<td>4.8</td>
</tr>
<tr>
<td>C. Moderate Activity</td>
<td>Golf (walking and carrying clubs)(^f)</td>
<td>5.5</td>
</tr>
<tr>
<td></td>
<td>Gardening(^b)</td>
<td>5.8</td>
</tr>
<tr>
<td></td>
<td>Table tennis(^b)</td>
<td>5.8</td>
</tr>
<tr>
<td></td>
<td>Hovering in helicopter(^a)</td>
<td>6.0</td>
</tr>
<tr>
<td></td>
<td>Swimming (breaststroke, 1 mph)(^b)</td>
<td>6.8</td>
</tr>
<tr>
<td></td>
<td>Basketball(^f)</td>
<td>7.3</td>
</tr>
<tr>
<td></td>
<td>Chopping wood(^b)</td>
<td>7.3</td>
</tr>
<tr>
<td></td>
<td>Tennis(^e)</td>
<td>7.6</td>
</tr>
<tr>
<td>D. Heavy Activity</td>
<td>Cycling at 10 mph, heavy bicycle(^b)</td>
<td>8.9</td>
</tr>
<tr>
<td></td>
<td>Basketball (recreational)(^f)</td>
<td>9.7</td>
</tr>
<tr>
<td></td>
<td>Bicycling (&gt;10 mph)(^f)</td>
<td>9.8</td>
</tr>
<tr>
<td></td>
<td>Climbing stairs at 116 steps/min(^b)</td>
<td>9.8</td>
</tr>
<tr>
<td></td>
<td>Jogging, 5 mph(^f)</td>
<td>9.8</td>
</tr>
<tr>
<td>E. Very Heavy Activity</td>
<td>X-country skiing(^e)</td>
<td>10.0</td>
</tr>
<tr>
<td></td>
<td>Jogging, 6.7 mph(^e)</td>
<td>13.5</td>
</tr>
<tr>
<td></td>
<td>Running (10 mph)(^e)</td>
<td>17.8</td>
</tr>
</tbody>
</table>

\(^a\) Littell and Joy (1969); average of measurements during flight in 3 different helicopters by the same 5 pilots or in utility aircraft (U-6A) by 4 pilots.

\(^b\) Webb (1973).

\(^c\) Kaufman et al. (1970).

\(^d\) Harding (1987).

\(^e\) Alpers et al. (1995).

\(^f\) Thompson and Veneman (2005).
1.9.2. Macronutrients

As stated previously, the major energy input into the human body consists of nutritional constituents known as carbohydrates (CHO), proteins (PRO), and lipids (FAT). The energy provided by the chemical bonding between carbon (C), hydrogen (H), oxygen (O), and nitrogen (N) (note: found in protein) is insufficient to provide the body with the energy required to maintain itself and any additional physical activity required to sustain life. Therefore, it can be surmised that the chemical energy from CHO, PRO, and FAT is transferred to a more suitable energy source in the production of the high-energy molecule adenosine triphosphate (ATP). Energy in food molecular bonds is chemically released within our cells and then conserved in limited quantities in the form of ATP, which consists of adenosine (adenine + ribose) and three inorganic phosphate (Pi) groups. Carbohydrate provides about 4 kcal (16.7 kJ) of energy per gram compared to about 9 kcal (37.7 kJ) of energy per gram of FAT. However, CHO is more accessible. The rate of energy release is partially determined by the choice of the primary fuel source. The enzyme ATPase acts on ATP to split off a phosphate (Pi), rapidly releasing high energy (7.6 kcal·mole⁻¹ of ATP). ATP is generated through three energy systems. The first two systems are anaerobic, energy is produced in the muscle cell without use of oxygen, and the latter system is aerobic, energy is produced in the muscle cell organelle, the mitochondria, with use of oxygen.

In the ATP-phosphocreatine system (i.e., ATP-PCr system), Pi is separated from phosphocreatine through the action of the enzyme creatine kinase. The Pi can then be combined with ADP to form ATP. This system is anaerobic, and its main function is to maintain ATP levels. The energy yield is 1 mole of ATP per 1 mole of PCr. During initial intense muscular activity, ATP is maintained as PCr declines as it is used to maintain ATP levels. However, after 3 – 15 seconds of maximal effort, ATP and PCr stores are depleted.

The glycolytic system involves the process of glycolysis, through which glucose or glycogen is broken down to pyruvic acid via glycolytic enzymes in the cytoplasm of the cells. When conducted without oxygen, the pyruvic acid is converted to lactic acid. Substrates for exercising muscle are glucose, available from blood glucose and liver glycogen, and muscle glycogen. To initiate glycolysis, muscle glycogen is broken down via glycogenolysis to glucose-1-phosphate and, in turn, to glucose-6-phosphate; glucose also converts to glucose-6-phosphate but at the cost of an ATP (one molecule of ATP per one molecule of glucose). One mole of glucose yields two moles of ATP, but one mole of glycogen yields three moles of ATP. The ATP-PCr and glycolytic systems are major contributors of energy during the early minutes of high-intensity exercise (the glycolytic system is limited to approximately 60 – 90 seconds in well-trained individuals). These anaerobic systems are limited in capacity, hence the term “anaerobic capacity” (whereas aerobic metabolism is measured as a rate).

The oxidative system is the primary means for energy production and involves breakdown of fuels with the aid of oxygen—this is true respiration or cellular respiration. This system yields more energy than the ATP-PCr or glycolytic system. Oxidation of carbohydrate involves: 1) glycolysis, 2) the Krebs cycle, and 3) the electron transport chain-oxidative phosphorylation. The latter two steps occur inside the cell organelles, the mitochondria, which in muscle are adjacent to the myofibrils and throughout the sarcoplasm. At the end of glycolysis, pyruvic acid converts to acetyl coenzyme A versus lactic acid in the anaerobic process. Acetyl CoA enters the Krebs cycle for oxidation and production of two moles of ATP. The remaining carbon (after
breakdown) combines with oxygen to form CO₂, which diffuses to the blood for transport to the lungs. Hydrogen from glycolysis and the Krebs cycle combines with coenzymes (NAD, nicotinamide adenine dinucleotide, and FAD, flavin adenine dinucleotide), which carry the hydrogen atoms to the electron transport chain where they are split into protons and electrons. The protons combine with oxygen to form water, and the electrons are transported through a series of reactions to provide energy for the phosphorylation of ADP to ATP. This process produces 39 molecules of ATP per molecule of glycogen (38 ATP per glucose); triglycerides from adipose cells and intramuscular fat deposits break down to glycerol and free fatty acids (FFA). FFA is released to the blood and diffuses into the muscle fibers for oxidation. Fat oxidation begins with beta (β) oxidation of FFA and then follows the same latter path as carbohydrate oxidation: the Krebs cycle and the electron transport chain-oxidative phosphorylation. The energy yield for fat oxidation is much higher than for carbohydrate oxidation, and it varies with the FFA being oxidized. Although fat provides more kilocalories per gram than carbohydrate, fat oxidation requires more oxygen than carbohydrate oxidation. The energy yield from fat is 5.6 ATP molecules per oxygen molecule used compared to carbohydrate’s yield of 6.3 ATP molecules per oxygen molecule. Oxygen delivery is limited by the oxygen transport system, so carbohydrate is the preferred fuel during high-intensity exercise. Protein oxidation is more complex because amino acids contain nitrogen, which cannot be oxidized. Protein contributes relatively little to energy production. Protein or fat can be converted to glucose via gluconeogenesis, and protein can be converted to fat via lipogenesis. Your muscles’ oxidative capacity depends on their oxidative enzyme levels, their fiber-type composition, and oxygen availability. The following section provides basic information regarding the description, function, and storage of CHO, PRO, and FAT in the human body.

### 1.9.2.1. Carbohydrates.
These hydrated carbon molecules (CH₂O), known as carbohydrates (CHO), are a major component of an individual’s daily dietary intake (DDI). The most significant, biologically, of these sugars is glucose (Figure 1.9.2.1-1) because of its central role in CHO metabolism. In fact, other CHO’s are converted to glucose before being processed through CHO metabolic pathways. Interestingly, counterintuitive to their primary role in energy metabolism, CHO’s are not the most abundantly stored energy source in the human body; approximately 0.6% (≈ 420 g) and 1% (≈ 1,700 kcal) are stored relative to total body weight and total calories as depicted in Figure 1.9.2.1-2. In addition to serving as a primary energy source, these bio-molecules are significant structures in the body as glycoproteins, glycopeptides, and glycolipids, creating the fundamental building blocks in cell membrane collagen, nerve cell myelin, hormones, and hormone receptors.

![Glucose Structure](image)

**Figure 1.9.2.1-1. Glucose Structure**
These sugars are commonly categorized into monosaccharides, disaccharides, and polysaccharides. Mono- (e.g., glucose, fructose, and galactose) and disaccharides (e.g., sucrose, maltose, and lactose) are the smallest of the sugar molecules, being a single- and double-chained sugar molecule, and are known as simple sugars. Polysaccharides are more than two monosaccharides chained together and are referred to as complex carbohydrates. Of primary interest with regard to human nutrition is glycogen, the polysaccharide that stores chained glucose molecules.
together in the liver and muscle tissue. Within the body, approximately ¼ of the glycogen stores are in the liver, while the remaining ¾ is reserved for muscle tissue. The storage of liver glycogen equates to ≈ 75 to 100 g (≈ 300 to 400 kcal) of stored reserved to maintain blood glucose levels, while muscle glycogen can range from ≈ 300 to 400 g (≈ 1,200 to 1,600 kcal). The physiological significance of these respective glycogen stores focuses on the energy supply for the central nervous system (CNS), which is the brain and spinal cord, and the muscular system. Consider that the brain utilizes circulating blood glucose as its primary fuel source to produce energy to function. As shown in Figure 1.9.2.1-3, 75% of circulating blood glucose is derived from glycogen breakdown (i.e., glycogenolysis) to supply the brain energy for its metabolic function, which accounts for the majority of blood glucose utilization, 45%, thus the significance of maintaining blood glucose levels for optimal functionality.

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Figure 1.9.2.1-3. Quantitative Overview of Circulating Blood Glucose Turnover

Normal blood glucose ranges between 60 to 110 mg · dL⁻¹, with an average concentration of 80 mg · dL⁻¹; however, after a meal glucose levels in the blood can rise as high as 120 to 140 mg · dL⁻¹ but fall back within the normal range within 1 to 2 hours. Blood glucose is a highly regulated process, with the primary controllers being insulin and glucagon, wherein insulin is responsible for glucose uptake and glucagon releases glucose into the blood. The balance between uptake and release of glucose
into the circulating blood is to prevent two conditions from occurring: hyperglycemia and hypoglycemia. When blood glucose levels are allowed to climb higher than the normal range, the condition of hyperglycemia exists and can cause some serious pathological issues. If untreated, hyperglycemia can cause cellular dehydration, loss of glucose in the urine causing osmotic diuresis and the depletion of water and electrolytes, and tissue damage (e.g., vascular injury associated with uncontrolled diabetes mellitus increases the risk of heart attacks, strokes, renal disease, and glaucoma). The overall implications of hyperglycemia are an important aspect to health; the primary concern from an operational standpoint is the physiological effects of hypoglycemia on performance. With the low blood sugar of hypoglycemia, the brain succumbs to lack of fuel availability and reduced oxygen utilization, ultimately leading to neuroglycopenia (i.e., lack of glucose to fuel the brain) and autonomic neural stimulation. The manifestation of the condition is, of course, dependent upon the individual and his/her perception of the symptoms. The characterization of autonomic and neuroglycopenic symptoms can be grouped and characterized as in Table 1.9.2.1-1, but one should realize that there is not specific onset in the chronology of the symptoms; simply, an individual will consistently feel the same symptoms from episode to episode. The impact of hypoglycemia on performance and preventative recommendations will be discussed in further detail in the Nutrition and Performance area of this section.

Table 1.9.2.1-1. Classification of Hypoglycemic Signs and Symptoms Based upon Effector Groups

<table>
<thead>
<tr>
<th>Autonomic Group</th>
<th>Neuroglycopenic Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Feeling of warmth</td>
<td>Confusion or difficulty thinking</td>
</tr>
<tr>
<td>Nausea</td>
<td>Inability to concentrate</td>
</tr>
<tr>
<td>Pounding of the heart</td>
<td>Difficulty speaking</td>
</tr>
<tr>
<td>Sweating</td>
<td>Fatigue</td>
</tr>
<tr>
<td>Tingling</td>
<td>Headache</td>
</tr>
<tr>
<td>Trembling or shaking</td>
<td>Dizziness</td>
</tr>
<tr>
<td></td>
<td>Tiredness</td>
</tr>
<tr>
<td></td>
<td>Blurred vision</td>
</tr>
<tr>
<td></td>
<td>Drowsiness</td>
</tr>
<tr>
<td></td>
<td>Hunger</td>
</tr>
<tr>
<td></td>
<td>Weakness</td>
</tr>
</tbody>
</table>

1.9.2.2. Protein. The significant role proteins (PRO) play in our daily dietary nourishment is reflected in the genesis of the word itself, proteos—primary or taking first place. The primary functional roles of PRO are as enzymes as biocatalysts in metabolic chemical reactions (e.g., phosphofructokinase), peptide hormones in the regulation of growth and metabolism (e.g., insulin, somatotropin), structural proteins in the muscles and connective tissue (e.g., actin, myosin, collagen), transport proteins providing a method of movement for various substance (e.g., hemoglobin, albumin), and immunoproteins as antibodies (i.e., immunoglobins).

The human body is composed of approximately 14% PRO. Based upon the 70-kg body weight (BW) example (Figure 1.9.2.1-2), the total PRO weight is 10 kg; 6 kg is considered to be metabolically active. The turnover, i.e., production rate vs. degradation rate, of the metabolically active PRO is estimated to be 3 to 5 g · kg of BW$^{-1}$ · day$^{-1}$ (Note: ≈ 1 pound of PRO for a 70-kg individual) and can account for 20% of an
individual’s BMR. Generally, PRO composition in humans simply consists of the same 20 amino acids (AA): 10 essential (EAA) and 10 nonessential (NEAA) (Table 1.9.2.2-1). An example of an amino acid is shown in Figure 1.9.2.2-1.

![Figure 1.9.2.2-1. Arginine Structure](image)

The traditional definition distinguishing the differences between EAA, (i.e. “indispensable”) and NEAA (i.e. “dispensable”) can be defined as those amino acids that must be consumed exogenously and those that can be produced endogenously, respectively. The debate exists that amino acid categorization can be further broken down into nutrition, metabolic, and physiologic function when determining if they’re essential, nonessential, or conditionally essential. That argument, however, is beyond the scope of the section; thus, the primary focus remains on the protein requirements for an individual.

<table>
<thead>
<tr>
<th>Essential Amino Acids</th>
<th>Nonessential Amino Acids</th>
</tr>
</thead>
<tbody>
<tr>
<td>Histidine</td>
<td>Phenylalanine</td>
</tr>
<tr>
<td>Isoleucine</td>
<td>Threonine</td>
</tr>
<tr>
<td>Leucine</td>
<td>Tryptophan</td>
</tr>
<tr>
<td>Lysine</td>
<td>Valine</td>
</tr>
<tr>
<td>Methionine</td>
<td>Tyrosine</td>
</tr>
</tbody>
</table>

1.9.2.3. Lipids. Lipids, also known as FAT, exist in the human body in different forms: triglycerides, free fatty acids (FFA), phospholipids, and sterols. Contrary to popular belief, FATs are considered a vital aspect to dietary nourishment because of the diverse ways they are utilized physiologically: a primary energy source, as essential components of cell membranes and nerve fibers, support and protection of visceral organs, production of steroid hormones, mediator for fat-soluble vitamins, and thermoregulatory processes. FAT is composed of one glycerol molecule linked with three FFA, which most often will be either long chain, saturated (e.g., palmitic or stearic) or monounsaturated (e.g., oleic) fatty acids. FAT can be derived either endogenously or exogenously. In its most abundant form, triglycerides, FAT can be delivered to the body endogenously via hepatic and adipose lipogenesis or exogenously through dietary intake. Therefore, minimum requirements for dietary intake are not strict due to its relative social overabundance. However, 3% to 5% of FFAs cannot be synthesized endogenously and are known as essential free fatty acids (EFFA) because they must be consumed through daily intake; those are the polyunsaturated fatty acids lenoleic, linolenic (Figure 1.9.2.3-1), and arachidonic fatty acids.
Triglycerides constitute the majority of FAT in the body and are stored in adipose tissue, which is ≈ 20% of total body weight equating to ≈ 127,000 kcal of stored energy reserve in our ideal 70-kg individual (Figure 1.9.2.1-2), making FAT the human body’s most concentrated fuel reserve. The significance of FAT as a primary energy source is exemplified through the 70-kg individual’s DEE distribution. Of the 2,000 kcal required for total caloric DEE, the BMR relies on 70% of those calories being derived from FAT, in a rested state. In other words, in a seated, normal rested state your body utilizes FAT as the predominant fuel source. For this reason, FAT is considered an important aspect of nutritional intake to performance, and recommendations will be provided within the nutrition and performance section.

1.9.3. Nutrition and Performance

The first part of the preceding section introduced the idea of nutrition relative to an individual’s needs, the concept of energy metabolism with regard to DEEs and DDIs, as well as the primary and secondary functions of the human body’s energy sources. The intent of this section is to put into practical terms the applied aspect of nutrition with regard to the operational environment and the military personnel affected by mission demands. In doing so, recommendations for basic dietary daily intake for weight management and aspects of pre-mission/pre-competition meal planning for peak performance will be outlined. Although the section’s language seems to be focused on athletic performance, one must remember that physiological stress can occur within the cockpit of an aircraft traveling at 400 knots on a hot day just as well as on the athletic field. Members of the United States Armed Services—Air Force, Army, Navy, Marines, and Coast Guard—must be considered nothing less than occupational athletes who are required to be in the best possible health and fitness, physiological and psychological, for them to have their peak performance at any given notice when duty calls.

1.9.3.1. Macronutrients. Before recommendations can be made regarding nutritional DDI, the first consideration should be to understand the bioenergetics of the metabolic pathways utilized and the energy source required to perform the work at peak performance. Metabolically, the energy systems to perform muscular work focus on the phosphagen, glycolytic, and oxidative systems. Ultimately, each one is responsible for the production of the high-energy phosphate bond molecule ATP. Through metabolism, the human body takes the chemical of the macronutrients
CHO, PRO, and FAT to create the more efficient, biologically active form of energy in ATP, which allows for a variety of work production; i.e., energy output (Figure 1.9.1-1).

There is only a small amount of ATP stored within the human body skeletal muscles, ≈ 5 mmol · kg wet weight, which only allows for a few seconds of work before more ATP is required. If the work being performed is short term, high intensity (i.e., ≈ 3 to 15 seconds), then the phosphagen system utilizes phosphocreatine (PCr) to resynthesize ATP rapidly; however, if the activity is to continue at the same intensity level, then the most probable outcome will be fatigue because of the lack of PCr availability. Yet, if the workload intensity were to decrease so as avoid fatigue and increase time of work duration (i.e., ≈ 60 to 180 seconds), then energy metabolism would transition into the glycolytic pathway, where glucose or glycogen is the primary fuel source in the production of ATP with the byproduct of lactic acid. Oxidative metabolic pathways, CHO and FAT oxidation, take primary control of energy production for activities longer than 3 minutes by oxidizing substrates such as glucose, muscle and liver glycogen, triglycerides (i.e., intramuscular, blood, and adipose tissue), along with negligible amounts of amino acids. Examples of activities for workloads that are considered short term, high intensity to long term, moderate intensity might range from performing an anti-G straining maneuver (i.e., AGSM) in a single bout (≈ 3 seconds), performing an F-16 qualification run at 9 G for 15 seconds, to flying offensive air combat maneuvers for a 1-hour sortie, respectively. The factors that determine the primary metabolic pathway of utilization are intensity, frequency, duration, volume, mode (or type of activity), sex, and current physical fitness status, as well as DDI and status of physiological energy reserves.

Optimal performance is based upon the balance of energy requirements, which is the balance between DDI versus DEE and the maintenance of optimal total body weight to include physiological energy reserves. If the balance is towards the negative, either due to insufficient DDI or an excessive DEE, these insufficiencies can lead to detrimental weight loss via fat or lean muscle mass or the depletion of vital glycogen stores. The losses of weight and energy reserves can be seen in poor performance in activities requiring strength and/or endurance and compromised immune, endocrine, or musculoskeletal functions. Avoidance of this energy requirement imbalance can be mitigated through the careful calculation of DEE components (Figure 1.9.2.1-2) utilizing RMR calculations (Table 1.9.1-2) and physical activity energy expenditure estimates (Table 1.9.1-3 or Table 1.9.1-4) plus the complimentary DDI based upon recommendations from the American College of Sports Medicine’s joint position stand paper written by the American Dietetic Association and Dieticians of Canada, as well as the Dietary Guidelines for Americans 2005 published by the U.S. Department of Agriculture (USDA) and Department of Health and Human Services (HHS).

1.9.3.2. Carbohydrates (CHO). The recommendation for CHO intake on a percentage range such as the one provided in the Dietary Guidelines for Americans 2005. The acceptable macronutrient distribution range (AMDR) for CHO is from 45% to 65% of DDI. Even though high CHO diets have been advocated in the past for athletes, the following examples show the pitfalls of that methodology. For example, when DDI is 4,000 to 5,000 kcal X d⁻¹, even a diet containing 50% of the energy from CHO will provide 500 to 600 g of CHO (or approximately 7 to 8 g X kg⁻¹ (3.2 to 3.6 g X lb⁻¹) for a 70-kg (154-lb) individual), an amount sufficient to maintain muscle glycogen stores from day to day. Conversely, when DDI is less than 2,000 kcal
X d\(^{-1}\), a diet providing 60% of the energy from CHO may not be sufficient to maintain optimal CHO reserves (4 to 5 g X kg\(^{-1}\) or 1.8 to 2.3 g X lb\(^{-1}\)) in a 60-kg (132-lb) individual. Therefore, a more appropriate range is from 6 to 10 g X kg\(^{-1}\) body weight X d\(^{-1}\) (2.7 to 4.5 g X lb\(^{-1}\) body weight X d\(^{-1}\)).

1.9.3.3. Protein (PRO). The same issue with CHO recommendation exists with PRO; for example, if PRO intake was 10% of DDI, total PRO ingestion (100 to 125 g X d\(^{-1}\)) could be greater than the recommended PRO intake for an individual. Currently, the PRO recommendation from the recommended daily allowance (RDA) is 0.8 g X kg\(^{-1}\) body weight and the USDA’s AMDR is 10% to 35% of DDI. Albeit, the aforementioned PRO recommendation might be appropriate for a healthy adult ≥ 18 years old, it may fall short for individuals under extreme endurance or muscular strength workload demands. For those endurance individuals, an applicable range consists of 1.2 to 1.4 g X kg\(^{-1}\) X d\(^{-1}\), while an individual needing high muscular strength might require more along the lines of 1.2 to 1.7 g X kg\(^{-1}\) X d\(^{-1}\).

1.9.3.4. Lipids (FAT). As stated earlier in the section, FAT is an abundant macronutrient, and recommendations between agencies and professional organizations do not vary significantly. The DDI for FAT can be between 20% to 35%, with ≈ 10% derived from polyunsaturated, monounsaturated, and saturated FATS each.

1.9.3.5. Water. Water, i.e., H\(_2\)O, is not considered a macronutrient; however, as the most prevalent substance in the human body (Figure 1.6.17-2), with ≈ 60% of the ideal 70-kg individual’s total body weight being of water; it must be considered as being significant to biological function. A reflection of water’s significance to physiological functions: it has been estimated that an individual can survive a loss of up to 40% of total body weight in CHO, PRO, and FAT, but a loss of 9% to 12% in total body weight in water can cause death.

During physical performance of activity, the human body will tend to lose water via sweat for a variety of reasons due to environmental conditions (i.e., ambient temperature, humidity, and wind), metabolic rate, elevated exertion levels, and clothing. As core temperature increases, the body will invoke its primary cooling effect and increase sweat rates in an attempt to cool itself. Thus, total body water losses increase, and concomitant electrolyte imbalances are induced, producing potentially poor performance and health issues (e.g., heat exhaustion, heat stroke, muscle cramps, rhabdomyolysis, acute renal failure, exercise-induced hyponatremia).

Prevention is the key; for an individual to prevent dehydration, one must know what euhydration is relative to the body. Hydration assessments consist of establishing a first-morning average body weight baseline value, then monitoring urine and body weight movements to calculate sweat losses and H\(_2\)O replacement needs or employing a urine specific gravity (USG) test to determine euhydration. The next best step in preventing or minimizing the further progression of a dehydrated state is to recognize the signs and symptoms of dehydration listed below as adapted from the American College of Sports Medicine’s Position Stand on Exercise and Fluid Replacement:
• Dehydration increases physiologic strain and perceived effort to perform the same exercise task and is accentuated in warm-hot weather.

• Dehydration can increase the likelihood or severity of acute renal failure consequent to exertional rhabdomyolysis.

• Dehydration and sodium deficits are associated with skeletal muscle cramps.

• Symptomatic exercise-associated hyponatremia can occur in endurance events.

• Fluid consumption that exceeds sweating rate is the primary factor leading to exercise-associated hyponatremia.

• Large sweat sodium losses and small body weight (and total body water) can contribute to exercise-associated hyponatremia.

• The greater the dehydration level, the greater the physiologic strain and aerobic exercise performance decrement.

• Critical water deficit and magnitude of exercise performance degradation are related to the heat stress, exercise task, and the individual’s unique biological characteristics.

• Dehydration (> 2% BW) can degrade aerobic exercise performance, especially in warm-hot weather.

• Dehydration (> 2% BW) might degrade mental/cognitive performance.

• Dehydration (3% BW) has marginal influence on degrading aerobic exercise performance when cold stress is present.

• Dehydration (3% to 5% BW) does not degrade either anaerobic performance or muscular strength.

• Hyperhydration can be achieved by several strategies but has equivocal benefits and several disadvantages.

• Dehydration is a risk factor for both heat exhaustion and exertional heat stroke.

Finally, the knowledge of modifying factors that affect how an individual responds to a dehydrated status might be the difference between prevention and a mishap. The modifying factors of dehydration can be grouped into three categories: age, sex, and nutrition. The considerations with age focus upon older adults and children; older adults are prone to age-related, decreased thirst sensitivity when dehydrated and slower renal responses to water, predisposing them to slower voluntary reestablishment of euhydration and increased risk of hyponatremia, respectively. While on the other end of the spectrum, prepubescent children have lower sweat rates than adults, in turn making thermoregulation more difficult in environmental conditions in
which adults can acclimatize. Generally, women have lower sweat rates than men and are at greater risk for developing symptomatic hyponatremia, but the etiology is not related to renal water and electrolyte retention. Every effort should be made to promote euhydration before dehydration occurs, and one of the better ways to do so is through meal consumption; eating not only promotes water intake but also sodium and potassium electrolyte replenishment. The diuretic effects of caffeine and alcohol are a concern, but only in so much as how much and when they are consumed. Caffeine consumption below < 180 mg · dL$^{-1}$ is not likely to increase daily urine output beyond the normal daily excretion or contribute to dehydration, unlike alcohol, which is a strong diuretic and should be only consumed during the post-activity meal.

1.9.4. **Meal Planning**

As the final aspect of nutritional recommendations, the topic of meal planning is of significance. The reasons for meal planning are quite simple: understand the basic concept of whole body metabolism and bioenergetics in the human body, identify and explain the function of the three main fuels source we use to create energy, make certain DDI and DEE are matched for optimal weight management, and reensure that fuel energy reserves are properly stored with complete fuel reserves to avoid the deleterious effects of hypoglycemia and dehydration on physical and cognitive performances. Regardless of whether or not the military warfighter is on the ground, in the air, or on the sea, the practical application of nutrition and performance affect everyone. From the pilots of tankers to fighters to reconnaissance; to the flight surgeons, nurses and technicians of the critical care aeromedical transport (CCAT) teams; to the high altitude parachutists, combat rescue officers, and special operation personnel; they all must have nutritional awareness. An awareness of one’s nutrition entails the fundamental building blocks of meal planning for mission readiness and preparedness and consists of the following core questions; *what*, *when*, *where*, *why*, and *how*.

1.9.4.1. **Pre-Activity Meal Planning.** An individual should approach pre-activity planning with a thoughtful focus on those core questions from an anticipatory perspective. Before activity, a meal or snack should provide sufficient fluid to maintain hydration, be relatively low in fat and fiber to facilitate gastric emptying and minimize gastrointestinal distress, be relatively high in carbohydrate to maximize maintenance of blood glucose, be moderate in protein, be composed of familiar foods, and be well tolerated by the individual. The size and timing of the pre-activity meal have an important relationship because most individuals don’t like to perform work on a full stomach; thus, a smaller meal would be consumed near the event to allow for gastric emptying, whereas larger meals can be consumed when more time is available. Certain amounts of CHO supplementation, ranging from ≈ 200 to 300 g of CHO for meals consumed 3 – 4 hours before the activity/work to be performed, have been shown to enhance performance. Although the above guidelines are effective, individual needs must be emphasized; individuals can know what works best for them by experimenting with new foods and drinks prior to and during practice events and planning ahead to ensure they will have access to these foods at the appropriate time. As far as pre-activity hydration, the individual should drink approximately 5 to 7 mL X kg$^{-1}$ body weight ($\approx 2$ to 3 mL X lb$^{-1}$) of water or a sport beverage at least 4 hours before exercise, allowing enough time to optimize hydration status and for excretion of
any excess fluid as urine. If the individual doesn’t produce urine, or the urine is dark or highly concentrated, he/she should slowly consume more fluid (e.g., \( \approx 3 \) to \( 5 \) mL \( X \) kg\(^{-1}\)) about 2 hours before the event.

1.9.4.2. **During-Activity Meal Planning.** During exercise, the primary goals for nutrient consumption are to replace fluid losses and provide CHO, \( \approx 30 \) to \( 60 \) g \( X \) h\(^{-1}\) for maintenance of blood glucose levels. These nutrition guidelines are especially important for endurance events lasting longer than an hour when the individual has not consumed adequate DDI before the activity, especially when considering the activity is taking place in an extreme environment (i.e., heat, cold, or high altitude). CHO intake should begin shortly after the onset of activity; consuming a given amount of CHO after 2 hours of exercise is not as effective as consuming the same amount in 15- to 20-minute intervals throughout the entire duration. The purpose for drinking during exercise is to avoid a \( H_2O \) debt >2% of body weight; thus, for optimal fluid replacement the individual needs to consider sweat rate, the duration, and opportunities to drink. The type, intensity, and duration of exercise and environmental conditions will alter the need for fluids and electrolytes. Additionally, drinks containing electrolytes, such as sodium and potassium, help to replace sweat electrolyte losses, whereas sodium stimulates thirst and fluid retention and CHO provides energy. The optimal fluid replacement drink should contain 6% to 8% CHO for activities lasting longer than 1 hr.

1.9.4.3. **Post-Activity Meal Planning.** After exercise, the nutritional goal should be to provide fluids, electrolytes, and CHOs to replace muscle glycogen, ensuring optimal recovery. The when (i.e., timing) and what (i.e., composition) of the post-activity food intake is dependent upon the intensity and duration of the workload performed (i.e., did glycogen depletion occur) and on when the next intense work will occur. For optimal glycogen resynthesis, the post-activity CHO ingestion should be \( \approx 1.0 \) to \( 1.5 \) g \( X \) kg\(^{-1}\) body weight (0.5 to 0.7 g \( X \) · lb\(^{-1}\)) during the first 30 minutes and again every 2 hours for 4 to 6 hours. PRO can be provided after exertional work to provide amino acids for repair of muscle tissue. Also, the type of carbohydrate consumed affects post-activity glycogen resynthesis; glucose and sucrose are both effective, and CHO intake of high glycemic index provides higher muscle glycogen levels 24 hours post-activity. Because most individuals don’t consume enough fluids during physically demanding activity to balance fluid losses, the session is completed with them in a dehydrated state. If adequate time is taken, intake of normal DDI will restore hydration status by replacing fluids and electrolytes lost during the activity. However, a more rapid and complete recovery from excessive dehydration can be accomplished by consuming \( \approx 16 \) to \( 24 \) oz of fluid · lb\(^{-1}\) of body weight lost during the activity.

1.9.5. **Special Considerations**

- Current Physiological Status
- Environmental Conditions
  - Hot and humid environments
  - Cold environments
  - Altitude
- Mission Requirements
References

Recommended Readings

Concepts
Energy metabolism

Vocabulary
- Basal metabolic rate (BMR)
- Blood glucose
- Dehydration
- Energy expenditure
- Macronutrients
- Physical activity
- Resting metabolic rate (RMR)
- Water
2. ATMOSPHERE

James T. Webb, Ph.D.

Variation in earth-bound environmental conditions places limits and requirements on our activities. Even at sea level, atmospheric environmental conditions vary considerably due to latitude, climate, and weather. Throughout the range of Air Force operations, crewmembers and their craft face even larger variations in atmospheric properties that require life support systems and personal equipment for survival and preservation of optimal function. Understanding the physical nature of our atmosphere is crucial to understanding how it interacts with human physiology and what protective measures must be employed.

The evolution of humans on the surface of the earth did not involve selection of traits for survival and optimal performance above the altitudes where most humans and their predecessors resided. Most humans lived at or near sea level throughout history. Our species typically lacks significant permanent adaptations to high altitude. Therefore, we exhibit significant and progressively reduced performance when exposed to altitudes above 10,000 ft. The effects covered here are acute. They occur over relatively short periods of exposure and are pertinent to the altitude exposures typical during Air Force missions. Other deleterious effects of altitude are slower to develop (e.g., high altitude pulmonary edema, acute mountain sickness). However, these usually will not have time to develop in a typical Air Force flying mission.

2.1. Constituents and Properties of the Atmosphere

In 1805 John Dalton provided the first analysis of the constituents of the atmosphere by percentage of each gas (Dalton, 1805).

2.1.1. Constituents of the Atmosphere

Discussion of the atmosphere as a whole required development of a definition of a standard atmosphere. The standard atmospheric pressure at sea level is accepted to be 760 mmHg, which is equivalent to 14.7 psi and 29.92 inHg at 15°C (59°F).

Constituents of the atmosphere we breathe are shown in Table 2.1.1-1 and these percentages are consistent throughout the atmosphere of interest to aerospace physiology. However, as altitude increases, the total pressure decreases yielding a decrease in partial pressure of each of the constituents of the atmosphere.

2.1.2. Atmospheric Zones

Two strategies for dividing the atmosphere into zones are presented here. The first and most generally used is based on physical characteristics of the atmosphere itself. The second is more useful to the aerospace physiologist and is based on the physiological needs of humans.
Table 2.1.2-1. Constituents of Earth’s Atmosphere

<table>
<thead>
<tr>
<th>Gas</th>
<th>Atmospheric Content (%)</th>
<th>Partial Pressure (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nitrogen</td>
<td>78.084</td>
<td>593.44</td>
</tr>
<tr>
<td>Oxygen</td>
<td>20.948</td>
<td>159.20</td>
</tr>
<tr>
<td>Argon</td>
<td>0.934</td>
<td>7.10</td>
</tr>
<tr>
<td>Carbon dioxide</td>
<td>0.031</td>
<td>0.24</td>
</tr>
<tr>
<td>Other gases</td>
<td>0.003</td>
<td>0.02</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>100.000</strong></td>
<td><strong>760.00</strong></td>
</tr>
</tbody>
</table>

*aClean, dry air at 15°C (59°F), sea level; mean of values every 15° between 15° N and 75° N (U.S. Standard Atmosphere, 1962)*

Temperature and its lapse rate, direction, and rate of change with respect to altitude, provide much of the basis for subdivisions of Earth’s atmosphere into regions as defined in Figure 2.1.2-1.

![Image of temperature and pressure changes in Earth's atmosphere](image)

**Figure 2.1.2-1. Zones of Earth’s Atmosphere (National Weather Service)**

The lowest zone, the troposphere, is the only region of Earth’s atmosphere capable of supporting human habitation without artificial support. Approximately 80% of the mass of the atmosphere and most of the weather phenomena occur in the troposphere.

The troposphere starts at the Earth’s surface and extends to the tropopause, between 26,000 and 48,000 ft (5 – 9 mi, 8 – 14.5 km) higher at the equator and lower at the poles. At its higher levels, above 20,000 ft (3.8 mi, 6 km), at least some degree of artificial support is required in the form of supplemental oxygen.

A linear decrease in temperature characterizes the troposphere from sea level (15°C) to the tropopause, typically at about 35,000 ft (6.6 mi, 10.7 km), where the temperature is normally about -55°C. Starting around 36,000 ft, the temperature of the...
atmosphere plateaus at \(-56.6^\circ C\) throughout the lower portion of the stratosphere (Figure 2.1.2-1; Appendix 1a). The environmental lapse rate through the troposphere is \(-2^\circ C\), or about \(-3.5^\circ F\), per 1,000 ft. The approximate change in temperature with increasing altitude can be predicted by:

\[
\text{Temp at Altitude} = \text{\(C_{\text{starting}}\)} \quad \text{\(- \left( \Delta \text{altitude} \times \frac{2^\circ C}{1,000 \text{ ft}} \right) \)}
\]

Variations in temperature, pressure, and humidity in the troposphere account for extreme differences in the environmental conditions we experience as weather.

The tropopause is the division between the troposphere and stratosphere. Aircraft jet engines perform with greater efficiency at lower temperatures, which is one reason cruise is planned near the tropopause where the temperature is lowest. The stratosphere starts just above the tropopause and extends up to 164,000 ft (31 mi, 50 km). Ninety-nine percent of the mass of the air is located in the troposphere and stratosphere. The temperature throughout the lower part of the stratosphere is relatively constant (Figure 2.1.2-1). Compared to the troposphere, this part of the atmosphere is dry and less dense. The temperature in this region increases gradually to \(-3^\circ C\), as the absorption of ultraviolet radiation creates a positive lapse rate.

Ultraviolet radiation reaching the lower stratosphere from the sun is responsible for the creation of ozone, the ozone layer, or ozonosphere. In the process of ozone production and in reactions with ozone, nearly all of the ultraviolet (UV) radiation is absorbed, including the most hazardous form to life forms, UV-C. Much of UV-B is also absorbed, although the UV-B reaching the surface is sufficient to be a major cause of sunburn and melanoma cancers. Most of the UV-A reaches the Earth’s surface. UV-A converts a precursor to vitamin D in human skin. Although flight in the upper troposphere and lower stratosphere involves exposure to more radiation than on the surface, no health risk is currently associated with routine flying operations. Flight above the stratosphere and space flight involve risk of exposure to significant levels of radiation.

The higher regions of the atmosphere, 50,000 ft and above, are so thin that pressure suits are required to sustain life. Temperature variations result from variable absorption of the sun’s energy in several forms; hence, thermal protection must be incorporated for any exposure in these regions. In the higher regions, flight of “air-breathing” aircraft (those that use atmospheric oxygen to burn fuel) becomes impossible and control surfaces are no longer effective.

Another typical subdivision of the atmosphere described above relates to the ability of humans to function based on the partial pressure of oxygen available and the need for artificial pressure to sustain life. These divisions are referred to as the physiological divisions of the atmosphere and are described in Table 2.1.2-2 below.
### Table 2.1.2-2. Physiological Divisions of the Atmosphere

<table>
<thead>
<tr>
<th>Physiological Division</th>
<th>Altitude and Pressure Range</th>
<th>Problems</th>
<th>Solutions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physiological Zone</td>
<td>0 - 10,000 ft 0 - 3,048 m 760 - 523 mmHg</td>
<td>Trapped gas expansion/contraction during changes in pressure results in middle ear or sinus blocks; shortness of breath, dizziness, headache, or nausea in unacclimatized individuals or with exercise</td>
<td>Reductions in performance or longer term acclimatization</td>
</tr>
<tr>
<td>Physiologically Deficient Zone</td>
<td>10,000 - 50,000 ft 3,048 - 15,240 m 523 - 87 mmHg</td>
<td>Oxygen deficiency progresses from minor reductions in cognitive and physical capabilities at 10,000 ft to death over about 25,000 ft (possibly lower) without supplemental oxygen</td>
<td>Supplemental oxygen allows good performance to about 35,000 ft with progressively less capability</td>
</tr>
<tr>
<td>Space Equivalent Zone</td>
<td>Above 50,000 ft &gt; 15,240 m &lt; 87 mmHg</td>
<td>Survival requires assisted PBA* or, above about 63,000 ft, a full pressure suit and delivery of 100% O&lt;sub&gt;2&lt;/sub&gt; to supply at least 140 mmHg O&lt;sub&gt;2&lt;/sub&gt;</td>
<td>Pressurized cabin or pressure suit with 100% O&lt;sub&gt;2&lt;/sub&gt;</td>
</tr>
</tbody>
</table>

*PBA = positive pressure breathing for altitude.

The negative physiological effects of increasing altitude and the need for increasing countermeasures can easily be understood when related back to the transport of O<sub>2</sub>. Figure 2.1.2-2 shows the relationship between the oxygen-hemoglobin dissociation curve (red line), the drop in atmospheric pressure (green line), and the physiological divisions of the atmosphere.

![Figure 2.1.2-2. Physiological Divisions of the Atmosphere](image-url)
2.1.3. Altitude

Altitude is measured in many different ways using different standards for different purposes (Table 2.1.3-1.).

<table>
<thead>
<tr>
<th>Type of Altitude</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>True Altitude</td>
<td>As measured with a tape measure from sea level to the aircraft</td>
</tr>
<tr>
<td>Mean Sea Level (MSL)</td>
<td>Average height of the surface of the sea</td>
</tr>
<tr>
<td>Pressure Altitude (PA)</td>
<td>Height read on an altimeter set to 29.92 inHg</td>
</tr>
<tr>
<td>Indicated Altitude</td>
<td>As read from a properly set altimeter</td>
</tr>
<tr>
<td>Above Ground Level</td>
<td>Altitude determined by subtracting the elevation of the ground below the</td>
</tr>
<tr>
<td>(AGL)</td>
<td>aircraft from the true altitude of the aircraft</td>
</tr>
</tbody>
</table>

True altitude is the standard to which other, more practical altitudes are compared. On low-altitude maps provided to USAF pilots, the height of physical features of the Earth, like mountains and airfields, is measured in feet above mean sea level (MSL). MSL is the average height of the surface of the sea for all stages of the tide over a 19-year period, usually determined from hourly height readings. With properly set, calibrated, and functioning altimeters, feet above MSL is the altitude viewed by the pilot on the aircraft altimeter when flying below 18,000 ft. Since altimeter settings vary with atmospheric pressure variations, the MSL altitude will only be correct if the correct pressure is set in the Kollsman window of the altimeter (Figure 2.1.3-1).

![Figure 2.1.3-1. Altimeter](image)

Pressure altitude (PA) is the height in the atmosphere at which a given value of standard pressure exists. With 29.92 inHg set in the Kollsman window of the altimeter (Figure 2.1.3-1), pressure altitude is displayed in feet on the altimeters of USAF aircraft at or above 18,000 ft. This is the standard setting when aircraft are at or above 18,000 ft, where altitudes are referred to as flight levels, in hundreds of feet (FL, altitude in feet divided by 100). For example, 20,000 ft is flight level 200 (FL200). Indicated altitude is
the altitude read on an altimeter and will be MSL below 18,000 ft and, typically, pressure altitude above 18,000 ft with 29.92 set in the Kollsman window.

Pilots are naturally quite interested in the height of their aircraft above the ground. Above ground level (AGL) altitude is the altitude determined by subtracting the elevation, in feet, of the ground below the aircraft from the true altitude of the aircraft. This is the distance between the pilot and the ground (or an obstacle).

Since some controllers in the U.S. leave off the “2” of 29.92, confusion can arise when U.S. pilots “assume” that, for example, 998 means 29.98 inHg when given by an air traffic controller overseas. That overseas controller may mean 998 millibar (mb), which, if misinterpreted, could mean a difference of about 400 ft.

Although inHg is the standard for altimeter settings and is a pressure indication, it is not normally used in aviation for describing total atmospheric pressure at a given altitude. Elevation is typically measured in feet and pressure in psia or mmHg. In parts of the world, other measures are standard and listed in Appendices 1 and 2.

2.1.4. Air Density, Pressure, and Temperature

Atmospheric pressure decreases exponentially with increasing altitude, reaching 50% of sea level density and pressure at approximately 18,000 ft (5.49 km). This relationship is affected in any specific locale by deviations from standard temperature and pressure. Figure 2.1.4-1 graphically shows how atmospheric pressure is affected by altitude. The curve depicts how each 10,000-ft increase in altitude results in less change in pressure: 0 – 10,000 ft changing by 237 mmHg, 10,000 – 20,000 ft changing by 173 mmHg, and 40,000 – 50,000 ft changing by only 54 mmHg. This relationship explains why pressure effects from trapped gases are more noticeable in the lower altitude ranges. See Section 2.2 (The Gas Laws) for implications of this pressure change.

![Atmospheric Pressure vs. Altitude](image-url)
Flight altimeters are still based on air pressure, although modern methods based on radar and on the Global Positioning System are coming into use. During takeoff, landing, and low-level phases of flight, aircraft altimeters are routinely set to the field altimeter setting (i.e., the local barometric pressure at the airfield). Using this setting accounts for variations in local atmospheric pressure, which varies depending on meteorological conditions. Otherwise, the altimeter reading may vary significantly from true altitude at one of the most crucial phases of flight: flying close to the ground.

Temperature variations from the standard temperature of 15°C also produce errors that affect terrain clearance. For instance, an aircraft flying at 5,000 ft in -40°C (e.g., Alaska in the winter) would be more than 1,200 ft lower than the indicated altitude after correction for local barometric pressure. Local barometric pressure in the United States is based on inches of Hg (inHg). Setting the current local barometric pressure in the Kollsman window on the altimeter will result in a reading of 0 ft at sea level. As the local pressure varies, altimeters must be adjusted up or down to yield the correct field elevation on an aircraft altimeter at a designated point on that airfield. Above 18,000 ft, altimeters are routinely set to 29.92 inHg to provide adequate and standardized clearance for aircraft altitude separation.

2.1.5. Light and Sound

Diffusion of light in the lower atmosphere accounts for the blue color of the sky as viewed from Earth’s surface, a phenomenon which significantly dissipates as low as about 50,000 ft where the blackness of space begins to become apparent.

The speed of sound is 761 mph (340 m/s; 1,116 ft/s) at sea level. It travels slower, 660 mph (295 m/s) at 50,000 ft, where the temperature is about 75°C lower. The speed of sound is a function of the square root of the temperature in °K (°C + 273). The speed of sound at a given temperature can be estimated by the following equation:

\[ C_{air} = 331.3 \frac{m}{s} \sqrt{1 + \frac{T}{273.15}} \]

where:
- \( C_{air} \) = speed of sound in air (in meters per second)
- \( T \) = temperature in °C

References
Dalton J. Experimental Enquiry into the Proportion of the Several Gases or Elastic Fluids, Constituting the Atmosphere. Memoirs of the Literary and Philosophical Society of Manchester 1805; 1:244-58.
Internet Resources; see Appendix 8.

Concepts
- Atmospheric zones
- Constituents of Earth’s atmosphere
- Physiological zone
- Physiologically deficient zone
- Standard atmospheric pressure
Vocabulary
   Above ground level (AGL)
   Indicated altitude
   Lapse rate
   Mean sea level (MSL)
   Pressure altitude (PA)
   Stratosphere
   Tropopause
   Troposphere
2.2. The Gas Laws

James T. Webb, Ph.D.
Lt Col Ryan W. Maresh, USAF, BSC, Ph.D.

1662 Boyle published the finding that at a constant temperature, the volume of gas is inversely proportional to its pressure (Boyle, 1662).

1787 Jacques Alexandre César Charles discovered the relationship between the volume of gas and its temperature (Gay-Lussac, 1802).

1802 Joseph Louis Gay-Lussac published the Charles findings that the volume of fixed mass of gas is directly proportional to its absolute temperature (Gay-Lussac, 1802).

1803 William Henry published his findings that the amount of a gas in a solution varies directly with the partial pressure of that gas over the solution (Davis, 2008).

1805 John Dalton presented his observations that the total pressure of a mixture of gases is equal to the sum of the partial pressures of each gas in the mixture (Dalton, 1805).

1833 Thomas Graham described the diffusion of a gas as being inversely proportional to the square root of its molecular weight (Graham, 1833). In 1834 he received the Keith Prize from the Royal Society of Edinburgh for this work.

The Gas Laws describe physical properties of all gases, including our atmosphere, and provide a basis for understanding how exposure to reduced atmospheric pressure affects our function.

2.2.1. Pressure-Volume Law (Boyle’s Law)

Robert Boyle (1627 – 1691) was an Anglo-Irish scientist noted for his work in physics and chemistry. In 1662, Boyle published the finding which states that at a constant temperature, the volume of a dry gas is inversely proportional to its pressure. P₁ and V₁ are the initial pressure and volume, and P₂ and V₂ are the final pressure and volume.

\[ P_1 \cdot V_1 = P_2 \cdot V_2 \text{ or } \frac{P_1}{P_2} = \frac{V_2}{V_1} \]

Solving this equation for the volume of a gas in a distensible container, such as a balloon, quantitatively describes trapped gas expansion with reduced pressure. Solving this equation to find the volume of a liter of dry gas expansion with reduced pressure. Solving this equation to find the volume of a liter of dry gas expansion with reduced pressure. Solving this equation to find the volume of a liter of dry gas taken from sea level to 20,000 ft and 40,000 ft, assuming unrestricted expansion, would result in the increase in volume shown in Figure 2.2.1-1.

The problem becomes more complicated by the inclusion of water vapor in the lungs and other spaces in the body as described in Section 3.3 on Trapped Gas. Briefly, the constant water vapor pressure of 47 mmHg must be subtracted. Thus, for wet gases, Boyle’s Law becomes:

\[ (P_1 - 47) \cdot V_1 = (P_2 - 47) \cdot V_2 \]
2.2.2. **Volume-Temperature Law (Charles’ Law)**

Jacques Alexandre César Charles (1746 – 1823) was a French inventor, scientist, mathematician, and balloonist. In 1783, he made the first balloon using hydrogen gas. Upon release it ascended to a height of nearly 3 km (2 mi). In 1787, he discovered the relationship between the volume of gas and temperature. Charles did not publish his findings, and Joseph Louis Gay-Lussac first published the findings in 1802, referencing Charles’ work. Therefore, it is known variously as Gay-Lussac’s Law or Charles’s Law or Charles and Gay-Lussac’s Law. However, for accuracy, it should be referred to as Charles’s Law, and can be expressed as:

\[
\frac{V_1}{V_2} = \frac{T_1}{T_2} \text{ or } \frac{V_1}{V_2} = \frac{T_1}{T_2}
\]

The temperature must be expressed in Kelvin degrees, where °K = °C + 273. Absolute zero, or -273°C, is 0°K.

Although Charles’s volume-temperature law is very important from an engineering and chemistry standpoint, the temperature of the human body varies little, limiting the law’s usefulness in physiology.

2.2.3. **Pressure-Temperature Law (Gay-Lussac’s Law)**

Joseph Louis Gay-Lussac is credited with describing the relationship between the pressure of a fixed mass of gas and its absolute temperature, when volume is held constant. This relationship can be expressed as:

\[
\frac{P_1}{P_2} = \frac{T_1}{T_2} \text{ or } \frac{P_1}{P_2} = \frac{T_1}{T_2}
\]

Like Charles’s Law, Gay-Lussac’s Law is important from an engineering and chemistry standpoint, but as the temperature of the human body varies little, it has limited usefulness in physiology. It is of importance when considering the variation in pressure of compressed gases, such as oxygen tanks, when the temperature varies.
2.2.4. Ideal Gas Law

Note that Boyle’s pressure-volume law describes changes in volume with respect to pressure when temperature is held constant. Charles’s volume-temperature law describes how volume changes with temperature when pressure is held constant. Gay-Lussac’s pressure-temperature law describes how pressure changes with temperature when volume is held constant. Changes in all three parameters (volume, pressure, and temperature) are better described by the Ideal Gas Law, which includes the three parameters in one equation with other factors to improve accuracy. The Ideal Gas Law includes the factors of volume, pressure, and temperature in a single equation, which explains the effects described by Boyle, Charles, and Gay-Lussac. Two added factors, \( n \) and \( R \), are constants if the total quantity of gas is constant. It is expressed as:

\[
P V = n R T
\]

where:
- \( P \) = pressure (kPa)
- \( V \) = volume (\( m^3 \))
- \( T \) = temperature (in Kelvin)
- \( n \) = number of moles
- \( R \) = universal gas constant = 8.3145 J/mol·K (or 0.08206 liters·atm/mole·K when \( P \) is in atm and \( V \) is in liters)

If the number of moles (quantity, number of molecules, of gas) is held constant, the effect of varying one factor can be seen on the other potential variables.

\[
P = \frac{T}{V} \quad \text{or} \quad V = \frac{T}{P} \quad \text{or} \quad T = PV
\]

In this case, it can also be written so as to compare initial and final states:

\[
\frac{P_1 V_1}{T_1} = \frac{P_2 V_2}{T_2}
\]

This arrangement of the Ideal Gas Law is often referred to as the Combined, or Universal, Gas Law.

2.2.5. Law of Partial Pressures (Dalton’s Law)

John Dalton (1766 – 1844) was an English chemist and physicist. In 1805, he described the total pressure \( (P_T) \) of a mixture of gases as equal to the sum of the partial pressures of each gas in the mixture.

\[
P_T = P_1 + P_2 + P_3 + \ldots + P_n
\]

Since the standard atmosphere at sea level is 760 mmHg, Dalton’s Law indicates that the sum of the partial pressures of the gases that make up the standard atmosphere must equal 760 mmHg as shown in Table 2.1.2-1.
The pressure of each gas in a mixture of gases is independent of the pressure of the other gases in that mixture. Multiplying the percentage of a gas in the mixture times the total pressure of the mixture yields the partial pressure of that gas (Table 2.1.2-1).

The standard atmosphere does not include water vapor pressure, primarily due to its variation in the Earth’s atmosphere between 0% and 100% relative humidity. That variation amounts to 0% to 6.2% of 760 mmHg, or 0 to 47 mmHg at body temperature, 37°C. See Section 3.3 on trapped gas about water vapor effects at high altitudes.

2.2.6. Law of Gaseous Diffusion (Graham’s and Fick’s Laws)

Gaseous diffusion is fundamental to the physiologic processes of lung and cellular respiration. It further applies to the process of denitrogenation, the removal of nitrogen from the body by breathing 100% oxygen.

Experiments of Thomas Graham (1805 – 1869), a Scottish chemist, showed that the diffusion of a gas in a liquid or gas is 1) inversely proportional to the square root of its molecular weight, and 2) directly proportional to the solubility coefficient of the gas. Thus, gases of lower molecular weight diffuse more rapidly than gases of higher molecular weight. A gas with greater solubility in its solvent, e.g., tissue or fluids, means more molecules of it will be available to diffuse as limited by the other factors.

Carbon dioxide is 24 times more soluble than O₂; however, when its diffusion rate is taken into consideration, CO₂ diffuses about 20 times more rapidly than O₂ through the alveolar-capillary barrier. Graham’s Law can be represented by:

\[ D \propto \frac{\text{Solubility}}{\sqrt{\text{MW}}} \]

where:
- \( D \) = diffusing capacity
- \( \text{MW} \) = molecular weight of a particular gas

Diffusion of a gas is also affected by the difference in concentration of the gas between two adjacent volumes. A larger difference in concentration produces greater diffusion. When considering the diffusion of gases within the body, the tissues through which the gases pass must also be taken into consideration. Fick’s Law of Diffusion governs the movement of O₂ and CO₂, as well as N₂, across the blood-gas barriers. Fick’s Law of Diffusion is represented by:

\[ \dot{V}_{\text{gas}} = \frac{A \times D \times \Delta P}{T} \]

where:
- \( \dot{V}_{\text{gas}} \) = volume of gas diffusing through the tissue barrier per unit of time (mL/min)
- \( A \) = surface area of the barrier available for diffusion (~70 m² in the lungs)
- \( D \) = diffusion coefficient of a particular gas
- \( \Delta P \) = partial pressure difference of the gas across the barrier
- \( T \) = diffusion barrier thickness (alveolar-capillary barrier is ~0.2 – 0.5 μm)
As an example of gaseous diffusion, Figure 2.2.5-1 shows what happens across the semi-permeable lung alveoli as a person begins to breathe 100% oxygen. Although this is a hypothetical figure, the principle of reaching equilibrium if diffusion were completed is shown. In the lung, equilibrium is likely not achieved due to the continuation of respiration. However, it can be seen that considerable N₂ diffuses from the capillary to the alveolus and considerable O₂ diffuses from the alveolus to the capillary. The solubility of CO₂ as a bicarbonate ion, HCO₃⁻ in capillary fluids, and O₂ uptake by hemoglobin in the capillaries complicate the values in this depiction, but the diffusion is in the direction shown.

**BEFORE DIFFUSION**

<table>
<thead>
<tr>
<th>Capillary Blood</th>
<th>Alveolus Air</th>
</tr>
</thead>
<tbody>
<tr>
<td>570 mmHg N₂</td>
<td>0 mmHg N₂</td>
</tr>
<tr>
<td>40 mmHg O₂</td>
<td>673 mmHg O₂</td>
</tr>
<tr>
<td>46 mmHg CO₂</td>
<td>40 mmHg CO₂</td>
</tr>
<tr>
<td>47 mmHg H₂O</td>
<td>47 mmHg H₂O</td>
</tr>
</tbody>
</table>

**AFTER DIFFUSION**

<table>
<thead>
<tr>
<th>Capillary Blood</th>
<th>Alveolus Air</th>
</tr>
</thead>
<tbody>
<tr>
<td>285 mmHg N₂</td>
<td>285 mmHg N₂ [increases]</td>
</tr>
<tr>
<td>357 mmHg O₂</td>
<td>357 mmHg O₂ [increases]</td>
</tr>
<tr>
<td>42 mmHg CO₂</td>
<td>42 mmHg CO₂ [increases]</td>
</tr>
<tr>
<td>47 mmHg H₂O</td>
<td>47 mmHg H₂O [no change]</td>
</tr>
</tbody>
</table>

**Figure 2.2.5-1. Gaseous Diffusion of N₂, O₂, and CO₂ between Alveoli and Capillaries after Beginning to Breathe 100% O₂**

Water vapor (H₂O) is at equilibrium throughout the process of respiration at the alveolar level shown in Figure 2.2.5-1. Hence, there is no change in the 47 mmHg partial pressure of water vapor in the alveoli at body temperature. This constant partial pressure of water vapor at body temperature becomes very important at very high altitudes because it represents a higher percentage of the gases present. At 63,000 ft, Armstrong’s Line, the total pressure is 47 mmHg.

**2.2.7. Law of Dissolved Gas Partial Pressure (Henry’s Law)**

William Henry (1775 – 1836) was an English chemist who, in 1803, published his findings that the amount of a gas in a solution varies directly with the partial pressure of that gas in contact with the solution, for a given temperature. This relationship explains why dissolved nitrogen transitions to a gas phase in blood and tissues during decompressions sufficient to result in supersaturation. The resulting bubbles of nitrogen, with minor amounts of oxygen, carbon dioxide, and water vapor, can cause decompression sickness.
The amount of gas dissolved in solution can be determined by the following:

\[ C_{gas} = S_{gas} \times P_{gas} \]

where:
- \( C_{gas} \) = concentration of dissolved gas
- \( S_{gas} \) = solubility of gas, for a given temperature
- \( P_{gas} \) = partial pressure of a particular gas

It’s important to remember that when a gas is in contact with a liquid, such as between the alveoli and blood in the pulmonary capillaries, the gas will dissolve in the liquid until equilibrium is reached between the two phases. At equilibrium, it is the partial pressures of the gas that are equal in the two phases, not the concentrations. Therefore, \( P_{liquid} = P_{gas} \), rather than \( C_{liquid} = C_{gas} \).

When considering the amount, or actual content, of nitrogen dissolved in the blood at 37°C and 760 mmHg, 0.0088 mL N\(_2\) will dissolve in 100 mL blood for every 1 mmHg P\(_{N_2}\) (Davis, 2008). Therefore, according to Henry’s Law, at a normal P\(_a\)N\(_2\) of 569 mmHg, approximately 5.01 mL N\(_2\) is carried dissolved in 100 mL of blood.

\[ C_{N_2} = S_{N_2} \times P_a N_2 \]
\[ C_{N_2} = \frac{0.0088 \text{ mL } O_2}{\text{mmHg}} \times 569 \text{ mmHg} = \frac{5.01 \text{ mL } N_2}{100 \text{ mL blood}} \]

While Henry’s Law is typically dealt with in terms of decompression sickness, the law also plays a role in respiration by describing the amount of the various gases that are dissolved in plasma. According to Henry’s Law, at 37°C and 760 mmHg, 0.003 mL O\(_2\) will dissolve in 100 mL blood for every 1 mmHg P\(_{O_2}\) (Davis, 2008). Therefore, at a normal P\(_a\)O\(_2\) of 100 mmHg, approximately 0.3 mL O\(_2\) is carried dissolved in 100 mL of blood.

\[ C_{O_2} = S_{O_2} \times P_a O_2 \]
\[ C_{O_2} = \frac{0.003 \text{ mL } O_2}{\text{mmHg}} \times 100 \text{ mmHg} = \frac{0.3 \text{ mL } O_2}{100 \text{ mL blood}} \]

### 2.2.8. Alveolar Gas Equation

As described in the previous sections, the percentage of oxygen is a consistent 21% of the total atmosphere throughout the altitudes in which aircraft operate. Accordingly, as the total pressure decreases with altitude, 21% of the shrinking total is less. Thus, as described by Dalton’s Law, the partial pressure of oxygen decreases with altitude.

The tables in Appendix 1 (altitude of exposure vs. alveolar partial pressure of oxygen) are based on the U.S. Standard Atmosphere (1962) geometric altitude. The following nomenclature is used throughout this document to describe the relationships between gases of importance to human physiology:
\( P_B \): Barometric pressure in mmHg
\( F_iO_2 \): The fraction of the total inspired air which is oxygen; the fraction of oxygen in atmospheric air is 21% (0.21)
\( P_iO_2 \): Partial pressure of inspired oxygen in the trachea
\( P_AO_2 \): Partial pressure of oxygen in the alveoli
\( P_ACO_2 \): Partial pressure of carbon dioxide in the alveoli, 40 mmHg at sea level, but decreases during exposure to lower total pressure due to hyperventilation caused by the lower \( P_AO_2 \)
\( P_AH_2O \): Partial pressure of water vapor at body temperature, a constant 47 mmHg
\( R \): The respiratory quotient (exchange ratio; R or RQ: \( \frac{CO_2 \text{ production}}{O_2 \text{ consumption}} \))

\( R \) increases with increasing altitude due to hypoxia-induced hyperventilation that increases \( CO_2 \) elimination, the same phenomenon observed during strenuous exercise with increased ventilation rate or during hyperventilation. The \( R \), normally about 0.83 at sea level, is also affected by diet due to the varying ratio of carbon to oxygen in various foods. Fat has a relatively high ratio of carbon to oxygen and requires more respiratory oxygen when metabolized by the body, thus lowering the \( R \) to 0.70. Metabolism of carbohydrates, which contain much more oxygen, yields an \( R \) of 1.0 and protein metabolism yields an \( R \) of 0.80. For a typical mixed diet, an \( R = 0.85 \) can be assumed.

Recall from Section 1.2.4, Alveolar Gas Tensions, that alveolar \( P_AO_2 \) can be calculated using the alveolar gas equation:

\[
P_AO_2 = P_iO_2 - \left[ P_ACO_2 \times \left(F_iO_2 + \left(\frac{1 - F_iO_2}{R}\right)\right)\right]
\]

where,
\[
P_iO_2 = (P_B - 47) \times F_iO_2
\]

At sea level pressure this becomes:
\[
P_iO_2 = (760 - 47) \times 0.21 = 149.7 \text{ mmHg}
\]
\[
P_AO_2 = P_iO_2 - \left[ P_ACO_2 \times \left(F_iO_2 + \left(\frac{1 - F_iO_2}{R}\right)\right)\right]
\]
\[
P_AO_2 = 149.7 - \left[ 40 \times \left(0.21 + \left(\frac{1 - 0.21}{0.85}\right)\right)\right] = 149.7 - 45.6 = 104.1 \text{ mmHg}
\]

Plotting the \( P_AO_2 \) values versus altitude breathing air or 100% oxygen using the equation above yields curves that depict the varying \( P_AO_2 \) over the range of most USAF missions (Figures 2.2.8-1 and 2.2.8-2).
Figure 2.2.8-1. Calculated $P_{A\ensuremath{O}_2}$ in mmHg as a Function of Altitude and Breathing Air (from Appendix 1a)

Figure 2.2.8-2. Calculated $P_{A\ensuremath{O}_2}$ in mmHg as a Function of Altitude and Breathing 100% Oxygen (from Appendix 1b)

Note for Figures 2.2.8-1 and 2.2.8-2: The dashed lines at 30 mmHg represent the approximate level of alveolar oxygen needed to maintain consciousness (Ernsting et al., 1999). The line in Figure 2.2.8-2 is based on data in Appendix 1b as adjusted to reflect no PBA (positive pressure breathing for altitude).
The minimum fraction of inspired oxygen \( (F_{i}O_{2NP}) \) delivered by a USAF narrow panel regulator (CRU 73/P) is a function of altitude in the cockpit/cabin. The regulator adds 100% oxygen to atmospheric air, diluting the air with oxygen. To calculate the \( F_{i}O_{2NP} \) delivered at a specific altitude (dotted line in Figure 2.8.8-3), the following formula can be used:

\[
F_{i}O_{2NP} = \% O_{2} \text{ added} + [0.21 \times (100 - \% O_{2} \text{ added})]
\]

At 25,000 ft in Figure 3.1.1-3, the % O\(_2\) added is about 37%, thus

\[
F_{i}O_{2NP} = 37 + [0.21 \times (100 - 37)] = 50\%
\]
References
Boyle R. A New Experiment Concerning an Effect of the Varying Weight of the Atmosphere upon Some Bodies in the Water. Philosoph Transact (1672) 7:5156. [In: Bert, 1878, p202, 311; Hitchcock translation, 1978].
Dalton J. Experimental Enquiry into the Proportion of the Several Gases or Elastic Fluids, Constituting the Atmosphere. Memoirs of the Literary and Philosophical Society of Manchester (1805) 1:244-58.
Internet Resources, See Appendix 8.

Recommended Readings

Concepts
Ideal Gas Law
Law of dissolved gas partial pressure
Law of gaseous diffusion
Law of partial pressures
Pressure-volume law
Volume-temperature law
Pressure-temperature law
Alveolar gas equation

\[ P_{iO_2} = (P_B - 47) \times F_iO_2 \]

\[ P_{A O_2} = P_{iO_2} - \left[ P_{A CO_2} \times \left( F_iO_2 + \left( \frac{1 - F_iO_2}{R} \right) \right) \right] \]

Vocabulary
Diffusion
\[ PV = nRT \]
\[ P_B \]
\[ F_iO_2 \]
\[ P_{iO_2} \]
\[ P_{A O_2} \]
\[ P_{A CO_2} \]
\[ P_{A H_2O} \]
3. ATMOSPHERIC ENVIRONMENTAL EFFECTS

3.1. Hypoxia

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3.1.1. Definition and Causes

Hypoxia is a state of oxygen deficiency in the blood, cells, or tissues sufficient to cause and impairment of function. More simply, hypoxia means “reduced oxygen” or “not enough oxygen.” A complete lack of oxygen is called anoxia. Although any tissue will die if deprived of oxygen long enough, the brain is particularly vulnerable to oxygen deprivation. Any reduction in mental function while flying can result in life-threatening errors. Hypoxia can be caused by several factors, including an insufficient supply of oxygen, inadequate transportation of oxygen, or the inability of the body tissues to use oxygen. Figure 3.1.1-1 summarizes general effects of hypoxia in relation to arterial oxygen saturation.

Figure 3.1.1-1. General Effects of Hypoxia on Arterial Saturation and Body Function (recreated from Roth, 1968)
3.1.2. Types of Hypoxia

3.1.2.1. Hypemic Hypoxia. Hypemic hypoxia occurs when the blood is not able to take up and transport a sufficient amount of oxygen to the cells in the body. This type of hypoxia is a result of a reduced oxygen carrying capacity of the blood, rather than a lack of inhaled oxygen, and can be caused by a variety of factors from a physical reduction of hemoglobin to altered forms of hemoglobin (these altered forms of hemoglobin that result in reduce oxygen carrying capacity can be collectively referred to as dyshemoglobins). The formation of dyshemoglobin by pharmaceuticals, recreational drugs, and toxic agents has been well characterized medically.

The most common cause of hypemic hypoxia is due to the inability of hemoglobin, the actual blood molecule that transports oxygen, to bind oxygen molecules, such as occurs during carbon monoxide poisoning. Carbon monoxide binds to hemoglobin in the same binding site as oxygen, forming carboxyhemoglobin (COHb) (Figure 3.1.2.1-1).

![Figure 3.1.2.1-1. Oxygen and Carbon Monoxide Binding to Hemoglobin](image)

This binding between carbon monoxide and hemoglobin is 200 times more effective than oxygen binding to hemoglobin, and the reaction is more rapid in the presence of hypoxia (West, 1962). The formation of carboxyhemoglobin (COHb) causes a left shift of the oxygen-hemoglobin dissociation curve, reducing the unloading of oxygen at the tissues (see Sections 1.2.6 and 1.2.7 for more details). The result is that an individual’s physiological altitude is higher than their true altitude due to the reduced oxygen carrying capacity and resulting hypoxia. Figure 3.1.2.1-2 shows the effect of increasing COHb concentrations and the relationship between physiological and true altitude (Judd, 1971).
Aircrew can be exposed to carbon monoxide in the exhaust of aircraft, ground equipment, or other vehicles on the flightline, in addition to an in-flight emergency involving smoke and fumes. Carbon monoxide levels of 1,000 ppm have been reported in the cabin compartment of U.S. Army CH-47A helicopters after firing 7.62 mm machine guns (Denniston, 1978). This level has the potential to raise blood COHb levels up to nearly 7% in non-smokers. In pressurized aircraft, any carbon monoxide in the air (i.e. from exhaust of other aircraft while taxiing or waiting to take off) can be circulated throughout the cabin. Figure 3.1.2.1-2 shows the relationship between an individual’s physiological altitude and their true altitude when exposed to various partial pressures of carbon monoxide conditions in ambient air (McFarland et al., 1944).
Treatment with 100% oxygen is an effective way of getting some oxygen to the tissues and to speed "wash out" of the carbon monoxide. Breathing 100% oxygen at ground level decreases the half-life of carboxyhemoglobin from approximately 5.5 hours to approximately 1 hour and 20 minutes (Kindwall, 1993). Hyperbaric oxygen treatment supplies oxygen in a pressurized chamber and is capable of providing oxygen at many times the normal amount in air. This is much more effective than 100% oxygen at sea level pressure and can further reduce the half-life of carboxyhemoglobin to approximately 20 minutes (Kindwall, 1993).

Another example of a condition that leads to hypemic hypoxia is decreased blood volume due to severe bleeding or blood donation. Blood can take several weeks to return to normal following a donation. Although the effects of the blood loss are slight at ground level, there are risks when flying during this time. U.S. Air Force aircrew are restricted from flying within 72 hours after donating blood or plasma (AFI 11-202v3, 2014). Certain conditions, such as anemia, can also cause hypemic hypoxia and lead to a higher physiological altitude (Table 3.1.2.1-1). "Actual" anemia is due to a physical decrease in the amount of hemoglobin present in the blood. This can occur due to iron or vitamin deficiencies, during chronic disease, in response to chemotherapy, genetics, during menstruation, or pregnancy (Mayo Clinic, 2016).
Table 3.1.2.1-1. Equivalent Altitudes for Anemic Patients

<table>
<thead>
<tr>
<th>Hb Level (g Hg/100 ml blood)</th>
<th>Acute Anemia (equivalent altitude in feet)</th>
<th>Chronic Anemia (equivalent altitude in feet)</th>
</tr>
</thead>
<tbody>
<tr>
<td>15</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>14</td>
<td>1,200</td>
<td>800</td>
</tr>
<tr>
<td>13</td>
<td>2,400</td>
<td>1,500</td>
</tr>
<tr>
<td>12</td>
<td>3,500</td>
<td>2,300</td>
</tr>
<tr>
<td>11</td>
<td>4,800</td>
<td>3,200</td>
</tr>
<tr>
<td>10</td>
<td>6,000</td>
<td>4,000</td>
</tr>
<tr>
<td>9</td>
<td>7,200</td>
<td>4,800</td>
</tr>
<tr>
<td>8</td>
<td>8,400</td>
<td>5,600</td>
</tr>
<tr>
<td>7</td>
<td>9,500</td>
<td>6,300</td>
</tr>
<tr>
<td>6</td>
<td>11,000</td>
<td>7,200</td>
</tr>
</tbody>
</table>

*Alaska Air Medical Escort Training Manual, Chapter 4—Hypoxia and Oxygenation, 2006.*

In addition to actual anemia, some medications cause the heme group to oxidize from Fe$^{2+}$ to Fe$^{3+}$ and form methemoglobin (a dyshemoglobin) which is no longer able to bind and release oxygen properly, resulting in functional, or “relative,” anemia (Figure 3.1.2.1-4). Recall that under normal physiologic conditions, the iron in hemoglobin is in an unstable Fe$^{2+}$ state that allows it to easily pick up and release O$_2$ for effective transport. It is only when the iron is transformed into the more stable Fe$^{3+}$ state that hemoglobin becomes too stable to actively participate in oxygen transport (Haymond, 2006). Other medications, such as sulphonamides (i.e. sulfa drugs like Bactrim™), have the potential to interact with hemoglobin and alter its structure such that its oxygen binding capacity is diminished or completely blocked (effects of medications are discussed in more detail below). Sulfa medications react with hemoglobin to form sulfhemoglobin (Figure 3.1.2.1-4), causing a conformational change in the hemoglobin molecule, prevent efficient oxygen transport, and cause relative anemia. Unlike carboxyhemoglobin, the interaction is irreversible. Carboxyhemoglobin (discussed above) also leads to relative anemia. Clinical presentation of individuals with acute hypoxia due to dyshemoglobin formation include cyanosis and decreased oxygen saturation and content, even with the administration of high concentrations of oxygen (normal oximetry readings may be misleading, such as with carbon monoxide poisoning).

![Sulfhemoglobin](image1)

![Methemoglobin](image2)

Figure 3.1.2.1-4. Altered Forms of Hemoglobin
Other altered forms of hemoglobin, such as sickle cell, that result from genetic mutations can also interfere with oxygen transport. There is research to support the fact that hypoxemia exacerbates the sickle cell condition (Claster et al., 1981). In recent years, there have been reports in the media regarding athletes with the sickle cell trait (i.e. are heterozygous) not competing at high altitude (such as in Denver, CO, elevation 5,280 ft) out of concern that the reduced oxygen pressure (i.e. mild hypoxia) and strenuous activity may lead to the hemoglobin altering configuration and becoming sickle shaped, obstructing blood flow, and lead to a cardiovascular crisis (Rayman et al., 2001). While individuals who are homozygous for sickle cell are medically disqualified from flying duties in the U.S. Air Force, individuals who carry the sickle cell trait can receive a waiver to fly. Non-flying personnel who are heterozygous carriers and travel to higher altitudes should also be monitored for possible negative health consequences, especially during periods of heavy activity.

As mentioned, the formation of dyshemoglobin by pharmaceuticals, recreational drugs, and toxic agents has been well characterized medically. Causative agents of dyshemoglobin formation vary widely in their mechanism of actions, with some having a direct effect (such as benzocaine), while others need to be metabolized by the liver via enzyme systems to the active creator of dyshemoglobin (Hartman et al., 2016). Further complexity is garnered in genetic predispositions and with varying enzyme system activity levels, specifically in the cytochrome P450 (CYP-450) family of enzymes in the liver. Genetic predisposition may be predicted in some cases, for example glucose-6-phosphate dehydrogenase (G6PD) testing, which is prevalent in the military for individuals deploying into high malaria regions. Genetic testing on enzymatic pathways may largely predict metabolite production and thus predict likelihood of adverse events; however, there is currently no feasible and economic way to carry out large scale testing to predict dyshemoglobin formation (i.e. CYP 2D6 transformation of lidocaine to its active dyshemoglobin forming metabolite (Hartman et al., 2014)). Genetic abnormalities such as sickle cell anemia (discussed above) or a persistent state of anemia due to blood loss or other conditions also tend to be comorbid conditions influencing the degree to which the symptoms may be clinically evident. Prudence dictates that at the very least we are able to educate our providers and pilots about the causative agents, but prediction of a probability of adverse effects leading to dangerous situations is beyond our current scope. Table 3.1.2.1-2 lists some of the known agents and common names that contribute directly or indirectly to dyshemoglobin formation (Wright et al., 1999).
### Table 3.1.2.1-2. Agents that Lead to Dyshemoglobin Formation

<table>
<thead>
<tr>
<th>Agent</th>
<th>Common Name or Drug Family Members</th>
<th>Medication use/purpose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aniline</td>
<td>Dyes/ink</td>
<td>Inactive ingredient</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>Lidoderm, lidocaine jelly, Rectocare, Magic Mouthwash, other OTC and Rx products for local analgesia</td>
<td>Local analgesic used to treat everything for back pain to anal fissures, to sore throats</td>
</tr>
<tr>
<td>Antipyrine</td>
<td>Auralgan</td>
<td>Ear pain associated with ear infection (currently off the market in the U.S.)</td>
</tr>
<tr>
<td>Benzocaine</td>
<td>Anbisol, oragel, auralgan, other OTC and Rx products for local analgesia</td>
<td>OTCs commonly for insect bites, mouth/tooth pain, Rx products for wide ranging analgesia</td>
</tr>
<tr>
<td>Chloroquine</td>
<td>Antibiotic</td>
<td>Malaria treatment and prevention</td>
</tr>
<tr>
<td>Dapsone</td>
<td>Antibiotic</td>
<td>Leprosy treatment, skin conditions such as severe acne</td>
</tr>
<tr>
<td>Metoclopramide</td>
<td>Reglan</td>
<td>Nausea and vomiting, and GI motility disorders</td>
</tr>
<tr>
<td>Nitrates, Nitric Oxide, Nitrites</td>
<td>Isosorbid, Imdur, nitroglycerine (tablets, sprays, sublingual formulations, pastes/creams/ointments), PDE-5 inhibitors (Cialis, Viagra, Levitra), certain muscle supplements, OTC “male vitality” supplements, illicit drugs (poppers/whippets)</td>
<td>Angina (chest pain), anal fissures, Erectile dysfunction, some muscle building supplements, some illegal drugs</td>
</tr>
<tr>
<td>Nitrofurazone</td>
<td>Antibiotic</td>
<td>UTI, Abdominal infections</td>
</tr>
<tr>
<td>Nitroprusside</td>
<td>Nitropress</td>
<td>Acute management of severe malignant hypertension</td>
</tr>
<tr>
<td>Phencypiridine</td>
<td>Pyridium</td>
<td>Analgesic commonly given for UTI associated pain on urination</td>
</tr>
<tr>
<td>Phenytoin</td>
<td>Dilantin</td>
<td>Seizure management</td>
</tr>
<tr>
<td>Prilocaine</td>
<td>Analgesic</td>
<td>Local analgesic for medical procedures or compounded into topical preparations for genital use</td>
</tr>
<tr>
<td>Primaquine</td>
<td>Anti-malaria</td>
<td>Treatment or prevention of malaria. Used extensively in malaria prophylaxis for returning deployers in the military</td>
</tr>
<tr>
<td>Sulfonamide</td>
<td>Antibiotic, Sepra, Bactrim, sulfamethoxazole-trimethoprim</td>
<td>Antibiotic commonly used in UTIs, skin and soft tissue infections, and other general infections</td>
</tr>
</tbody>
</table>

* OTC = over-the-counter; Rx = prescription; GI = gastrointestinal; UTI = urinary tract infection;
Other agents that are not as common in use include: acetanilid, alloxan, arsenic, benzene derivatives, chlorates, chlorobenzene, dinitrophenol, dinitrotoluene, hydroxylamine, menadione, naphthalene, nitroalkanes, nitrochlorobenzene, parquat/diquat, phenacetin, phenol, and trinitrotoluene. Various toxins, pesticides, smoke and carbon monoxide, and numerous other chemicals and medications may also contribute to dyshemoglobin formation, and as such this list is intended as a guide and not meant to be all encompassing.

Currently, management of causative agents of dyshemoglobin formation is focused on judicious use of the agents by Flight Medicine providers and clinicians, as well as strict adherence by aircrew to the no self-medication rule. Education on the types of prescriptions and over-the-counter medications is the primary preventative mechanism to reduce the potential for adverse events. Factors that must be considered in selection of a potential causative agent must be at a minimum: 1) seriousness of condition, 2) do treatments exist that are effective and may not cause any potential for harm, 3) duration of treatment, and 4) potential for operating in conditions where subclinical presentation becomes of concern.

In the absence of monitoring for dyshemoglobin, it is difficult to predict how individual patients will react to an agent. Thus, for providers and patients alike, consideration of all the above factors must be rendered before a treatment is selected. The responsibility of full disclosure to the provider and the necessity for the patient to report all medications including over the counter medications and supplements (dietary, and herbal) is essential for desirable outcomes of treatment to exist. Given the environments which military personnel perform in, it is possible that even subclinical manifestations of dyshemoglobin formation may prove dangerous, especially if in conjunction with other forms of hypoxia (i.e. hypobaric hypoxia) or physical exertion.

3.1.2.2. Stagnant Hypoxia. Stagnant means "not flowing," and stagnant hypoxia results when the oxygen-rich blood from the lungs isn’t moving, for one reason or another, to the tissues that need it. An arm or leg going to sleep because the blood flow has accidentally been shut off is one form of stagnant hypoxia. This kind of hypoxia can also result from shock, the heart failing to pump blood effectively, or a constricted artery. During flight, stagnant hypoxia can occur when pulling excessive +Gz, as discussed in Section 7.1, Physiologic Effects of Acceleration. Stagnant hypoxia is also called ischemic or circulatory hypoxia.

3.1.2.3. Histotoxic Hypoxia. The inability of the cells to effectively accept or utilize oxygen is defined as histotoxic hypoxia. "Histo" refers to tissues or cells, and "toxic" means poison. In this case, plenty of oxygen is being transported to the cells that need it, but they are unable to make use of it due to poisoning or impairment of the mitochondrial enzyme system. In histotoxic hypoxia, the cells are hypoxic without the individual experiencing hypoxemia (i.e. PaO2 is normal). This impairment of cellular respiration can be caused by alcohol, narcotics, carbon monoxide, and poisons such as cyanide (a metabolite of many nitrate medications). The impact of these are discussed in more detail in Section 4.2. Figure 3.1.2.3 shows the location of inhibition within the mitochondrial enzyme system of selected agents (Manzo-Avalos and Saavedra-Molia, 2010; Medh, n.d.; Santa Cruz, 2016; Ott, n.d.; Miles, 2003).
3.1.2.4. Hypoxic Hypoxia. Hypoxic hypoxia, commonly called altitude hypoxia, is a result of insufficient oxygen available to the lungs, or low $P_{\text{AO}_2}$. A blocked airway or drowning are obvious examples of how the lungs can be deprived of oxygen, but the reduction in partial pressure of oxygen at high altitude is an appropriate example for aircrew. Although the percentage of oxygen in the atmosphere is constant, its partial pressure decreases proportionately as atmospheric pressure decreases. As the airplane ascends during flight, the percentage of each gas in the atmosphere remains the same, but there are fewer molecules of each, including oxygen, available to diffuse through the alveolar membranes of the lungs. This decrease of oxygen molecules can lead to hypoxic hypoxia, also called altitude hypoxia. Appendix 1 shows the alveolar oxygen levels resulting from exposure to altitude breathing air up to 45,000 ft (Appendix 1a), breathing 100% oxygen from 25,000 to 50,000 ft with positive pressure breathing for altitude (PBA) (Appendix 1b), and breathing 100% oxygen from 50,000 to 70,000 ft with PBA (Appendix 1c). Pressure breathing requires the crewmember to “reverse breathe,” since the pressure supplied inflates the lungs with no effort from the crewmember during inhalation. Exhalation during PBA is accomplished by forcing air out instead of relaxing and can be very tiring. With training and experience, an individual can pressure breathe “against” a 50-mmHg pressure for a short time, but even a 30-mmHg gradient will cause fatigue, and higher levels require assisted PBA with a counterpressure jerkin that helps to restrain chest expansion and reduce the effort involved during exhalation (Ernsting et al., 1999).
High altitude flying can place an aircrew member in danger of becoming hypoxic if insufficient oxygen is delivered to compensate for the reduced partial pressure of oxygen in the cabin/cockpit. Without sufficient oxygen, the brain and other vital organs become impaired. During the onset of hypoxia, an early symptom may be euphoria (Table 3.1.7-1), a carefree feeling, which could affect recognition and judgment, delaying a check of the oxygen system. Increased depth of breathing is usually an early sign or symptom, possibly accompanied by oxygen want, or air hunger.

3.1.2.4.1. Stages of Hypoxic Hypoxia. The stages of hypoxic hypoxia are categorized by the level of symptoms experienced as a result of reducing arterial oxygen saturation as shown in Table 3.1.2.4-1.

<table>
<thead>
<tr>
<th>Stage</th>
<th>Altitude (feet)</th>
<th>Breathing Air</th>
<th>Breathing 100% O2</th>
<th>Arterial O2 Saturation (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Indifferent</td>
<td>0 to 10,000</td>
<td>34,000 to 39,000</td>
<td>95 to 90</td>
<td></td>
</tr>
<tr>
<td>Compensatory</td>
<td>10,000 to 15,000</td>
<td>39,000 to 42,500</td>
<td>90 to 80</td>
<td></td>
</tr>
<tr>
<td>Disturbance</td>
<td>15,000 to 20,000</td>
<td>42,500 to 44,800</td>
<td>80 to 70</td>
<td></td>
</tr>
<tr>
<td>Critical</td>
<td>20,000 to 23,000</td>
<td>44,800 to 45,500</td>
<td>70 to 60</td>
<td></td>
</tr>
</tbody>
</table>

The Indifferent Stage (sea level to 10,000 ft) relates to the Physiological Zone discussed in Section 2.1.2 and is often associated with little to no physiological impact. However, at altitudes as low as 4,000 ft, there is a slight increase in heart rate and pulmonary ventilation, which is often not noticed by a person at rest (TB Med 505, 2010).

One of the first physiological systems affected by a reduction in the partial pressure of oxygen is the visual system, as the retina is the most oxygen-sensitive tissue in the body (NASA/SP-2010-3407, 2010). This impact on vision is more noticeable with regards to rod function and night vision. This is especially true when using any night vision devices (NVDs). Increasing the rod and cone threshold was described as the main effect on hypoxic hypoxia (Miller and Tredici, 1992). Scotopic vision (vision during reduced illumination; see Section 1.4) at 10,000 ft was reported to be decreased by 20% and 35% at 13,000 ft without supplemental oxygen, while delayed dark adaptation and impaired color vision has been reported to begin around 4,000 – 5,000 ft (TB Med 505, 2010; NASA/SP-2010-3407, 2010). Figure 3.1.2.4.1-1 shows some of the visual decrements associated with increasing altitude.
The subtle effects of a lack of adequate oxygen on vision are readily apparent during training in a darkened chamber at 18,000 ft once supplemental oxygen is supplied. It is difficult to detect these negative effects without such a demonstration, but at least some negative effects have been documented at or above 10,000 ft (Balldin et al., 2007).

Physical performance, primarily maximal aerobic performance, is negatively affected starting at 4,000 ft. Figure 3.1.2.4.1-2 shows the performance decrements that occur after 10 days of altitude training as event duration and elevation increases. For example, an event that lasts 20 – 30 minutes at sea level will take approximately 7% longer at an altitude of 2,300 m (about 7,500 ft). Events lasting 2 – 3 hours will average 17% longer (Fulco et. al., 1998). Acclimatization does not improve maximal physical performance at high altitude. Long-term residence (i.e. months) may help improve it, but prolonged physical performance at altitudes starting around 3,000 meters will remain impaired relative to sea level performance (TB Med 505, 2010). For easier reference, 3,000 meters is approximately 10,000 ft, the upper limit of the Indifferent Stage.
The primary reason for this decreased performance with increasing altitude is related to the decrease in maximal oxygen consumption ($\dot{V}_{O_2,\text{max}}$) that occurs with ascent to higher altitude. This relationship is shown in Figure 3.1.2.4.1-3.
At altitudes above 8,000 ft, the risk of acute mountain sickness (AMS) also increases and can have significant impact on individual performance and unit capabilities. AMS affects both men and women and can occur in individuals of all ages (West et al., 2007). While AMS is not typically a concern in aviation, it should be considered for unacclimatized military members deploying to high altitude. Stateside, military members rarely encounter altitude stress and many are therefore unprepared for the effects. Rapid ascent (less than 24 hrs) to altitudes between 6,000 – 8,000 ft results in 10% – 20% of soldiers experiencing at least mild forms of AMS (USARIEM 94-2, 1994). Symptoms often occur within the first 24 hrs after ascent, peak in severity by 72 hrs, and then gradually subside within 7 days (Figure 3.1.6-2). One of the challenges of dealing with AMS is that it afflicts otherwise healthy individuals, without any reliable way to predict susceptibility other than prior altitude exposure. An individual’s physical fitness does not influence susceptibility or provide protection against AMS (West et al., 2007).

The Compensatory Stage (10,000 – 15,000 ft) of hypoxic hypoxia relates to the Physiologically Deficient Zone discussed in Section 2.1.2 and the name suggests that humans can compensate for the reduction in \( S_aO_2 \). However, the degree of hypoxia is sufficient to preclude adequate physiologic and cognitive function despite increases in breathing depth and rate, which provide some compensation. The increased ventilation associated with this stage is due to the hypoxic ventilatory response, as \( P_aO_2 \) drops below 60 mmHg and the peripheral chemoreceptors are activated. As \( S_aO_2 \) is starting to move to the steeper part of the oxyhemoglobin dissociation curve, small changes in \( P_aO_2 \) result in larger decreases in \( S_aO_2 \) and can result in rapid onset of symptoms. Cognitive performance is affected more than psychomotor performance and there is increased difficulty performing tasks requiring mental alertness or assimilation of new information. Reaction time, finger dexterity, and arm-hand coordination are also degraded (TB Med 505, 2010). Hence, persons in control of aircraft are required to breathe supplemental oxygen at altitudes above 10,000 ft. Figure 3.1.2.4.1-4 illustrates some of the decrements that start in the Compensatory State and extend into the next stage of hypoxic hypoxia, the Disturbance Stage (for reference: 2,000 m ≈ 6,500 ft; 6,000 m ≈ 19,700 ft).
In the **Disturbance Stage** (15,000 – 20,000 ft), physiologic compensatory responses, such as increased respiration and heart rate, are no longer capable of providing adequate oxygenation of tissues to maintain normal function. The $S_aO_2$ is now on the steeper part of the oxyhemoglobin dissociation curve, and small changes in $P_aO_2$ result in even larger decreases in $S_aO_2$ than they did in the Compensatory Stage (Figure 2.1.2-2 and Figure 3.1.1-1). During this stage, the chances of self-recovery continue to diminish due to increasing cognitive impairment.

The **Critical Stage** (20,000 – 23,000 ft) indicates a critical reduction in functional capability leading to incapacitation and ultimately a loss of consciousness when $P_aO_2$ drops below 30 mmHg (Figure 3.1.1-1).

Table 3.1.2-1, Causes of Hypoxia, summarizes the relationship between phases of respiration, problems encountered, and the common term for the type of hypoxia produced.
<table>
<thead>
<tr>
<th>Phase of Respiration</th>
<th>General Problem</th>
<th>Examples of Causes (Specific Problems)</th>
<th>Common Term for Type of Hypoxia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ventilation</td>
<td></td>
<td>Reduction in $P_{A}O_2$</td>
<td>Hypoxic hypoxia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1. Breathing air at reduced barometric pressures</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>2. Strangulation, respiratory arrest, laryngospasm</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>3. Severe asthma</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>4. Breath-holding</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>5. Hypoventilation</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>6. Breathing gas mixtures with insufficient $O_2$ pressure or %</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>a. Environments with reduced $O_2$ available; smoke and fumes</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>b. Malfunctioning oxygen equipment at altitude</td>
<td></td>
</tr>
<tr>
<td>Diffusion</td>
<td></td>
<td>Reduction in gas exchange area</td>
<td>Hypoxic hypoxia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1. Pneumonia</td>
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<tr>
<td></td>
<td></td>
<td>2. Drowning</td>
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<tr>
<td></td>
<td></td>
<td>3. Atelectasis</td>
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<td>4. Emphysema/COPD (chronic obstructive pulmonary disease)</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>5. Pneumothorax</td>
<td></td>
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<td></td>
<td></td>
<td>6. Pulmonary embolism</td>
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<td></td>
<td></td>
<td>7. Congenital heart defects</td>
<td></td>
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<td></td>
<td></td>
<td>8. Physiological shunting</td>
<td></td>
</tr>
<tr>
<td>Transportation</td>
<td></td>
<td>Reduction in oxygen-carrying capacity</td>
<td>Hypemic hypoxia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1. Anemia</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>2. Hemorrhage, blood donation or loss</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>3. Hemoglobin abnormalities</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>4. Drugs and chemicals (smoke and fumes; sulfanilamides, nitrites, cyanide, carbon monoxide)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Reduction in systemic blood flow</td>
<td>Stagnant hypoxia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1. Heart failure</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>2. Shock</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>3. Continuous positive pressure breathing</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>4. Acceleration (G forces)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>5. Pulmonary embolism</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>1. Exposure to extremes of environmental temperatures</td>
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<td></td>
<td></td>
<td>2. Postural changes (esp. following prolonged sitting or bed rest)</td>
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<td></td>
<td></td>
<td>3. Tourniquets (including restrictive clothing, straps, etc.)</td>
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<tr>
<td></td>
<td></td>
<td>4. Hyperventilation</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>5. Embolism by clots or gas bubbles</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>6. Cerebral vascular accidents</td>
<td></td>
</tr>
<tr>
<td>Utilization</td>
<td></td>
<td>Metabolic poisoning or dysfunction</td>
<td>Histotoxic hypoxia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1. Respiratory, enzyme poisoning or degradation</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>2. CO; smoke and fumes</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>3. Cyanide</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>4. Alcohol</td>
<td></td>
</tr>
</tbody>
</table>

Source: AFP 160-5, Table 4-1.
3.1.3. Environmental Factors that Affect Severity of Hypoxia

The partial pressure of oxygen available is related to altitude and breathing gas as shown in Figures 2.2.8-1, 2.2.8-2, and 2.2.8-3 and in Appendix 1. Between 30,000 and 40,000 ft, automatic pressure-demand oxygen regulators are designed to deliver 100% oxygen with a slight “safety” pressure, 3 to 4 mmHg above ambient, to prevent inboard mask leakage. Use of 100% oxygen at these altitudes was recognized in 1944 as providing for “maximum efficiency” (Figure 3.1.3-1).

![Figure 3.1.3-1. “Use of Oxygen and Oxygen Equipment” T.O. No. 03-50-1 (1944)](image)

At altitudes above 40,000 ft, the positive pressure delivered to the mask increases with increasing altitude, as shown in Appendix 1b and 1c. This additional pressure increases the total pressure of 100% oxygen delivered to the mask and is called positive pressure breathing for altitude.

*PBA may include the addition of a counterpressure vest to provide external pressure on the thorax during pressure breathing.*
Rate of ascent/rapid decompression is a factor, with greater severity if the decompression is rapid, meaning between 2 and 15 s (Davis et al., 2008). A rapid decompression compounds the effect of hypoxia by potentially creating a diffusion gradient, which drives oxygen from blood back into the lungs. A rapid decompression from a cabin altitude of about 18,700 ft to an ambient altitude of 45,000 ft with a 5-psid pressurization system (7.15 psia to 2.15 psia) while breathing 37% oxygen (minimum delivered by the USAF narrow panel regulators, Figure 2.8.8-3) provides such a situation. The cruise \( P_{A}\text{O}_2 \) before the rapid decompression would be approximately 79 mmHg, equivalent to breathing air at about 5,500 ft. This is not a problem for a relatively inactive crewmember. However, the first few breaths of the 37% oxygen in the mask and hose after a rapid decompression to 45,000 ft would yield a \( P_{A}\text{O}_2 \) far below 30 mmHg. This is insufficient to maintain consciousness if maintained for more than a few seconds (Ernsting, 1978). Several breaths may be required for the residual mixture in the supply line to be replaced with the higher concentration of oxygen being supplied at the regulator. Indeed, the blood reaching tissues shortly after such a rapid decompression would have less oxygen than the tissues, causing removal of oxygen from the tissues. For these reasons, the EPT following rapid decompression to altitude while breathing air is less than shown in Appendix 1a. Appendix 1a is based on a decompression typical of aircraft climb rate, 1,000 to 10,000 ft/min. It is likely that respiration rate would increase significantly immediately following a rapid decompression due to the stress of an emergency. This would hasten delivery of 100% oxygen to the crewmember and may prevent unconsciousness for those wearing masks at the time of decompression. Those who quickly don masks set to deliver 100% oxygen will have the same reversal of the usual direction of diffusion until they fill their lungs with oxygen. It is critical in training to stress that each individual must take care of himself or herself first!

A rapid decompression (2 – 15 seconds) to 60,000 ft, even if breathing 100% oxygen prior to the decompression and with 60 mmHg of PBA after the decompression, would yield a \( P_{A}\text{O}_2 \) less than 55 mmHg, equivalent to breathing air at about 13,000 ft (Appendix 1a and 1c). Although 70 mmHg of PBA would increase the \( P_{A}\text{O}_2 \) significantly at 60,000 ft, assisted positive pressure breathing (PPB) (using a chest counterpressure device) would be necessary to avoid extreme difficulty in regulating breathing.

A rapid decompression can present additional dangers beyond hypoxia. A rapid decompression may cause lung overinflation with more than 80 mmHg of transthoracic pressure. A breath-hold during a rapid decompression could exacerbate the condition, leading to a pulmonary air embolism: transfer of gas from the lung to the circulatory system (not ebullism). Differential transthoracic pressure of up to 80 mmHg (Luft and Bancroft, 1956) has been tolerated without injury and without assisted PPB including a counterpressure jerkin. Air embolism in the arterial system may lead to several symptoms resembling acute neurologic decompression sickness (DCS). Onset of symptoms from an air embolism is much faster than from DCS, and treatment by immediate descent while breathing 100% oxygen should be followed by hyperbaric oxygen (HBO) treatment as necessary.

Slow decompression has its own characteristic risk; it is insidious. There is no loud noise or fogging of the air to draw attention. As pressure is slowly lost, signs and symptoms develop slowly. It is possible to suffer the debilitating effects of hypoxia without recognizing their presence in the absence of cabin pressure warning systems. This has dangerous and sometimes fatal consequences.
Duration at altitude is a factor because remaining at altitudes between about 20,000 and 30,000 ft after a fast decompression (less than about 3 minutes) increases the chance of loss of consciousness due to depletion of tissue oxygen reserves. Also, physiologic compensatory mechanisms are limited. At these altitudes, a timely decision to descend should prevent altitude-induced loss of consciousness if other remedies are insufficient to provide adequate oxygenation.

A higher ambient temperature and/or an increase in humidity correspond to decreased air density and, therefore, a small reduction of the partial pressure of oxygen. Higher temperature increases body metabolism, which increases oxygen demand. Although both effects are small, their combined effect may increase the severity of hypoxia.

There is considerable variation in individual tolerance to hypoxia. Factors that are known to affect hypoxia tolerance are the level of physical fitness and several other factors that affect the ability to utilize available oxygen. Good physical fitness offers some protection from hypoxia, particularly if physical activity is involved in the hypoxic episode. The increased perfusion and ventilation capabilities of athletic-level fitness provide more pathways for oxygen delivery and utilization. Note however, that if alveolar PO$_2$ drops below pulmonary capillary PO$_2$ as described in the rapid decompression discussion above, there are more pathways for oxygen to leave the blood, too. Physical fitness provides no immunity to hypoxia.

Since physical activity involves elevated oxygen consumption, a lower oxygen partial pressure will adversely affect maximal exercise capability. Increased respiration rate induced by peripheral oxygen chemoreceptors will partially reverse this effect, in part by reducing the partial pressure of carbon dioxide in the alveoli, allowing a little more oxygen to be present.

Metabolic rate is affected by emotional state. A highly agitated, excited, angry, or scared individual will require more oxygen for optimal function, and mild hypoxia could become worse under those emotional conditions. Hyperventilation can also further complicate the physiological reaction.

Medication and drugs can affect metabolic rate, oxygen utilization, respiration rate, and other factors that bear on susceptibility to hypoxia. Alcohol has long been recognized as having a synergistic effect with hypoxia. Smoking reduces available hemoglobin by 4% to 7% because carbon monoxide in cigarette smoke combines with hemoglobin. The result is a physiological altitude higher than aircraft cabin altitude (Figure 3.1.4-1). Prescription medications may also interfere with cellular respiration. These effects are negligible at low altitude but reduce the reserves of the system in hypoxic environments. For this reason, aircrew must have all medications, whether prescription or over-the-counter, approved by a flight surgeon.

Acclimatization to altitude is a multifaceted process involving increased ventilation, a shifting of the oxygen-hemoglobin dissociation curve (Section 1.2) to the right to facilitate oxygen delivery at the tissue level, increased red blood cell count (higher erythrocyte count and hematocrit), increased vascularization (more capillaries per unit volume of tissue), and an increase in muscle myoglobin concentration. Some improvement in function can be observed in a few days due to the first two items, but the remaining changes take months to reach steady state (Figure 3.1.3-2). However, even acclimatization of several weeks is insufficient to compensate for the oxygen requirements of mild exercise above about 18,000 ft (Hb saturation about 72%) (Davis et al., 2008).
3.1.4. Smoke and Fumes

Smoke and fumes in an enclosed space such as a cockpit or aircraft cabin can induce hypoxia via interference with any of the phases of respiration (Table 3.1.2-1).

- Ventilation: by displacing the oxygen
- Diffusion: by interfering with alveolar function
- Transportation: by interruption of hemoglobin’s capability to transport oxygen
- Utilization: CO poisoning, etc.

The result of smoke inhalation, primarily from the inhalation of carbon monoxide and the formation of carboxyhemoglobin (COHb), is a physiological altitude that can be significantly higher than the aircraft cabin altitude. Figures 3.1.2.1-2 and 3.1.2.1-3 show the relationship between increasing COHb and physiological altitude. Exposure to environmental pollutants such as smog, sulfur dioxide, and hydrogen sulfide can also increase the presence of sulfhemoglobin in the blood (discussed above) and reduce the amount of oxygen available (Martin, 2005).

Aircraft checklists for smoke and fumes typically direct crewmembers to select 100% oxygen (ON) and then take steps to isolate the cause and take appropriate action. Oxygen systems that deliver less than 100% oxygen do so by diluting with ambient (cabin) air, which will be contaminated in a smoke and fumes environment. For aircraft in which crewmembers wear an oxygen mask throughout flight, the same procedure would apply but may emphasize checking the oxygen delivery system to ensure it is not the source of the problem.
3.1.5. Symptoms of Hypoxia

Since symptoms of hypoxia can be different for each individual, the ability to recognize hypoxia can be improved by experiencing and witnessing the effects of it during an altitude chamber flight or with a reduced oxygen breathing device, which uses carefully calibrated mixed gas to provide a reduced partial pressure of oxygen. Table 3.1.5-1 summarizes the most common symptoms of hypoxia experienced by aircrew.

<table>
<thead>
<tr>
<th>Objective (Signs, Observable in others)</th>
<th>Subjective (Symptoms, Self-Observed)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cyanosis (blue fingernails and lips)(^a)</td>
<td>Air hunger or oxygen want</td>
</tr>
<tr>
<td>Decreased reaction time</td>
<td>Apprehension (worried or nervous)</td>
</tr>
<tr>
<td>Euphoria (unusually happy) (^a) or belligerence</td>
<td>Dizziness</td>
</tr>
<tr>
<td>Impaired judgment</td>
<td>Fatigue</td>
</tr>
<tr>
<td>Increased respiration (increased depth/rate of breathing)(^a)</td>
<td>Headache</td>
</tr>
<tr>
<td>Mental confusion(^a)</td>
<td>Hot and cold flashes</td>
</tr>
<tr>
<td>Muscle incoordination(^a)</td>
<td>Lightheaded or dizzy sensation</td>
</tr>
<tr>
<td>Unconsciousness</td>
<td>Nausea</td>
</tr>
<tr>
<td></td>
<td>Numbness</td>
</tr>
<tr>
<td></td>
<td>Tingling in fingers and toes</td>
</tr>
<tr>
<td></td>
<td>Visual impairment (blurred/tunnel vision, dimming of light or color, etc.)</td>
</tr>
</tbody>
</table>

\(^a\)Could also be self-observed.

With increased oxygen starvation, the extremities become less responsive and flying becomes less coordinated. The symptoms of hypoxia vary with the individual, but common symptoms include objective signs and subjective symptoms and some manifestations that may fall in both categories.

As hypoxia worsens, the field of vision begins to narrow, and instrument interpretation can become difficult. Even with all these symptoms, the effects of hypoxia can cause a crewmember to have a false sense of security and be deceived into believing that everything is normal. The treatment for hypoxia is more oxygen, either by flying at lower altitudes and/or use of supplemental oxygen.

3.1.6. Time of Useful Consciousness (TUC) or Effective Performance Time (EPT)

Everyone is susceptible to the effects of hypoxia, regardless of physical endurance or acclimatization. When flying at high altitudes, it is paramount that oxygen be used to avoid the effects of hypoxia. As altitude increases above 10,000 ft, the symptoms of hypoxia increase in severity, and the effective performance time rapidly decreases above 20,000 ft (Figure 3.1.6-1 and Appendix 1a). The solid line in Figure 3.1.6-1 reflects the trendline of the data and therefore varies slightly from the values in the inset table, which represent the lower end of average published times.
The terms “effective performance time” (EPT) and “time of useful consciousness” (TUC) describe the maximum time the crewmember has to make rational, life-saving decisions and carry them out at a given altitude without supplemental oxygen. It is not the amount of time until loss of consciousness. It is important to note that TUC is a very rough guide, affected by many factors other than altitude. It cannot be used to predict how long an individual will be “useful” in any given situation. The variability in TUC means that observers must be diligent during chamber training flights to ensure that once TUC has been reached for any participant, proper procedures are initiated. With timely and adequate oxygen supplied to crewmembers who reached their TUC prior to initiating recovery procedures themselves, unconsciousness and potential brain damage can be avoided.

The rapid decrease in TUC with increasing altitude can be explained by the shape of the oxygen-hemoglobin dissociation curve (Figures 1.2.6-1 and 3.1.6-2). From sea level to 10,000 ft, the oxygen-hemoglobin dissociation curve is relatively flat. At altitudes greater than 10,000 ft, the oxygen-hemoglobin dissociation curve becomes steeper and small changes in $P_{A}O_2$ lead to much larger decreases in hemoglobin saturation. The result is that the $C_aO_2$ drops and there is no longer adequate oxygen availability to the tissues.

At 10,000 ft, the $P_{A}O_2$ while breathing air is about 62 mmHg, which yields an arterial oxygen saturation of about 87%. This is in the range of oxygen saturation with deleterious effects on performance (Crow and Kelman, 1971; Denison et al., 1966). While TUC provides a rough guide, the assumption should never be made that at a given altitude a given individual’s TUC will match the “book value.” Too many factors contribute, and particularly in a slow decompression, the onset of hypoxia may go
unnoticed for many minutes. In this case, much of the TUC is already gone by the time recognition makes corrective action possible.

Figure 3.1.6-2 shows the relationship between barometric pressure (green), time of useful consciousness (black), hemoglobin saturation (red), and alveolar partial pressure of oxygen (P_AO_2) (blue).

![Graph showing relationship between barometric pressure, TUC, hemoglobin saturation, and P_AO_2](image)

**Figure 3.1.6-2. Comparison of Barometric Pressure, TUC, Hemoglobin Saturation, and P_AO_2**

### 3.1.7. Hyperventilation

Hyperventilation is an increase in ventilation beyond that driven by blood carbon dioxide levels, the normal driver for rate and depth of breathing. It is not “too much air” or “too much oxygen.” Respiratory alkalosis results when ventilation exceeds the metabolic need to eliminate CO_2, which can lead to changes in cellular respiration and blood acid-base balance. The increase in breathing rate and/or depth causes the carbon dioxide level in the blood to be reduced below normal. The resulting hypocapnia leads to cerebral vasoconstriction, and hence cerebral stagnant hypoxia, and can result in unconsciousness (Gibson, 1979). Figure 3.1.7-1 shows the change in cerebral blood flow for a given drop in arterial PCO_2. If unconsciousness occurs, the respiratory system will regain control of breathing by slowing ventilation until CO_2 levels return to normal.
Figure 3.1.7-1. Effect of Hypocapnia on Cerebral Blood Flow (modified from Gibson, 1979)

Hyperventilation can be caused by emotional or psychological stress (anxiety, fear, excitement), physical or physiological stress (pain, motion sickness), environmental stress (altitude, heat, vibration, positive pressure breathing), or by improperly sized or fit equipment (increased breathing resistance, restricted chest expansion, shallow breathing). Hyperventilation is a normal response to hypoxic hypoxia as the body attempts to increase the PAO₂ and therefore PAO₂, by reducing PACO₂ in accordance with the alveolar gas equation discussed earlier. This increase in ventilation can occur even at moderate altitudes, and may occur even if the pilot is using supplemental oxygen. Hyperventilation occurs more often among pilots than is generally recognized or acknowledge (Pilot’s Handbook, 2016). While it rarely incapacitates an individual, it can cause the on-set of symptoms that mimic many of the symptoms of hypoxic hypoxia and lead to alarm in an individual. Common signs and symptoms of are listed in Table 3.1.7-1.

Table 3.1.7-1. Symptoms of Hyperventilation

<table>
<thead>
<tr>
<th>Objective (Signs, Observable in others)</th>
<th>Subjective (Symptoms, Self-Observed)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Decreased reaction time</td>
<td>Headache</td>
</tr>
<tr>
<td>Impaired judgment</td>
<td>Visual impairment</td>
</tr>
<tr>
<td>Euphoria</td>
<td>Lightheaded or dizzy sensation</td>
</tr>
<tr>
<td>Pale, clammy appearance</td>
<td>Tingling in fingers and toes</td>
</tr>
<tr>
<td>Muscle spasms&lt;br&gt;^a</td>
<td>Numbness</td>
</tr>
<tr>
<td>Tetany&lt;br&gt;^a</td>
<td></td>
</tr>
<tr>
<td>Drowsiness&lt;br&gt;^a</td>
<td></td>
</tr>
</tbody>
</table>

^aCould also be self-observed.
Since many of the symptoms of hypoxia and hyperventilation are identical or similar, in-flight hyperventilation is difficult to diagnose from symptoms alone and typically has to be diagnosed by exclusion (Gibson, 1979). In addition, the on-set rate for symptoms due to hyperventilation is typically gradual and slower than that for hypoxia symptoms. Recovery is also slower due to the need for the amount of carbon dioxide to build back up in the blood and restore homeostasis. These characteristics can confuse diagnosis and potentially delay treatment. As such, aircrew should not attempt to diagnosis or differentiate between the two conditions, but rather follow their checklist procedures. Some differences in the manifestation of hypoxia and hyperventilation symptoms are shown in Table 3.1.7-2, but in an emergency or uncertain environment, time should not be wasted attempting to distinguish between them.

Table 3.1.7-2. Differences between Symptoms of Hypoxic Hypoxia and Hyperventilation

<table>
<thead>
<tr>
<th>Signs and Symptoms</th>
<th>Hypoxic (Altitude) Hypoxia</th>
<th>Hyperventilation&lt;sup&gt;b&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset of symptoms</td>
<td>Rapid (altitude dependent)</td>
<td>Gradual</td>
</tr>
<tr>
<td>Muscle activity</td>
<td>Flaccid, limp</td>
<td>Spasm</td>
</tr>
<tr>
<td>Appearance</td>
<td>Cyanosis</td>
<td>Pale, clammy</td>
</tr>
<tr>
<td>Tetany</td>
<td>Absent</td>
<td>Present</td>
</tr>
</tbody>
</table>

<sup>a</sup>From DeHart (1985).
<sup>b</sup>See discussion of hyperventilation in Section 1.2.10.

Although the usual causes and mechanisms of action are different, because hypoxia is a deadly condition while hyperventilation is self-limiting, treating the two can and should be achieved by following one procedure, discussed below in Section 3.1.8 and in Appendix 4. However, if hyperventilation occurs where there is reasonable assurance hypoxia is not involved, e.g., at sea level, control of rate and depth of breathing will treat the condition. In addition to slowing the breathing rate, breathing into a paper bag or talking aloud helps to overcome hyperventilation.

3.1.8. Treatment of Hypoxia and Hyperventilation

Hypoxia training in an altitude chamber or with a reduced-oxygen breathing device can teach an individual how to recognize signs and symptoms and initiate well-established recovery steps.

3.1.8.1. Aircraft Boldface Procedures. At the most elementary level, the treatment of hypoxia involves providing oxygen. Following the BOLD FACE procedures provides oxygen and restores normal breathing rate and depth, thus treating both hypoxia and hyperventilation simultaneously and without delay. Two procedures are shown, particular to the type of oxygen regulator used (aircrew should always follow the procedures specific to their aircraft):
1. **CRU-73/A and CRU-68/A Narrow Panel Regulators**: All 3 switches up (“gangload”)

   - **SUPPLY**: ON
   - **OXYGEN**: 100% OXYGEN
   - **EMERGENCY**

   **CRU-93/A and CRU-98/A Narrow Panel Regulators**: All 3 switches up

   - **SUPPLY**: ON/PBG
   - **OXYGEN**: 100% OXYGEN
   - **EMERGENCY**

**F-22 BRAG Valve Panel**

   - **OBOGS**: ON
   - **SUPPLY**: Normal
   - **MIXTURE**: Max (ensures maximum O₂ will be available to the crewmember’s supply line within seconds, assuming the system is operational and properly preflighted by the crewmember)

2. **MASK**: ON.

   It is natural to think that this step should be first. However, for those who do not regularly wear masks, especially if they do not have the quick-don type of emergency delivery, critical time is lost putting on the mask without any oxygen flowing through it. By taking the actions in Step 1, O₂ will already be flowing and available for breathing during the mask fit and attachment.

3. **Check Regulator and Connections.**

   Steps 2 and 3 ensure that max/100% oxygen will be available to the crewmember. This step detects an unconnected hose or malfunctioning regulator or mistakenly gangloading one regulator and hooking up to another.

4. **Control rate and depth of breathing.**

   This step is needed to prevent hyperventilation, especially with a pressurized source of breathing gas, and to eliminate the cause of preexisting hyperventilation (cure).

5. **Notify Aircraft Commander, Lead, or Other Flight Members.**

   Depending on the effectiveness of the previous steps and the speed of the decompression, notifying someone else is important in case the hypoxia gets worse and because the cause may have gone unnoticed by others, such as failure to pressurize on ascent or smoke and fumes.

6. **Descend to below 10,000 ft MSL.**
3.1.8.2. Treatment Anomalies. Providing aid to someone who is unconscious under likely hypoxic conditions dictates a similar course of action beginning with the administration of supplemental oxygen. Although providing oxygen to an extremely hypoxic individual is essential for recovery, symptoms may, at first, seem to get worse, with the individual feeling dizzy and nauseated, and even trying to remove an oxygen mask. In severe cases, breathing may cease entirely for many seconds after a deep breath of oxygen. These symptoms are called oxygen paradox and usually pass quickly.

3.1.8.2.1. Oxygen Paradox – General Characteristics. The cause of oxygen paradox is believed to be hypocapnia resulting from hyperventilation during hypoxia. The resulting drop in plasma CO\textsubscript{2} leads to 1) cerebral vasoconstriction, which further diminishes blood flow to the brain, and 2) plasma carbon dioxide pressure that is too low to drive the respiratory reflex. During hypoxia (P\textsubscript{a}O\textsubscript{2} < 60 mmHg), oxygen drives respiration (the hypoxic ventilatory response). The first breath of oxygen eliminates this drive, and many seconds pass before carbon dioxide builds up enough to again drive respiration. Oxygen administration also affects capillary flow control, leading to systemic vasodilation and transient reductions in blood pressure leading to dizziness and nausea.

3.1.8.2.2. Oxygen Paradox – Visual Symptoms. During the USAF hypoxia training profile, students participate in a night vision demonstration at 18,000 feet. During the demonstration, students look at the center of a color wheel (Figure 3.1.8.2.2-1) and note any changes in vision (i.e. decreased peripheral vision, difficulty distinguishing colors, changes in color brightness/intensity). After 5 minutes breathing ambient air, students correct by replacing their oxygen mask and breathing 100% oxygen. Immediately after going back on 100% oxygen, some students report experiencing either a 3D effect of the color wheel (i.e. the words or Zs jump out at them) or other strange visual effects (some state they completely lose color vision) for a couple of seconds.
When examining potential explanations for the above visual phenomenon, the most likely candidates for locations are the retinal and choroidal vasculature, and the cerebral cortex. The effect of altitude on intraocular pressure (IOP) and its subsequent potential to affect retinal blood flow are not well characterized, and therefore are not considered in this analysis. Auto-regulation of retinal blood flow has been shown to be mostly unchanged, even with moderate increases in IOP, so there is some validity in ignoring this parameter (Alm and Bill 1972). Additionally, there is an age dependent decrease in choroidal blood flow (Grunwald et al., 1998); however, this is likely not a variable that needs consideration here given the presumed subject demographics of most military aviators. Therefore, the discussion to follow will consider the changes in blood gases as the primary variables.

Generally speaking, the inner retinal layers of non-foveal retina are supplied directly by retinal vasculature, whereas the more metabolically active outer retina, where the photoreceptors are located, is supported primarily by the choroid. Retinal blood flow is estimated at about 40 – 53 µL/min (Wang et al., 2009), whereas choroidal blood flow has been estimated at 500 – 2,000 mL/min/100 g (Riva et al., 2011) (notice unit changes). This matches well with the metabolic demands of the supported tissues, since the outer retina has one of the highest metabolic rates seen in the human body. Also, the high choroidal blood flow has been hypothesized to play a critical role in thermoregulation of the outer retina as well. The “20/20” part of the eye, the foveola, has no retinal vasculature and is supplied solely by the underlying choroid.

Considering the oxygen paradox theory outlined above, we do know that retinal arterioles undergo marked vasoconstriction in healthy human subjects when the partial pressure of O\textsubscript{2} is increased after breathing 100% O\textsubscript{2}, leading to a substantial decrease in retinal blood flow (Jean-Louis et al., 2005). If this had an impact, it would presumably affect inner retina predominately (i.e. ganglion cells, etc.), not photoreceptors. Choroidal blood flow is not measurably impacted by hyperoxia (Geiser et al., 2000), which would indicate that photoreceptor metabolism and signaling is probably unaffected as well.

Choroidal thickness does decrease after hyperventilation and subsequent hypocapnia (Ozcimen et al., 2015), which may indicate reduced blood flow through the choroid. Hypocapnia has been shown to decrease average retinal blood flow as measured by Doppler frequency domain optical coherence tomography (FD-OCT) (Shahidi et al., 2014).

As a result of these effects, the hypocapnia condition induced by hyperventilation causes decreased choroidal blood flow, which would impact the ability of the photoreceptors to recycle their photopigment and fire electrical signals to the brain for visual processing. Combine this with the inner retinal blood vessel constriction and reduction of blood flow in response to both the hypocapnia and then the O\textsubscript{2} administration, a potential disruption in signal propagation, both between retinal layers and downstream from the retina, may exist. This would be transient and expected to resolve once the system is regains equilibrium.

As an aside, in the hypercapnia condition both retinal and choroidal blood flow is increased. Specifically for the retina, an increase of just 1mmHg in CO\textsubscript{2} partial pressure results in a 3% rise in retinal blood flow (Tsacopoulos and David 1973). The change in interstitial retinal pH influences the vasomotor response during hypercapnia (Tsacopoulos and Levy 1976), as well as the endothelial interaction between nitric oxide (NO) and prostaglandins affecting vascular tone. Lactate may also play a role in this function by mediating the release of these factors (Hein et al., 2006). 90% of the
glucose utilized by the retina is converted to lactate and 70% of \( O_2 \) consumption in the retina is due to the oxidation of glucose to \( CO_2 \) (Riva et al., 2011). Anaerobic production of lactate in the retina is 2 – 3 times higher than aerobic production (Winkler et al., 2004). Another factor that may contribute to the issue, but is not well characterized, is the transition from light to dark and vice versa, as there are reports that subfoveal choroidal bloodflow may decrease by 15% after a transition from light to darkness (Longo et al., 2000).

Given all of the above, while the retinal impact of hypoxia should not be minimized, the cortex remains the most likely candidate for the site of the visual system dysfunction. Light is transformed into electrical signals by photoreceptors in the retina, undergoes some level of modulation in the retina, and is then transported to the lateral geniculate nucleus (LGN) via ganglion cell axons, and then further to the primary visual cortex (V1), which in turn communicates with extrastriate visual cortex areas. These processes are highly specialized and organized, and specific areas of the brain have been mapped to specific visual functions. In this case, area V4 of the extrastriate cortex is primarily involved in the “what” (as opposed to the “where”) part of our visual system. V4 contributes to both color vision and stereopsis (Pasupathy, 2015), which seem to be the primary areas of vision affected. The lack of retinal signal modulation for stereopsis suggests that higher level visual processing is affected in that regard. If interruption of retinal photoreception was the culprit, it would be expected that there would also be a change in other visual phenomenon, such as perceived brightness and visual acuity in addition to color vision and stereopsis.

3.1.9. Recognition and Prevention of Hypoxia

Experiencing hypoxia with prior knowledge that it will happen allows trainees to recognize their individual symptoms and practice self-treatment to limit the severity of symptoms and prevent loss of consciousness. Since individual symptoms tend to be consistent on subsequent hypoxic episodes (Cable, 2003; Files et al., 2005), awareness can cue a crewmember to the possibility of hypoxia so the crew can take the appropriate emergency procedural actions (see Checklists in Appendix 5). Altitude chamber training provides an opportunity for crewmembers to recognize their own symptoms and practice corrective measures. The chamber training also allows crewmembers to observe objective signs of hypoxia in other trainees, further reinforcing their understanding of the dangers of hypoxia while in safe, controlled conditions.

Since the root cause of hypoxia is insufficient oxygen at some phase of respiration (ventilation, diffusion, transportation, or utilization), prevention involves ensuring all phases of respiration are functioning adequately. Hypoxic hypoxia is the most common form of hypoxia in aviation and is commonly avoided by adequate cabin/cockpit pressurization. Aircraft that do not have adequate pressurization for hypoxia prevention provide oxygen systems which, if properly supplied, maintained, checked, and operated, ensure that crewmembers have adequate oxygen at the alveolar level. Exceptions would be operation of aircraft at such high altitudes that the aircraft systems’ capabilities and/or aircrew personal equipment are overextended. Such results could occur if some newer generation fighters are allowed to be operated above 50,000 ft without personal protective equipment and aircrew training designed for this environment. New unpressurized tilt-rotor aircraft have the potential to allow seven crewmembers to breathe off a system designed for four.
Hypoxia training to ensure crewmembers are better capable of recognizing their own hypoxia symptoms, ensuring they have a working knowledge of the aircraft oxygen systems, and following the appropriate aircraft checklists and command guidance should prevent most hypoxia-related physiologic incidents. Aircrew should remember to always preflight oxygen equipment, both aircraft and personal, and perform before takeoff and climb/level off checks.

References


Use of Oxygen and Oxygen Equipment. T.O. No. 03-50-1. 1944.


**Recommended Readings**


**Internet Resources, See Appendix 8.**


**Concepts**

Time of Useful Consciousness

Symptoms of hypoxia

Treatment of hypoxia

**Vocabulary**

Acclimatization

Hyperventilation

Hypoxia

Oxygen paradox

Stages of Hypoxic Hypoxia
3.2. Oxygen Toxicity

James T. Webb, Ph.D.

As life forms that have evolved in an atmosphere containing less than about 35% oxygen for the past few million years, we had no need to maintain the biochemistry to handle higher levels of oxygen. Oxygen toxicity results from too much oxygen for too long and is related to the partial pressure of oxygen, not the oxygen percentage. Oxygen partial pressure and duration of exposure are variables that affect development of oxygen toxicity symptoms. After several hours of breathing 100% oxygen at sea level (760 mmHg partial pressure of oxygen), symptoms of substernal discomfort associated with oxygen toxicity will develop in most people. However, a National Aeronautics and Space Administration (NASA) study reported that breathing 100% oxygen at 11,500 ft (493 mmHg partial pressure of oxygen) did not result in any symptoms of oxygen toxicity (Webb et al., 1991). Therefore, breathing 100% oxygen above 12,000 ft (a partial pressure of oxygen less than 493 mmHg) is not likely to result in symptoms of oxygen toxicity (Figure 3.2-1). Aside from some discomfort associated with breathing dry aviator’s oxygen and fire hazards associated with use of 100% oxygen, acceleration atelectasis is the main concern (see Section 7.1).

![Figure 3.2-1. Human Time-Tolerances: Oxygen Partial Pressures (Space Handbook, 1959)](image)

Prevention of hypoxia involves increasing the partial pressure of oxygen in the breathing gas and, at altitudes above 30,000 ft, adding positive pressure breathing. Under these conditions, oxygen toxicity is not likely to occur because the partial pressure of oxygen does not exceed that of sea level air. During unpressurized flight below 10,000 ft and in pressurized cabins during flight below 35,000 ft, oxygen masks are not used unless required, and oxygen toxicity is not an issue. For those missions and type aircraft where oxygen masks are used under those conditions, the amount of
supplemental oxygen delivered is insufficient to result in oxygen toxicity. Therefore, oxygen toxicity should not be an issue in Air Force operations.

Some Air Force personnel have been crewmembers on NASA missions where 100% oxygen was used prior to launch at sea level pressure. No effects of oxygen toxicity were recorded for those several-hour exposures. After launch the partial pressure of oxygen was greatly reduced due to the reduction in total pressure to 5.0 psi (259 mmHg). Again, there were no reports of oxygen toxicity symptoms even during long, multiday exposures (Pool, 1998).

References

Recommended Readings
Internet Resources, See Appendix 8.
3.3. Trapped Gas

James T. Webb, Ph.D.

There I was, a guy-in-the-back (GIB pilot) of an F-4D coming back from the bombing range at 12,000 ft with no problems…until descent. I began to feel like one of my left upper teeth was about to explode. Clearing my ears did no good. If I’d had a pair of pliers, I think I would have tried to pull it out. The pain couldn’t have been worse. I told the front seat pilot who slowed the descent, but we were low enough on fuel that he had to come down. The pain was so bad I banged my head against the canopy with the thought that pain somewhere else might lessen its effect. No good. I even gave serious thought to ejecting, as if that would help, but didn’t reach for the handle. Good choice. After the precautionary landing and interview with the flight surgeon, the pain eased and I was sent to a specialist. I had the dreaded nasal polyps. They blocked a maxillary sinus, preventing equalization of pressure during descent to higher atmospheric pressure. The trapped gas had equalized at 12,000 ft, and during descent, the sinus cavity was compressed, putting pressure on a nerve innervating a tooth. Pulling the tooth wouldn’t have helped. Surgery did, and it never recurred.

3.3.1. Water Vapor and Gas Expansion

The mechanical effects of expansion and contraction of a trapped physiologic gas follow Boyle’s Law closely due to the relatively constant temperature of human tissue where gases are located. During decompression (ascent), trapped gases expand because a differential pressure develops as the external pressure decreases. Many trapped gases remain essentially constant in volume due to their structure, e.g., sinuses and the middle ear. Instead of responding by increasing volume, these trapped gases exert a differential pressure on surrounding tissues, which can cause severe, potentially disabling pain and potential physical damage to tissues.

The constant pressure (47 mmHg) of water vapor at body temperature plays an increasing role in gas expansion during ascent as the partial pressures of the other expanded gases decrease.

As a result, the constant water vapor pressure of 47 mmHg must be subtracted from the pressure inside body cavities. Thus, for wet gases, Boyle’s Law becomes:

\[(P_1 - 47) * V_1 = (P_2 - 47) * V_2\]

For example, water vapor makes up only 1% of the volume of a trapped gas bubble at 6 atm (65 ft of sea water (fsw)) and 6% at sea level but 33% of a trapped gas bubble at 40,000 ft. Due, in part, to the water vapor occupying more of the available pressure in alveoli, \(P_{A\text{O}_2}\) becomes extremely low above 45,000 ft. Even with 30 mmHg of additional pressure applied to the lungs via pressure breathing for altitude (PBA) at 50,000 ft and 60 mmHg of PBA at 60,000 ft, the \(P_{A\text{O}_2}\) is the same as breathing air above 18,000 ft. Breathing 100% oxygen with 70 mmHg of PBA at 60,000 ft will provide a \(P_{A\text{O}_2}\) equivalent to breathing air at about 15,000 ft, assuming no pressure loss due to mask leakage and sufficient training to allow proper control of breathing. PBA and its effects are discussed in the section on personal equipment effects.

At this writing, no pulse oximetry results have been obtained from humans using current oxygen equipment during exposure to 60,000 ft in an altitude chamber while breathing 100% oxygen with 70 mmHg of PBA (Appendix 1c).
At 63,000 ft, Armstrong’s Line, water boils at body temperature. The ambient pressure is 47 mmHg, the same as the partial pressure of water at body temperature. An unprotected human would experience vaporization of tissue water, or ebullism. (Ebullism and embolism are sometimes confused due to the similarity of the terms. Embolism results from respiratory air being forced into the circulation by an overpressure in the lungs.) The altitude or pressure at which ebullism occurs varies with the temperature and pressure in specific tissues. As an example, peripheral tissues are at a lower temperature than internal tissues, and embolism could occur at a lower pressure (higher altitude). Similarly, the higher blood pressure in the arterial system would result in ebullism at lower pressure (i.e., a higher altitude than 63,000 ft). Any artificial increase in pressure around the body lowers the potential for ebullism. A pressure suit increases pressure around the body. PBA keeps the lung at a higher pressure by delivering pressurized breathing gas but does not protect the rest of the body. Current equipment is inadequate to provide sufficient partial pressure of oxygen to tissues above 60,000 ft, even with assisted pressure breathing for altitude (APBA). APBA involves the use of a counter-pressure jerkin worn to allow 60 mmHg of pressure to be tolerated for more than a couple minutes.

3.3.1.1. Effects of Trapped Gases During Pressure Change. Since the volume of a sphere is a function of the cube of its radius, \( V = \frac{4}{3}\pi r^3 \), a relatively small change in diameter represents a large volume change. This is shown in Figures 3.3.1.1-1a and 3.3.1.1-1b). However, it is the pressure differential, not volume change, that results in most of the effects listed below.

![Figure 3.3.1.1-1a. Change in Volume and Diameter of a DRY Gas Sphere at Different Pressures, Relative to Sea Level](image-url)
Figure 3.3.1.1-1b. Change in Volume and Diameter of a WET Gas Sphere at Different Pressures, Relative to Sea Level

3.3.2. Physiological Effects of Trapped Gas

3.3.2.1. Expansion of Trapped Gastrointestinal (GI) Gas. There is always some gas in the large intestine, and during a GI illness, there may be gas in the small intestine, also. Intestinal gas is produced by resident bacteria in the gut. These bacteria are important in maintaining health and normal gut function, which is why antibiotic treatment often produces GI side effects. However, the bacteria produce gases including methane and sulfur dioxide as by-products. In accordance with the gas laws, bubbles of gas in the GI tract expand with altitude. Due to the location within the digestive system, expulsion of this gas may be difficult. This can cause discomfort, which may be severe. In extreme conditions, the gas expansion can lead to vasovagal (neurocardiogenic) syncope. Syncope occurs due to peripheral vasodilation (decreased sympathetic tone) leading to reduced cardiac filling and bradycardia (increased vagal tone) that result from a neural reflex following stimulation of the cardiac C fibers of the vagal nerve (Chen-Scarabelli and Scarabelli, 2004). There is a sudden drop in heart rate and blood pressure. The result is lack of blood flow to the brain and a loss of consciousness. For this reason, students with severe abdominal pain should not be allowed to stand in the altitude chamber without assistance. Treatment is to expel the gas, either through flatus or belching. If unable to effectively expel the gas, the chamber or aircraft must descend to a higher pressure. Prevention involves limiting intake of high-fiber foods before flights. This is in direct contradiction to general health advice to eat more fiber on a regular basis. Increasing fiber consumption for health benefits should be done slowly to allow the gut to adjust, and fiber intake should be limited for 6 hr prior to an unpressurized flight. Keep in mind that foods consumed in the previous 12 – 24 hours may also contribute to gas expansion in the intestinal tract. Gas trapped in the stomach will also expand, but tends to be more easily expelled (by belching) since there is a more immediate opening to the outside environment. The most common cause for stomach gas is intake of gas in the form of carbonated (soda pop) or aerated (milk shake) beverages.
3.3.2.2. Ear Block (barotitis media). An ear block may be defined as an acute or chronic traumatic inflammation of the middle ear produced by a pressure differential (either positive or negative) between the air in the tympanic cavity and contiguous air spaces and that of the surrounding atmosphere (Figure 3.3.2.2-1). To equalize the pressures during descent where a pressure differential typically develops, an aircrew member must physically do something to aid equalization across the tympanic membrane (the ear drum). The Valsalva maneuver increases the nasopharyngeal pressure against a closed Eustachian orifice to force air into the middle ear. To perform the Valsalva maneuver, pinch your nose and blow while your mouth is closed. You should be able to feel your ears “pop” as the increased pressure forces open the Eustachian tube. Too frequent or forceful Valsalvas can overpressure the middle ear and also lead to a feeling of fullness and/or pain. Performing the Valsalva maneuver with a cold or severe allergies can also force bacteria into the middle ear and lead to an ear infection. The Frenzel maneuver may also be performed by thrusting the jaw forward to open the Eustachian tubes, thereby providing a path for equalization of pressure. Both of these maneuvers introduce air to the middle ear.

![Figure 3.3.2.2-1. Anatomy of the Ear](image)

An increase in pressure within the middle ear can put pressure on the balance and orientation organs of the inner ear. If there is uneven pressure between the left and right sides of the head, a condition known as alternobaric vertigo can occur.

The Valsalva or Frenzel maneuvers may also be used while breathing air after breathing 100% oxygen to avoid pain from delayed ear block. They do so by introducing air containing 78% nitrogen that is not absorbed by surrounding tissues. The differential pressure involved in delayed ear block is middle ear atelectasis and is caused by absorption and utilization of 100% oxygen by the surrounding tissue. Atmospheric nitrogen is not utilized by body tissues, and introducing it to the middle ear reduces the differential pressure between the middle ear and ambient pressure.

Delayed ear blocks sometimes occur after breathing enriched or 100% oxygen. The gas trapped in the middle ear may be very high in oxygen, which is slowly absorbed by the surrounding tissue. This results in negative pressure in the middle ear compared with the external ear. The nitrogen portion of the ambient gas introduced by the Valsalva or Frenzel maneuvers is inert, and if equalization is performed several times.
during the hours after flight the composition of gases in the middle ear will be returned to normal.

The Valsalva and Frenzel maneuvers are only effective if the Eustachian tubes are not blocked. A cold or upper respiratory infection (URI) causes secretion of mucous and swelling of the tissues surrounding the Eustachian tubes, including the adenoids at the oronasal opening of the Eustachian tubes (Figure 3.3.2.2-2). The mucous and inflamed tissues can block the Eustachian tubes and prevent their normal function of equalization between the atmosphere and the middle ear, hence the recommendation to avoid flying with a cold.

Figure 3.3.2.2-2. Adenoids

While middle ear block is the most common type, it is possible to get an external ear block. This occurs when a tight-fitting ear plug without a vent is worn through pressure changes. Such ear plugs will always have a vent if they are to be used in flight, but the vent may become blocked with ear wax or other debris. Expanding gas will push past the plug on ascent without difficulty, but on descent the contracting gas will pull the plug in deeper into the external ear canal and will result in a tight seal with a negative pressure on the external surface of the tympanic membrane. It is important to distinguish this from middle ear block because the Valsalva maneuver is the wrong treatment. The Valsalva maneuver results in increased pressure in the middle ear, which in the case of the external ear block increases the pressure differential across the tympanic membrane and can cause its rupture. The Frenzel maneuver works for both middle and external ear block and should be stressed as the best way to treat ear block for those wearing tight-fitting ear plugs.

Although rare in aviation, another possible injury associated with the middle ear is tympanic rupture. The tympanic membrane may rupture when the pressure difference between the middle ear and the ambient environment exceeds approximately 260 mmHg (roughly 5 psi) (Stewart, 2006). Nearly all tympanic membranes will rupture with the pressure difference exceeds 750 mmHg (14 psi).

3.3.2.3. Sinus block (barosinusitis). A sinus block is an acute or chronic inflammation of one or more of the nasal accessory sinuses produced by a pressure difference (usually negative) between the air in a sinus cavity and the surrounding atmosphere. Sinus cavities include the maxillary (cheekbones), frontal and
sphenoid (forehead), and mastoid (the bony projection behind the ear) (Figure 3.3.2.3-1).

**Figure 3.3.2.3-1. Sinuses**

Pressure is usually equalized through drainage passages, but a cold or URI may cause swollen mucous membranes and/or secretion of mucous, blocking the small drainage passages and preventing pressure equilibration. The condition is characterized by pain in the affected region; this pain can develop suddenly and be so severe that the individual will be incapacitated. Treatment in flight is limited to the Valsalva maneuver discussed under ear block and the use of short-acting antihistamine nasal spray. Antihistamine/vasoconstrictor nasal sprays are considered an emergency measure and should never be used preventively, as these drugs cause a rapid buildup of tolerance resulting in swollen mucous membranes, leading to greater, rather than lesser, risk of sinus block. If used, you must be able to taste it in the back of your throat to ensure that it has fully reached the sinus openings and the openings to the Eustachian tubes. A sinus block may also result in referred tooth pain during descent due to pressure on nerves leading through sinuses to the teeth (Figure 3.3.2.3-2).

**Figure 3.3.2.3-2. Neural Involvement in Referred Tooth Pain**
3.3.2.4. **Tooth pain (barodontalgia).** Tooth pain can occur due to trapped gas exerting positive or negative pressure on surrounding tissue. Rarely, a bubble can be seen on x-ray in the vicinity of an affected tooth, but more often there is no obvious source of pressure. It is believed that microbubbles trapped in small fractures or under a recent filling may be enough to exert pressure on the nerve in the tooth pulp. Tooth pain can also be caused by maxillary polyps that block sinus equalization, resulting in sinus block that can exert pressure on a nerve leading to a tooth (Figure 3.3.2.3-2).

3.3.2.5. **Lung over-inflation (pulmonary barotrauma).** Lung overinflation due to breath-holding or inadequate equalization of pressure during decompression, can result in serious problems, such as pulmonary embolism (air in arterial circulation), pneumothorax (air in pleural cavity), or pneumomediastinum (air in the mediastinum). In animal studies, intrapulmonary pressure differentials of 80 – 100 mmHg (1.5 – 1.9 psi) occurring in 0.1 – 0.2 seconds has lead to alveolar rupture (Clark, 2008). Pulmonary overpressure may occur if a rapid decompression happens during breath-holding, swallowing, or yawning. Fortunately, the chances of the glottis being closed, and maintained in such a state, during a rapid decompression is extremely rare (Luft and Bancroft, 1956).

3.3.3. **Treatment**

When treating gas expansion problems, it is important to keep in mind the direction of travel. In general, the Valsalva maneuver should never be performed during ascent, as it adds gas to the cavities that are trying to expel it. Below are general guidelines for treating gas expansion problems during flight:

3.3.3.1. **Ear and Sinus Pain**

- Pain on **descent**
  - Level off and try a Valsalva
  - Climb to relieve pressure
  - Decrease descent rate
  - Consider using Afrin® or other vasoconstrictor (approved for emergency get-me-down use only—must report to flight surgeon
  - Declare an IFE (in-flight emergency)?
  - Land as soon as practical

- Pain on **ascent**
  - Do not Valsalva
  - Descend and land as soon as practical

3.3.3.2. **Tooth Pain**

- Descend and see a flight surgeon/dentist
3.3.3.3. Gastrointestinal Tract Pain

- Release the gas (belch or pass flatus) and/or descend
- Massage lower abdomen using the heal of your hand from the upper right to lower left (helps moves bubbles along the large intestine towards the colon)

References

Concepts
- Expansion of trapped gastrointestinal (GI) gas
  - Armstrong’s Line
  - Delayed ear block

Vocabulary
- Eustachian tubes
- Valsalva maneuver
- Sinus block
- Pulmonary overexpansion
- Barodontalgia, tooth pain
- Ebullism
3.4. Altitude Decompression Sickness

James T. Webb, Ph.D.

1670 Sir Robert Boyle published his findings concerning bubbles he observed in animal tissue resulting from decompressions of animals using a pneumatic pump (Davis, 2008).

1672 Boyle describes the escape of bubbles in liquids during decompression as causing alteration in circulation (symptoms) (Bert, 1878).

1906 H. von Schrötter describes symptoms experienced in a steel chamber after ascending to 8,994 m (29,500 ft) in 15 minutes. The symptoms closely resembled those occurring when caisson workers decompressed to surface pressure (caisson disease) (von Schrötter, 1906).

1908 J.S. Haldane's seminal paper on decompression sickness prevention during caisson work (Boycott et al., 1908).

1917 First clear reference to altitude DCS in the literature where Henderson described a detailed theory in which he postulated that it would be possible to get decompression sickness from altitude exposure (Henderson, 1917).

1931 A description of pain in the knees experienced in a hypobaric chamber while doing a step exercise at 9,160 m (30,000 ft; 233 mmHg) was most likely a manifestation of DCS, unrecognized as such at the time (Barcroft et al., 1931).

1938 Boothby and Lovelace reported a case of transient paraplegia in a fellow physiologist (Dr. J.W. Heim) during an ascent to 10,670 m (35,000 ft) while on oxygen; the paraplegia disappeared upon repressurization to ground level. This case illustrated the potential for serious neurological DCS at altitude (Boothby and Lovelace, 1938).

ca1939 Armstrong researched the effects of decreased barometric pressure on the aviator and described bubble formation that he experienced himself while at altitude in the hypobaric chamber (Engle and Lott, 1979): “I wound up with the classical symptoms. My hands began to feel stiff and slightly sore and I massaged them, trying to improve the circulation. Then I noticed a series of small bubbles in the tendons of my fingers. I could actually feel them, and by manipulating my finger along my tendon I could squirt these bubbles back and forth. I was certain in my own mind they represented aeroembolism, but it was not positive proof.”

1947 Behnke postulated the existence of “silent bubbles,” which are present in tissues and blood, do not cause symptoms (Behnke, 1947).

1959 The School of Aviation Medicine (which had just moved from Randolph Air Force Base (AFB) to Brooks AFB) became part of the USAF Aerospace Medical Center.

1963 On 21 Nov 1963, President John F. Kennedy visited Brooks AFB to dedicate a new complex of buildings added to the USAF School of Aerospace Medicine (USAFSAM), including an altitude research chamber where the photograph below was taken (Figure 3.4-1). He was assassinated less than 24 hr later (Stepanek and Webb, 2008).
Merrill Spencer publishes a method to grade intravascular bubbles, venous gas emboli (VGE) heard during ultrasonic monitoring of the heart following decompression (Spencer, 1976).

Labatory function of USAFSAM incorporated into the Armstrong Laboratory at Brooks to become one of the four "super labs" of the Air Force Systems Command (AFSC) on 13 Dec 1970 (AFSC Historical Publication).

The Air Force Research Laboratory, which was created to incorporate all AF laboratories, including the Armstrong Laboratory, in a single AF laboratory, was formally activated on 22 Oct 1997 (AFSC Historical Publication).

Cancellation of high-altitude research in the USAF facilities at Brooks City-Base.

Decompression sickness (DCS) was referred to in the 1940s and earlier as the bends, dysbarism, caisson disease, compressed-air illness, staggers, etc. Altitude decompression sickness refers to that malady occurring during some aviation and space endeavors in which gas bubbles are formed during decompression from surface or vessel pressure to a subatmospheric pressure. Bubbles can form in fluids at low levels of supersaturation if forces act to pull objects apart that are in close proximity, a process called tribonucleation (Ikels, 1970).

DCS can result when pressure is reduced on body fluids saturated with inert gas. At ground level, tissues and blood are always saturated with nitrogen, an inert gas. Since nitrogen gas is not metabolized by the body tissues (inert), its concentration in the tissues is only a function of its partial pressure in the breathing gas and its solubility in the body tissues. When the tissues become supersaturated with nitrogen during decompression to altitude, the nitrogen may not diffuse into the capillaries and be exhaled before it forms bubbles. This process is analogous to the bubbles formed when a carbonated beverage is opened, resulting in a pressure reduction that occurs faster than the carbon dioxide can diffuse out of the fluid, also forming bubbles. The bubbles of nitrogen (containing some carbon dioxide, oxygen, and water vapor) can interact with
the surrounding tissue and blood by exerting local pressure. The pressure can slow or block blood flow and stimulate responses by sensory nerves. Bubbles in venous circulation are usually cleared effectively in the lungs. Rarely, bubbles pass through the lungs, entering the arterial circulation, and can cause serious symptoms as arterial gas emboli.

Extravascular bubbles, tissue bubbles, can cause symptoms. The existence of bubbles in and of itself is not DCS; DCS refers to the maladies caused by the bubbles. DCS symptoms are highly diverse.

3.4.1. Symptoms of DCS

The symptoms of DCS can cause distraction and may interfere with optimal function. They can also be more serious, involving respiratory or neurologic function, resulting in severe loss of performance, abort of a flight mission, and requirement for hyperbaric oxygen therapy (HBO or HBOT) to achieve resolution. There are four categories of DCS symptoms:

- **Limb Pain.** Typically joint or muscle pain (70% - 84% of all altitude DCS symptoms) (Balldin et al., 2002, 2004; Ryles et al., 1996); most common DCS symptom.

- **Skin.** Mottling, pins and needles, tingling, prickling (about 13% of all altitude DCS symptoms) (Ryles et al., 1996); second most common DCS symptom.

- **Neurologic.** Cold sweat, dizziness, edema, inappropriate or sudden onset of fatigue, headache, light headedness, loss of consciousness, motor and/or sensory loss, nausea, tremor (shakes), vertigo (1% - 8% of all altitude DCS symptoms) (Balldin et al., 2004; Ryles et al., 1996; Clark, 1992).

- **Respiratory (pulmonary).** Cough, dyspnea (difficult or labored breathing), substernal distress (tightness and/or pain in chest, especially during inspiration) (about 3% of all altitude DCS symptoms) (Balldin et al., 2002; Ryles et al., 1996).

DCS symptoms have been categorized as Type I, pain-only symptoms, and Type II, serious symptoms, since introduction of the nomenclature in 1960 by Golding et al. (1960). This categorization was created to separate the symptoms into groups based on their response to treatment. The symptoms developed during or after decompression from caisson work on the Dartford Tunnel in England. This system was somewhat analogous to the four-table treatment scenarios used at that time by the U.S. Navy as described by Donnell and Norton (1960). The four hyperbaric treatment tables (I – IV; I-A and II-A without oxygen available) involved treating increasing symptom severity with more aggressive hyperbaric profiles. The dichotomous Type I/II separation of symptoms does not provide sufficient information for U.S. Air Force physicians to adequately diagnose altitude DCS and prescribe treatment, although it is still in common use for describing diving DCS (Moon and Sheffield, 1997). The U.S. Navy uses the Type I and Type II categories of DCS for the Master Diver to determine treatment of diving DCS. The Master Diver provides HBO treatment as needed for USN divers in the absence of a physician, creating a need for clear guidelines regarding treatment. USAF physicians determine treatment for altitude DCS based on the

Each symptom should be stated with the time interval from its onset to the commencement of treatment. “Progressive, limb-pain DCS occurring 1 hr prior to commencement of treatment” would provide essential information to a USAF flight surgeon for use in determining treatment, unlike “Type I DCS.” The timeline of any relapsing or progressive symptoms should be further described to include an indication of change and level of intensity. In addition, the response to recompression should be indicated (complete recovery, incomplete recovery, or none) to guide any further treatment options.

3.4.1.1. **Symptom Evolution.** A report from research chamber studies using well-documented symptom onset and resolution information (Muehlberger et al., 2004) discussed DCS resolution in 1,096 cases of DCS observed during research exposures at Brooks. Of that group, 76 cases (6.9%) were treated with HBO to resolve symptoms or as a precautionary measure. The remaining 1,020 cases were not treated with HBO and completely resolved before arrival at ground level or before treatment was deemed necessary. All but one of the 15 cases involving precautionary treatment also involved HBO treatment to resolve another symptom. This indicates that most altitude DCS symptoms resolve during descent and do not require any additional treatment.

3.4.1.2. **Altitude vs. Diving DCS.** Due to frequent misconceptions regarding the similarities and differences between altitude and diving DCS, Table 3.4.1.2-1 is provided to clarify the major differences between the two environmental hazards (Pilmanis et al., 2004).
Table 3.4.1.2-1. Major Differences between Diving and Altitude Decompression Sickness

<table>
<thead>
<tr>
<th>Altitude DCS</th>
<th>Diving DCS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Decompression starts from a ground level tissue $N_2$ saturated state.</td>
<td>1. Upward excursions from saturation diving are rare.</td>
</tr>
<tr>
<td>2. Breathing gas is usually high in $O_2$ to prevent hypoxia and promote</td>
<td>2. Breathing gas mixtures are usually high in inert gas due to oxygen toxicity concerns.</td>
</tr>
<tr>
<td>denitrogenation</td>
<td></td>
</tr>
<tr>
<td>3. The time of decompressed exposure to altitude is limited.</td>
<td>3. The time at surface pressure following decompression is not limited.</td>
</tr>
<tr>
<td>4. Pre-mission denitrogenation (preoxygenation) reduces DCS risk.</td>
<td>4. The concept of preoxygenation is generally not applicable.</td>
</tr>
<tr>
<td>5. DCS usually occurs during the mission.</td>
<td>5. DCS risk is usually greatest after mission completion.</td>
</tr>
<tr>
<td>6. Symptoms are usually mild and limited to joint pain.</td>
<td>6. Neurological symptoms are common.</td>
</tr>
<tr>
<td>7. Recompression to ground level is therapeutic and universal.</td>
<td>7. Therapeutic chamber recompression is time limited and sometimes hazardous.</td>
</tr>
<tr>
<td>8. Tissue $P_{N_2}$ decreases with altitude exposure even while breathing air.</td>
<td>8. Tissue $P_{N_2}$ increases with hyperbaric exposure while breathing air.</td>
</tr>
<tr>
<td>9. Metabolic gases become progressively more important as altitude increases.</td>
<td>9. $N_2$ dominates.</td>
</tr>
<tr>
<td>10. There are very few documented chronic sequelae.</td>
<td>10. Chronic bone necrosis and neurological damage have been documented.</td>
</tr>
</tbody>
</table>

3.4.2. Treatment of DCS

Since many physicians have no training in recognition or treatment of altitude DCS, it is important for aircrew to be aware of the symptoms of DCS and the need to seek medical attention from informed personnel. Altitude DCS is typically resolved during descent to a lower altitude while breathing 100% oxygen in accordance with current USAF directives. Continued breathing of 100% oxygen on the ground for 2 hr is usually effective treatment for mild cases of DCS that do not resolve completely during descent (Krause et al., 2000). The reason for resolution of symptoms with this procedure is twofold: 1) the gas emboli (bubbles) are subjected to increased pressure during descent, which will reduce their size and effect (Muehlberger et al., 2004); and 2) breathing 100% oxygen partially denitrogenates blood and tissues. This reduces the potential for bubble growth and results in shrinkage of existing bubbles in tissues adjacent to capillaries, where the diffusion gradient will favor nitrogen leaving the tissue and entering the denitrogenated blood.
3.4.3. Hyperbaric Oxygen Therapy

The following are the USAF procedures if DCS is suspected:

- 100% oxygen
- Descend as soon as practical
- Declare an in-flight emergency (IFE)
- Land at the nearest airfield with qualified medical assistance (military flight surgeon or civil aeromedical physician) available

Hyperbaric oxygen therapy is the standard of DCS care, and it is successful in treating DCS symptoms that do not resolve before landing or which involve neurologic or pulmonary (respiratory) symptoms. The additional pressure of the hyperbaric treatment further reduces the size of existing bubbles. Breathing of 100% oxygen during the HBO treatment ensures no further nitrogen is delivered to the tissues and helps to oxygenate tissues where bubbles may have blocked delivery of oxygenated blood. HBO treatment of DCS, whether from altitude or hyperbaric exposures, has been documented to be more successful if begun as soon as practical after symptoms appear. When symptoms are reported later, treatment is not as effective. The nature and severity of the symptoms dictate the specific hyperbaric profile for treatment and may require multiple treatments for complete resolution. The USAF School of Aerospace Medicine’s Hyperbaric Medicine Division (USAFSAM/FEH) serves as the primary source of information and consultation on treatment of DCS for the USAF.

Treatment of USAF altitude chamber reactors is guided by USAFSAM/FEH directives based on time since treatment and the symptoms at time of treatment. Some treatment scenarios involve ground level oxygen and the rest utilize hyperbaric oxygen therapy. HBO treatment scenarios include modified USN Treatment Table 5 and 6 profiles to 60 fsw breathing 100% oxygen with air breaks to avoid oxygen toxicity. The profiles last from 135 to 285 minutes not including descent time. Treatment Table 6A to 165 fsw for 319 minutes is employed for treating air embolism and only rarely for DCS cases that do not resolve with Treatment Tables 5 or 6. At 165 fsw, air or a nitrox mix is used to prevent oxygen toxicity. Due to the renitrogenation that takes place at 165 fsw and the relatively small reduction in bubble size with the extra pressure (see Figure 3.3.1.1-1b), Table 6A is generally not considered a good choice for treatment of DCS. The algorithm to assist in treatment shown in Table 3.4.3-1 was developed at the USAFSAM Hyperbaric Medicine Division.
Table 3.4.3-1. Management of Altitude Decompression Sickness

<table>
<thead>
<tr>
<th>Carefully assess DCS symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Joint pain or skin symptoms only; A-D; not, 2.</td>
</tr>
<tr>
<td>A. Symptoms present in less than 2 hr?</td>
</tr>
<tr>
<td>Surface level oxygen</td>
</tr>
<tr>
<td>Worsen or fail to improve?</td>
</tr>
<tr>
<td>Treatment Table 5</td>
</tr>
<tr>
<td>Worsen or fail to improve?</td>
</tr>
<tr>
<td>Treatment Table 6</td>
</tr>
<tr>
<td>Worsen or fail to improve?</td>
</tr>
<tr>
<td>Consider extensions or Treatment Table 6A and tailing dives until resolve or symptoms plateau</td>
</tr>
<tr>
<td>B. Symptoms present in 2 to 6 hr?</td>
</tr>
<tr>
<td>Treatment Table 5</td>
</tr>
<tr>
<td>Worsen or fail to improve?</td>
</tr>
<tr>
<td>Treatment Table 6</td>
</tr>
<tr>
<td>Worsen or fail to improve?</td>
</tr>
<tr>
<td>Consider extensions or Treatment Table 6A and tailing dives until resolve or symptoms plateau</td>
</tr>
<tr>
<td>C. Symptoms present &gt; 6 hr?</td>
</tr>
<tr>
<td>Treatment Table 6</td>
</tr>
<tr>
<td>Worsen or fail to improve?</td>
</tr>
<tr>
<td>Consider extensions or Treatment Table 6A and tailing dives until resolve or symptoms plateau</td>
</tr>
<tr>
<td>D. Symptoms present &gt; 36 hr?</td>
</tr>
<tr>
<td>Reconsider diagnosis of DCS</td>
</tr>
</tbody>
</table>

2. Neurologic, pulmonary, or cardiac symptoms

<table>
<thead>
<tr>
<th>Treatment Table 6</th>
</tr>
</thead>
<tbody>
<tr>
<td>Worsen or fail to improve?</td>
</tr>
<tr>
<td>Consider extensions or Treatment Table 6A and tailing dives until resolve or symptoms plateau</td>
</tr>
</tbody>
</table>

Of the nearly 1,000 cases of DCS observed in subjects during 20 yr of altitude DCS research chamber activity at Brooks AFB (City-Base), 89 subjects were treated with HBO by the Hyperbaric Medicine Division, all with complete resolution of symptoms. The remaining cases were successfully treated with 2 hr of ground level oxygen (Krause et al., 2000) or with no treatment.

Disposition of aircrew who develop DCS is described fully in Air Force Instruction (AFI) 48-123v3. The large majority of cases are treated successfully and returned to flying status. Grounding for 72 hr after any treatment is standard, although any residual symptoms receive further evaluation and potential waiver or permanent grounding.
3.4.4. Factors Affecting Incidence of DCS

The factors that affect DCS incidence are extensive and involve both the environment and the individual exposed. The four primary factors that affect DCS incidence are altitude, time at altitude, level of activity, and prebreathe time.

3.4.4.1. Altitude. The higher the altitude, the higher the incidence of DCS once the threshold altitude is achieved. The threshold altitude for 5% DCS during 4-hr zero-prebreathe exposures with mild exercise was shown to be a little under 20,000 ft (see Webb et al., 2003; Pilmanis et al., 2003). Above 20,000 ft, the incidence of DCS increases exponentially to 80% by 25,000 ft. Research (Webb et al., 2003; Haske et al., 2002) has indicated that exposures above 20,000 ft without prebreathe would involve significant DCS risk if not limited in duration. DCS has occurred below 20,000 ft, although the incidence is so low as to be operationally insignificant (Webb et al., 2003).

Figure 3.4.4.1-1 shows results of 113 male and 80 female 4-hr exposures with mild, upper-body, ambulatory exercise (Webb et al., 2003). Each subject was exposed one time, allowing a probit curve to show the relationship between altitude and incidence (Webb et al., 2003). DCS occurs below 20,000 ft, although the incidence is so low as to be operationally insignificant with the probit curve showing less than 1.03% DCS at 18,000 ft and less than 0.01% DCS at 14,400 ft. The incidence of venous gas emboli is also shown as a corollary of exposure severity versus altitude, although VGE detection as a predictor of DCS is poor. The sigmoidal relationship between exposure altitude and DCS incidence (Figs. 3.4.4.1-1 and 3.4.4.1-2) illustrates why exposures to 35,000 and 40,000 ft with up to 90 minutes of prebreathe do not result in 100% DCS (Webb et al., 2001; Pilmanis et al., 2003). Although much higher onset rates of DCS occurrence were observed at successively higher exposure altitudes up to 25,000 ft (Figure 3.4.4.1-1; Webb et al., 2003), no DCS was observed within 15 minutes. The onset curves did indicate a higher rate of symptom onset with increasing altitude.

![Figure 3.4.4.1-1. VGE and DCS During 4-hr, Zero-Prebreathe Exposures of 193 Male (113) and Female (80) Subjects Performing Mild Exercise and Breathing 100% Oxygen During Exposure](image-url)
3.4.4.2. Time at Altitude. The longer the exposure time, the higher the incidence of DCS. However, there is a lag time before symptom onset that makes very brief exposures relatively safe, e.g., rapid decompression followed by immediate descent on 100% oxygen (Webb and Pilmanis, 1995), as would occur in a rapid decompression in which aircrew successfully follow their emergency procedures.

Risk factors of both altitude and time at altitude (other factors kept constant) reveal a sigmoidal relationship with DCS incidence when plotted as in Figure 3.4.4.1-2.

3.4.4.3. Level of Activity. Level of activity has a significant effect on DCS risk depending when it is done (before, during, or after exposure to altitude) and what level of activity is performed.

3.4.4.3.1. Before exposure. Pre-exposure exercise can have a beneficial effect if accomplished during prebreathe (see Prebreathe – Exercise During Prebreathe below).

3.4.4.3.2. During exposure. Higher levels of activity during exposure to reduced pressure result in higher levels of DCS and can be a very significant factor in overall incidence (Gray and Masland, 1946; Pilmanis et al., 1999; Webb and Pilmanis, 1995; Webb et al., 2001, 2010aandb). This does not appear to be related to the type of activity (isometric vs. dynamic, arm vs. leg) (Pilmanis et al., 1999) or whether walking is part of the activity (Webb et al., 2005). Also, lower levels of activity result in onset curves, which may not indicate the DCS risk levels even after 4 hours of exposure as shown in Figure 3.4.4.3-1. The total incidence of DCS while decompressed appears to be related to the oxygen consumption (metabolic rate) during the highest 1 minute of activity repeated during an exposure as shown in Figure 3.4.4.3-2 (Webb et al., 2010a).

3.4.4.3.3. After exposure. Exercise following a decompression exposure was shown to involve no more risk than resting after exposure (Webb et al., 2002b). However, exercise-induced pain may be misdiagnosed as DCS pain, or vice versa in the case of latent/delayed DCS. It is therefore advised to avoid strenuous exercise for 12 hours after altitude chamber training.

Figure 3.4.4.1-2. Sigmoidal Relationship of Increasing Risk Factor with Increased DCS Risk (Webb and Pilmanis, 1995)
Figure 3.4.4.3-1. Cumulative DCS Incidence in Altitude DCS Research Exposures to 30,000 ft for 4 hours Following 1 hour of Resting Prebreathe While Performing Different Activities

Figure 3.4.4.3-2. Relationship of Oxygen Consumption to DCS Incidence With All Other Conditions Constant (29,500 – 30,000 ft; 4-hr exposure; 1-hr prebreathe)
3.4.4.4. **Prebreathe or Denitrogenation.** Reduction in the level of nitrogen dissolved in body fluids and tissues reduces the potential for supersaturation, bubble formation, and resulting symptoms of DCS. Nitrogen can be eliminated from the body by breathing 100% oxygen before decompression, a process variously called prebreathe, preoxygenation, or denitrogenation. This is a method of reducing DCS risk and is common practice when other methods are not practical. Prebreathe works because there is more nitrogen dissolved in the blood flowing into the lung capillaries than there is in the adjacent alveoli. The alveoli, when breathing 100% oxygen, contain no nitrogen, allowing diffusion of nitrogen from the capillaries into the alveoli where it is exhaled. For several current operational mission scenarios it is required (Air Combat Command Instruction (ACCI) 11-459; AFI 11-409; Army Regulation (AR) 95-1; Field Manual (FM) 3-04.301; Internet Resources, see Appendix 8).

3.4.4.4.1. **Prebreathe time.** Longer prebreathe, or denitrogenation time, results in less DCS, although each additional hour of prebreathe produces less protection than the hour before it (Waligora et al., 1987; Webb et al., 2002a). Generally, greater DCS risk is mitigated by longer prebreathe, but the relationship is not linear (Figure 3.4.4.4.1-1). Although effective, the time consumed while prebreathe works also becomes an operational factor due to crew duty day limitations and thermal considerations depending upon conditions where the prebreathe occurs. During U-2 operations, a pressure suit is required because loss of pressurization at operational altitudes without such protection would not allow successful recovery. The pressure suits are movement restrictive and are not normally pressurized during flight. Since the U-2 pressurization system is inadequate to provide protection from DCS at operational altitudes without pressurization of the suit, there is a requirement for at least 1 hour of prebreathe. Some individuals require more prebreathe time to avoid serious symptoms, limiting the crew duty day remaining and potentially limiting their operational effectiveness (Bendrick et al., 1996). The plot of preoxygenation time versus DCS risk is exponential (Figure 3.4.4.4.1-1), indicating that each additional period of prebreathe is less effective than the previous period. One study showed that complete protection from DCS at 30,000 ft was only achieved after 8 hours of prebreathe (Waligora et al., 1987).

![Figure 3.4.4.4.1-1. Exponential Relationship of Reduced DCS Risk with Increasing Prebreathe Time (Webb and Pilmanis, 1995)]
3.4.4.4.2. In-flight denitrogenation. In lieu of breathing 100% oxygen prior to takeoff, breathing enriched oxygen while airborne at a cabin altitude below which DCS is a concern, staged decompression, can provide efficient denitrogenation (Webb et al., 2000). The practical upper altitude limit for efficient use of this method appears to be 16,000 ft. Merely living at an altitude well above sea level provides some protection because the partial pressure of N₂ in the body is reduced. Adler (1964) summarized Haldane’s (1908) work on staged-ascent denitrogenation by stating that greater denitrogenation efficiency occurs during ascent as a result of increasing the pressure gradient of nitrogen from inside to outside the body. Testing for susceptibility to DCS in chambers situated at 4,700 ft resulted in only one rejection in 28 trainees exposed on multiple occasions. The 4% rejection rate was far below the 35% DCS incidence reported using chambers situated near sea level (Cheetham, 1947). Clark et al. (1960) discussed denitrogenation by living at an altitude of 10,000 ft (525 mmHg) or after 2 days at 14,160 ft. Balke (1959) stated that residence at 14,160 ft for 2 days followed by decompression to 38,000 ft resulted in [Grade 2 DCS joint pain] slight, easily tolerable pains that disappeared toward the end of the 1-hr test involving five deep knee bends performed at regular intervals. “The protective effect of the partial denitrogenation at an altitude of 14,000 feet was also confirmed in experiments in which the subjects were exposed to altitudes between 42,000 to 56,000 feet for a total time of 30 to 40 minutes.” “There were no symptoms of decompression sickness.” “Comparative experiments at sea level had shown that 4 to 6 hours of breathing 100% oxygen did not offer complete protection against decompression sickness.” A staged-decompression has been used since early 2007 on the International Space Station (ISS). Another staged-decompression procedure has been proposed for use during Moon and Mars exploration using the crew exploration vehicle and habitat at the stage pressure (Lange et al., 2005).

3.4.4.4.3. Exercise during prebreathe. Making a given prebreathe duration more effective can be accomplished by increasing heart rate, stroke volume, and ventilation rate by exercise of all major muscle groups during prebreathe with 100% oxygen. Exercise also results in vasodilatation, which increases perfusion to muscle and skin. The consequent increased rate of denitrogenation has been shown to be effective in experimental conditions (Webb et al., 1996) as well as in the operational U-2 environment (Hankins et al., 2000). It was also used, with modification by NASA, to prepare for extravehicular activity (EVA) from the ISS (Woodruff et al., 2000). Beginning in 2001 and until a staged-decompression procedure, in-flight denitrogenation, was developed and used beginning in early 2007, the exercise-enhanced prebreathe procedure was used during 21, two-member EVAs (42 individual EVAs) from ISS (Derby J, Personal communication, 12 Jul 2007; Webb et al., 2010b).

3.4.4.4.4. Break in prebreathe. It was thought for some time that a very short break in prebreathe would have significant effects on DCS incidence, requiring a “make-up” prebreathe. Recent studies showed that a 10-, 20-, or 60-minute break in prebreathe significantly increased DCS incidence under laboratory conditions involving 4-hr exposures (Pilmanis et al., 2010). It was suggested “...that a safe limit does exist...,” perhaps of 5-minutes, 3-minutes, or 1-minute duration.
3.4.4.5. Other Factors Affecting Incidence of DCS

3.4.4.5.1. Rate of ascent. A higher rate of ascent has been implicated as causing more DCS, although a study by Pilmanis et al. (2003) did not find such a relationship when 5,000 fpm and 80,000 fpm were compared during decompressions to 40,000 ft. Although the 80,000-fpm decompressions were much faster, they did not qualify as rapid (2 – 15 seconds), and no conclusion could be drawn as to the effect of a "rapid" decompression on DCS incidence. A very slow rate of ascent while breathing 100% oxygen would provide effective denitrogenation and thus confuse the results of a comparison with the more typical rate of ascent of 5,000 fpm used in most USAF chambers and altitude DCS research.

3.4.4.5.2. Repeated exposure. A study of repeated exposures comparing one 2-hr continuous exposure with four 30-minute exposures separated by either 0 (bounce recompressions) or 2-hr ground times showed the repeated exposures to have significantly less DCS risk (Pilmanis et al., 2002). Unlike diving, repeated exposures to altitude involve repeated denitrogenation, which reduces the nitrogen in the body and therefore the DCS risk.

3.4.4.5.3. Flying after diving. Nitrogen absorbed by the body tissues while scuba diving increases risk of DCS during an altitude exposure after diving if the interval between the dive and flight is insufficient to allow the additional nitrogen to be expired (Bassett, 1982; Vann et al., 2004). Bruce Bassett's technical report, Decompression Procedures for Flying After Diving, and Diving at Altitudes Above Sea Level (Bassett, 1982) reviewed studies conducted at the request of the USAF Aerospace Rescue and Recovery Service, Military Airlift Command, in 1976. The hyperbaric and hypobaric chambers at Brooks were used to test several schedules involving no-decompression dives followed by altitude exposures of 10,000 ft and 16,000 ft (Phase I) and 8,500 ft and 14,500 ft (Phase II). Data from the 160 subject-exposures resulted in recommendations for changes to the U.S. Navy schedules for altitude exposures following diving. The U.S. Navy Standard Air Decompression Tables were revised based on the results.

3.4.4.5.4. Time of Day and Temperature of Exposure. Definitive studies have yet to be done, and conclusive data are not available.

3.4.5. Individual Susceptibility

Some individuals are resistant and others susceptible to development of altitude DCS. However, they can only be identified by subjecting them to several altitude profiles that have sufficient DCS risk to allow differentiation of susceptibility. Since this is impractical, prediction of an individual's susceptibility based on anthropometric or physiologic parameters was attempted. Although groups of individuals with the following characteristics were shown to be more or less susceptible to symptom development (Webb et al., 2005), using such characteristics to predict susceptibility of any one individual has been unsuccessful:
3.4.5.1. **Body Mass Index.** Higher body mass index (BMI) in both males and females was shown to correlate with higher susceptibility to DCS ($P < 0.001$; Webb et al., 2003).

3.4.5.2. **Maximal Oxygen Uptake.** Lower maximal oxygen uptake (lower physical fitness) in both males and females was shown to correlate with higher susceptibility to DCS ($P < 0.001$; Webb et al., 2003). Since aerobically less fit individuals generally have a lower level of vascularization than those more physically fit, they may not be able to denitrogenate as quickly as the more physically fit.

3.4.5.3. **Body fat.** The logic behind the conventional wisdom that individuals with higher body fat are more susceptible to DCS symptoms is based, in part, on the fact that “…at body temperature the fat of mammals dissolves at least five times as much nitrogen as water or as blood and blood plasma” (Vernon, 1907). Anecdotal evidence and some retrospective studies have supported this view. A recent study reported that the 79 female subjects with higher body fat (highest third) had a higher incidence of DCS than the 83 with lower body fat (lowest third) ($P < 0.03$; Webb et al., 2003). The 479 male subjects so divided did not show any difference in DCS incidence. It must be noted that all subjects were chosen to closely match the USAF aircrew and NASA astronaut corps in body fat percent; thus, the body fat percentages observed were lower than the general population: females, $22.2\% \pm 4.4\%$ and males, $16.8\% \pm 5.1\%$.

3.4.5.4. **Age.** Increased age has been cited several times as contributing to DCS susceptibility (see review by Behnke, 1971) and was recently shown to affect males and females in the age range of USAF personnel in different ways (Webb et al., 2003), but it was only significant when the oldest 5-yr group of males was compared to the youngest 5-yr group of males ($P < 0.03$). Increased age is related to lower metabolic rates and generally lower physical fitness, possibly having a negative influence on rate of denitrogenation.

3.4.5.5. **Weight.** Weight was shown to be a factor only in males, with the heaviest third being more susceptible to DCS ($P < 0.01$; Webb et al., 2003).

3.4.5.6. **Gender.** Although some studies concluded that females are more susceptible to DCS, others have stated no difference was found. The recent extensive review of prospective research chamber experiments with both male and female subjects showed that gender is not a factor in susceptibility to DCS ($P > 0.23$ with 45% DCS during 309 female exposures and 50% during 550 male exposures). Females were shown to be more resistant to development of bubbles as detected by ultrasound and echo-imaging of the right atrium and ventricle (Webb et al., 2003).

3.4.5.7. **Height.** Height was not shown to be a factor in DCS susceptibility (Webb et al., 2003).

3.4.5.8. **Dehydration and Previous Injury.** Although dehydration has been suggested as contributing to DCS risk (Cockett et al., 1965), no definitive studies have been accomplished that address that variable. Alcohol consumption frequently
results in some degree of dehydration, complicating the anecdotal link of DCS risk with alcohol. Bridge et al. (1944) did not find an effect of previous injury on DCS location.

During World War II (WWII), variations in DCS susceptibility of an individual from day-to-day were greater than between individuals, making efforts to identify DCS-susceptible or DCS-resistant individuals impractical (Gray et al., 1947). Even with more detailed information available now under laboratory conditions with evaluation of more parameters, it is not possible to reliably predict an individual's susceptibility based on anthropometric or physiologic measures (Webb et al., 2005) due to the same day-to-day variations in individual susceptibility.

3.4.6. Prevention of DCS

During the early part of WWII, the incidence of hypoxia and DCS was high due to frequent unpressurized flight above 30,000 ft in the B-17 and B-24. Adequate pressurization of aircraft was shown to be the best answer to both DCS and hypoxia as demonstrated with development and employment of the pressurized-cabin B-29. Hypoxia is normally prevented by proper use of adequate oxygen equipment below 40,000 ft. However, DCS risk remains a current problem above about 20,000 ft. Some of the reasons for DCS risk to be a current issue are operational in nature:

- Occasional loss of pressurization in adequately pressurized aircraft followed by the rare incidence of continued flight at altitudes exceeding about 20,000 ft can lead to DCS.
- Development of aircraft designed to cruise above 50,000 ft, but with inadequate pressurization systems, presents conditions leading to risk of DCS, e.g., the U-2 high-altitude reconnaissance aircraft and the F-22.
- Flight in pressurized aircraft at or above 20,000 ft operated unpressurized for operational reasons or parachute operations can provoke DCS if operational guidelines (AFI 11-409, High Altitude Airdrop Mission Support Program) are not followed.
- Flight in unpressurized aircraft at or above 20,000 ft may result in DCS. The CV-22 is unpressurized and can cruise at 25,000 ft. Without prebreathe, the crew may be at risk of DCS depending on level of activity and duration of such cruise.

Barring use of adequate pressurization, other methods of preventing DCS are likely to impact operational requirements. Such methods include procedures that reduce the risk factors discussed earlier by:

- Limiting altitude (see Figure 3.4.4.1-1)
- Limiting time at altitude (see Figure 3.4.4.3-1)
- Limiting activity while decompressed (see Figure 3.4.4.3-1)
- Using preoxygenation (see Figure 3.4.4.4.1-1)
The decision to utilize measures to reduce DCS risk should be based on an accurate prediction of the risk during a planned operational scenario. Previously, such predictions were possible only when research findings were available that corresponded closely to the scenario in question. Such cases were very rare. A better method of prediction was needed.

3.4.7. Prediction of DCS

Many attempts have been made to model DCS, whether based on hyperbaric or hypobaric exposures. Commercial success with diving computers was achieved with equations that calculated the level of nitrogen absorbed from increased partial pressure of nitrogen in the breathing gas over time. The same approach has been attempted for modeling altitude exposures with some success.

An early attempt related the partial pressure of nitrogen in the tissue before decompression ($P_T N_2$) to the total barometric pressure after decompression ($P_B$) as a ratio. It is referred to here as the tissue ratio (TR). This model results in a metric for level of supersaturation during zero-prebreath exposure and has been used as a guide for NASA operations in space (Conkin et al., 1987) and in planning for Moon/Mars exploration (Lange et al., 2005).

$$TR = \frac{P_T N_2}{P_B}$$

As an example, an ascent to 18,000 ft would yield $569/380 = 1.5$, which is considered quite safe based on USAF studies from the early 1980s to 2002 (Webb et al., 2003).

Although Table 2.1.2-1 shows the atmosphere of Earth as having a sea level partial pressure of $N_2$ of 593.4 mmHg, the alveolar value of 569 mmHg of $N_2$ is derived by subtracting the alveolar partial pressures of $O_2$, $H_2O$, and $CO_2$ (Appendix 1) from the sea level total pressure ($760 – 104 – 47 – 40 = 569$). Since the partial pressure of $N_2$ in the blood, and therefore the tissues, will reach equilibrium with the partial pressure of $N_2$ in the alveoli, $P_T N_2 = P_A N_2$.

Unfortunately, the TR equation only describes the supersaturation present at the beginning of an exposure and does not describe the risk at any other point during the exposure. The added and important factor of prebreathe time complicates the equation, as does activity level, while decompressed. Therefore, it is of no practical use in the USAF environment and is only mentioned for background.

The need for a much better model was addressed by Pilmanis et al. (2004) following development and validation with human trials. It is the only altitude DCS model tested using accepted validation procedures. Before this model was developed, estimates of DCS risk were based on research chamber exposure scenarios, which were likely to be quite different from the planned operational exposures. The development of the model included a mathematical model of bubble formation and growth together with a log-logistic statistical model using the extensive Air Force Research Laboratory (AFRL) Altitude DCS Research Database (Kannan et al., 1998).

\[ P_A N_2 = P_B - P_A O_2 - PH_2O - P_A CO_2 \]
The model is called the Altitude DCS Risk Assessment Computer (ADRAC) model. It is available via the AFRL web site (Internet Resources, see Appendix 8).

3.4.8. DCS Incidence: Research vs. Operational

As long as there are missions that require airmen to transit the atmosphere at altitudes that may induce decompression sickness, there will be a need to understand and apply research in high-altitude physiology (see Webb, 2010a and b). Translating research results to operational DCS risk has consistently resulted in some confusion. The research community routinely reports a much higher DCS risk than operational reports of DCS or DCS-related mission aborts indicate is present. Figure 3.4.8-1 depicts how the high incidence of DCS observed during research exposure to the U-2 cockpit environment simulating a high-altitude reconnaissance mission actually translates into few operational reports or aborts. In this hypothetical example, each step, from research subject exposures performing mild exercise including walking to resting pilots with high incentive to complete a mission, involves halving the incidence. Note: The graph is a possible explanation to the disparity between research and operational DCS risk. It has not been tested and is the author’s “best guess” as to the causes of the differences.

![Graph showing DCS incidence vs level of activity](image)

**Figure 3.4.8-1. Hypothetical Explanation of the Difference between Research Results and Operational Risk of DCS during a 4-hr Exposure to 30,000 ft with a 1-hr Prebreathe**

**Research Results with Moderate Exercise.** Human volunteers are directed to report even the slightest change in well-being during the exposure, and this results in about 80% DCS reported by subjects in Brooks studies under the conditions described in Figure 3.4.8-1. Report of even very mild symptoms is necessary to protect research subjects from more serious symptoms that could occur with continued exposure.

**Research Results with Mild Exercise** shows a reduction in DCS incidence consistent with a reduced level of effort. **Research Results with Ambulatory Rest**, merely walking a few steps every 10 – 20 minutes, produced even less DCS (Webb et al., 2010a; also
see Figure 3.4.4.3-1). Research Results with Seated Rest is what pilots do during cruising flight, albeit including some arm movement, which only uses two-thirds as much energy as slow walking. This activity is estimated to yield about 20% DCS (hashed bar) based on a report in peer review (Webb et al., 2010a) that shows a linear relationship between DCS incidence and the highest 1 minute of oxygen consumption during a 15- to 20-minutes sequence of activity tested.

The next descriptions are a theoretical explanation of the differences between existing research findings and operationally reported DCS. Probably only about half of the symptoms reported by resting research subjects would be Noticeable by operational crew doing their jobs. Thus, only about 8% of operational missions may involve any noticeable DCS and even then a perception that they are not important or not DCS may prevail. Only about half of the noticeable symptoms would likely involve Mild Impairment of operational function. Perhaps only about half of those would be severe enough to result in an Operational Function Compromised situation. Although that 2% symptom incidence should be reported, the high level of mission orientation may yield a lower number, especially since the symptoms would usually disappear during descent. Probably only about half of those symptoms that should be reported actually result in an Abort by a mission-oriented crewmember reluctant to do so (Bendrick et al., 1996), affecting probably less than 2% of the missions. Included here are the aspects of peer pressure, inconvenience of treatment, and even career protection. Additional rebreathe or use of exercise during rebreathe (Webb et al., 1996, 2010b; Hankins et al., 2000) by susceptible individuals may reduce the abort risk to less than 1%.

3.4.9. Summary

The research done on altitude DCS during WWII and in the early years of the space program provided answers and raised questions that were addressed from the 1980s to 2005 by research at USAFSAM and AFRL facilities at Brooks AFB (City-Base). Many of those findings are referenced here to provide further resources. Production of the first altitude DCS risk assessment computer model during that period represents a large advance in the capability to predict DCS risk under many conditions. Although rare in current aircraft operations, the potential for DCS to interfere with optimal human performance remains significant. It is incumbent on aerospace physiologists to use the knowledge gained in the past 60+ years to prevent DCS from being a limiting factor in future aerospace applications.
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**Recommended Readings**


**Concepts**

Altitude vs. diving DCS
Decompression sickness (DCS)
Four categories of DCS symptoms
   - Limb pain
   - Skin
   - Neurologic
   - Respiratory
Four primary factors that affect DCS incidence
Standard of DCS care

**Vocabulary**

Body mass index (BMI)
Break in prebreath
Exercise during prebreath
Hyperbaric oxygen (HBO) therapy
In-flight denitrogenation
Maximal oxygen uptake
Prebreath, preoxygenation
Rate of ascent
Repeated exposure
Sigmoidal relationship
Venous gas emboli (VGE)
4. HUMAN PERFORMANCE EFFECTS

4.1. Human Performance Optimization

4.1.1. Aviation Psychology and Behavioral Models of the Stresses of Flight

Andrew D. Woodrow, Lt Col, USAF (Ret), BSc and David R. Jones, MD, MPH

Human performance optimization is a systematic approach to identifying the areas of performance that are central to successful outcome. It is the human factors analogue to the Five Rings Model developed by Colonel (ret) John Warden III and was used to translate his ideas into actual combat planning for operations in the 1991 Persian Gulf War. Colonel Warden believed that nation-states operate like biological organisms composed of discrete systems. In a perfect world these systems function in harmony and the organisms survive and flourish. However, certain systems controlled other systems and were thus significant, while other elements might appear to be vital, they were actually not important for sustaining the organism. Warden believed that like a biological organism a nation could be stunned. Military action could produce strategic paralysis (Chun, 2006). This model, identifying the centers of gravity in a system, is a good analogy for the concept of human performance optimization. Understanding the biologic components of the human operator and analyzing the interface within system are important first steps in identifying the centers of gravity and weaknesses of human systems integration. Humans become vulnerable to sensory assault or become neutralized when technology overwhelms with signals or input that cannot be processed effectively. Ultimately, the goal of human performance optimization is to sustain the psyche and physiological well-being of the operator while moving toward the objective goal. Planning for performance optimization uses the same system mindset in maximizing the likelihood for success in terms of performance outcome. This planning process must include a thorough understanding of the stressors that render the human vulnerable to performance decay. This chapter will focus on the psychology and behavioral aspects linked to the flight environment.

As we consider the psychological stressors of flight, we need to keep one fact clearly in mind. Men and women who enter aviation as a career or an avocation bring to the arena of the air all the strengths and frailties of the human race, all the varied elements of heredity, nature, nurture and experience that form our separate strengths and vulnerabilities. Flight, with its many joys, dangers, rewards, and demands upon body, mind and spirit, can but add to the inevitable life experiences, good and bad, that the years bring to each of us (Jones, 1986).

Just as a good flight surgeon must first be and continue to be a good physician, so all those contemplating excellence in the support of the mental health, resilience, and effectiveness of a flyer in the realms of human factors and safety must first be and continue to be accomplished and able in their respective professions. Thus qualified, we may care for all aspects of the health of aviators as they proceed through their lives and careers. We can expect no less of ourselves than we do of the flyers.
4.1.1.1. Sources of Stress. The flight environment introduces physiological stress—heat, cold, noise, vibration, circadian and dietary disruptions, g-forces, multitasking workloads, sleep deprivation, cramped cockpits, and many others. Flight also introduces psychological stressors that affect effective performance and flight safety. Experiments in flight simulators that measure the time to respond to signals presented under various conditions (e.g., heat, noise, workload, etc.) have demonstrated degraded performance. For obvious reasons, a crew would not be exposed to real hazards for the purpose of measuring responses; however, accident investigations and anonymous aviation safety reports reveal a consistent downward trend in performance when facing danger or uncommon stress in flight.

A stressor is any event or situation that is perceived by an individual or group as a threat that leads either to adaptation or initiation of a stress response. Simply said, a stressor is a stimulus—the cause—and stress is a response—the effect. In aviation, the mere activity of achieving takeoff speed and configuring the aircraft for flight is stressful due to the time compression and minimal response sets available once one passes the decision speed for takeoff: continue or abort?

Dr. Hans Selye, one of the first to study the effects of stress, coined the term “eustress” to explain the positive, desirable stressors that keep life interesting and help to motivate and inspire. Events such as going to college, getting married, starting a new job, or becoming a parent can be happy, joyous, stress-producing occasions. Eustress also involves successfully managing stress even if one is dealing with a negative stressor. Eustress implies that a certain amount of stress is beneficial and, in fact, necessary to maintain overall health and performance.

“Distress” typically refers to the negative effects of stressors that drain us of energy and may individually or collectively surpass our capacity to cope. In the aviation environment, this can be linked to acute (short-term) stressors or extended stressful conditions (long deployments or other adverse life situations). Very often when we speak of stress, we are referring to distress. For the purposes of this chapter, however, we shall consider stress in relation to its effects on performance.

Humans register stress in several ways. Biometric instruments are routinely used to identify changes in heart rate, respiratory rate, sweat rate, and even pupil size. Unlike these biometric measures, hormone levels seem to differentiate levels of stress even among highly experienced aircrew (O’Hare, 1990). In one study of U.S. Air Force instructor pilots and students, urine samples were taken before and after training flights for 4 mo. Neurotransmitter levels taken after flights classified as either emergency or precautionary showed a marked increase in both instructors and students (Krahenbuhl, 1985). As a practical measure, stress hormones may not be first on the list of markers to use in a flying squadron; however, behavior and performance (outcome) are well accepted and can be graphed out for analysis.

Most readers of psychology are familiar with the Yerkes-Dodson Law (Figure 4.1.1.1-1; based on Proctor & Zandt, 1994, Figure 9-9) depicting the relation between arousal and level of performance. It is well accepted that performance reaches a peak when a certain level of stress is included with the task. The height of this peak depends upon whether the task is complex or simple. For example, a pilot flying a holding pattern in severe weather is likely to have a higher optimum level of arousal than when the same pilot is leveled out and simply checking the cockpit before a routine landing.
The link between the physiological condition and the stress performance curve is important to consider. For instance, it is believed that if a crewmember is fatigued and experiencing low blood sugar, the effect of stress on performance will be greater than in a fit, rested person. The nature of our human condition is to cope or adapt to the environment; autonomic adjustments allow for adjustments to physical stress, but adaptation to psychological stress is much less pronounced and harder to measure. Despite the reasonable ease of measuring heart rate in flight and the natural correlation between heart rate and situational arousal, it is important to consider concomitant factors such as workload (physical and mental) as well as responsibility on the flight deck when assessing the overall effects of stress on performance. Considering the “whole person” of a flyer, one must also be aware of any acute or chronic life-situational stressors outside the aviation arena: those of family, career, health, finances, or legal problems, for instance.

Operational errors in day-to-day flying are the central concern for aviation physiologists or human factors experts. However, we must also consider the cumulative effects of ongoing stressors. Significant long-term health problems identified in medical records of personnel follow a career of high-stress operations, such as in air traffic control. The incidence of hypertension or elevated blood pressure in controllers has been found to be six times that of pilots (O’Hare, 1990). One question must be asked when considering stress in the workplace: “Is it the work environment, the workload, or the types of tasks that induce a stress response?”

### 4.1.1.2. Stress and Performance Modeling

A conceptual model has been used to explain the stress/performance relationship (Driskell & Salas, 1991). Their model considers four elements:
• The presence of a specific stimulus (warning light, alarm buzzer)
• Appraisal of the stimulus (how the individual or team evaluates the threat, situation, and resources)
• Development of performance expectations (often built through simulation/training)
• Psychological/behavioral/physiological effects

Driskell and Salas identified that the effects of stress on performance are mediated by the type of task and the severity of the stress. As an example, the perspective of an emergency break-away during aerial refueling has a different connotation from the perspective of the boom operators than the pilot in the receiver aircraft. Both have a critical task to complete: one is relying on the uploading fuel to continue the flight and safely return to home base, while the other may be in the middle of a long line of receivers and more concerned about damaging the boom, resulting in an aborted mission. Complete understanding of the stress/performance relationship must consider both chronic and acute stress, as well as the action of the entire crew and not simply individual performance. The theoretical models developed to address the stress/performance relationship cannot be universally applied without a thorough knowledge of the mission and crew composition.

Confounding the understanding of stress in the individual pilot is the general lack of understanding of the processes that a group or team (e.g., a cockpit flight crew) goes through to accomplish tasks during a mission. Group dynamics during a task-intensive scenario may be more relevant to errors than individual proficiency. In several key studies presented in realistic simulator environments, the subordinate team member often yielded to authoritative members of the team when under stress (Foushee & Helmreich, 1988). This is especially troubling if the junior member has a more complete picture of the situation but is unwilling or unable to share his/her situational awareness with the captain or other senior members of the crew.

Overcoming stress in the workplace is not as simple as removing the “offending part.” Operational conditions and environments are not easily reengineered; however, one remedy for stress that affects performance is to redesign the task or its cockpit presentation. Automation has reduced the workload for many in the aviation environment, including aircrew and air traffic controllers. The introduction of glass (computerized) cockpits in the later part of the 20th century has reduced some workloads and provided a more effective suite of information displays for the operator.

Stress cannot be engineered completely out of the system, so a second consideration is the selection of personnel for positions prone to stress. Military selection processes take place before potential flyers, air traffic controllers, and other flight-essential personnel enter active flight operations. These selection sites are few (e.g., the Air Force Academy, certain training bases, NASA-Johnson Space Center), so only a few mental health practitioners are likely to become involved in this process. Briefly, the Air Force “selects-out” those considered unfit for such training due to clinical or personality-related issues. Some “select-in” considerations apply to particularly demanding mission requirements in which personnel must not only be fully qualified but the best qualified (e.g., special operations, USAF astronaut candidates). These criteria involve matters of motivation (both emotional and rational), ability (physical, cognitive,
autonomic, neuropsychological, and physiological), and stability (personality, temperament, interpersonal relations). Such matters may be approached both scientifically and intuitively and form an integral part of the mental health of aviators (Jones & Marsh, 2001; Santy, 1994).

4.1.1.3. Stress Assessment and Management. There is limited knowledge of the meaning of current measures used in stress assessments. The correlations between physiological and psychological stress are not often correlated well with the broad set of circumstances a crewmember might be found in over a career. A stress performance screening test would have to account for a myriad of stressors and link to aviation-centric decision making. Developing a set of circumstances that would cover particular subject matters under varied conditions could still not reasonably capture all conditions and all candidates.

The third and most reasonable approach to stress reduction is through training. Stress management techniques and stress exposure interventions are the two most common methods (Johnston, 1997). Most stress management programs focus on the comfort of the individual rather than actual changes in performance. For example, in SCUBA training the trainee is inserted into a troubleshooting situation and presented a series of options to resolve the problem. In a manner similar to clinical desensitization to phobic objects through successive approximation followed by guided relaxation, this training process helps the individual become comfortable in one scenario. The student experiences a stressor and practices coping strategies under conditions of graded exposure in a controlled setting prior to confronting the actual stressful experience. This approach provides feedback and coaching while the student is adjusting his/her perception of the ability to cope with the target stressor, rather than reacting with an uncontrolled “blow-up” to the surface. The objective is to help the trainee use a particular skill set to enhance performance in the presence of a defined, predictable stressor. The combined approaches seem to be the best solution set for work in complex, dangerous environments.

Application of stress management techniques in the human factors realm is most effective when linked to performance issues. The practitioner of aviation physiology and human factors should understand the nature of the environmental stressors and the tasks of each crewmember in completing a successful mission before constructing a solution set for reducing stress. Intervention must be supported by research relevant to the environment of concern. Safe, effective human performance is the goal; stress management is the tool.

4.1.1.4. Psychophysics and the Environmental Stress of Flight. Long before man took to flight, scientists searched for a way to measure the sensations that physical stimuli evoke in man. Gustav Fechner, in 1860, proposed in his book Elemente der Psychophysik that the sensations arose from an interaction between the physical world and the psychological worlds (Blake, 2006). In a modern approach, how would you go about describing the quantity of sensation to the physical intensity of a stimulus evoking a sensation? Is there a formula that could be used to describe the intensity of the input? One example in aviation is visible light. Say you display a spot of light with an intensity of “100 units,” a value that is easily visible to the unaided eye. Next, increase the intensity of the light to the first point where the observer notices the change in intensity; let’s call the new intensity level “110 units.” The difference will be called the threshold. Next question: Would the threshold be a constant across the
visible spectrum?' If you repeated the demonstration, this time starting with an intensity of "1000 units," it is unlikely you would notice a difference of "10 units" due to the starting point (intensity) of the stimuli. The structure and function of the eye and visual processing center has distinct limits to the intensity required/allowed for activation on both ends of the spectrum. Ernst Weber, a contemporary of Fechner, noted that the threshold of stimulation is not constant but tended to be in constant proportion of the initial stimulus value. The concept of absolute and difference threshold are key elements in the study of perception. Absolute threshold is essentially the point of transition between undetectable and detectable signals. This is well appreciated for those who have had an audiology evaluation; the least perceptible sound identified is the absolute threshold of your hearing. The difference threshold is the minimum amount of change of a stimulus in order to produce a noticeable change in perception. Psychophysical equations are based on these principles.

4.1.1.5. Psychophysics and Reduced Human Performance.
Understanding human perception is not complete without an appreciation of the nature of the psychophysical stimuli associated with the modality and physiological processes that register the presence of a signal and nature of the stimulus. Each of the sensory modalities- vision, hearing, tactile, smell and taste- is specially designed to pick up signals in the environment relevant to particular cues and events. For example, the smell of oil following a ‘popping’ sound and visual cue of smoke may be all the stimuli you need to determine there is a mechanical fault in the system. Understanding the complexity of picking-up these signals requires further study. Sensory transduction is the conversion of physical energy into electrical signals within biological (sensory) receptors. In every aviation environment, there are myriad physical signals that guide and distract. System design helps to filter some of these cues from the human operator, but there are times when physical input across the sensory spectrum is unfiltered and overwhelming. A quick review of some examples may assist in understanding the phenomena.

The visual system, the key physiological process in so many tasks, if overwhelmed by physical cues (e.g. photons transmitting extremely bright light to the eye) is unable to process additional visual input until some form of filtering is in place. For example, a warning light displayed on a panel can be “washed” out if bright light from an outside source (e.g. a laser) floods the visual field. Auditory cueing is, likewise, a fragile sensor that can be easily overwhelmed with competing sounds on the flight deck. Consider the case of airborne vibrations passing by the external ear and converting to mechanical actions at the ossicles of the middle ear. The “sound” registered in the brain is really a result of a complex series of transductions. The resultant electrical changes in the hair cells release a chemical transmitter which is picked up by auditory nerve fibers and ultimately carried from the cochlea to the central nervous system to be catalogued as “sound.” The pressure waves that lead to microscopic movements of tissues bones and hairs have a direct impact on one’s feelings, emotions, and behaviours. Think about an aircraft warning alarm tone. In order to be effective, the sound must be registered as “urgent” but not overwhelming. Additionally, the sound cannot be masked by other ambient noise and should not present false cues or be mistaken for other auditory signals. Experience can aid in developing strategies to process conflicting or a high volume of signals, but physiological and psychological limits to physical input received in a span of time are the barrier to human performance. In another psychophysical example, motion
sickness symptoms in a virtual environment (e.g. simulator or cinema) represent a novel, but important form of visually-induced cybersickness. Sensory conflict results when there is a struggle between the visual, vestibular, and/or proprioceptive systems while participating in a fixed-based simulator. In a study of visually induced motion sickness using the combined motion cues of pitch, roll, and yaw, it was implied that increasing sensory conflict magnitude leads to increased levels of motion sickness (Keshavarz, 2011). Even when the stimuli were removed, the persistence of motion sickness was evident. Although the motion sickness decreased rapidly after stimulus offset, the symptoms persisted for several hours; almost two-thirds of the subjects reported that they felt worse 1 hour after the exposure, and some had symptoms up to three hours post-event. In an aircraft or synthetic environment, if the sensory nervous system detects a discrepancy in sensory inputs and registers a sensory conflict, a cascade of psychophysical decay could result in diminished capacity to complete the mission.

4.1.1.6. Behavioral Health in Aviation. Since early flight, and especially sustained duration missions, behavioral effects of the aviation environment have been among the primary concerns of researchers studying human performance. Performance measures have been an essential part of the development of the field as there is no better indicator of psycho-physiological integrity and capability. Human behavior, unlike physiological measures, is a complex component to assess. Although some forms of physiological monitoring has emerged in aviation (e.g oxygen saturation levels in F-22 pilots), real-time assessment of the cognitive state of aircrew is problematic. Russo identified that judgment and decision-making are critical operational functions that ideally use higher-order cognitive pathways to weigh the cost versus the benefits of particular actions (Russo et al., 2005). In a laboratory, neurophysiological functions can be monitored using techniques like the positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) to measure brain activity changes at specific cortical and subcortical sites. Monitoring systems designed for aviation are still years away; the robust nature of the device and signal fidelity must meet specifications yet to be defined. If, however, there was technology available to measure the relationship between efferent visual systems during active cognitive process and performance (e.g. measure eye blinks during information acquisition) there could be strategies developed to ensure time-on-task is correlated with physiologic stress.

The research and development requirement to identify behavioral health effects in aviation for evidence-based coordination between design engineering, human systems integration considerations, and behavioral health imperatives assumes a new level of importance with highly automated and remotely operated systems.

References

Concepts
Stress management
Yerkes-Dodson Law
Physiological stress
Psychophysical stress

Vocabulary
Stressor
Absolute threshold
Difference threshold
4.1.2. Human Factors in Aviation

Andrew D. Woodrow, Lt Col (Ret), USAF, BSC

“A classic tale of Human Factors: Icarus, the son of Daedalus was imprisoned in the Labyrinth on Crete to punish Daedalus for helping Theseus to kill the monster called the Minotaur and to escape with King Minos’ daughter, Ariadne. Daedalus knew that Minos controlled any escape routes by land or sea, but Minos could not prevent an escape by flight. So Daedalus used his skills to build wings for himself and Icarus. He used wax and string to fasten feathers to reeds of varying lengths to imitate the curves of birds' wings.

“When their wings were ready, Daedalus warned Icarus to fly at medium altitude. If he flew too high, the sun could melt the wax of his wings, and the sea could dampen the feathers if he flew too low.

“One airborne, Icarus became exhilarated by flight and ignoring his father’s warning, he flew higher and higher. The sun melted the wax holding his wings together, and the boy fell into the water and drowned. Daedalus looked down to see feathers floating in the waves, and realized what had happened. He buried his son on an island which would be called Icaria, and the sea into which Icarus had fallen would ever after be called the Icarian Sea” (Thompson, 1999).

Over the century since manned flight began in earnest, great progress has been made in both the engineering and human integration of man-to-machine systems. The earliest investigations of the tragic loss of life and materials were often centered on engineering aspects of flight or operations. Once the reliability of the machine is confirmed, many investigators still fall short of dealing with human performance issues. The study of human factors causes the reader to pry deeper into the interaction of the operator with the machine. Most accident investigation teams have precise engineering perceptual frameworks and are more comfortable working with data, yet many human factors elements are linked to circumstantial evidence that cannot be duplicated in a laboratory.

The Royal Flying Corps (UK) was the first to harness human factor specialists in the form of flight surgeons during the First World War. The aeromedical factors identified during the early investigations have become foundational to our current human factors rubric. As manned flight expanded from military to commercial aviation, human factors became a greater concern. Materials continue to improve, but the human retains weak points that are often the source of error.

To fully embrace human factors as a profession or function of safety, one must approach the field with a systematic progression. The process begins with descriptive survey and observational methods (What did you see?), to controlled laboratory experiments (Can we replicate behavior in a simulator?), and back to a field experiment to validate the findings. Human Factors (HF) is an applied science, and as such, the practitioner must use scientific reasoning to guide the activities of any investigation. The same scientific method used in a chemistry lab is used in the HF field; continuous refinement and development of theory based on observations is critical to satisfactory outcome.
Identification, classification, and analysis of human error is the cornerstone of reliability and human error in complex systems. Errors are typically linked to inadequacies of system design and are grouped into three categories (Proctor and van Zandt, 1994). The first is linked to task complexity: the limitation of the human to process information or the capacity to recall, calculate, or attend to information presented from the system. The second is linked to error-likely situations: the predisposition of the operator to making an error due to inadequate training, procedures, or poor supervision. The third category is linked to individual differences: the susceptibility to stress, inexperience, and attitudes can produce as much as a tenfold increase in human error probability. For example, the closest relationship with overall number of errors made on a flight deck was found in the communication category “acknowledgement”: the acknowledgement of one crew member that a piece of information has been received is a critical part of the feedback loop. Reinforcement of communication between two or more crew members reflects that a harmonious flight deck atmosphere is less likely to produce errors than a tense, strained one (O’Hare and Roscoe, 1990). Most recently, the flight deck has expanded to an ever-broadened geography including the mission control element, the sensor operator, the battlefield commander, and other aerial weapons systems all working in unison to achieve a singular mission objective. Data that support the case for human factors must be grounded on solid scientific method, as this study demonstrated through the use of cockpit voice transcripts, simulator reenactments, and role play with aircrew followed by observation on a flight deck. Analysis of 4,000 mishaps linked to pilot-causal factors converged on a single conclusion about human performance: judgment errors (e.g., decision-making, goal-setting, and strategy-selection errors) were associated with major accidents whereas procedural and response execution errors were linked to minor accidents (Wiegmann and Shappell, 1997). One challenge of the current generation of aviators is employing “air sense” without the experience of flying an aircraft. Numerous studies have attempted to pin down the importance of experience in decision-making and skill-based activities. Expert pilots are likely to use flexible task management during emergencies and procedural task management during routine flying (Kennedy et al., 2010). Often, novice pilots are not aware of the decisions to be made and may struggle sorting information that is “important and relevant.” Even as crew stations are designed and customized with a video-gamer-inspired control interface featuring hands-on-stick-and-throttle (HOTAS), rudder pedals and high back chair, the mission of flying a sortie for 20+ hours at altitudes above 50,000 feet and with multi-million dollar payloads is beyond a typical gamer’s intellectual experience level. The challenge of remotely piloted vehicles may level the playing field between experienced pilots and novices when it comes to task management, as the interplay of working memory and sensory-motor input is reversed.

There is virtually no limit to the dimensions that can be captured in human factors analysis and, likewise, no limit to the fields that HF can be applied. Human physiology can be a useful link to understanding the human interface within a system. Color perception, auditory cues, sensory stimulation, neurological limitations, and even basic strength and endurance can each have a role in understanding why errors occur and how to develop methods to mitigate errors. For example, when evaluating color cue on displays, it is important to note that the effect of color on task performance is very situation specific. A number of considerations may influence the effectiveness of multifunctional displays. Color coding on a multifunction display must account for symbol density (number of symbols displayed), discrimination of color (contrast and
redundancy), viewing time (dependent on other tasks and location of display), and skill level of user (Weiner and Nagel, 1988). Additionally, there are cultural cues that are relevant to understanding response and interpretation of colors and symbols on a display. The linkage between physiology, psychology, and operational experience (e.g., training) is evident in this example.

The fundamental processes involved with operating complex systems have changed widely over the first century of manned flight. Classic human factors models lined up useful but disparate processes in an attempt to explain error. Bainbridge describes the five main cognitive functions involved in a human-machine interface: discriminating a stimulus, perceiving whole parts of sensory input, naming the input, choosing an action and comparison across a range of processes (Garland et al., 1999). From this, it is clear that no human response is made in isolation. The influence of the environment and individual capacity and experience are at the bedrock of our role as human factors practitioners. Specifically, in the context of aircrew decision making prior to or during a flight, the practitioner should draw a distinction between the influence of strong situations on decision making and other, related influences that can cause or add to strong situations such as organization, personality, and physiological factors, and specific social psychological phenomena (e.g., informational social influence). For example, when examining the decision to ‘press on’ into hazardous weather conditions, one must determine if the importance of the mission; a rescue/recovery is likely to influence the perceived pressure to continue versus a redeployment to home station following a routine sortie. Approximately 75% of tactical decision errors that led to accidents investigated by the National Transportation and Safety Board (NTSB) were identified as plan continuation errors. These errors occur when a pilot inappropriately continues with a plan of action in the face of cues that suggest plan revision (Orasanu et al., 2001). A related phenomenon is a perceived feeling to “get there” and pilots report that they are sometimes overly influenced by this vague, unspecified pressure to get to their destination.

References
4.1.3. Human Factors Training

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Human factors is that branch of science and technology that includes what is known and theorized about human behavioral and biological characteristics that can be validly applied to the specification, design, evaluation, operation, and maintenance of products and systems to enhance safe, effective, and satisfying use by individuals, groups, and organizations (Christensen et al., 1988).

Our little knowledge of human beings has been gleaned at a person-to-person level and by day-to-day contact with working associates in aviation. Much was learned over the years by observing and talking to other more experienced instructors of aviation and science, both good and bad. If good tutors in human factors awareness had been available 100 years ago, would we be facing the same struggle with the man-machine interface as the 21st century human factors expert? It is rare indeed to find teachers addressing this subject in formal class anywhere, when secondary education ignores it as a subject to be taught. Flying training organizations do try harder to promote human factors awareness in instructors, beyond the content of student pilot multicrew cooperation or crew resource management (CRM) courses. Notwithstanding, aerospace physiology instructors usually need to find out and learn “the hard way” about human factors and reactions in the operational environment, not leaving behind the lessons found in text books but building on the theoretical to the practical application. Where does one then learn about human factors? The dearth of human factors information and guidance has prompted much work since 1985 to fill a glaring gap on the subject. Training in the field of HF covers a broad variety of specialty areas, from ergonomics and anthropometry, engineering and design, cognitive psychology and physiological response to the environment. The HF specialist uses scientific processes to analyze the human component as a subsystem of the whole operation. As early as the 19th century, researchers have been studying the interaction between man and machine. Ernst Weber established a quantitative measure, now known as Weber’s Law, which made possible the expression of one’s ability to tell that two stimuli differ in magnitude. Weber’s Law (Weber, 1978) is expressed as:

\[
\frac{\Delta I}{I} = K
\]

where:

- \(\Delta I\) = the amount of change between 2 stimuli needed to tell that the two stimuli differ in magnitude
- \(I\) = the intensity of a stimulus
- \(K\) = a constant

Applied in the aviation environment, the designer of warning signals might consider the intensity of a warning light against all lighting conditions in a cockpit to ensure the difference between signals is identified by the operator. Or, similarly, a warning tone must be presented against the other environmental noise to ensure the tone is heard.
In aviation physiology, aircrews are taught new skills based on both motor theory and cognitive theory. Neuromuscular patterns of activity are established during physical practice of tasks, like selecting regulator settings during hypoxia recovery. Secondly, mental practice allows the individual to establish a mapping sequence of action before actual physical practice begins. The cognitive components of a task that are rehearsed apparently reinforce the physical components of a practiced task. Unfortunately, training environments are not often identical to the operational environment, and this may lead to a problem of transferring skills from one to the other. Any form of training – simulators, emergency procedures, equipment operation – should aim to produce positive transfer. Negative transfer is the result of a previously learned stimulus-response reaction being reassigned so that stimuli that require one response now require another. For instance, if controls or displays are reversed, or control switches are configured differently, the likelihood of negative transfer is higher. When developing demonstrations in aerospace physiology, it is critical to consider the operational environment and match the training materials and devices as closely to the flight environment as possible. One limiting factor is the broad population serviced in aerospace physiology. When discussing and practicing oxygen discipline, for example, one objective is to establish aircrew confidence and competency in the use of oxygen equipment. This is complicated when neither the oxygen equipment nor the environment it is employed in aerospace physiology matches the operational environment, e.g., joint helmet-mounted cueing system (JHMCS) and environment the aircrew will face outside of training (Figure 4.1.3-1). In a related analysis of human factors problems confronting hospital staff are not limited to medical-centric issues (e.g. the difficulty of the patient’s condition) but rather to the difficulties with the tools and work environment in the clinical arena. Unfortunately, the culture of health care workers leads them to take these sorts of obstacles for granted as “just part of the job.” Tucker and Edmondson have shown that this sort of “patching” behavior lets workers complete their tasks but prevents health care organizations from learning about their vulnerabilities (Tucker, 2003). The concepts of education and training are pivotal in development of human-factors-based curricula in every environment.

In consideration of the distinct lanes of education and training, the practitioner of human factors must put into context the tasks of every crew member. Memorizing Dash-1 or an objective list of facts is a form of learning that has a very important place in flying training. This level of “hard-fact memorization” is effectively accomplished via computer-based training or individual study. Soft skills, or the ability to execute critical thinking or crew decision-making activities, are often the linchpin to success at a point in the mission when facts are not enough. Instructor-based training maintains its relevance largely because it is the most effective medium to teach soft skills. As important as memorization of facts and procedures is to flight safety, the soft skills of critical thinking and team building seem to be the definitive point in successful mission execution. How can we effectively educate aircrew on complex soft skills such as adaptability, critical thinking, mission organization and collaboration--traits closely tied to common break points related to mission execution? Instructor-based, classroom training provides aircrew with the opportunity to practice and receive feedback from an instructor who can observe and evaluate performance in real time.

A key to successful learning is the application of lessons. Quickly turning a lesson into an experience in the aircraft reinforces knowledge retention. According to the University of Waterloo’s “Curve of Forgetting” study, students lose 50 to 80 percent of what they’ve learned after one day. This figure jumps to 97 percent after a month.
Education and training, then, are actually two distinct aspects of the instructional process. Education is most associated with broad-based sets of knowledge, values, attitudes, and skills. The concepts presented under the banner of education are foundation stones for the aircrews to build specific job skills upon. Training, on the other hand, is aimed at developing specific skills, knowledge, or attitudes. The skills developed during instruction can be organic, as in language-based, physical, intellectual, or social. The objective measure of success in human factors training must include acquisition of both the knowledge (education) and the skill (training) to use the information.

Figure 4.1.3-1. JHMCS Helmet

Review of hundreds of accident reports reveals that, while human error, particularly operator error, is the most frequently cited causative factor, it is so often contaminated by other errors and events that the magnitude of its contribution may be less than publicized. If errors of intent were personal choice, that is, they involved a known violation, the analysis of human factors could be considered in terms of whether the act is the normal approach of the individual based on competence, attitude, or ignorance or an unusual circumstance that involved no apparent background cause. Human factors taxonomies are clear to point out that few errors can be ascribed to the individual alone without other personnel or material involvement. So the human factors practitioner preparing to teach a lesson in error mitigation must consider selecting examples that point out errors of intent committed for no apparent reason as well as errors not by intent, committed with no extenuating circumstances. An example of the first might be low flying a residential area by a trained, competent, experienced pilot who is well aware of the hazards; essentially a premeditated violation of flight rules. An example of the second might be a gear-up landing by a pilot of comparable training, experience, and ability that occurred under optimal conditions with no extenuating circumstances and that the pilot did not recognize or accept even after the aircraft scraped to a halt on the runway. The inadvertent actions of omission or commission that lead to this second kind of error defy evaluation except in terms of dynamic psychoanalytical concepts. It is only human factors accidents caused by these kinds of error that might be considered pure or uncontaminated operator-error events. Short of recording every action of the aircrew in flight and reviewing the actions following each mission, the errors committed are often uncorrected.
In order to best address the concept of human factors, it is helpful to start with a definition. The framework of an effective definition must be founded on performance factors related to humans, and it must be crafted for specific environmental challenges. Understanding where the performance breakdowns occur further amplifies the character of errors and may lead to performance improvements. Human factors must be approached as a multidisciplinary activity. Professor Edwards declares that “Human Factors is concerned to optimize the relationship between people and their activities, by the systematic application of human sciences, integrated within the framework of systems engineering” (Civil Aviation Authority).

The best way to illustrate the importance of studying human factors is through examples of air disasters.

- In 1977, two B-747s collided while on the runway at Tenerife, with a loss of 585 passengers and crew. A breakdown in normal communication procedures and misinterpretation of verbal messages were considered factors (ICAO Circular 153-AN/98) (Figure 4.1.3-2).

- A series of three B-737s crashed due to “rudder-over” conditions, uncommanded rudder deflection causing loss of control and impact with the ground. A good example of human factor failures may exist amidst a flawed design in a single model within two different airlines; nothing changes over a period of years until another failure triggers a design review.

- In 1987, an MD-80 crashed on take-off in Detroit. The pilots had not set the flaps, thus violating standard operating procedures. Also, the take-off configuration warning did not sound, for undetermined reasons (NTSB/AAR 88-05) (Figure 4.1.3-2).

Figure 4.1.3-2. Tenerife and Detroit Accidents
Human factors experts can assist in the analysis of accident investigation findings and often contribute to recommendations of remedies. In the context of human factors design-induced errors, procedural flaws, or inadequate warnings or training, the interest in understanding the linkage between the human and the system has heightened the need to refine the education and focus of today’s human factors experts. According to some, forensic human factors has been part of investigations since the first aviation fatality, Lt Thomas Selfridge, a U.S. Army pilot killed after crashing a Wright Flyer in the early days of aviation. The analysis led to the search for better crashworthiness and survivability, such as the routine wear of helmets and restraints (Gilson, 1999). Nearly every manufacturing specialty uses human factors techniques to review safety and operating procedures. The cost of evaluating system safety has been a tough sell until recently. Mega-volume production of automobiles, for instance, has resulted in teams of human factors experts assigned to production staff. Many of the techniques used in the early days of HF analysis have been replaced by computer modeling such as finite element modeling for injury analysis. Additionally, the use of simulations has been commonplace in aviation for decades.

Simulation has grown to be a method used to increase exposure to tactile and procedures training. Human factors simulation is not limited to aviation. The most notable is the inclusion of simulations in the medical field. Not unlike a pilot practicing an approach to an unfamiliar airfield, HF engineers and medical experts have devised methods of modeling medical techniques using highly sophisticated tools. Clearly, the more a technique can be minimized, the better off the patient may be in recovery. The main advantage of minimal invasive surgery is to avoid the trauma linked to the opening of the patient’s body. In the case of laparoscopy, a video camera and few surgical instruments are introduced inside the abdomen through small openings. This technique has the advantage of being less invasive and, therefore, shortens the stay of the patient at the hospital. However, minimal invasive surgery requires specific training due to the difficulty of moving a three-dimensional tool by looking at a two-dimensional video image, which creates a problem of hand/eye coordination. Furthermore, the manual dexterity is strongly reduced due to the shape of surgical instruments. A recent model developed by the Institut de Recherche contre le Cancer de l’Appareil Digestif (Delingette and Avache, 2005) is to provide computer software allowing:

- The teaching of the liver anatomy from three-dimensional computer-generated images
- The gesture training of a surgeon by simulating the interaction of various surgical tools with the organs of the abdomen
- The planning of the resection of hepatic segments from the computed tomography scan images of a patient

There are several advantages of a computer-aided simulator over current training techniques (mechanical simulator, training on cadavers or animals). Such a simulator would give an objective evaluation of a surgeon’s dexterity combined with a more intensive training activity (Figure 4.1.3-3). It would allow the simulation of rare pathological cases and could simulate the interaction with several organs. HF plays a major part in the refinement of equipment and software development.
Figure 4.1.3-3. Surgical Simulation Provides Highly Realistic Training

Simulation adds value and reduces cost, but the key to efficiency lies within the transfer of skills. Are the skills and knowledge acquired in simulations of value to actually handling an aircraft or other technical equipment? Roscoe and Williges (1980) developed the transfer effectiveness ration (TER) in an attempt to quantify the answer to this question. The ratios were defined for pilot training in the following way:

\[ \text{TER} = \frac{A_c - A_s}{S} \]

where:
- \( \text{TER} \) = transfer effectiveness ratio
- \( A_c \) = aircraft time required to reach criterion performance, without access to simulation
- \( A_s \) = aircraft time required to reach criterion performance, with access to simulation
- \( S \) = simulator time

As Roscoe and Williges (1980) indicated, the TER is the ratio of aircraft time savings to the expenditure of simulator time; it reveals how much aircraft time is saved for every unit of simulator time invested. Positive transfer from simulators is reliant upon environmental realism and solid instruction. Simulation has consistently shown an increase in effectiveness and a reduction in costs for many aspects of flight training. Coupled with actual flight, the transfer from simulator training was almost always superior to simulator-only training (Hayes, 1992). Moreover, self-paced simulator training is superior to lock-step instruction. Once simulator work is complete, the HF instruction continues in the aircraft (Figure 4.1.3-4).
How can the actions of an aircrew be evaluated outside the cockpit task training in the squadron? Monitoring in-flight activities may be an effective way to replay the mission as part of a training program. Mishap footage is common in classroom presentations to highlight the outcome of a mission that resulted in an accident. Typically, that footage is from an external camera, head-up display with an outside view of the flight or computer-generated images based on recorded flight data. Embedded training or testing is a concept familiar to any pilot who has reviewed gun camera footage following a mission. Flight recordings used for pilot performance assessment could be used for human factors analysis on ground-based display devices, permitting operational skills to be evaluated following actual or simulated missions. In aerospace physiology, when a video of aircrew or aircraft performance is selected for a training segment, the instructor must be thoroughly familiar with the circumstances of the mission. Developing training programs that address situational awareness, sociopsychological, and other human factors that influence aircrew performance requires comprehensive study of error, causes of error, and methods of reducing incidence or severity of human error in complex systems.

How can we keep human factors pertinent to training? Many aircrews have only a limited understanding of human factors and cockpit or crew resource management. The meanings are not explicit in the words used as a panacea for failures in human intercommunication skills. Is there a need for new terms to describe what is really intended by human factors, as applied to the various aspects of the aviation scene? The International Civil Aviation Organization (ICAO) has produced some documentation in the form of digests on the subject of human factors concepts including some material relevant to training and evaluation and also on selection processes that could be of help in general terms. CRM and line-oriented flight training concepts were also addressed in one of the early ICAO digests and could be useful to new and not-so-new aerospace physiology instructors. In the current status of HF instruction, there still seems to be a certain lack of comprehensive “human factors” material that addresses the evaluation function and the interrelationship between aircrew and other elements of the flight environment. More specific research in human factors-related topics provides good insight to the problem of human-machine interface. One example is the impact of automation on the flight deck (see Sections 4.4 and 4.5). One universal understanding is that in a high workload situation, fewer attentional resources are available to monitor automated systems. Reliance and compliance are the terms associated with the...
manner in which humans use automation. Compliance refers to a user following cues presented by the automation; we are familiar with the use of a navigation system that maps directions to an unknown location. In the absence of ‘double-checking’ the cue, the user has *complied* with the system. There are obvious consequences to this behavior if the automation is faulty. Reliance refers to the use of automation when it is silent; any time there is no audio or visual alert from the automation. Going back to the navigation example, consider the driver who is relying on the GPS to alert the next way-point. If the automation is silent, and the driver continues passed the way-point, then the driver has *relied* on the system. Evidence from a study on workload, age and automation indicates that both younger and older adults tend to rely on automation more than they comply, perhaps because of the nature of false alarms and misses. (McBride, 2011) It is recognized that, to an extent, formal training in human factors matters is better served through university instruction and supplemented by technical experience in the field. Much has been written on the subject by specialists at large and by regulatory authorities such as the Federal Aviation Administration (FAA) and universities with expertise in the study of human performance. It is imperative that all practitioners of aerospace physiology increase individual knowledge and practical application of human factors in all aspects of the human performance arena.

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Recommended Reading


Human Factors and Aerospace Safety [journal]. Cranfield University, Bedford.


Concepts

Physiological stress
Psychophysical stress
Stress management
Yerkes-Dodson Law

Vocabulary

Absolute threshold
Difference threshold
Stressor
4.1.4. **Crew Resource Management / Maintenance Resource Management**

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4.1.4.1. **The Aviation Community.** The pioneering efforts to tackle crew-related communication and resource utilization issues on a flight deck stemmed from a series of commercial aviation accidents in the 60's and 70's that pointed to a common failure among professional flight crew: a lack of coordination and communication during critical segments of flight. From those beginnings, case studies, classroom lectures and simulator training programs formed what is now referred to as Cockpit Resource Management. Cockpit resource management (CRM) as defined by Dr. John Lauber, former member of the National Transportation Safety Board (NTSB), as “the use of all available resources, information, equipment, and people to achieve safe and efficient flight operations” (Lauber, 1984). The emphasis of CRM principles as applied in commercial aviation did not initially translate to military or general aviation, but the philosophy of identifying key elements in decision-making, briefing and debriefing, and maintaining situational awareness during the mission remains the bedrock of CRM training curriculum. The 1980’s saw a rapid uptick in the application of crew resource management programs across commercial and military aviation. The wealth of information generated by the commercial aviation industry led to advanced studies in academia and collaboration with military researchers. In the decades since formal cockpit/crew resource management programs were added to the training portfolio, aircraft maintenance, nuclear power industry, and medical teams have begun employing similar programs to enhance performance. The goal of any CRM program is to improve performance of a team, and as Dr. Lauber stated, “achieve safe and efficient operations.” The formal record of investigations into aircraft accidents, such as those conducted by the NTSB, provides chilling documentation of instances where crew coordination failed at critical moments (Helmeich, 1999):

- A crew, distracted by the failure of a landing gear indicator light, failing to notice that the automatic pilot was disengaged and allowing the aircraft to descent into a swamp.

- A co-pilot, concerned that take-off thrust was not properly set during a departure in a snowstorm, failing to get the attention of the captain with the aircraft stalling and crashing into the Potomac River.

- A crew failing to review instrument landing charts and their navigational position with respect to the airport and further disregarding repeated Ground Proximity Warning System alerts before crashing into a mountain below the minimum descent altitude.

- A crew distracted by non-operational communication failing to complete checklists and crashing on take-off because the flaps were not extended.

- A breakdown in communication between a captain, co-pilot, and Air Traffic Control regarding fuel state and a crash following complete fuel exhaustion.
• A crew crashing on take-off because of icing on the wings after having inquired about de-icing facilities. In the same accident, the failure of a flight attendant to communicate credible concerns about the need for de-icing expressed by passengers.

The paradox of a "WILCO" attitude coupled with a culture of blame is a universal framework of accidents and incidents on the flight line and on the flight deck. A more accurate and constructive way to express the situation is to say that most accidents are the product of human factors issues, either at the level of the individual, or the level of the organization, or more probably both. This acknowledges the complexity of the situation, and the contribution from both the fallible operator and the system within which he or she works. Most often, the decisions that lead to a reportable incident are the result of a string of events; the end point is not always a tragic accident, but reveals the weakness in the human-system interface. Consider the commercial aircraft, a Boeing 737, landing in Louisville, Kentucky on a midsummer evening:

The airport environment during the taxi in was surrealistically lit with frequent surface lightning strikes. Ground refueling for the short trip to Lexington was delayed by the Captain until the convection activity had passed overhead. Crew preparation began with calls to the Dispatcher and Meteorologist to plan a route around the thunderstorms. All concluded that a deviation to the northwest would be prudent. The planned route, combined with some good crew weather radar technique, allowed for a smooth flight to the Lexington area. During descent, the aircraft broke out of the lowest layer of clouds at 6,000 feet. The airport and runway picture was orientated properly and matched the crews Lexington "frame." The ILS was inoperative; the crew presumed it was lightning damaged. The Captain looked cross cockpit at the Distance Measuring Instrument (DME) and saw nine miles, just about the proper distance. The control tower was closed due to the late hour, which required the crew to key their VHF microphone several time on tower frequency to turn on the runway lights. The runway lights came on immediately and the B-737 landed…at Frankfort, Kentucky. The reliance on seat of the pants decision making strategies and stress increase the probability of decisional error and breakdown in situational awareness of the crew.

Decision making, on the flight deck or on the flight line, is part of a judgment structure. This judgment is a correlation of past events with present circumstances to make future predictions. When a crew shares the same schema, or mental model, their performance can look well-coordinated and linked to a behavior continuum, like performing a series of checklists during taxi and take-off. Events that are not part of the standard operating procedure tend to lead to performance biases—that is, actions based on previous experiences. Expert crews generally perform well in a time-compressed, non-standard situation because they have a larger, more developed cognitive schema for the task at hand and are more efficient at "solving the puzzle." A less experienced crew has smaller, still forming schema and consequently share a less developed team behaviour pattern. Pre-CRM trained air carrier pilots had a notorious “bullet proof” attitude about stress, especially among younger crew (Helmreich, 1989).
In a military example, a well-experienced C-130 crew was executing a training drop on a drop zone (DZ):

The C-130 was conducting tactical training and flown by an operational crew with an Instructor Pilot (IP) and Instructor Navigator (IN) on the flight deck to supervise and monitor the flight. The low-level portion of the sortie was to include a practice parachute drop on a prescribed drop zone (DZ). The crew planned the approach to the DZ from the southwest down a valley with an exit to the north. The terrain presented a rock buttress straight ahead of the DZ which required a turn nearly all the way west before reversing to the north. The crew did not realize their predicament and followed the plan. The Hercules requires about 9 seconds to roll from 45 degrees of bank one way to 45 degrees of bank the other way—all the time the mountain was filling their visual field. The crew continued to try and haul the aircraft around the corner, following the briefed plan. The crew could have continued the left turn after the DZ and escaped back up the valley that they had just flown down, but instead struck the ground 28 seconds after passing the DZ with 10 degrees of right bank and 15 degrees nose high. Forward speed was 87 KTS and the rate of descent was 1,600 feet per minute. All crew were lost on impact.

This operational crew was conducting training; they were not a student crew who needed close supervision. Moreover, they were actually overseen by an experienced IP and IN, and the planned route was in an area that had been used many times before. What role does complacency play in this scenario? The plan for this sortie was undoubtedly flawed, but no one spoke up. How does crew resource management training lead to open questioning of a potentially unsafe act or situation?

Amongst other things, CRM teaches flight crews how to examine communication skills, interpersonal duties such as leadership and coordination, effective team formation, problem-solving, decision-making and maintaining situational awareness. Over the last few decades, CRM training programs have focused on the development of specific skills in the “non-technical” areas of communication, situation awareness, problem solving, and stress management. These skills are now seen as critical operational countermeasures with respect to error occurrence and management, and accordingly have the potential to mitigate the effects of unfamiliarity on crew performance (Thomas, 2006). The airlines tried to familiarize the cockpit and cabin crew with the above factors to encourage them to perform effectively as a team. Despite this, there has been an unrelenting division of aircrew based on crew position in times of routine operation and emergencies (Chute, 2002). In order to identify the problems that exist on the flight deck, a method of data collection had to be established. In May 1975, the Federal Aviation Administration (FAA) implemented a confidential, voluntary, non-punitive safety reporting program in an effort to increase the flow of information from aircrew regarding actual or potential discrepancies and deficiencies. As could be predicted, crews and controllers were concerned about the use of the data by the agency responsible for enforcement of regulations. Because of those fears, the FAA turned to the National Aeronautics and Space Administration (NASA) to serve as an objective, disinterested party for data collection, analysis and dissemination (FAA, 1976).

The Aviation Safety Reporting System (ASRS) was designed to stimulate a large flow of information concerning errors and operational problems in commercial air
operations. It was recognized that a voluntary system could not, in all probability, provide a comprehensive description of the frequency or prevalence of problems in the aviation environment. NASA implemented ASRS in April 1976, and in the first 7 years there were 35,000 reports logged from pilots, controllers, and armed forces (Billings, 1984). Clearly, a system like ASRS has proven to be an invaluable resource of information about behavioral problems in aviation. For example, in a single year (1990), there were 8,780 altitude deviations uncovered; that averages one each hour, 24-hours a day, 365 days a year. The advent of the National Air Traffic Control System (NATS) in the United Kingdom brings coordination between aircrew and terminal services to the 21st Century by delivering enroute air traffic services and airport optimization services. Advances in these services should have the effect of improving coordination, but does not eliminate the intra-cockpit distractors that lead to errors. In the altitude deviation example, the reason identified for a majority of the errors was cockpit duties and distractions resulted in only one flight crew member monitoring the ATC frequency (George, 1993).

Similarly, controller workload and frequency congestion are factors which affect the ability of controllers to closely monitor pilot read backs. The statement “Most accidents are the product of ‘Human Error’” is a convenient layman’s label, and can be misleading. The term “error” tends to be used in a pejorative and negative sense in this context. More importantly, it tends to carry with it an implication of “blame.” Also, it can have the effect of placing the focus too narrowly on an individual—often the individual at the sharp end, the frontline operator (e.g. pilot, driver, etc.), whose actions were temporally closest to the final outcome. Although such an approach may be seductive and temporarily convenient, it is neither appropriate nor helpful. In failing to take the investigation back beyond the individual operator, the context of the accident will be lost, the responsibility of others obscured, and a proper root cause analysis rendered impossible. Focusing blame on the individual leaves little room for appropriate countermeasures; disciplinary action or exhortations to “take greater care” are unlikely to prevent a recurrence. Errors are rarely a result of a catastrophic lapse of concentration, so identifying the root cause often reveals a subtle breakdown in procedures.

Procedural drift refers to the tendency to become complacent with procedures, and in some cases willfully infringe rules and procedures. Where clear rules and procedures are not available, aircrew often draw on personal experience to identify a suitable course of action. When the solution is offered by a subordinate crew member, the sociotechnical aspects of the cross cockpit hierarchy may come into play and interrupt the communication. The focus of research to date has been on the timing of communication between crew. Generally there is a predefined time in the sequence of a checklist or troubleshooting when the cross-flow occurs. The impact on task performance is directly influenced by the communication process. New research is addressing the movement from two-person crew to single crew or fully automated flight. These task models highlight the necessity to define how CRM will be supported in a broader Air Traffic Control/Terminal Operations model (Cahill, 2014).

Cross cultural influences on effective CRM have been a topic of interest in the last decade. As difficult as it was to broaden the communication channels between a senior flight deck officer and his subordinates, the divide within certain cultural mores is evident through accident data. Accident rates differ among global regions; Asia and Africa have higher accident rates (5.1 and 8.0 accidents/million departures, respectively) than either America or Europe (1 – 1.5 accidents/million departures).
underlying causal factors also show differences. In Asia and Africa, failures in crew resource management are the most frequent circumstantial factor in accidents. An analysis of accidents involving aircraft from Taiwan (Republic of China) using the Human Factors Analysis and Classification System found that poor CRM was related to subsequent errors in decision-making, perceptual errors, and violations in procedures. (Li, 2008). These error categories showed a 30- to 40-fold increase in their likelihood of occurrence in the presence of poor CRM. Much of the literature and training that addresses CRM comes from the Western culture orientation. There are fundamental differences in the mental models of Western culture. Westerners tend to adopt a function-oriented model; stimuli are grouped in terms of their purpose and connected to a task-oriented operating concept where specific actions are performed to achieve well-defined results. This typically results in a preference for a sequential approach to undertaking tasks (e.g. checklists and standard operating procedures mentality). The Asian preference is for an integrated, thematic approach; stimuli are grouped in terms of common, generic interrelationships and a task-oriented operating concept contradicts their preferred method of working.

The fatal crash of Asiana Airlines Flight 214 at San Francisco International Airport on 6 Jul 2013 was caused by the pilots mismanaging the descent toward the airport and not aborting the landing to try again, the National Transportation Safety Board reported. Contributing factors included the complexities of the Boeing 777’s auto-throttle along with inadequate pilot training and monitoring. Although not directly listed as causal, the experience of the crew created a cockpit gradient that may have influenced the outcome; the Captain was very senior in time and experience compared to the First Officer, and culturally this is the kind of set-up that can delay communication. Touching on flight crew communication, the NTSB highlighted:

Cockpit Voice Recorder (CVR) recordings also indicated that the Pilot Flying (PF) did not tell the Pilot Monitoring (PM) that he was stressed about performing the approach without the benefit of an electronic glideslope to aid him in managing the airplane’s vertical path. If the PF had expressed his concern, the PM might have discussed the availability of cues like the altitude range arc or suggested alternate methods for conducting the approach, such as flying a VNAV approach that would have provided precise vertical guidance via the flight director. The PF was likely reluctant to acknowledge his lack of confidence in this area, and, as a result, the flight crew missed an opportunity to discuss ways that they could have effectively managed the airplane’s vertical path (NTSB, 2014).

Dynamic environments and the activities within those environments pose the additional challenge of coping with a degraded situation, such as the Asiana B-777 flying below the glide slope without the support of typical navigation aids. CRM principles must be embedded to a range of crew activities. There are myriad examples of crews being rushed, for example, during the approach phase that results in a reportable incident. Include in the mix of cockpit duties, the dynamics of a multi-cultural crew and unpredictable nature of passengers, and the recipe for disaster assembled. Aircraft manufacturers are now designing 800 plus passenger aircraft and an increasing number of international airlines employ cockpit and cabin crews recruited from different countries and distinct cultures. In those circumstances, the need for inter-cultural mixed CRM training arises. Furthermore, in view of the increasing use of multi-national crews
by major international airlines, the need for universal inter-cultural training programs is also increasing (Metscher, 2009).

In a parallel to the cultural considerations on the flight deck, crew familiarity has been implicated in the degradation of crew performance. For instance, even the two-person crew of the modern flight deck can be seen as susceptible to “groupthink,” a term coined to describe the negative effects of team cohesion on effective decision making (Thomas, 2006). Safety-critical contexts of command and control further highlight the importance of crew coordination.

In a study that reviewed the effect of crew familiarity on error rate and type, error occurrence was found to be significantly higher for unfamiliar crews. The protocol set up 154 flight sectors with crew rostering reflective of typical short-haul operations. In the data set, a total of 509 individual error events were observed, with an average rate of 3.31 errors per flight sector. The average number of errors per flight sector operated by unfamiliar crews was 4.09, compared with an average of 2.96 errors per flight sector operated by familiar crews. The types of errors made by unfamiliar crews differed significantly when compared with those made by familiar crews. Unfamiliar crews were found to make proportionately less errors relating to flight controls, checklist use, standard calls, and monitoring and cross checking, and proportionately more errors relating to paperwork and external communication (Thomas, 2006). Coupling the understanding of cultural mores, the familiarity of crews, and the construct of CRM, the training programs of the future will prepare crews for a broad variety of eventualities in dynamic environments.

Whether the environment is a flight deck, tarmac, or maintenance facility, the interruptions to checklist procedures can be detrimental to the cognitive and complex tasks. A study of task interruption looked at the factors that modulate how command and control (C2) operators cope with task interruptions in complex dynamic situations. In the study, team members were interrupted during a high work load scenario, and during the interruption a display was removed, communications were disabled, and the participant was asked to assess the state of the situation at the time of the interruption (Tremblay, 2012). Analysis of interruption-based performance measures revealed a benefit of teamwork over individual work when interrupted in a complex dynamic environment. The generalization of the findings would indicate that communication in a C2 role is enhanced by having more than one person monitoring and coordinating activities in a high work-load environment, but the size of a crew, the nature of the interruption, and the training-based experience of the crew must be considered.

4.1.4.2. The Medical Community. Concentration and communication are a familiar pairing in the medical community, and like the aviation environment, there are protocols and checklists to follow (Figure 4.1.4.2-1). The introduction of CRM to the operating theater has been on-going for well over a decade. Aviation-based CRM, however, cannot be imported wholesale into health care organizations (Musson, 2004). Training and procedures have to be customized to the industry. Likewise, short didactic training sessions are unlikely to effect a cultural change in health care. As the process of integrating CRM in aviation has been a multi-generational approach, the success of a singular CRM tool in the medical arena has not been universally received. However, in some locations, the entire concept of CRM was introduced into clinical areas with success. Haller and colleagues introduced a 2-day CRM training program taught by aviation experts that was designed to improve teamwork and communication skills to the obstetrical units of a large academic hospital. Over a year of observation after
training, there was a positive change in the team and safety climate in the hospital, as well as improved stress recognition (Haller, 2004).

Figure 4.1.4-1. Common CRM Traits: Assertiveness, Conflict Resolution, Feedback, Problem Solving

In this program, communication and team skills were emphasized with introduction of a pre-procedure briefing. Compliance with the brief/debrief “time out” local policy rose from 6.7% pre-CRM to 99% four months following CRM training (Ricci, 2012). Other groups have introduced preoperative briefings to the operating room with a consequent reduction in surgical interruptions and delays. This outcome benefits not only the patient, but also reduces costs associated with malpractice and improves the sense of purpose across the specialty team.

Not unlike advanced flight simulators, the medical community is now utilizing advanced simulators to demonstrate CRM. High-fidelity simulation-based training complements medical training in patient care settings. Moreover, simulation-based training reduces medical error, enhances clinical outcomes, and reduces the cost of clinical care. Health care providers are increasingly trained in specific medical skills and difficult team procedures, and crew resource management is used to address aspects of human error, such as failures in interpersonal communication, decision making, and leadership. Just as in aviation scenario training, medical CRM provides a set of error countermeasures with three successive lines of defense: 1) avoidance of error, 2) trapping incipient errors before they are committed, and 3) mitigating the consequences of the errors that nonetheless occur (Drooh, 2012). Although it is difficult to measure outcomes of training, experienced intensive care unit (ICU) nurses, board-certified intensivists, and ambulance drivers reported the training to be useful; the general consensus among those who received the CRM training was a feeling more confident about their skills after practicing the logistics of the transport process, with an increased awareness of medical, technical, or purely transport-related issues. This is a very good parallel to the practice of CRM in aviation and other environments.

4.1.4.3. The Maintenance Community. The utility of CRM is not lost on the aircraft maintenance community. A total of 18% of all USAF-reportable aviation mishaps are attributable to aircraft maintenance actions (Figure 4.1.4.3-1). These maintenance-related mishaps cost an average of $25 million per year. Increased ops tempo, limited resources, and other factors put increasing strain on maintenance organizations. Maintenance Resource Management (MRM) traces its roots back to cockpit resource management (CRM). The objective of MRM training is to improve understanding and application of the key human factors skills that improve combat capability, as well as safety. Mishap analysis of maintenance-related human factors
has been generally overlooked because the impact of maintenance malpractice may not be linked, proximally, to a mishap. These missed opportunities may foster an environment where preventable mishaps threaten the people, planes, and missions.

Figure 4.1.4.3-1. Systems Specialists with Team Approach vs. the Result of Poor Cross Check on Tech Data

The Air National Guard were first in the Department of Defense to propose a solution to reduce aircraft mishaps by developing a program of training and tools to help leaders at all levels target aircraft maintenance-related human factors issues. The objectives of a maintenance resource management program must define the problem of preventable maintenance mishaps, focus on the six human factors areas contributing to maintenance mishaps, identify the root causes of maintenance-related mishaps, examine the top aviation maintenance errors and give real-world examples of each, analyze interactive case studies of a real-world, maintenance-related, fatal mishaps, and provide tools for reducing the risk of human factor-related mishaps. By no coincidence, effective MRM parallels similar programs by focusing on maintenance organizations effectively working together, increasing individual and unit coordination to enhance mission effectiveness by minimizing maintenance preventable errors, maximizing maintenance resources, and optimizing risk management at all level of the organization.

The high profile disasters of the Space Shuttles Challenger (28 Jan 86) and Columbia (01 Feb 03) led a NASA team of government employees and civilian contractors to take a hard look at their organizational culture. Post-mishap investigation revealed that line-level maintenance workers had identified the causal factors in the mishaps, but that they were not able to communicate their information/concerns to the people in a position to accept the risk or correct the deficiency. As is often the case in large organizations, mid-level management was reluctant to communicate “bad” news; in this case, there were potential deficiencies that needed attention prior to launch. Although the senior leadership preached safety first, in reality, the mid-level management inactions sabotaged mission success and led to tragedy.

As a result of the shuttle disasters, two NASA contractors implemented organizational “Time-Out” programs that serve as excellent models to encourage a proper safety culture that permeates all levels of the organization. Unfortunately, it took the tragic loss of 14 astronauts and two expensive national assets. Instead of re-learning the same harsh lessons, we can learn from NASA’s misfortunes and incorporate a management style that gives the line-workers—the ones with the closest eyes to the mission—a way to communicate safety concerns. The ultimate test of crew
coordination comes in a situation when the crew is separated by time or distance; such is the circumstance that exists between the controllers and engineers at the Johnson Space Center and the crew on board the International Space Station (ISS).

Early in 2015, the ISS crew was alerted to a potential ammonia leak (NASA, 2015). The alarm was raised at 4 a.m. ET, when Mission Control saw pressure changes that could have been caused by an internal leak in the station’s coolant system, which uses water on an inside loop and toxic ammonia on an outside loop. Crew members put on emergency masks, powered down the systems on the station’s U.S.-built segment, moved into the Russian segment and closed a connecting hatch. The ammonia coolant system services only the U.S. side of the station, so controllers said there was no risk to the crew.

A leak of toxic chemicals inside the station’s cabin is one of the top three anomalies that the space station’s crew has been trained to handle. The other potential anomalies are rapid depressurization and an onboard fire or smoke event. The crew and mission control team actually practice the response plan on the ground before launch and again once in orbit. The coordination of actions, troubleshooting, and recovery options are critical elements tied to the success of the mission.

In a worst-case scenario, the crew could be forced to board the Soyuz capsules that are attached to the station and fly back to Earth, leaving the orbital outpost empty. The CRM effort is well-practiced and focused on the kinds of emergencies that could harm the crew; the CRM effort is focused on dealing with the issue, and then if the issue is unresolved, a back-up plan (like evacuation of the ISS) is elected.

4.1.4.4. **Summary.** Traditionally, cockpit resource management emphasized an awareness of pilot (crew) attitude and personality factors. The objective of the formal training programs was on more effective communication; often fairly intangible skills. As programs have developed and been introduced to a wider audience, there has been a shift toward more trainable skills that help a team manage resources more effectively. The translation of general CRM skills is now translated into operational procedures and task-based actions. The CRM skill-based training can be complex and many of the issues are not yet resolved by research or practical application. The skill-based training should be focused on task-specific events (e.g. scenarios), built on some amount of practice (e.g. recurrent drills), and balanced between both technical and human behavior. Development of efficient teams and integrating CRM into a safety culture is not a destination; fully embracing the concepts discussed in this chapter are but a sample of elements that are part of the daily journey toward a complete crew.
References
Federal Aviation Administration Advisory Circular; Aviation Safety Reporting Program. FAA AC 00-46A, March 1976.

Recommended Readings

Concepts
Components of Crew Resource Management
Utilization of CRM Principles outside of Aviation
Development of Effective Teams

Vocabulary
Efficient flight operations
Human error
Interpersonal communication
Decision making
Maintenance resource management
4.1.5. **Human Factors Associated with Remotely Pilot Aircraft**

Andrew D. Woodrow, Lt Col, USAF (Ret), BSC and Maj Dorian Williams, USAF, BSC

4.1.5.1. **Emergence of the Remotely Piloted Aircraft.** In the decades since unmanned aerial vehicles (UAVs) or remotely piloted aircraft (RPAs) were assigned operational duties in the Department of Defense, great challenges for the human systems integration field have been tackled and, in some cases, conquered. Missions have expanded well beyond defense and now include border patrol, homeland security, crop management, and the entertainment industry. This rapid growth has outpaced our understanding of the boundaries of human integration, creating new challenges for recruiting, training programs, and operations. As the field continues to expand, the implication that there are “no humans” integrated in the RPA system has been the first hurdle to cross. In reality, there are often more people involved in the direct operation of an RPA system than some manned systems. The US Air Force identified this and between 2008 and 2014 more than tripled the number of its active-duty pilots flying RPAs, including the General Atomics MQ-1 Predator, the MQ-9 Reaper, and the Northrup Grumman RQ-4 Global Hawk, as well as a number of operational prototypes. The increase in demand and the introduction of more capable platforms that carry multiple payloads have resulted in a significant increase in the workload for RPA pilots and sensor operators, but insufficient research and training tied to human system interface has stifled optimal growth. The scope of RPA-specific human factors concerns is not terribly different than that of conventional manned-aircraft. The good news is that there are cross-over human factors issues between manned and unmanned systems that impact design, training, and certification, so there is baseline research available for RPA. One significant difference between the manned-to-unmanned systems is how the operator receives and processes input from the aircraft. In particular, the challenges of telemetry latency, lag instruments, flying without sensory cues, the relationship of pitch sensitivity to airspeed, and vertical speed analyses. Some of these elements have been examined through the Military Flight Operations Quality Assurance (MFOQA) process in an effort to improve the ability to quantify the associated challenges and hazards identified by flight crews. The progress made in engineering crew stations and human interface for the RPA is the focus of this section.

4.1.5.2. **“Stick Forward, Houses Get Bigger; Stick Back, Houses Get Smaller.”** No discussion of human flight is complete without a review of sensory input that aids the pilot in keeping the aircraft pointed in the right direction. Very specific to the RPA community is the lack of sensory feedback to the operator. The issue of instrumentation/sensory feedback as a factor in Air Force RPA mishaps raises several interesting points. Compared with crews of manned aircraft, the RPA operator is relatively sensory deprived, lacking peripheral visual, auditory, and proprioceptive cueing. However, the effect of this sensory deprivation has not been well researched. Sensory awareness is our most vital characteristic; unduplicated at the same level of accuracy or sensitivity by computers. Orientation to one’s environment is tied closely to the ability to navigate within that environment; otherwise, a sense of place must be complimented by a sense of direction. The human typically relies on physical signals (visual, auditory, and proprioceptive) to assess status in a dynamic environment. In the case of the vestibular system, processing physical stimuli functions as an inertial
navigation system and is associated with postural stability, balance, and the stabilization of the eyes to enable visual acuity in the presence of head movement and locomotion. In the absence of vestibular cues, motor performance is controlled by cognitive processes and based primarily on the discrete-continuous dimension (Stott, 2011). Tasks like pressing a key or throwing a switch are discrete because the action has a beginning and an end. Actions like steering an aircraft or operating a slewing camera are continuous. An activity such as gathering data from sensor input, applying commands for the aircraft to maneuver, and waiting for feedback through visual cues involves a series of discrete actions. The open skills demonstrated in the RPA battlefield are performed in a dynamic environment, and the skills require rapid adaptation to changing demands of the mission. Most importantly, when comparing feedback typically received on a flight deck compared to the ground control station (GCS) (Figure 4.1.5.2-1), one must analyze motor programs through a new lens or scope. Motor programs are regarded as an abstract plan for controlling specific classes of movements; particular movements that require muscle movement are influenced by force, duration, and timing of contractions. In a stationary GCS, the operator is presented with a broad variety of visual and some auditory cues, but the body is devoid of proprioceptive cues normally associated with aircraft position. Instead, instrumentation and skill-based habit patterns must take the lead on orientation cuing. Review of engineering, operations, and human integration allows on-going, in-depth study of leading indicators of errors and the development of mitigation strategies.

Figure 4.1.5.2-1. MQ-9 Ground Control Station

4.1.5.3. Highly Complex Even at Ground Speed Zero. Research and operational experience has been growing, to include supervisory control, flight control interface, workload and automation, cognition and the impact of large RPA team, and even spatial disorientation. In many respects, the development of the RPA systems has suffered from similar growing pains as early aviation; the obvious difference has been the loss of airframes has not been accompanied by a loss of aircrew lives. Learning a new system and applying terrestrial skills to an aircraft that operates remotely seems to challenge the conventional pilot training model. In a typical scenario over the battlefield,

MQ-1/9 RPA aircrews are communicating in real time with joint terminal attack controllers (JTAC), ground forces, supported units, air traffic control (ATC), and other air assets. During an actual engagement, RPA crews communicate directly with the JTAC via secure radio onboard the aircraft or
through a number of other alternate means. Many RPAs are capable of providing real-time full-motion video (FMV) to the JTAC, the tactical operations center (TOC), the ground forces commander, or any other ground forces through remotely operated video-enhanced receiver (ROVER) or other remote video terminals (RVT). Emerging technologies will allow ground forces to provide enhanced targeting information back to RPA via NextGen RVT (James, 2014).

Complex mission sets that include precision weapons effects (e.g., Close Air Support, Search and Recovery), multiband communications, and crew interaction highlight that the integration and training of the human-in-the-loop become increasingly important. Diverse target sets and unknown conditions stress the crews in their ability to maintain situational awareness beyond conventional capabilities. In the context of workload, the distribution and preparation of RPA crews is central to the effectiveness of the mission. Data from the Office of the Under Secretary of Defense for Acquisition, Technology, and Logistics indicates that RPA flight hours rose from a couple thousand in 1996 to approximately 115,000 in 2005 (OSD, 2004). In particular, flight hours on the RQ-1/MQ-1 Predator tripled in the period from 2003 to 2007 (Brook, 2007). After an initial spike in aircraft crashes, safety statistics indicate a tremendous decrease in the number of lost aircraft. Aside from reliability of the machine, the reliability of the operators is a growth industry that is central to the studies undertaken by flight medicine, aviation physiology, human factors engineers, and aviation psychologists.

4.1.5.4. Recruiting for Success in the RPA. One area that is not typically reviewed under the banner of human factors is manpower. However, if the cadre recruited and trained for RPA missions self-eliminate or is not retained after the first assignment, the value of the effort is lost; this is where human factors analyses may help to frame the process of pilot selection and training. The thrust of the manpower analysis effort has been central to the concern over recruiting and retention of RPA crews. When Undergraduate RPA Training (URT) was established, the Air Force chose to use selection criterion that had demonstrated predictive validity for manned aircraft training rather than develop a tailored approach. Not long after URT was fielded, questions about the validity of the standard screening exams were raised. More recently, as part of a long-term research program to improve the assignment of officers to aircrew training specialties, a research group focused on factors unique to the RPA work environment as opposed to those applicable to both manned and unmanned aviation. These reflected both positive and negative aspects of the RPA work environment (Carretta, 2013). Examples included:

1. The opportunity to work with cutting edge aircraft technology
2. The opportunity to take lethal action against enemy combatants when authorized to do so
3. Participating in combat operations from a remote location
4. Work performed in shifts which may entail rotating, overnight, and/or long shifts
5. Potentially limiting the amount of time available to spend with friends and family

6. Requirement to work as a member of a team

7. Activity level is typically low (vigilance problem), with periods of intense activity

8. Working in a controlled environment, where one is required to simultaneously monitor multiple information sources

The quality of life for crews of any flight platform is central to the overall operational effectiveness; this is an aspect negatively affecting the life of RPA pilots as the mission requires a unique balance between war fighting roles with personal lives. While the RPA platforms and their support personnel are forward based at or near conflict zones, the pilots and systems operators are typically based stateside and live at home (Figure 4.1.5.4-1). While RPA ground crews are closer to dangerous environments, they seem to cope better with the stress than home-based crews.

Figure 4.1.5.4-1. RPA Ground Crews vs Home-Based Crews

This scenario, called “deployed on station,” exposes RPA crews to the combat experience alongside their personal lives, and has been shown to negatively affect morale. Compounding the challenge to morale, excessive workload causes dissatisfaction among RPA pilots. In 2008, the Air Force determined the optimum number of RPA pilots for some of the units but has not updated this ratio to reflect the growing mission diversity and workload. For example, in the mid 2000s the crew ratio for the MQ-1 Predator was determined to be 10:1; ten crews supporting a Predator RPA on a 24 hour mission. Since then, the Air Force has introduced the MQ-9 Reaper, which carries more weapons and sensors and can fly longer missions, but the crew ratio for the Reaper is still 10:1. Longer mission, more complex system, yet the same crew ratio. The GAO found that in most units, the ratio is significantly lower than this goal (GAO, 2014). “Low crew ratios diminish combat capability and cause flight safety concerns” the report stated, adding that “the Air Force not only operates below the optimal ratio, it hasn’t met even the minimum crew ratio.” In 2012, the Air Force filled only 82 percent of its RPA training slots, while virtually all manned aircraft slots were filled. As of early 2013, according to a report by Air Force Col. Bradley T. Hoagland, a command pilot, the Air Force Academy had only 12 volunteers for its 40 RPA training
slots (Carroll, 2013). High work demands on RPA pilots limit the time they have available for training and development and negatively affects their work-life balance.

4.1.5.5. The Role of Gaming in Recruiting and Training. The established role of human error as a significant and consistent cause of aircraft accidents sets the stage for an in-depth analysis of post-accident data. Although some elements of RPA operations are autonomous, the human integration across the mission profile remains central to the successful employment of the aircraft. The comparison of video gamers and RPA pilots/systems operators has been at the forefront of performance comparisons. In several ground control stations, the prevalent use of flat-screen monitors, keyboard and mouse control inputs, online “chat” functions across several platforms, and a game-like flight stick/throttle, easily makes the comparison with the traditional video game environment. A wide variety of studies have concluded that non-pilots with minimal piloting training could be just as competent as manned aircraft pilots in operating unmanned aerial systems (Schrieber, 2002). It is also suggested that video game players (VGP) can track more targets and may benefit from superior stimulus response mapping in visual tasks. Additionally, experienced VGPs enjoy enhanced spatial skills, improved psychomotor skills, and quicker reaction times. Perhaps most importantly, in the context of an aviation application, these skills seem to transfer to other cognitive tasks and environments. Boot et al. (2008) found experienced VGPs exhibited a variety of cognitive differences when compared to non-VGPs, such as the ability to track targets moving at increased velocities, superior ability to detect subtle variations in objects stored in short term memory, faster attention switching between tasks, and improved efficiency in a mental rotation task. This is an important data point to consider in the pilot screening process. The Initial Flight Training (IFT) program conducted by the Air Force aims at establishing a baseline of basic flying skills once the candidate has demonstrated accepted minimum standards for airmanship using a standardized computer battery.

A 2011 study compared pilots to video game players on selected performance outcomes in an attempt to discover whether VGPs possess superior UAV-relevant cognitive skills when compared to manned aircraft pilots (McKinley, 2011). The data suggest that pilots do not hold a performance advantage over VGPs in landing a simulated Predator aircraft, although both groups performed significantly better than the control group. Pilots outperformed VGPs, but the only statistically significant difference was the glide slope root mean squared error (RMSE) parameter. The data also suggest the VGP group, on average, performed significantly better when compared to both pilots and control group subjects on the “Motion Inference” task (McKinley, 2011). This task refers to the ability to “perceive and process both the motion of an object and the estimate trend information so as to predict its position at a future point in time even when direct line-of-sight cannot be maintained continuously.” Successfully controlling an aircraft is only one aspect of what makes a successful pilot in the military. Modern aviators of manned aircraft are trained to communicate with Air Traffic Control, ground units, and other aircraft through multiple complex radio conversations; effectively, the pilot is responsible for mission planning, mission execution, aircraft recovery, and completion of any other post-mission requirements which require a thorough understanding of the aircraft, the environment, rules and regulations regarding operations, and the overall mission. For the RPA pilot/crew, this task-set may also include employment of weapons, which requires a full understanding of the rules of engagement—far more important in the field of battle than in a video game.
The current generations of RPA crews are also facing the challenge of scaling up to an environment that engages tens of aircraft in a single mission. Research has demonstrated operator performance controlling even a single RPA is significantly degraded when heavy demands are imposed by payload operations. This would suggest the ability of an operator to attend to multiple aircraft might be severely compromised under non-idealized conditions, especially if an aircraft is malfunctioning or damaged (Wickens, 2002). This places the operator in a task loading situation that may require monitoring multiple task sequences simultaneously while maintaining intense communication with other control teams. The importance of task characteristics including time spent, time to proficiency, and consequences of error and the ability to quantify error are central to team task analysis.

4.1.5.6. Crew Resource Management in RPA. The curriculum of crew resource management (CRM) programs introduced in the 1970’s initially evolved from traditional crew-based flight decks to single seat aircraft. The underlying objectives remain the same: utilize available resources to maximize performance and effectiveness. The extensive literature draws a distinction between teamwork and task work, where teamwork refers to the team’s efforts to facilitate interaction among team members in the accomplishment of team tasks. In contrast, task work refers to the team’s efforts to understand and perform the requirements of the job, tasks, and equipment to be used (Arthur, 2012). If a system is designed to reduce the task work (e.g. through automation), there remains a challenge to sustain adequate teamwork across the mission. Although automation has relieved RPA crews of some amount of task work, the approach to autonomous systems does not have the same outcome across all crews. Workstation design and layout can also contribute to the effectiveness of crew teamwork and accomplishment of team tasks (Figure 4.1.5.6-1).

Figure 4.1.5.6-1. MQ-1/9 Operator Control Station with Obvious Design Limitations

The terms used in workload and automation tasks are compliance and reliance. Compliance refers to a user following alerts presented by the system; for example, a navigation system that provides turn-by-turn directions to a driver. If the driver follows
the direction without first cross-checking, the user is said to be compliant with the system. Reliance refers to the use of automation when it is silent (e.g. no alert signals). The same driver, now on an unfamiliar route and waiting on the navigation system to prompt each turn, will likely miss the waypoint because he is relying on the system (McBride, 2011). Both younger and older crews tend to rely on automation more than they comply with system prompts. Dixon (2007) found that false alarms tend to suppress both compliance and reliance behavior; imperfect automation systems tend to elicit more supervisory roles in crews, and this role may increase the workload since monitoring automation is an additional task demand.

Synthesizing the RPA team takes many of the same skills sets as legacy aircraft platforms. Effective team performance, then, relies on shared mental models. Team members should have mental models of the technology employed and the aircraft systems. If individuals comprehend how their actions interact with those of other crew members, then task strategies, contingency plans, and organized knowledge of the system status leads to the coordination of an action. The synchrony and sequencing of individual behaviors in a segment of time and space are necessary for effective performance to achieve team goals (McGrath, 1999). The feedback loop commonly witnessed on a flight deck is complicated in the RPA mission since there are several key members of the mission are separated by distance and time. The team modelling is further complicated by the requirement to manage, monitor, and support multiple numbers and types of RPAs conducting a variety of missions over a single battlefield at the tactical level. An example from an Air Operations Center (AOC) depicts this complex crew effort:

The team in the AOC needs to understand the risk associated with flying in certain conditions. The on-site weather officer at the ground control station (GCS) advises the operators in the GCS to bring the RPA back to home station because the weather is deteriorating. At the same time, the commander in the AOC might direct the GCS to remain on station to complete the mission and provide the ground commander the support that has been requested. The GCS then informs the commander that they will follow the order to stay on station, but not without risk of losing the aircraft. The AOC team did not realize the situation with deteriorating weather, and once it was communicated, the AOC might say, “By all means, head for home!” (DeJoode, 2006).

4.1.5.7. Human Systems Integration. The leading method for tying the elements of human performance together in this, and other complex systems, is Human Systems Integration (HSI). Coupling legacy human factors considerations with a contemporary understanding of engineering the human into a system, HSI brings together a variety of specialties to evaluate specific domains relevant to the challenges of the RPA system. The domains include personnel selection, training program syllabus, environmental safety, habitability, survivability, and human factors engineering. Effectively, HSI aims to describe a model that identifies the elements that can be manipulated to optimize performance. A leading expert in the area of RPA-human interface, Anthony Tvaryanas, describes the benefit of HSI this way:
A program forced to accept shortfalls in cockpit design (human factors engineering domain) could respond by augmenting training (training domain) or selecting more capable or experienced aircrew (personnel domain). In addition, by providing an understanding of the underlying structure (“the anatomy”) and processes (“the physiology”) of human performance within complex systems, the HSI model has utility for clinically oriented aerospace medicine practitioners responsible for anticipating or diagnosing degraded performance (Tvaryanas, 2006).

Operational issues can be traced to critical gaps in Mission Management (MM) capability (Table 4.1.5.7-1). Each issue and referenced MM capability has a proposed “lane” for reconciling the issue; all have a link to human factors and human systems integration.

<table>
<thead>
<tr>
<th>Operational Issues</th>
<th>Mission Management Gaps</th>
<th>Technology, People, Processes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Manning &amp; Selection</td>
<td>Insufficient/Inflexible Automation</td>
<td>Develop Flexible Automation &amp; Improve Processing</td>
</tr>
<tr>
<td>Airspace Deconfliction &amp; Management</td>
<td>Poorly-Designed Operator Control Stations (OCS)</td>
<td>Properly Architect OCS &amp; Enhance HMI</td>
</tr>
<tr>
<td>Collateral Damage</td>
<td>Limited Communications Systems</td>
<td>Create Robust Communications Architecture</td>
</tr>
<tr>
<td>Contested Environment Operations</td>
<td>Inadequate Selection &amp; Training</td>
<td>Develop Targeted Selection &amp; Effective Training</td>
</tr>
</tbody>
</table>

### 4.1.5.8. RPA Accidents and Analysis.

One of the major problems in any aviation system is the inevitable accident. In the RPA community, the nature of the mission itself, such as sustained loiter times, multichannel communications, and frenetic battlefield, puts an extreme load on the crew’s capacity. In order to fully appreciate where errors occur, there must be a consistent system employed to identify the problems. Classifying errors associated with mishaps and accidents has been reasonably successful using the Human Factors Analysis and Classification System (HFACS). Initial review of RPA mishaps by the Defense Service Board identified human factors, including 1) limited experience level of RPA operators and maintainers, 2) inadequate overall professional development of RPA personnel, and 3) the need to better address takeoff and landing errors (SecDef, 2004). Of the 221 RPA mishaps occurring during the period of fiscal years 1994 – 2003, 17.2% involved the RQ-1 Predator, 57.5% the RQ-2 Pioneer, 1.8% the RQ-4 Global Hawk, 11.3% the RQ-5 Hunter, 9.0% the RQ-7 Shadow, and 3.2% miscellaneous or unspecified RPAs. Overall, 60.2% of mishaps involved operations-related human causal factors (Tvaryanas, 2006).

The USAF uses a systematic process to identify, classify, and analyze human error—the Department of Defense Human Factors Analysis and Classification System (DoD HFACS) (see also Section 8.4). According to Air Force Instruction 91-204, DoD HFACS is the primary model used to analyze the interface between the person and other elements in the system: software, hardware, and the environment. DoD HFACS presents a systematic approach to error analysis and is the primary tool USAF accident investigators use to report and analyze DoD mishaps. The method used to catalogue the HFACS is a series of nanocodes, shorthand references for consistent reporting and ease of searching databases of mishaps. Investigators insert applicable HFACS nanocodes.
nanocodes into the mishap report for all findings identified in a mishap sequence. This model is designed to present a multidimensional approach to error analysis. A few of the most common nanocodes cited in RPA mishaps include:

- **PE205 Automated System Creates an Unsafe Situation**: This is a factor when the design, function, reliability, symbology, logic or other aspect of automated systems creates an unsafe situation.

- **PE206 Workspace Incompatible with Operation**: This is a factor when the workspace is incompatible with the task requirements and safety for an individual.

- **OP007 Purchasing or Providing Poorly Designed or Unsuitable Equipment**: This is a factor when the processes through which aircraft, vehicle, equipment or logistical support are acquired allows inadequacies or when design deficiencies allow inadequacies in the acquisition.

Surprisingly, skill-based errors rank in the highest of reasons for accidents in the RPA community. These are essentially errors that confound basic flight skills and entail highly automatized psychomotor behaviors that occur without significant thought (Wiegmann, 2003). The majority of the skill-based errors identified were procedural errors, where the technique employed by the operator unintentionally set them up for the mishap. There are currently vast differences between the services in the selection and training of RPA operators. The Air Force initially used experienced pilots who already had at least one operational tour of duty in another aircraft. By contrast, the Army and Navy/Marines have used enlisted personnel who are generally non-pilots and are given a RPA-specific training program. Time will tell whether previous experience in a manned aircraft is beneficial to RPA operators. Human Factors is not just about humans, it is also about how features of people’s tools, tasks, and working environment systemically influence their performance.

Very specific to the RPA community is sensory feedback to the operator. The issue of instrumentation/sensory feedback as a factor in Air Force RPA mishaps raises several interesting points. Compared with crews of manned aircraft, the RPA operator is relatively sensory deprived, lacking peripheral visual, auditory, and proprioceptive cueing. However, the effect of this sensory deprivation has not been well researched. The prediction and planning of RPA missions at the team level are often based on dynamic data. Time-sensitive targets, weather changes, and RPA resources (fuel load and weapon stores) are part of that dynamic information flow. Future design of RPA cells and operations are reliant on continued analysis of the human factors associated with the cognitive site-picture of every member of the team. Clearly a simple chapter on RPAs only scratches the surface of a very complex system. In order to fully appreciate the nature of the RPA mission, one must be immersed into the growing literature related to systems design and human factors related to the overall RPA system.
References


Concepts
Deployed on Station
Discrete-Continuous Actions
Sensory feedback
Shared mental models
Workload distribution

Vocabulary
Crew Resource Management (CRM)
Ground Control Station (GCS)
Human Systems Integration (HSI)
Human Factors Analysis and Classification System (HFACS)
Launch and Recovery Element (LRE)
Mission Control Element (MCE)
Remote Split Operations (RSO)
Remotely Piloted Aircraft (RPA)
Sensor Operator
Undergraduate RPA Training (URT)
Unmanned Aerial Vehicle (UAV)
Video Game Player (VGP)
4.2. Self-Impose Stressors

Dr. Mary T. Brueggemeyer, Lt Col, USAF, MC, FS; Capt James A. Dreibelbis, USAF, BSC; and Lt Col Ryan W. Maresh, USAF, Ph.D.

4.2.1. Self-Medication

4.2.1.1. Introduction. The ever-increasing availability of medications of all types creates an environment of risk for our aircrews. The aerospace physiologist must be knowledgeable about over-the-counter (OTC) medications approved for aircrew use, nutritional and herbal supplements, and the process for approval by the flight surgeon for usage. Lists of medications are useful, but the guiding principle for any medication usage is the potential for impairment of psychomotor function, vigilance, or disturbances of the special senses. In addition, the underlying condition for which medication is sought must be minor and not interfere with flight duties or the use of life support equipment. This chapter will review current USAF policy for OTC medication usage and issues and challenges with nutritional and herbal supplements and provide resources for future reference and study.

4.2.1.2. Current USAF Policy for OTC Medication Use in Aircrew. Table 4.2.2.1 lists OTC medications approved for occasional use by aircrew without flight surgeon approval. The condition must be minor and self-limited and not interfere with flying duties or life support equipment. Occasional use is defined as 72 hours of continuous use unless otherwise stated. Medications are listed by the generic name with examples of trade names given in parentheses. Any questions about a medication or condition should be referred to a flight surgeon.

**Table 4.2.1.2-1. OTC Medications Approved for Aircrew Use without Flight Surgeon Approval**

<table>
<thead>
<tr>
<th>Oral Medications for Occasional Use for a Maximum of 72 hr Continuous Use</th>
<th>No More than 2 Doses Per Week for Heartburn Symptoms Lasting &lt;48 hr</th>
<th>Topical Medications for Occasional Use (Condition Must not Interfere with Flying or Life Support Equipment)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acetaminophen (Tylenol)</td>
<td>Famotidine (Pepcid)</td>
<td>Hemorrhoid medications (Preparation H, Tucks)</td>
</tr>
<tr>
<td>Ibuprofen (Motrin, Advil)</td>
<td>Omeprazole (Prilosec)</td>
<td>Topical antifungals (Tinactin, Lotrimin)</td>
</tr>
<tr>
<td>Aspirin</td>
<td>Ranitidine (Zantac)</td>
<td>1% hydrocortisone cream (Cortaid)</td>
</tr>
<tr>
<td>Naproxen (Naprosyn)</td>
<td></td>
<td>Vaginal anti-fungals (Monistat)</td>
</tr>
<tr>
<td>Antacids (Mylanta, Maalox, Tums, Rolaid)</td>
<td></td>
<td>Anti-infectives (Polysporin, Neosporin)</td>
</tr>
<tr>
<td>Bismuth (Pepto-Bismol)</td>
<td></td>
<td>Analgesics (BenGay, IcyHot, Aspercreme)</td>
</tr>
<tr>
<td>Fiber (Metamucil, Fibercon)</td>
<td></td>
<td>Benzoyl Peroxide</td>
</tr>
<tr>
<td>Simethicon (Gas-X)</td>
<td></td>
<td>Chlorhexidine (Peridex)</td>
</tr>
<tr>
<td>Docusate (Colace)</td>
<td></td>
<td>Salicylic Acid (warts)</td>
</tr>
</tbody>
</table>

4.2.2. Nutritional Supplements

Current USAF policy on the use of nutritional supplements is found in the Surgeon General policy letter dated 28 Oct 1999. The only supplements specifically prohibited for use are anabolic steroids and hemp oil. Ephedra (Ma Huang) was removed from the market by the Federal Drug Administration (FDA) in 2004 following numerous reports of death and serious injury.

Official lists of prohibited or approved dietary, herbal, and nutritional supplements for aircrew use do not exist. All supplements (including pre- and post-workout products) must be reviewed and approved/disapproved by the flight surgeon and documented in the medical record. An SF600 overprint is available for this documentation (see Appendix 8: Internet Resources). Evaluation of a supplement product should include a review of all the ingredients, identification of intended effects, proof of efficacy, unintended side effects, potential for risk of sudden incapacitation, decrements in psychomotor functioning and higher senses, easily detectable adverse reactions, and compatibility with performance in sustained flying operations in austere environments. The risks from nutritional and herbal substances stem from the lack of regulation in the industry, as well as the lack of rigorous, controlled studies. Substances may not have consistent amounts of product, may not be efficacious, may contain pharmaceutical products or near relatives of pharmaceutical products, may have a narrow difference between therapeutic and toxic doses, and may contain contaminants such as heavy metals.

Although there is not an official list of approved or disapproved substances, some substances are known to have effects that could adversely impact aircrew performance. Familiarization with these substances will aid in the education of aircrew and avoidance of risk. These substances are listed in Table 4.2.2-1.
Energy drinks are a subset of nutritional supplements that should be evaluated by the flight surgeon before aircrew use. Energy drinks contain substances that range from caffeine to ephedra-like substances to psychotropic substances. Aircrew can be exposed to risk from the stimulant substances in these energy drinks along with other aspects of their diet. Aircrew should be educated on proper usage of caffeine as a counter-fatigue agent and avoidance of dangerous stimulants that may be in energy drinks.
drinks. The flight surgeon should be aware of and evaluate the energy drinks available on base and in the snack lounges of the flying squadrons. The physiologist should focus on educating aircrew about the potential risks and benefits of nutritional supplements, especially as they apply to the flying environment. In addition, the benefits of a healthy diet, adequate sleep, and exercise should be stressed. Common substances found in energy drinks are listed in Table 4.2.2-2.

<table>
<thead>
<tr>
<th>Table 4.2.2-2. Substances Commonly Found in Energy Drinks</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Caffeine</strong></td>
</tr>
<tr>
<td>Guarana</td>
</tr>
<tr>
<td>Inositol</td>
</tr>
<tr>
<td>Taurine</td>
</tr>
<tr>
<td>Super Citramax</td>
</tr>
<tr>
<td>(Hydroxy Citric Acid, Garcinia Cambogia Extract)</td>
</tr>
<tr>
<td>Glucuronolactone</td>
</tr>
<tr>
<td>Carnitine</td>
</tr>
<tr>
<td>Yohimbe</td>
</tr>
<tr>
<td>Panax Ginseng</td>
</tr>
</tbody>
</table>

### 4.2.3. Aeromedical Considerations of Supplements

There has been an influx of supplementation use for aircrew, as well as maintainers, security forces, air traffic controllers, and other fields in recent years. Nutritional/dietary supplements and ergogenic aids have become the cornerstone of modern day health and fitness. Many of these different supplements have a variety of potential benefits for the everyday individual, or even ground “War Fighters,” but they can also pose adverse effects. These detrimental effects can be exacerbated to a much greater extent in the aviation community, especially when compounded by even mild dehydration.

Many of these supplements have a nutritional benefit as long as the recommended dosage and use requirement is met. They can promote weight loss, stimulate metabolism to burn fat, increase strength and lean body mass, increase energy levels, promote focus, and develop increased nutrient and oxygen delivery to muscular tissue to improve stamina during times of exertion (Baechle and Earle, 2008; Warfighter, 2009). The potential side effects of certain supplements containing ephedra, or ephedra substitutes, can include tachycardia or ventricular arrhythmia/fibrillation, syncope, thermogenic reactions altering metabolism and body temperatures, hypertension, adrenal issues (rhabdomyolysis), dehydration, cramping,
nausea, and possible death if taken improperly (Baechle and Earle, 2008; Warfighter, 2009).

Caffeine is the most widely used stimulant in the world and very prevalent in the aviation community. It comes in many forms, but most commonly consumed in coffee, tea, energy drinks, and soda. Caffeine is readily absorbed through the gastrointestinal tract, reaching peak concentration approximately 15 minutes to 2 hours after ingestion. Caffeine can remain in the bloodstream for 1.5 – 9 hours, with a mean half-life of about 5 hours (some oral contraceptives can increase, in some cases doubling, caffeine’s half-life) (Vanderveen, 2001). Caffeine is not cleared by the liver as it passes from the GI tract to the circulation and once in the bloodstream, is freely distributed within the intracellular tissue water throughout the body, including passing through the blood-brain barrier. Caffeine is readily reabsorbed by the renal tubules, with metabolism happening primarily in the liver. Repeated caffeine ingestion does not alter its absorption or metabolism; however, a tolerance to its physiological effects does occur (Vanderveen, 2001). The maximum recommended dosage is no more than 400 mg/day (Nawrot et al., 2003; Mayo Clinic, 2014). Due to individual differences, this may be too high for some individuals (Warfighter, 2009).

Caffeine has been shown to increase alertness, psychomotor skill, vigilance, act in a thermogenic capacity, and aid in physical performance (can be seen in caffeine doses of 100 – 200 mg) (Warfighter, 2009). Caffeine doses of 3 – 9 mg/kg of bodyweight has been shown to increase performance in short-duration, high-intensity exercise while doses of 450 mg have been shown to improve aerobic endurance in aerobically trained athletes (Baechle and Earle, 2008). Consumption of over 600 mg/day has been shown to lead to significant side effects, such as tachycardia, tremors, insomnia, nervousness, gastrointestinal upset, chest pain, and arrhythmias (Jellin and Gregory, 2011). Other side effects can include cardiovascular fibrillation, increased body temperature, anuria, and moderate to severe dehydration (Baechle and Earle, 2008; Warfighter, 2009). Aviators who consume more than the recommended dosage can experience physiological issues relating to changes in cardiovascular output and decreased hydration levels. Although caffeine falls into the “yellow” zone of safe usage, both aircrew and ground personnel is required to use discretion (Warfighter, 2009). Increased cardiac output can cause changes in stroke volume and regulatory blood flow to areas of the body. This may affect the G-tolerance of the aviator as well as visual acuity during certain phases of flight.

Because of the diuretic nature of caffeine, hydration levels can fluctuate immensely if water is not also consumed. Flying in hot environments may result in a longer time to acclimatize, decreased task performance, and lowered situational awareness of the individual. Flying in a cold environment does not preclude aircrew from caffeine-associated dehydration. Due to the body’s thermoregulatory response to maintain temperature, as well as increased water lost during expiration, individuals can dehydrate quicker when exposed to hypothermic-like conditions. Water is key when trying to balance caffeine intake in order to limit adverse effects, with a recommended minimum of 2 quarts (1.9 liters) per day and up to 12 quarts (11.4 liters) per day based on work intensity (AFTTP(I) 3-2.26; AFI 48-151).

Another popular nutritional supplement consumed by many individuals involved in the fitness realm is creatine. Although creatine can promote muscular strength and endurance, promote hypertrophy/body composition, increase cardiovascular and respiratory levels (during intense/short-bout exercise), increase fatigue resistance, and improve mental stimulation (Baechle and Earle, 2008), it can also cause muscle
cramping, reduction in hydration levels, and kidney dysfunction (Warfighter, 2009). These negative responses can increase when the body is exposed to altitude conditions. The major cause of concern for aviators taking creatine is when the “Loading Phase” is utilized. This phase can last anywhere from 5 – 10 days and the daily intake is 4 – 5 times the daily recommended dose (recommended dose 2 – 5 g/day) (Warfighter, 2009). Creatine monohydrate is the most common form of nutritional supplement creatine and the loading phase dosages can be as high as 20-25 g/day for 5 days. These first 5 days will result in increased muscle mass as well as increased water retention (Baechle and Earle, 2008). This phase is initiated to increase creatine circulation throughout the blood stream and induce an influx of the body’s internal (free) creatine and creatine phosphate (CP) to be delivered to the muscle fiber in the form of ATP for energy production. Many nutritional supplements that contain creatine also contain other ingredients (i.e. caffeine) but still recommend the loading phase. Because of the extremely high levels of synthetic creatine combined with other ingredients being ingested during this phase, flight surgeons have banned any aircrew member from using creatine products due to the increased prevalence in dehydration, heat intolerance, and muscle cramping. Ground personnel are “allowed” to use creatine products without flight surgeon approval as it is considered a “yellow” zone supplement by the DoD, but discretion and moderation is strongly advised (Warfighter, 2009). Hypertension and gastrointestinal disturbances are also very common (Warfighter, 2009). Increased blood pressure and gas build up can cause a variety of physiological issues inside the aircraft. Other concerns that have been reported are aching, cramping, and burning of the lower extremities which can impact the proficiency of the anti-G straining maneuver (AGSM) which is vital to the pilot training and fighter jet communities. Excessive weight gain can also be a side effect of the loading phase causing an overall decrease in health, fitness, and G-tolerance of the aviator.

Bitter orange (citrus aurantium) is another prominent nutritional supplement and found in the form of workout powders and pills. Bitter orange is a source of synephrine, a stimulant often used to increase weight loss. Synephrine is an alkaloid containing six different isomers, making it difficult to define which are present in bitter orange products. Some of these isomers closely resemble or act as an alternative to ephedra because of the adrenergic effects it has on the body.

Many of the supplements containing bitter orange have a thermogenic effect on the body. They speed up metabolism to burn more calories both at rest and during exercise. They can induce greater fat loss (lipolysis) and also act as an appetite suppressant, especially when combined with caffeine (Baechle and Earle, 2008). Because of its thermogenic nature, potential side effects include tachycardia, hypertension, syncope, stroke, and cardiac arrest which can be even more detrimental in aviation setting if taken for any reason (Warfighter, 2009). These symptoms can lead the aviator to experience undue physiological stresses, decreased situation awareness and G-tolerance, and leave them more prone to spatial disorientation.

Popular supplements like Cellucor C4®, BSN Hyper Shred®, and Beast 2 Shredded® contain either bitter orange extracts or a form of synephrine. There are many more supplements on the market containing these compounds, so it is crucial labels are read properly before purchase and consumption. Some of these supplements may contain warnings to use with caution especially for those in the military, law enforcement, or related career fields. The chemical compounds found in synephrine can cause a positive reading for amphetamines and/or stimulants in a drug test, which is why these products should never be taken by aircrew.
Ephedra (Ma Huang) is a banned substance and any supplement containing it should be avoided completely. Ephedra has been associated with heart attack, cardiac arrhythmia, stroke, visual impairment, seizure, cerebral hemorrhage, loss of consciousness, and even death (Warfighter, 2009). A case report concerning a U.S. Army pilot showed results cardiac arrest and indicated permanent neuropsychological deficits (Warfighter, 2009). An additional study on highly trained infantry soldiers indicated exertional heatstroke. Other reported side effects include psychosis, rhabdomyolysis, and blindness due to encephalopathy because of the physiological demands placed on the individual during flight while taking the supplement (Warfighter, 2009). At no time should aircrew ever consider the use of ephedra as a nutritional supplement.

Melatonin is another popular dietary aid. Melatonin is a naturally produced, light-sensitive hormone and antioxidant that is secreted into the plasma of the circulatory system. Peak secretion levels (50 – 200 pg/ml) occur at nighttime with minimal secretion levels during daytime and can help combat sleep disorders and reset the body’s circadian rhythm to allow for easier time-zone acclimation (Warfighter, 2009). It has been shown to be effective with resetting sleep/wake cycles and responding accordingly to zeitgeber cues within that time-zone. Melatonin may help relieve daytime fatigue associated with jet lag and can aid in fighting insomnia. Despite these potential benefits, there are some known side effects to supplemental melatonin use. Some of the reported side effects include reduction in body temperature, central nervous system effects (headaches, seizures, nightmares), hyper- or hypotension, and gastrointestinal issues (Warfighter, 2009).

While melatonin use by military aviators is prohibited, use within civil aviation is not as tightly regulated and civil aviators have used melatonin to help deal with their altered sleep schedules or sleep restrictions that result from their respective flying schedules. In general, there is no FAA prohibition on the use of melatonin. Guidance for aviation medical examiners, and aircrew, is generally that the use of melatonin is acceptable as long as it does not interfere with flying duties. The major potential concern with the use of melatonin is the significant amount of drowsiness that may be experienced when taking this supplement. Melatonin use may also decrease in G-tolerance for aviators of high performance aircraft due to residual drowsiness, desynchronized circadian rhythms, and potential hypotensive effects. As previously stated, melatonin should never be used by any aircrew member, as it is on the DoD banned substance list (“red” category) for aviators (Warfighter, 2009). Ground personnel may be able to take melatonin, but should consult with the medical personnel prior to use.

Nitric oxide (NO) has become a common ingredient found in many pre-workout and dietary supplements because of its vasodilation effects, which increases blood and nutrient flow to both skeletal and myocardial tissue. This can help acclimatization to altitude because of the increased NO in the lungs, plasma, and red blood cells. Studies have shown that native highland populations have higher resting levels of NO in these areas than lowland populations or visitors (Beall et al., 2012). The elevated NO and its reaction with hemoglobin (Hb) can be identified as integral components in adaptation to both acute and chronic hypobaric hypoxia exposure (Janocha et al., 2011).

L-Arginine, an amino acid precursor to nitric oxide production, falls under the same classification as nitric oxide. The rationale behind this ergogenic aid is primarily related to its vasodilation properties. It also aids in the production of creatine in the liver and also thought to increase the release of growth hormone. The recommended dose
of L-Arginine is 5 – 9 g/day and usually combined with creatine in dietary powders or pills (Warfighter, 2009). Some aviation concerns of nitric oxide and/or L-Arginine supplements include cardiac palpitations, headache, and elevated blood pressure. Please consult with the flight surgeon before taking any products contain “nitric oxide” ingredients.

Whey or soy protein, casein, branched-chain amino acids, amino acid supplements, beta alanine, essential fatty acids, and multivitamins are probably the most common nutritional supplements available today. According to the Human Performance Resource Center classification list, each of these nutritional aids fall under the low risk, or “green” and “yellow,” categories (Warfighter, 2009). Most whey or soy proteins contain anywhere from 20 – 50 grams of protein per serving and are most effective when consumed within 60 – 90 minutes post-exercise (Baechle and Earle, 2008). Combinations of whey, soy, or casein aid in muscle repair and regeneration, increase muscle fiber cross-sectional area and density, increases in strength, and hypertrophy. Branched-chain amino acids, beta alanine, and other amino acid supplements are naturally occurring molecules within the body and are safe to consume without prior flight surgeon approval. Each of these dietary aids fall under the “green/yellow” range of safe consumption but are recommended to take in moderation or talk to the flight surgeon for more information. The benefit of these is to increase strength, force output, power, endurance, lean body mass, and to repair muscle tissue during the rest phase. Be sure to monitor intake dosages, as over-ingesting these supplements can lead to fatigue, decreased dexterity, motor and coordination issues, balance disorders, spatial disorientation, and seizures.

Essential fatty acids are vital and important to the diet of the aviator. Omega-3 (alpha-linolenic acid) and Omega-6 (alpha-linoleic acid) fatty acids are considered polyunsaturated fats (PUFA) and provide a great nutritional benefit to the individual. Omega-3 fatty acids are often found in fish, nuts, plants, nut and plant-based oils, and fish-oil supplements. Omega-6 fatty acids are found in meat, poultry and eggs, as well as nut and plant-based oils. Omega-9 fatty acids, or monounsaturated fatty acids (MUFA), are a type of oleic acid found in nuts, fruits, avocados, olives, and oils. Each of these will aid in protection against high cholesterol, heart disease, stroke, cancer, and improve bone health.

Vitamins, minerals, and antioxidants (i.e. Centrum One A Day®) are supplements that can be consumed without prior flight surgeon approval. They aid in energy production from the breakdown of macronutrients (CHO, fats, & proteins), aid in tissue repair, support reproductive function, promote neural and brain function, and increase immune function (Warfighter, 2009). As with all supplements, more is not necessarily better or safe, so it is important to closely monitor daily intake levels, as each have different toxicity levels which can lead to multiple side effects. Dietary fibers, probiotics, and prebiotics can also be taken by the aviator. These benefits of can help protect against gastrointestinal diseases, hypertension, diabetes, heart disease, cholesterol, immune function, and various types of cancer.

Ginkgo biloba is common in many dietary aids. Its primary function is to improve memory and concentration when taken in doses of 120 – 600 mg/day (Warfighter, 2009). There have not been any serious reports in the aviation community but possible issues can lead to mild gastrointestinal problems, headaches, dizziness, constipation, and increase risk of hemorrhage.

Ginseng (panax), meaning “all-healing,” has been known for its broad range of restoration purposes. It is stated to aid as an immune booster, increase stamina and
performance, improve erectile dysfunction, hypertension, diabetes, and reduce stress when doses for 200 – 600 mg/day are followed (Warfighter, 2009). Reported side effects include hypoglycemia, headache, insomnia, gastrointestinal disturbances, anxiety and cardiovascular effects, but not enough information has been documented within the aviation community. Even though ginkgo biloba and ginseng can be safely consumed, it is recommended to consult with the flight surgeon before use for further information.

Aircrew are at risk from the synergistic, as well as cumulative, effects of the stimulant-like substances in pre-workout powders and pills, dietary supplements, and energy drinks when added to the other components of their diet. Aircrew should be properly educated (i.e., from reputable scientific literature/flight surgeons, not health magazines, gym pamphlets, or other users) on the correct usage of these supplements and avoid the dangerous stimulants that may be in these products. Each of these dietary and nutritional supplements will affect every individual in a different capacity, so it is very difficult to determine the body’s physiological response at altitude. Any new use (either product or dosage) should be ground-tested to ensure the user is aware of how the supplement will affect them prior to getting in an aircraft.

4.2.4. Cigarettes, Smokeless Tobacco, and e-Cigarettes

4.2.4.1. Cigarettes and Carbon Monoxide. Smoking tobacco has particular significance to the flyer, as there are both short- and long-term harmful effects from smoking cigarettes, cigars, and pipes. The effects have been well documented in the 2004 Surgeon General's Report on the health consequences of smoking and in sources detailing the adverse effects on aircrew performance (Bronson, 1979; Robinson and Wolfe, 1976). These overall health consequences are not the focus of our discussion here, but rather the more immediate concerns to the aviator in the flying environment.

Carbon monoxide (CO) is a colorless and odorless gas produced by the incomplete combustion of organic matter. Smoke from one cigarette can contain up to 21,400 µg of CO and can saturate approximately 1 – 1.5% of an individual’s hemoglobin (Steinfeld, 1972; Judd, 1971). The CO binds to hemoglobin (nearly 200 times more readily than O₂), displacing oxygen, to form carboxyhemoglobin (COHb) (see Section 3.1 for more discussion).

The effect of CO on the human body is both cumulative and persistent. Initially, a cigarette smoker can inhale an average concentration of CO into the lungs of 400 ppm, or 0.04% (Goldsmith and Landaw, 1968). Continued smoking produces the COHb levels previously mentioned. Since the estimated half-life of COHb in the body while breathing room air is approximately 5.5 hours, the effect of smoking is long lasting (Kindwall, 1993; Varon and Marik, 1997). Average smokers have roughly 5.7% COHb levels, with users who smoke 20 – 30 cigarettes a day (1 to 1½ packs a day) can be as high as 10% (Judd, 1971). Due to the half-life of COHb, moderate smokers can still have COHb levels as high as 4.5% after 8 to 15 hours of deprivation. In the flying environment, 3 – 7% COHb negatively impacts visual acuity, night vision, depth perception, and cognitive performance (Denniston et al., 1978).

Research has shown that the adverse effects of cigarette smoking on oxygen transport may be more pronounced at high altitudes and impact an individual’s ability to adapt to reduced oxygen pressure, as the lower oxygen pressure makes it easier for CO to bind hemoglobin (Steinfeld, 1972; West, 1962). The binding of CO to hemoglobin
alters oxygen interaction with hemoglobin and results in a left shift of the oxygen-hemoglobin dissociation curve, decreasing oxygen release and availability to the tissues (see Sections 1.2.6 and 1.2.7 for more discussion on oxygen transport) (Denniston et al., 1978). This effect is critical for smoking crewmembers, even those that fly in pressurized aircraft at cabin altitudes between 6,000 and 8,000 ft. Smoking crewmembers flying at a cabin altitude of 7,000 ft with carboxyhemoglobin (COHb) levels of 5% and 10% will have a physiological altitude of approximately 11,000 ft and 13,500 ft, respectively (Judd, 1971). Table 4.2.4.1-1 shows approximate physiological altitudes for COHb levels 5% and 10% as derived from Figure 3.1.2.1-2 from the work of Judd.

Table 4.2.4.1-1. Approximate Physiological Altitude at a Given COHb Level

<table>
<thead>
<tr>
<th>Cabin Altitude (ft)</th>
<th>0% COHb</th>
<th>5% COHb</th>
<th>10% COHb</th>
</tr>
</thead>
<tbody>
<tr>
<td>6,000</td>
<td>6,000</td>
<td>10,500</td>
<td>13,000</td>
</tr>
<tr>
<td>7,000</td>
<td>7,000</td>
<td>11,000</td>
<td>13,500</td>
</tr>
<tr>
<td>8,000</td>
<td>8,000</td>
<td>12,000</td>
<td>14,000</td>
</tr>
</tbody>
</table>

Because of the reduced oxygen carrying capacity of hemoglobin in smokers, smoking crewmembers must be aware that their performance may become impaired earlier, and potentially more severely, than a non-smoking crewmember. Even if a decrease in physical performance is not noticeable, cognitive skills, vision, and fine motor skills may be, and fatigue may set in quicker. These subtle decrements may become more pronounced during night operations or tasks that demand a high mental function or duration. Smoking aircrew should be aware that their physiological altitude may also put them above the altitude that Air Force Instruction (AFI) 11-202, Volume 3, General Flight Rules, requires the use of oxygen.

While the focus of discussion here has been on the impact of smoking tobacco, aircrew are exposed to other sources of carbon monoxide, such as from ground equipment, vehicles, and aircraft exhaust. As discussed in Section 3.1, carbon monoxide levels of 1,000 ppm have been reported in the cabin compartment of U.S. Army CH-47A helicopters after firing 7.62 mm machine guns (Denniston, 1978). This level has the potential to raise blood COHb levels up to nearly 7% in non-smokers. Regardless of the source, the physiological effects are the same as discussed above. Exposure to CO, from either smoking or the environment, makes aircrew more prone to hypoxia (see Section 3.1 for more details). In flight, aircrew can experience hypoxic-like symptoms as a result of their raised physiological altitude, exertion levels at altitude, and lowered time of useful consciousness (TUC) at that given altitude.

In addition to the CO effects, smoking introduces other substances into the body that are a concern to aircrew (as well as other negative health consequences not discussed here). Nicotine, a potent vasoconstrictor, is found in concentrations of 200 to 2,400 µg per cigarette (Steinfeld, 1972), and concentrations in blood of smokers has been reported to be 0.1 – 10 µg/ml, depending on a number of variables in addition to smoking habits (Ingenito et al., 1971). When you smoke, nicotine is absorbed into the bloodstream and acts on various targets throughout the body, such as the central nervous system and the cardiovascular system. The effects can last for several hours after nicotine inhalation, as the half-life of nicotine is approximately 2 hours, and it can accumulate in the body for 6 – 8 hours over the course of the day with regular use (Benowitz, 1988). Levels of nicotine in the blood are similar between users of different tobacco products (i.e. smoking, smokeless, gum).
Nicotine, regardless of source, has shown to increase heart rate and blood pressure, increase the prevalence of coronary and peripheral vascular disease, increase cardiac contractibility, and increase cardiac oxygen consumption (Fenton and Dobson, 1985; Benowitz and Gourlay, 1997; Ramakrishnan et al., 2011). Other effects include cutaneous vasoconstriction reducing skin temperature, hemodynamic and platelet dysfunction, increase in cholesterol, tremors, ulcers, and endocrine and hormone issues (Benowitz, 1988).

Nicotine effects the body by enhancing the release of various neurotransmitters. While nicotine acts as a central nervous system stimulant, its effects on the cardiovascular system may be of more concern to aviators. Once in the bloodstream, nicotine acts on cardiovascular tissues to increase heart rate and blood pressure. Cardiac output is increased by increasing heart rate by 10 – 15 beats per minute and increasing myocardial contractility (Fenton and Dobson, 1985; Benowitz and Gourlay, 1997). The vasoconstrictor properties of nicotine also leads to constriction of coronary arteries, even at low doses (Benowitz and Gourlay, 1997; Young et al., 1988). The increased cardiac oxygen consumption, coupled with the reduced oxygen content of the blood due to elevated levels of COHb, and the increase in heart rate means that the heart has to work harder to meet demands. The systemic vasoconstriction effects of nicotine can lead to a 5 – 10 mmHg increase in blood pressure, further increasing the work demands of the heart (Benowitz and Gourlay, 1997). Other cardiovascular effects of nicotine result in cutaneous vasoconstriction, which can lead to reduction in fingertip skin temperature, and vasoconstriction in the cerebral circulation (Benowitz and Gourlay, 1997; Black et al., 2001; Ingenito et al., 1971). The result of this cerebral vasoconstriction is cerebral stagnant hypoxia.

In skeletal muscle, however, nicotine actually leads to vasodilation (Benowitz and Gourlay, 1997). For the aviator, especially in high-G aircraft, this potentially reduces venous return, and at a minimum may mean that the pilot must work harder while performing the anti-G straining maneuver.

In addition to the cardiovascular effects, nicotine has been shown to shown to alter retinal function, due to both reduced retinal blood flow and direct action on the retinal cells. Vision is effected due to lack of blood supply to the ocular vasculature and retinal tissue, causing aviators to experience decreased visual acuity, contrast, and depth perception as well as an increase difficulty in dark adaptation. Varghese et al. have also shown that nicotine affects the functional properties of retinal neurons (Varghese et al., 2011). Visual impairments, increased rate of tracking errors, visual coordination, and performance errors were also reported (Nesthus et al., 1997).

4.2.4.2. Smokeless Tobacco. “Chewing,” “dipping,” or smokeless tobacco, has become a real issue of concern in recent history for aircrew, especially in younger populations. It is important to state that smokeless tobacco is not a safe alternative to smoking cigarettes, but yet it is a common habit for many aviators. Chew, dip, snuff, and snus are common types of smokeless tobacco based on how the tobacco is actually processed and packaged after being cultivated. Smokeless tobaccos still contains a variety of chemicals including nicotine, polonium, waste by-products, and other cancer-causing carcinogens. While these carcinogens pose a range of health concerns, we will not address the overall health issues here but rather focus on the effects that the use of these products can have on in the flying environment, independent of the overall health impact.
Unlike smoking tobacco, smokeless tobaccos do not pose the same risk when it comes to carbon monoxide. For the flyer, the concern has more to do with the nicotine present in tobacco. While nicotine is absorbed more slowly from smokeless tobacco than from cigarette smoke, the peak values in venous blood are similar between the delivery methods (Benowitz, 1988). However, unlike nicotine delivered from smoking, nicotine levels from the use of smokeless tobacco reach a plateau during, and last until after, use, even after removal from the mouth (Benowitz, 1988).

The amount of nicotine absorbed from one serving of smokeless tobacco is 3 – 4 times the amount delivered by one cigarette and will last much longer, in turn, transporting more nicotine to the bloodstream (Benowitz, 1988). Many of these products contain fiberglass or woodchips to make microscopic tears into the inner lip membrane and gums. This will allow the nicotine to be processed directly into the bloodstream at a much slower capacity than smoke inhalation.

Some studies have reported that the nicotine absorbed will help individuals with anxiety and arousal, stimulating a calming or relaxation process (Benowitz, 1988). While this may be true, the side effects of smokeless tobacco pose large threats to the aviation community. Because of the nicotine and other carcinogens in smokeless tobacco, vasoconstriction in vital organs will still be observed. Refer to the discussion on nicotine above for more details.

4.2.4.3. Electronic Cigarettes and Vaping. Electronic cigarettes (e-cigarettes) and vaping have been gaining popularity, especially with younger users. Although this phenomenon is still relatively new, some data have suggested that e-cigarettes and vaping still contain harmful chemical and not just harmless flavors and water vapors as purported by the industry. Atomized propylene glycol and glycerin that may be altered with additives contribute to the flavors, colorings, and variable nicotine content in these products. It is true that e-cigarettes deliver only about 20% nicotine, which is less than that of a regular cigarette, but the toxins within the vapors still have potential to damage lung tissue (Tarantola, 2014). This means the average “vaper” is more likely to huff on the e-cig longer and more often than a regular cigarette. When heated, the solvents used to dissolve the nicotine and flavorings can be converted into carcinogenic compounds known as carboxyls. The vapors contain ultrafine particles (chromium, nickel, mercury, lead) which can be delivered into the circulatory system. It has been reported that only 20 – 27% of these particles will diffuse into the blood, compared to 25 – 35% from smoking a regular cigarette (Grana et al., 2014). It has also been documented that the amount of vapors (i.e. carcinogenic carboxyl compounds) will vary based on the amount of the solvent solution content in the cartridge and the battery output of the e-cigarette itself (Kosmider et al., 2014). The e-cigarette performance has been shown to be virtually identical to that of regular cigarettes in terms of exhaled nitric oxide rates. Nitric oxides cause the smooth muscles in the lungs and heart to relax, which can lead to reduced lung function and increased risk of heart attack. There has been no statistically significant data reporting the benefit of e-cigarettes or vaporing over that of regular cigarettes. The fact remains that e-cigarettes are synthetic chemical cocktails that we know little about and aviators need to be cautious as to the side effects on the myocardial and lung tissue from exposure to e-cigarettes or vaping.
4.2.4.4. **Tobacco Cessation.** Many nicotine replacement modalities include gum, sunflower seeds, mints, or chewing on straws to ease the mental reliance upon the oral fixation. Tobacco cessation medication also include nicotine gum and trans-dermal patches. Each base will offer tobacco cessation programs for individuals that want to quit using tobacco products. In the U.S. Air Force, Health Promotions (HP) (formerly the Health and Wellness Center (HAWC)) can provide information in the form of pamphlets, DVDs, and individual consultations. Due to the aeromedical implications and overall health issues, it is recommended fliers do not use any tobacco products. To avoid inflight effects of nicotine withdrawal, aircrew should go through the cessation program during a non-flying or duties not including flying (DNIF) status. Please consult with flight surgeons, physiologists, and health promotions representatives for more education regarding the cessation programs.

4.2.5. **Alcohol**

*Note: Unless otherwise annotated, the following information was taken from Air Force Pamphlet (AFP) 160-5, Physiological Training, 23 January 1976.*

The ingestion of alcohol, liquor, wine, or beer is common in most social cultures. Ethyl alcohol is the active ingredient in these beverages and acts as an anesthetic drug, which depresses the brain. Also present are volatile substances that slow down the rate at which the body disposes of ethyl alcohol. Alcohols are poisonous to the body; ethyl alcohol is the least poisonous and most tolerated by the body.

4.2.5.1. **Acute Effects of Alcohol.** The intoxicating effects of alcohol are brought about in two major actions on the brain; one effect is a change in the proportion of two chemicals called neurohormones, which affect the brain. These neurohormones, serotonin and norepinephrine, are believed to control mood and alertness. After consumption of small amounts of alcohol, a crewmember may lose normal cautionary attitudes and become reckless, possibly before he/she even notes a change in skill or performance.

Another effect of alcohol is a reduction in the ability of the brain cells to utilize oxygen. Hypoxia enters the mental picture, causing judgment and performance to be impaired. Alcohol also acts as a relaxant and anesthetic, removing a person’s inhibitions and lessening his/her worries. In larger amounts, this relaxation progresses to actual unconsciousness and eventually leads to death due to respiratory paralysis.

The concentration of alcohol in the blood and brain depends on three factors: 1) the amount consumed, 2) the rate of absorption from the stomach and small intestine, and 3) the rate of its metabolism by the body.

The rate of absorption depends on many factors: the type and quantity of food in the stomach, the degree of hydration of the body, the concentration of alcohol in the beverage and the type of beverage with which it is mixed, how fast the alcohol is consumed, body weight, and the individual variation in the absorptive characteristics of the stomach.

The rate of metabolism or digestion of alcohol in the body is relative constant, regardless of the amount of alcohol present in the body (McFarland and Barach, 1973). Two to 10% of the alcohol is excreted through the lungs and kidney and the remainder is oxidized by the liver. It takes about 1 hour to eliminate 0.33 oz (9.8 mL) of pure ethyl alcohol from the body. This is the amount of ethyl alcohol in 0.67 oz (19.6 mL) of 100-
proof liquor or in 6 oz (170 mL) of beer. Table 4.2.5.1-1 gives an idea of the amount of alcohol found in common alcohol beverages.

<table>
<thead>
<tr>
<th>Beverage</th>
<th>Typical Serving (oz)</th>
<th>Alcohol Content (oz)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Table Wine</td>
<td>4.0</td>
<td>0.48</td>
</tr>
<tr>
<td>Light Beer</td>
<td>12.0</td>
<td>0.48</td>
</tr>
<tr>
<td>Aperitif Liquor</td>
<td>1.5</td>
<td>0.38</td>
</tr>
<tr>
<td>Champagne</td>
<td>4.0</td>
<td>0.48</td>
</tr>
<tr>
<td>Vodka</td>
<td>1.0</td>
<td>0.50</td>
</tr>
<tr>
<td>Whiskey</td>
<td>1.25</td>
<td>0.50</td>
</tr>
</tbody>
</table>

*Pilot's Handbook of Aeronautical Knowledge, Figure 17-8. 2016.

The metabolism of alcohol cannot be expedited by any readily available method or remedy. Walking, drinking black coffee, breathing 100% oxygen, or taking cold showers are common folklore methods that do not eliminate alcohol from the body.

4.2.5.1. Blood Alcohol Level. The physiological effects of alcohol depend on the level of alcohol in the blood. Several terms have been used to express blood alcohol level (BAL): milligram percent (mg%), grams alcohol per 100 milliliters whole blood (gm/100 mL), and percent of alcohol in the blood.

The use of the term "mg%" is a misnomer, since it refers to the number of milligrams alcohol in 100 mL whole blood. To convert from mg% to percentage of alcohol in the blood, one must first convert to gm/100 mL, then multiply by 100.

For example, an individual whose blood alcohol level is 200 mg% would have an alcohol level of 200 mg/100 mL whole blood or 0.2 gm/100 mL whole blood.

\[
200 \text{ mg}\% = \frac{200 \text{ mg alcohol}}{100 \text{ mL whole blood}} = \frac{0.2 \text{ g alcohol}}{100 \text{ mL whole blood}}
\]

Since the density of blood is approximately 1 g/mL, 100 mL blood weighs approximately 100 gm. Therefore,

\[
\frac{0.2 \text{ g alcohol}}{100 \text{ g whole blood}} \times 100 = 0.2\% \text{ alcohol in the blood}
\]

With 0.05% to 0.10% of alcohol in the blood, mild intoxication is present, which will lower altitude tolerance even if symptoms are not observed. With 0.10% to 0.15% alcohol in the blood, everyone is affected to some degree. In most states, an automobile driver with this level would be considered intoxicated (in most states, the legal limit for operating a motor vehicle is 0.08%; the limit for civil aircrews is 0.04% (FAR 91.17)). At a blood alcohol level of 0.15% to 0.20%, performance deteriorates and there are marked symptoms. At 0.30%, acute intoxication and lack of coordination occur, and consciousness is lost. Table 4.2.5.2-1 summarizes the effects at various blood alcohol levels. Keep in mind that the effects vary by individual.

Unconsciousness and possible death may result at levels of 0.40% to 0.50%. For an average 160 pound (72.6-kg) man, 2 oz (58.8 mL) of 100-proof whiskey consumed in 1 hour will produce 0.05% in the blood. If a person weighing 160 pounds (72.6 kg) were to consume approximately four-fifths of a quart of whiskey in 1 hour, it
would be fatal. Since the effects of alcohol are compounded by altitude, 10,000 ft (3,048 m) of altitude doubles the effects of alcohol on the body.

<table>
<thead>
<tr>
<th>Blood Alcohol Level</th>
<th>Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.01 – 0.05% (10 – 50 mg%)</td>
<td>Average individual appears normal</td>
</tr>
<tr>
<td>0.03 – 0.12% (30 – 120 mg%)</td>
<td>Mild euphoria, talkativeness, decreased inhibitions, decreased attention, impaired judgement, increased reaction time</td>
</tr>
<tr>
<td>0.09 – 0.25% (90 – 250 mg%)</td>
<td>Emotional instability, loss of critical judgement, impairment of memory and comprehension, decreased sensory response, mild muscular incoordination</td>
</tr>
<tr>
<td>0.18 – 0.30% (180 – 300 mg%)</td>
<td>Confusion, dizziness, exaggerated emotions (anger, fear, grief), impaired visual perception, impaired balance, staggering gait, slurred speech, moderate muscular incoordination</td>
</tr>
<tr>
<td>0.27 – 0.40% (270 – 400 mg%)</td>
<td>Apathy, impaired consciousness, stupor, significantly decreased response to stimulation, severe muscular incoordination, inability to stand or walk, vomiting, incontinence of urine and feces</td>
</tr>
<tr>
<td>0.35 – 0.50% (350 – 500 mg%)</td>
<td>Unconsciousness, depressed or abolished reflexes, abnormal body temperature, coma, possible death from respiratory paralysis (≥ 450 mg%)</td>
</tr>
</tbody>
</table>

*Pilot’s Handbook of Aeronautical Knowledge, Figure 17-8. 2016.

4.2.5.3. Chronic Effects and Hangover. Of the many chronic effects of alcohol, those usually having long-term effects on health are found in people suffering from alcoholism. Chronic effects such as vitamin, mineral, and protein deficiency and a fatty liver are caused by improper diet and the direct effect of alcohol. Excess of body carbohydrates, cirrhosis of the liver, and alcoholic psychosis are other effects the heavy drinker may suffer.

The effect of a hangover probably constitutes a more significant flight safety hazard than does the mild intoxication state of alcohol ingestion. It is unlikely that an Air Force flyer would attempt to fly an aircraft while intoxicated. However, the same flyer 8 to 18 hours later, with a hangover, may not abort a flight although he/she is less efficient and less physically capable than normal.

The symptoms of a hangover are not entirely due to alcohol ingestion. Many are due to the activities that often accompany overindulgence in alcohol: excessive tobacco smoking, loss of sleep, improper diet, etc. Alcohol is a known cause of dehydration, resulting in many of the hangover symptoms. Alcohol takes moisture from the cerebral spinal fluid that surrounds the brain. Loss of fluid causes tension on the supporting structure of the brain, producing a headache. Dehydration can be increased by exposure to low atmospheric pressures, breathing dry oxygen, and thermal stress. In the early stages of dehydration, judgment and emotional changes are observed, and each can seriously interfere with the flyer’s ability to carry out tasks safely and efficiently.

For the aviator, it is important to remember that the effects of alcohol can act synergistically with other stressors and reduce safety of flight. Recall from Section 3.1,
Hypoxia, alcohol interferes with the cells’ ability to function and leads to histotoxic hypoxia. The general rule of thumb, as cited in the common literature, is that 1 oz (29.6 ml) of alcohol can raise an individual’s physiological altitude 2,000 feet (Clumpner, 2011; Dennie and Bayley, 2002; Pilot’s Handbook, 2016). This most likely comes from the early work of McFarland and others that said “the alcohol in 2 – 3 cocktails taken at 6,000 – 8,000 feet cabin altitude would tend to have the effects of four or five cocktails at sea level” (McFarland, 1973). While this does not seem like much, when coupled with even mild carbon monoxide exposure and/or mild hypoxic hypoxia (i.e. being at 6,000 – 8,000 feet cabin altitude), the synergistic nature of the stressors increases the histotoxic hypoxia effect and may be sufficient to put you behind the aircraft. Another aviation consideration is that alcohol causes disorientation due to its combined effects on the lower brain and inner ear (see discussion in Section 1.5). The effect on the inner ear may be identified by involuntary reflex eye muscle movement 36 to 48 hours after heavy drinking, even after blood alcohol levels have returned to zero. Holloway (1994) reviewed 155 empirical studies from 1985 to mid-1993 and concluded that since alcohol sensitivity can vary from time to time, person to person, and situation to situation, the setting of a “safe blood alcohol content will always be arbitrary, being based on a low, but non-zero, incidence of effects below that level.” Campbell and Bagshaw (2002) indicated 0.03% BAL “…increases the likelihood of an individual having any form of accident.” They also pointed out that impairment of judgment could be measured at 0.05% BAL and that loss of performance could occur after only one drink and by the products of alcohol metabolism.

Time is the only factor that will alleviate a hangover. Other symptomatic relief can come from eating a well-balanced meal, which provides nonalcoholic carbohydrates to the liver, and by consuming large quantities of nonalcoholic fluids to reduce dehydration. Breathing 100% oxygen has no direct effect on a hangover.

The way to avoid flight safety problems resulting from a hangover is to educate the flyer to the hazard that persists for many hours after alcohol ingestion. There is no objective test to measure the effects of a hangover. The effects on the eye, inner ear, fluid balance, etc. require 12 hours of “bottle to throttle” separation for truly safe flight conditions to exist. AFI 11-202v3 specifically states that aircrew members will not fly “if any alcohol is consumed within 12 hours prior to takeoff (or assuming aircraft control for UAS [unmanned aerial systems]) or if impaired by alcohol or any other intoxicating substance, to include the effects or after-effects.” While Federal Aviation Administration (FAA) regulations are less restrictive, with Federal Aviation Regulation (FAR) 91.17 stating that “no person may act or attempt to act as a crewmember of a civil aircraft within 8 hours after the consumption of any alcoholic beverage, or while under the influence of alcohol,” the FAR does include a specific BAL restriction, stating “or while having 0.04% by weight or more alcohol in the blood” (FAR 91.17). The old adage about “an ounce of prevention is worth a pound of cure” is especially true in the three-dimensional world of extra stresses in aviation. The person affected may determine overt symptoms are gone, but one of the most dangerous aspects of this problem is determining when and where the physiological compromise stops so that safe flight can be accomplished.
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**Recommended Readings**


DISTRIBUTION STATEMENT A. Approved for public release. Distribution is unlimited.
Vocabulary
Blood alcohol level (BAL)
Branched-Chain Amino Acids (BCAA)
Carbon monoxide (CO)
Carboxyhemoglobin (COHb)
Hemoglobin (Hb)
Monounsaturated Fatty Acids (MUFA)
Nitric Oxide (NO)
Polyunsaturated Fatty Acids (PUFA)
Over-the-counter (OTC) medications
4.3. Fatigue and Fatigue Countermeasures

James C. Miller, Ph.D., CPE

4.3.1. Human Fatigue and its Effects

In any human-machine system, including weapons systems, the most unpredictable component in the system is the human. After training and currency, the greatest contributor to that human variability is fatigue. Good human-machine system design exploits human strengths and protects the system from human weaknesses. This is a fundamental concept in human factors engineering (HFE). The human brings to a system much more powerful pattern recognition capabilities and decision-making skills than can be provided in software. However, the human also brings much more performance variability to a system than one finds in software and modern hardware.

4.3.2. Sources of Variability

Incomplete training and lack of currency are sources of human variability. When novices are learning to operate a complex system, they display a learning curve. Initially, their performance is quite poor and variable, but they learn the basics quickly. Next, their performance is noticeably better, on average, but still more variable than desired. Finally, as they approach the expert user level, their average performance is quite good and it varies only a small amount. Similarly, when expert users become “rusty” in the operation of a complex system, their performance may be more variable than desired until they return to their expert level.

Additionally, one of the primary hallmarks of human fatigue is performance variability. This is due to large amplitude, moment-to-moment fluctuations in attentiveness associated with fatigue. One’s average performance may be acceptable, but there are brief periods when responses are extraordinarily delayed or absent (“mental lapses”). A good example of a mental lapse is missing your exit from the freeway. We often call this “distractibility” and note that fatigued system operators are more easily distracted than nonfatigued operators.

4.3.3. Sources of Fatigue

Sleepiness and fatigue are different human states. You may recall times when you were fatigued but not sleepy. However, when we have asked workers for numeric ratings of sleepiness and fatigue across many days and nights of demanding work, their two sets of ratings have correlated very strongly. Thus, it appears that in our day-to-day activities we perceive the two states as changing in parallel most of the time. For most purposes, you may view sleepiness and fatigue as being almost the same thing. We sort the generators of fatigue into the five categories: physical, circadian, acute, cumulative, and chronic.

4.3.4. Circadian Effects

There are normal, inherent, unavoidable, 24-hr rhythms in human cognitive and physical performance. Most of these circadian rhythms oscillate between a high point late in the day to a low point in the pre-dawn hours with a peak-to-trough amplitude of
about 5% to 10% of their average value. Human circadian rhythms are slightly longer than one cycle per day but are normally slaved, or entrained, to exactly one cycle per day by external time cues (often referred to by the German word for time cues, zeitgebers), especially the daylight-darkness cycle.

Circadian-related fatigue falls into two major categories. The first is fatigue or sleepiness associated with attempting to work or function at times that coincides with the circadian trough. Personnel working on a night shift often experience this type of fatigue. The second is fatigue associated with a circadian rhythm that is disrupted because of some type of schedule change. Shift workers experience this type of fatigue for several days/ nights after rotating to a new work/rest cycle (shift lag), and travelers experience this type of fatigue after traveling to a new time zone (jet lag). Note that the problem is not just that the rhythms are peaking and troughing at the wrong times with respect to external cues; they also fall out of synchronization with each other. These different types of loss of synchrony—with external cues and internally—both seem to contribute to feelings of malaise.

4.3.4.1. Jet Lag. The feelings of malaise and fatigue that accompany a time zone change that is faster than about one time zone per day. Jet lag occurs during the period of resynchronization of circadian rhythms to new external time cues, especially the daylight-darkness cycle.

4.3.4.2. Shift Lag. The feelings of malaise and fatigue that accompany a change from day work to night work and vice versa. Shift lag occurs during the period of attempted resynchronization of circadian rhythms to new external time cues. Compared to jet lag, the attempt to resynchronize to a night work and day sleep schedule occurs more slowly and is much less successful because the main time cue, the daylight-darkness cycle, tends to inhibit resynchronization.

4.3.5. Types of Fatigue

4.3.5.1. Acute Fatigue. Acute fatigue builds up normally and unavoidably within in one waking period, but recovery from acute fatigue occurs as the result of one good-quality, nocturnal sleep period.

4.3.5.2. Cumulative Fatigue. Cumulative fatigue builds up across several waking and duty periods when there is inadequate recovery (due to inadequate sleep) between the duty periods. Recovery from cumulative fatigue cannot be accomplished in one good-quality, nocturnal sleep period.

4.3.5.3. Chronic Fatigue. Chronic fatigue may set in after 1 to 2 weeks of cumulative fatigue. The symptoms of chronic fatigue include:

- The desire to sleep
- Apathy
- Substantial impairment in short-term memory or concentration
- Muscle pain
- Multi-joint pain without swelling or redness
- Headaches of a new type, pattern, or severity
- Unrefreshing sleep
- Post-exertional malaise lasting more than 24 hours

These symptoms are similar to those of chronic fatigue syndrome (CFS); however, unlike CFS, the cause is known (continuing cumulative fatigue) and it occurs much sooner than the 6-month diagnostic requirement for CFS. The Air Force Safety Center has in the past called chronic fatigue “motivational exhaustion.” While this label accounts for only one of several possible symptoms of chronic fatigue (apathy), it describes well the attitude that one observes in a person with chronic fatigue. It is possible that the long-term presence of chronic fatigue in an individual is one of the causes for the illnesses associated with chronic night work or shift work (para 4.3.9).

4.3.5.4. Physical Fatigue. There are a number of physiological costs associated with physical effort. Physical fatigue, physiological costs are metabolic in nature and may include, among others:

- Elevated whole-body metabolism associated with nonsedentary work, like jogging
- High levels of specific muscle anaerobic metabolism associated with lifting or with the maintenance of a single posture for a long time
- Relatively high heart muscle (myocardial) metabolic demands due to the combination of poor physical conditioning and high physical workloads
- Increased potential for the triggering of central nervous system sleep systems (falling asleep on the job) associated with sleep disruption

Fatigue may also lead to injury. An acute physical stress that exceeds connective-tissue limits may lead to a sprain or strain of a joint. Lesser physical stresses, repeated for days, months, or years, may cause cumulative or repetitive stress injuries. Sedentary work in the absence of exercise and nutritional limitations may lead to morbid obesity and to cumulative trauma of the back. Excessive aerobic effort, especially in a hot environment like a hot aircraft on the ramp, may lead to heat exhaustion and to myocardial ischemia, raising the possibility of heart muscle damage.

On the other hand, exercise increases heart and lung fitness while reducing stress, anxiety, and insomnia. It also raises endorphin levels—the natural “mood elevators” produced by the brain in response to physical exercise. Endorphins reduce pain, relax muscles, suppress appetite, and produce feelings of well being. As a result, sleep will be deeper and more restful. Even something as simple as brisk walking can have a positive effect, if done regularly. The best time to exercise for maximum sleep efficiency is at noon, or between 5 p.m. and 7 p.m. Excessive exercise (beyond one’s capacity) that occurs within several hours before trying to sleep can disrupt sleep.
Physical fatigue does not seem to interact much with cognitive fatigue. However, circadian rhythms do affect physical strength and endurance. These usually vary about 10%, with a peak in the late afternoon/early evening and a trough in the hours around dawn.

4.3.6. Nature of Fatigue

Fatigue is ubiquitous, pervasive, and insidious. By ubiquitous we mean that fatigue affects everybody. There are individual differences: a few people are truly more resistant to fatigue effects than others. Many other people feel, without basis, that they are more resistant to fatigue effects than others. This misperception may cause them to form ill-advised intentions and/or to make bad decisions.

By pervasive, we mean that fatigue affects everything we do, physically and cognitively. Again, there are individual differences. In the physical domain, there are those who are inherently able to train to much greater levels of strength and endurance than the rest of us. This may also be true in the domains of cognition and attention: some people seem inherently less susceptible cognitively than most others.

By insidious, we mean that often when we are fatigued, we are quite unaware of how badly we are performing. Most people have experienced the attention lapse associated with mild fatigue when they miss a freeway exit or realize suddenly that they don’t remember the last mile or two driven on the highway. Similarly, most people recovering from a period of physical, emotional, or cognitive stress have uttered the phrase, “I didn’t realize how tired I was!”

Understanding these aspects of fatigue, it is easy to see how we may become tricked into conducting safety-sensitive jobs such as flying, driving, operating weapons, and making command and control decisions when we are too fatigued to be safe. If we think that we are more resistant to fatigue than we really are, and if we don’t realize that we are very fatigued, then we slog on toward the goal while making poorer decisions, accepting more risk, and being more easily distracted than we should. This is not an intelligent approach to operations, though it has been the accepted approach on many occasions.

According to the National Sleep Foundation, lack of sleep is associated in the short term with irritability, impatience, anxiety, and depression. These problems can upset job and family relationships, spoil social activities, and cause unnecessary suffering. In the long term, shift workers may experience more stomach problems (especially heartburn and indigestion), menstrual irregularities, colds, flu, weight gain, and cardiovascular problems than day workers.

4.3.7. Quantitative Estimation of Fatigue

Fortunately, the biological changes and rhythms that cause fatigue-induced declines, lapses, and variability in human performance are relatively lawful and predictable. There are quantitative models and simulations, implemented in software, that allow us to estimate and predict the timing and severity of fatigue episodes, given some information about when and how much people sleep. A world-class applied model (or simulation) was developed primarily with Department of Defense funding. The Sleep, Activity, Fatigue, and Task Effectiveness (SAFTE) applied model integrates quantitative information about (1) circadian rhythms in metabolic rate, (2) cognitive performance recovery rates associated with sleep and cognitive performance decay
rates associated with wakefulness, and (3) cognitive performance effects associated with sleep inertia to produce a three-process applied model of human cognitive effectiveness.

The Fatigue Avoidance Scheduling Tool (FAST™) is based upon the SAFTE applied model. FAST™ was developed initially as an Air Force product under the Small Business Innovation Research program to deal specifically with Air Force scheduling issues. At this writing, it is a Windows® program that estimates the average effects of various work-rest schedules on human cognitive performance based on information supplied about an individual’s work and sleep patterns. These data may be entered into the computer in any of several formats.

Alternatively, there are two other world-class, quantitative models that may be used to help predict fatigue occurrence in shift work. One is the System for Aircrew Fatigue Evaluation (SAFE), which focuses mainly on aviation issues (Civil Aviation Authority, 2005). The other is the Fatigue Audit InterDyne™ (FAID), which focuses on estimating the risk of fatigue-induced errors (http://faid.interdynamics.com/). FAID is authorized for use as a tool in the implementation of fatigue risk management systems in Australian civil transportation sectors (aviation, rail, highway, maritime) and military operations. It has also been used in specific implementations in the U.S., Canada, the UK, South Africa, and Southeast Asia.

At a more simplistic level, the level of fatigue in personnel can be assessed by using the USAFSAM Fatigue Scale, developed in the early 1980s and used in many laboratory and field research studies:

1. Fully alert; wide awake; extremely energetic
2. Very lively; responsive; but not at peak
3. Okay; somewhat fresh
4. A little tired; less than fresh
5. Moderately tired; let down
6. Extremely tired; very difficult to concentrate
7. Completely exhausted; unable to function effectively

A score of 6 or 7 should disqualify an individual from performing safety-sensitive work such as driving vehicles, operating aircraft, using a weapon, making command-and-control decisions, etc. The fatigue scale may be used repeatedly to look for trends. Additionally, a one-time assessment of sleepiness may be accomplished with the Epworth Sleepiness Scale:
**Epworth Sleepiness Scale**

How likely are you to doze off or fall asleep in the following situations, in contrast to just feeling tired? Though you may have not done many of these things recently, please estimate their effect on you the best you can.

Use this scale, and enter one number on each line:

0. Would *never* doze  
1. *Slight* chance of dozing  
2. *Moderate* chance of dozing  
3. *High* chance of dozing

a_____ Sitting and reading  
b_____ Watching TV  
c_____ Sitting inactive in a public place; for example, a theater or meeting  
d_____ As a passenger in a car for an hour without a break  
e_____ Lying down to rest in the afternoon when circumstances permit  
f_____ Sitting and talking to someone  
g_____ Sitting quietly after lunch without alcohol  
h_____ In a car while stopped for a few minutes in traffic

A sum of 10 or greater is cause for concern if safety-sensitive tasks are to be performed.

**4.3.8. Biology, Night Work, and Shift Work**

In terms of human biology, night work is a crime against nature. We cannot see well in the dark. Our metabolism slows overnight until it reaches a low point, usually during the pre-dawn hours. In the dark, the pineal gland at the base of the brain releases the hormone melatonin, which, in turn, makes us feel drowsy. At night, the likelihood compared to daytime that we will sleep when lying down comfortably with our eyes closed is very high. Our brains and bodies are designed to sleep at night and to work during the day. Thus, when an operation requires staffing 24 hours per day, 7 days per week (24/7), there is no “good” shift work schedule to be found because some personnel simply will be forced to work at night. However, there are an infinite number of possible shift work schedules. The principle-based approach to scheduling described in the AF Research Laboratory’s shift work scheduling manual (Miller, 2006) constrains the infinite number to those schedules that are simple, practical to implement, and least harmful to worker health, job performance, and attitude. Thus, the constraints should help produce the least-injurious schedule for a given operation.

**4.3.9. Safety and Productivity in 24/7 Operations**

According to the National Sleep Foundation, people who are sleep deprived think and move more slowly, make more mistakes, and have difficulty remembering things. These negative effects lead to lower job productivity and can cause accidents. One of the leading shift work research centers combined the findings from numerous field studies conducted in companies engaged in 24/7 shift work (Folkard and Tucker, 2005).
The results of their efforts show you what to expect in terms of safety and productivity when shift work is used. The combined data from field studies showed that:

“Risk [of injuries and accidents] was found to increase in an approximately linear fashion across the three shifts, …18.3% on the afternoon shift…30.4% on the night shift, relative to that on the morning shift.” (Figure 4.3.9-1, from eight studies)

“…’real-job’ speed and accuracy measures are only above average between 0700 hrs and 1900 hrs; at all other times efficiency is likely to be relatively impaired, especially so during the early hours of the morning” (Figure 4.3.9-2, from three studies).

![Relative Risk Graph](image_url)

**Figure 4.3.9-1. Relative Risk of Injuries and Accidents across the Three Shifts**
(data from 8 field studies; redrawn from Folkard and Tucker, 2003)
There is an important caveat with respect to this and subsequent graphs concerning the relative risk of injury and accidents in industry: it is highly likely that injuries and accidents are underreported in industry. In theory, the use of a relative risk measure accounts for that underreporting. However, if the underreporting pattern varies across shifts and/or across days of the week, then the correction may not be valid.

Field-study data also indicated that, in the short term, acute fatigue may be offset to some degree by work breaks (Figure 4.3.9-3). Following a 15 minute break after 2 hours of continuous work, “risk [of injuries and accidents] rose…approximately linearly, between successive breaks … risk had doubled by the last 30 minute period.” There was “…no evidence that this trend differed for the day and night shifts.” (Of course, there is a higher absolute risk during the night shift, as shown in Figure 4.3.12-1). This risk pattern argues for a high frequency of rest breaks. However, if the handing-over of a task from worker to worker involves high risk, then the risk-related benefit of frequent breaks may be lost.

Also according to the National Sleep Foundation, the risk of workplace accidents and automobile crashes rises for tired shift workers, especially on the drive to and from work. People think that opening the car windows or listening to the radio will keep them awake. However, studies show that these methods do not work. In fact, attempts to use these countermeasures should be taken as a signal of fatigue and the need to pull over immediately. Sleepiness at the end of a shift should signal the need for a nap before driving home. Allow 5 to 20 minutes for sleep inertia (feelings of grogginess and/or sleepiness that occur immediately after waking up) to wear off, as needed.
Follow these steps to arrive home safely:

- Carpool, if possible; have the most alert person do the driving.
- If sleepy, stop to nap, but do so in a locked car in a well-lit area. Allow 5 to 20 minutes for sleep inertia to wear off, as needed.
- Take public transportation, if possible.
- Drive defensively.
- Don't stop off for a "night cap." Alcohol, combined with fatigue, increases the risk of a fatigue-related accident quite sharply.

These actions may often be viewed as impractical or unnecessary because night workers almost always drive home safely after their night shift. Similarly, drunk drivers almost always drive without accidents. However, just as the Air Force does not condone drunk driving, we should not condone the idea of sending a highly fatigued driver out the gate of an Air Force base and into traffic or a long commute on a boring highway. A recent court case provided an example of what can happen to a fatigued night worker on his way home (Escoto, 2001). Nabors Drilling Co. employee Roberto Ambriz fell asleep at the wheel on Texas State Highway 490 near the city of Raymondville about 20 minutes after ending his graveyard shift in March of 1998. His
pickup truck went into the oncoming lane, striking a Dodge pickup being driven by Martin Rodriguez. Rodriguez and his three passengers died at the scene, and Ambriz died from his injuries 2 days later. A decision was won against the employer for failure to train employees who work graveyard shifts about the risks of driving after working a graveyard shift.

4.3.10. Practical Recommendations

Air Force personnel and supervisors should act to improve the practicality of the preventive actions above whenever possible. For example, the use of public transportation may delay the onset of daytime sleep for night workers substantially. Thus, supervisors should consider providing and requiring the use of a carpool with a rested, trained driver for night workers with relatively long commutes. Similarly, supervisors should consider providing and requiring the use of pre-drive napping quarters for night workers and fatigued workers who are ending their shifts. Supervisors should also consider added training sessions for night and rotating-shift workers concerning defensive and intoxicated driving.

The following recommendations may be applied as fatigue countermeasures in the 24/7 workplace (also, see Miller, 2006):

- Schedule a good-quality break of at least 15 minutes once every 2 hours or more frequently.
- Whenever safe and acceptable, insert a nap into the duty day. Don’t worry about nap length – any sleep is good. Allow at least 30 minutes after the nap before performing safety-sensitive jobs.
- Keep duty periods relatively short – no more than 8 hours long.
- Schedule no more than three night shifts in a row.
- Schedule at least 24 hours of uninterrupted rest after night shifts.
- Schedule days off in continuous periods of at least 3 days.

The following recommendations may be applied at the individual level as general fatigue countermeasures:

- Reduce stress as much as possible.
- Exercise to stay fit.
- Keep mentally stimulated.
- Eat properly.
- Stop smoking.
The following recommendations may be applied at the individual level as sleep aids:

- Establish a bedtime ritual.
- Take a warm bath before bed.
- Noise: If you can’t avoid noise, try soft earplugs or create soft white noise by running a fan or air conditioner or setting the tuner of a radio between two stations.
- Light level: Use dark fabric to block windows or the rim of a door. Or, try eyeshades.
- Temperature and humidity: Try to maintain a comfortably cool environment (about 70°F) and 60% - 70% humidity. A humidifier or dehumidifier may also provide soft white noise.
- Security: As part of your bedtime ritual, check door locks and close windows.
- Clocks: Hide illuminated clocks from view. If you’ve established a regular sleep schedule, you may not even need an alarm to wake you up.
- Cleanliness: Keep the bedroom clean and free of clutter. Piles of clothes, reports, and bills induce feelings of stress.
- Nightclothes: Choose loose-fitting, soft garments that breathe, in the right weight for your bedroom’s temperature.
- Bed sheets and pillows: Use bed sheets that are clean, cool, and comfortably soft. Use a good pillow that puts you in a healthy sleep posture.

References
Escoto F, et al. vs. The Estate of Robert Ambriz et al., Cause No. 00-81, Raymondville, Willacy County, Texas, 197th Judicial District, 16 November 2001.
Civil Aviation Authority. Aircrew Fatigue: A Review of Research Undertaken on Behalf of the UK Civil Aviation Authority. CAA PAPER 2005/04, Research and Safety Analysis Section, Safety Regulation Group, October 2005; Internet Resources, See Appendix 8
Fatigue Audit InterDyne (FAID); Internet Resources, See Appendix 8

Recommended Readings
National Sleep Foundation website; Internet Resources, See Appendix 8

**Concepts**
- Fatigue avoidance scheduling tool (FASTTM)
- Sleep, activity, fatigue, and task effectiveness (SAFTE)
- Human factors engineering (HFE)

**Vocabulary**
- Acute fatigue
- Chronic fatigue
- Chronic fatigue syndrome (CFS)
- Circadian
- Cumulative fatigue
- Jet lag
- Physical fatigue
- Shift lag
4.4. Human Systems Integration

Robert M. Lindberg and William Kosnik, Ph.D.

4.4.1. Human Systems Integration Perspective

Every Air Force system involves airmen from weapons to medical to human resource management systems; there are no unmanned systems within the Air Force. In today’s complex warfighting environment, and even more so in the future, airmen require the same, if not greater, attention and care as any acquired, modernized, or procured system (traditionally thought of as the hardware, software, and data products). The complexity associated with achieving human performance, as a component of total system performance, compared to even the most sophisticated military technology and concept of operations (CONOPS) mandates a robust human systems integration (HSI) program integrating nine crosscutting domains (Table 4.4.1-1). From this perspective, HSI must span the entire spectrum of force activities, from concept refinement, research and development, personnel selection, through acquisition and fielding, to operational employment and disposal.

Table 4.4.1-1. Human Systems Integration Domains

<table>
<thead>
<tr>
<th>Human Factors</th>
<th>Environment</th>
<th>Training</th>
<th>Occupational Health</th>
<th>Habitation</th>
<th>Survivability</th>
<th>Safety</th>
<th>Personnel</th>
</tr>
</thead>
<tbody>
<tr>
<td>the comprehensive integration of human capabilities and limitations (cognitive, physical, sensory, and team dynamic) into systems design to optimize human interfaces to facilitate human performance in training operation, maintenance, support, and sustainment of a system.</td>
<td>in the context of HSI, environment includes the conditions in and around the system and the concepts of operation that affect the human’s ability to function as a part of the system as well as the requirements necessary to protect the system from the environment (e.g., radiation, temperature, acceleration forces, all-weather ops, day-night ops, laser exposure, air quality within and around the system, etc.).</td>
<td>the instruction and resources required providing personnel with requisite knowledge, skills, and abilities to properly operate, maintain, and support a system.</td>
<td>the consideration of design features that minimize risk of injury, acute and/or chronic illness, or disability and/or reduce job performance of personnel who operate, maintain, or support the system.</td>
<td>factors of living and working conditions that are necessary to sustain the morale, safety, health, and comfort of the user population that contribute directly to personnel effectiveness and mission accomplishment and often preclude recruitment and retention problems.</td>
<td>the ability of a system, including its operators, maintainers, and sustainers, to withstand the risk of damage, injury, loss of mission capability, or destruction.</td>
<td>the application of systems engineering and systems management in conducting hazard, safety, and risk analysis in system design and development to ensure that all systems, subsystems, and their interfaces operate effectively, without sustaining failures or jeopardizing the safety and health of operators, maintainers, and the system mission.</td>
<td>the human aptitudes, skills, and knowledge; experience levels; and abilities required to operate, maintain, and support a system at the time it is fielded.</td>
</tr>
</tbody>
</table>
4.4.2. Warfighting Capability

Militarily useful weapons systems achieve superior warfighting capability through total system performance (Figure 4.4.2-1). In an operational context, warfighting capability is best represented by probability to kill (Pk), operational availability (Ao), and operator reliability (Rop), better known as human performance. Central to Pk, Ao, and Rop is who, what, when, where, and how the weapons systems will be used (CONOPS); through which technologies (hardware, software, and data products); and by what group of airmen performing the mission.

![Total System Performance](image)

**Figure 4.4.2-1. Total System Performance (adapted from Bost, 2005)**

To deliver militarily useful, operationally suitable, and effective weapons systems while delivering total systems performance and minimizing total ownership cost, the Air Force is building out a solid human performance and HSI program as directed by Health Services (AFDD 2-4.2, 2002), Operation of the Defense Acquisition System (DoDI 5000.2, 2003), and Operations of Capabilities Based Acquisition System (AFI 63-101, 2005). HSI explicitly integrates the nine crosscutting domains (Figure 4.4.2-2). This integration, in simple terms, leads to these outcomes (i.e., human-machine interface design; knowledge, skills, and abilities of airmen; work distribution between airmen and the technology; and whether airmen are qualified, rested, motivated, vigilant, and healthy) during all phases of military operations. Leading to and underpinning human performance are human capabilities and competencies, work to be performed, and human fitness for duty.
Figure 4.4.2-2. Building Blocks of Human Systems Integration (adapted from Bost, 2005)

4.4.3. What is Human Systems Integration?

Many people view HSI as being primarily concerned with the interface between the human and machine and therefore synonymous with human factors engineering. This view actually encompasses only a single domain within HSI. In the broadest sense, HSI is an effort to make human performance, within the context of total systems performance, a top priority in materiel and nonmateriel solutions. HSI is a process to ensure systems are affordably conceived, designed, and developed to optimize combat capability. This total systems approach focuses on human performance and ensures capabilities, limitations, opportunities, and risks are identified and managed throughout the process within the Defense Acquisition, Technology, and Logistics Life Cycle Management Framework (Figure 4.4.3-1). HSI starts when the Joint Capabilities Integration and Development System (JCIDS) process begins as indicated in Figure 4.4.3-1. If a nonmateriel solution (e.g., change in tactics, techniques, and procedures) is selected, HSI considerations would appear in the Doctrine, Organization, Training, Materiel, Leadership, Personnel, and Facilities (DOTMLPF) change requirement document (DCR) rather than in an initial concept document (ICD).
An effective HSI program requires planning, integration, and timely application. First, acquisition planning groups such as high performance teams (HPTs) and integrated product teams (IPTs) must incorporate the nine functional HSI domains into program requirements. Traditionally, the HSI domains (Table 4.4.1-1) have been employed as separate entities, typically using a “stovepipe” approach where each discipline is applied individually in the acquisition process. Proper implementation of HSI by HPTs and IPTs involves trade-off analyses to achieve an optimal design. This provides a common basis upon which to make knowledgeable decisions.

Second, HSI integrates the three components of a weapons system – hardware, software, and personnel (DoD 5001.1, 2005, E1.29) – to optimize total system performance. Past experience has shown that acquisition programs have frequently failed to consider the human component as part of the system, resulting in poor task allocation between hardware, software, and human performance.

Third, HSI is applied ideally during the JCIDS process, well before the concept refinement phase is begun, where it is mostly likely to positively affect total system performance and life cycle costs. Early application of HSI provides the best opportunity to maximize return on investment (ROI) and system performance. In reality, though, HSI must often be applied to legacy systems that are well along the life cycle chain. As shown in Figure 4.4.3-1, however, HSI may be applied anywhere in the system life cycle. Aerospace physiologists (APs) are most likely to encounter fielded weapons systems in the sustainment and operations phase of the life cycle. It is here that the AP may apply HSI to identify performance gaps, failures, and incompatibilities that may limit optimal functioning or cause hazardous conditions.

4.4.4. How Is HSI Applied?

For new weapons systems, the AP may be required to perform an HSI assessment at the beginning of the acquisition cycle to ensure compliance with human-centered design goals. In this instance, a bottom-up approach is taken, starting with an analysis of human performance requirements at each of the HSI domains. A
A comprehensive plan should be developed to address each HSI domain requirement. The HSI plan should support each phase of the life cycle (concept refinement, technology development, system development and demonstration, production and deployment, and operations and support). Requirements are derived from the operational user’s identification of gaps in current capability through a mission task analysis. These gaps can be filled through changes in doctrine, manpower, training, and materiel solutions. Trade-off analyses are conducted to ensure that the final solution addresses the user’s needs by the most effective, safe, and affordable means possible. Requirements developed in the HSI plan are transmitted to system developers through a series of progressively more detailed documents such as the initial capabilities document (ICD), capabilities development document (CDD), capability production document (CPD), and request for proposals (RFP) that support the acquisition process. HSI requirements are addressed in capability documents with associated key performance parameters (KPP) and key system attributes (KSA). This allows HSI requirements to become measurable, which is necessary for effective implementation in the acquisition stream.

4.4.5. HSI – Why Now?

The Air Force currently faces challenges that will affect the way it prepares for, conducts, and wins conflicts in the 21st century. Financial constraints are forcing the Air Force to become far more efficient than ever before with both materiel and human resources. The Air Force can no longer rely on the cost savings of a smaller force for the recapitalization of legacy systems. This current climate is forcing the Air Force to look more closely at balancing the contributions and costs of airmen, policy, and technology (Retelle and Chatelier, 2005). Considering the significant mission and personnel challenges the Air Force will face in the future, along with the unyielding threat environments, it is imperative that the human element of Air Force systems be continually addressed and optimized.

HSI enables the Air Force to leverage its human and technological resources to meet the challenges of the 21st century. By building the human into systems at every level—through capability definition, acquisition, systems development, training and education, operations, and maintenance—HSI can make the most of existing resources throughout the life cycle of its weapons systems. It is essential that HSI be considered early in, as well as throughout, the Defense Acquisition, Technology, and Logistics Life Cycle (Retelle and Chatelier, 2005).
4.4.6. The Aerospace Physiologist’s Role in Human Systems Integration

The mission of Aerospace Physiology is to enhance warfighter performance through aerospace medical training, education, research, and consultation. By its very nature, Aerospace Physiology is “human centered.” Aerospace Physiology has faced HSI domain issues throughout the history of aviation in a continual effort to maximize warfighter performance. Nevertheless, in the past there has been a tendency to apply HSI on a piecemeal basis with little consideration of the entire process. Costly modifications, unsafe systems and operations, excessive life cycle costs, and technology poorly suited to the warfighter have plagued AF weapons systems due to a lack of or improper HSI application. Furthermore, as missions become more complex and the flight environment more demanding, the systematic application of HSI is more critical than ever to ensure optimal performance and seamless integration of systems across the entire enterprise.

Organizations must focus attention within the JCIDS (CJCSI 3170.01, 2007) and the operation of the Defense Acquisition System (DoDI 5000.2, 2003) processes as illustrated in Figure 4.4.3-1. To do so, HSI requires highly qualified practitioners, such as aerospace physiologists, who can effectively and affordably integrate and evaluate human capabilities in new and existing weapons systems. More specifically, aerospace physiologists center attention on total system performance by integrating trade-offs and identifying capability gaps within and across the HSI domains of manpower, personnel, training, human factors engineering, environment, safety, occupational health, survivability, and habitability.

Aerospace Physiology is one of many career fields that make up the HSI community of practice (Figure 4.4.6-1). These fields contribute specialized knowledge of the human – physiology, cognition, psychology, behavior – along with knowledge of engineered systems – engineering, computer science, and mathematics – to optimize total system performance. The AP facilitates the integration of human and machine by applying HSI principles to the engineering systems that serve to sustain and augment human physiological processes. As such, HSI principles and practices may be applied to virtually every aspect of Aerospace Physiology as described in this handbook.
4.4.7. HSI as a Process Model of Human Performance

Aerospace Physiology grew out of efforts to cope with the demands of a harsh flying environment. When analyzed from a human performance perspective, the missions of aerospace physiology conform to an HSI process model. This handbook provides many examples of Aerospace Physiology missions that reflect the application of the nine HSI domains. For example, successful human flight required finding solutions to mitigate hazards of altitude, cold, acceleration, and other factors. In HSI these hazards are associated with environmental domain issues. Several sections in this handbook document efforts to solve these environmental problems that result in illnesses (performance gaps) such as hypoxia, decompression sickness, hypothermia, and motion sickness.

Similarly, the combat environment has placed additional demands on the integrity and performance of the airman. Aerospace Physiology has responded to these threats by developing performance enhancement systems such as cockpit displays, NVGs, head-up displays (HUDs), anti-G suits, and protective devices such as ejection
equipment and laser eye protection. These solutions have emerged from requirements stemming from the human factors engineering, survivability, and safety domains.

An increasingly hostile combat environment has forced other aircrew adaptations as well. Advancements in speed, altitude, maneuverability, and endurance in high-performance aircraft have increased the amount of spatial disorientation, vibration, noise, fatigue, and stress that must be tolerated by the aircrew. Aerospace Physiology has developed countermeasures to cope with these problems by tapping into the domains of training, occupational health, and habitability. Solutions have come in the form of situation awareness training, noise and vibration reduction devices, fatigue countermeasures, nutritional supplements, performance enhancers, and crew resource management training. The challenging flight environment and the need for protection and sustainment systems have driven the need to constantly select, train, and allocate qualified personnel to the mission. These activities highlight the domains of manpower, personnel, and training. These examples demonstrate that HSI continues to be the way of doing the business of Aerospace Physiology and that future application of HSI is necessary to provide trained, healthy, motivated, and capable airmen to the AF mission.

4.4.8. Capability Gap Analysis

One area where aerospace physiologists can provide leadership is capability gap analysis as mandated in Aerospace Medical Operations (AFI 48-101, 2005). Most often, the duties of the AP are required at the operations and sustainment phase of the system life cycle, that is, with fielded systems. However, no matter where the system resides in its life cycle, deficiencies and cost inefficiencies can be identified through a capability gap (Cap Gap) analysis. A Cap Gap analysis improves the aerospace team’s understanding of the mission and human performance shortfalls in mission execution. This analysis is performed within each of the nine HSI domains by applying a rigorous set of performance metrics to identify performance gaps. Although it would not be expected that APs become experts in Cap Gap analysis, it would be advantageous for APs to have a working knowledge of the main issues to more fully understand how HSI may be applied to identifying problems at the mission level.

APs understand both the limits of human performance and methods for improving performance. They can, therefore, provide valuable inputs into the capabilities-based assessment process in terms of:

1. Addressing the limits of human performance (tasks, conditions, standards).
2. Conducting analyses on nonmaterial solutions and formulating DCRs.
3. Ensuring HSI is addressed in high-level system measures of effectiveness (MOEs) in ICDs when a material solution is selected.
4. Performing a Cap Gap analysis to determine the performance impact of a deficiency and its mitigation.
As discussed in this handbook, physiological imbalances or deficiencies can lead to performance degrading outcomes. These deficiencies are viewed as performance gaps in HSI terms. A Cap Gap analysis consists of the following steps:

1. Assess performance.
2. Identify capability gap.
3. Measure performance gap to quantify effect.
4. Find root causes of gap with respect to the nine HSI domains.
5. Perform trade-off analyses.
6. Implement best solution to mitigate gap.

The next two sections provide examples illustrating the use of a Cap Gap analysis in aerospace physiology operations.

**4.4.9. Capability Gap Analysis in Unmanned Aircraft System Operations**

The problem of fatigue in remotely piloted unmanned aircraft systems (UASs) serves as an apt example of a Cap Gap analysis (Miller et al., 2008; Tvaryanas and Thompson, 2006). UAS crewmembers must often endure extended duty days, reduced crew size, and varying shift schedules (Walters et al., 2002) to accomplish long-endurance UAS missions. Stressful, long-duration missions often reduce operator effectiveness because of fatigue. A capability gap analysis can be used to assess the root causes of fatigue by applying the HSI process model. This approach offers a comprehensive method of identifying the sources of fatigue as well as a means for formulating remediation.

The problem of fatigue in shift workers can be attributed to both physiological factors (difficulty adjusting the circadian rhythm to a new work/rest schedule, strenuous physical activity) and institutional factors such as personnel selection, scheduling, training, and manpower (Miller et al., 2008). The human performance pyramid (Figure 4.4.9-1) may be used to identify the relevant domains associated with fatigue using a top-down approach. A human performance gap (fatigue in this case) can be attributed to fitness for duty and workload issues. Fitness for duty, in turn, implies that the workforce is not adequately rested or vigilant. Workload issues might indicate an unbalanced workload distribution. These issues point to HSI domains of personnel, manpower, training, environment, occupational health, and safety. The relationship between fatigue and these domains is discussed in more detail below.
4.4.10. Personnel and Training

UAS operators must have the necessary qualifications to operate the UAS. New personnel (i.e., sensor operators) are being trained for UAS operations that do not come from the traditional pilot/officer recruitment and training programs. Less emphasis is placed on manned pilot experience. Therefore, performance will be affected given the change in scope and initial experience level. Because few data exist on the performance aspects of UAS operators, the necessary knowledge, skills, and abilities may be underestimated, resulting in an excessive workload that may contribute to fatigue. Inadequate training may also add to workload demands and operator ineffectiveness.

4.4.11. Manpower, Safety, and Occupational Health

The aerospace physiologist may be most familiar with problems of fatigue stemming from issues of shift work. Although the advent of UASs may have generated the prospect of regular duty hours, this has not turned out to be the case. In fact, the long-endurance capability of UASs has necessitated round-the-clock staffing of ground control stations (GCSs). Inadequate staffing can result in longer shifts or shorter work/rest cycles. Shift work has a disruptive effect on circadian rhythms, as discussed in Section 4.3, which can exact a heavy toll on health and safety through acute and chronic fatigue.
4.4.12. Trade-Off Analysis

A trade-off analysis can be used to vary the mix of solutions from several domains to find the most workable and cost-effective remediation. This may involve analyzing the benefits of adding resources from one domain to replace a solution from another domain that may be more costly. For example, one solution for mitigating fatigue may involve adding more experienced personnel or changing the ratio of work to rest days (manpower) rather than adjusting circadian rhythms (occupational health). Another solution might be to automate tasks to reduce the workload. Changes in habitability might also be considered. Family or other outside commitments could be adjusted to reduce stress and increase sleep periods. The aerospace physiologist should also be aware that trade-offs are not always a win-win situation. Often meeting performance requirements in one domain comes at a cost in another domain. Therefore, it is important to keep in mind that design decisions should be made around overall HSI requirements and not to specific domains.

4.4.13. Fatigue Mitigation Strategies

A successful Cap Gap analysis will point to potential remediation solutions. In a recent study on fatigue in UAS operations, Miller and colleagues suggested this mitigation strategy:

The root problem for this population was not the shift system features themselves, but rather a lack of adequate manpower to provide sufficient recovery opportunities. Thus, at best, all that can be recommended are preventive and compensatory measures. While it is desirable to minimize the number of consecutive night shifts, it is a reasonable alternative to continue the present schedule with multiple night shifts in succession and provide exposure to bright light during the night shift. While this will require modification of the GCS work environment (i.e., human factors engineering and habitability domains) and may not be immediately feasible, this feature should be considered in all future GCS design iterations. Other recommendations include educating supervisors and crewmembers as well as their spouses (i.e., training domain) on circadian rhythms, sleep disorders, the impact of shift work on family and social life, alertness strategies, safe driving, nutrition, physical activity, and coping with stress. Supporting medical personnel should ensure they have up-to-date knowledge of sleep disorders and shift maladaptation syndrome (i.e., training domain) and provide tailored medical surveillance of shift workers (i.e., occupational health domain).

Finally, supervisors should implement methods to mitigate the danger of post-shift fatigue on driving safety by providing organizationally-sponsored car pools and offering work locations for post-shift naps prior to driving home (i.e., safety and habitability domains) (Miller et al., 2008).

By examining the mitigation of fatigue from the perspective of the different HSI domains, the AP will be led to the best possible solution(s). Applying the optimal solution is an HSI best practice that has the advantage of distributing the cost of the mitigation to achieve the least impact on the overall life cycle cost.

Human performance is optimized when human error is reduced to a minimum. As such, HSI may be used as a model for analyzing performance failures as well as for performance optimization. Nowhere is the analysis of human error more important than in mishap investigations. A thorough mishap investigation is essential to finding the causes of an accident and for preventing recurrences.

The root causes of mishaps can occur at any level of a mission operation, from the operator to higher levels in the chain of command. Traditionally, a mishap investigation focuses on the events immediately prior to the accident, but in reality the cause of an accident can be traced through many levels of the organization. An analytical model that recognizes the complexity of the causation chain has been instituted by the DoD. The DoD Human Factors Analysis and Classification System (HFACS) is the primary tool to identify the causes of mishaps across all levels of the causation chain. A description of HFACS and its value to performing mishap investigations is presented in Section 8.4. HFACS classifies mishap causes as a series of hazardous conditions or behaviors that can occur at four different levels of the aviation enterprise (Reasons, 1990). These conditions, starting from the operator level, consist of:

1. Unsafe acts by the operator
2. Preconditions for unsafe acts
3. Unsafe supervision
4. Organizational breakdowns

A failure at any or all levels of the enterprise can result in a mishap. Consequently, preventative measures might need to be applied at several levels, not just at the operator level. A closer look at mishap analysis shows how the HSI process model can be used to help identify the sources of failure. Figure 4.4.14-1 gives the breakdown of the four classes of failure conditions. At the most basic level mishaps may be caused by errors associated with the operator. These acts include skill-based errors, judgment and decision errors, and perception errors. From the human performance process model (Figure 4.4.2-2.), these errors flow down from the human capabilities and competencies block to the knowledge, skills, and abilities block and finally to the training and personnel domains. Unsafe acts ultimately can be traced to deficiencies in training or selection.

The second level of failure conditions, Preconditions for unsafe acts, can involve environmental factors, individual states, or personnel factors. Environmental factors include stressors such as noise, thermal stress, or darkness. Adverse physiological states, such as fatigue, or psychological, cognitive, or perceptual factors can constitute predisposing conditions for failure. Such failures may be addressed as environmental, training, occupational health, human factors engineering, or personnel issues.

Errors at the supervisory level can also set up conditions for failure. Inadequate oversight, poorly planned violations, or supervisory violations are latent conditions that can lead to mishaps. The last tier of failure conditions can occur at the organizational level. Problems stemming from personnel, manpower, safety, survivability, or
occupational health policies can filter down through the chain of command to set up preconditions for operator failure. By touching on all nine domains, the mishap investigator can be sure that all possible sources of failure conditions will be considered at every level of the organization.

Figure 4.4.14-1. DoD Human Factors Analysis Classification Scheme for Mishaps Investigation (adapted from Wiegmann & Shappell, 2001)

4.4.15. Benefits of Human Systems Integration

The HASC Report (2006) notes that better program results can be achieved by an approach that focuses on long-term cost reduction. By applying a robust HSI program early in system development and acquisition, the program manager can maximize the overall return on investment in several important ways. Implementation of effective HSI practices and concentration on reducing the overall life cycle budget will tend to optimize system performance, reduce life cycle costs, provide more usable systems, and minimize occupational health hazards and opportunities for mishaps.

The Department of Defense cites that optimizing total system performance and minimizing the cost of ownership throughout a system’s life cycle are the primary benefits of HSI. By adhering to HSI principles, the following benefits will also be realized:
• HSI becomes institutionalized as a “way of doing business” within the Defense Acquisition, Technology, and Logistics Life Cycle Management Framework.

• Requirements, information, issues, limitations, opportunities, and concerns from collaboration among the functional domains (Manpower, Personnel, Training, Human Factors Engineering, Environment, Safety, Occupational Health, Habitability and Survivability) will be applied from a system’s pre-concept (inception) to disposal (grave) perspective.

• A human-centered approach to acquisition will be provided.

• The usability of systems through a focus on human-machine/technology interfaces will be improved.

• Warfighter and mission capabilities will be enhanced.

• System design will be optimized through an analysis of alternatives (AoAs), trade-off studies, and HSI tool use.

For Aerospace Physiology to fulfill its mission, it must continue to develop systems that support and enhance warfighter capabilities. The warfighter requires weapons systems that can be used effectively, safely, and without a large retraining component. The adoption of HSI principles and practices will ensure that Aerospace Physiology remains at the forefront of optimizing warfighter performance.

4.4.16. Training Opportunities

• AF Institute of Technology (AFIT) Courses @ http://www.afit.edu/ls/index.cfm
  • SYS 160, Introduction to Human Systems Integration Course
  • SYS 161, HSI in Systems Capabilities Requirements Course
  • SYS 162, HSI Roadmap Course (Under Development)
  • SYS 260, HSI in Defense Acquisition Management (Under Development)

• HSI Community of Practice (CoP)

References
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Bost JR. Presentation to 2005 Business Managers’ Conference, Defense Acquisition University.
CJCSI 3170.01F, Joint Capabilities Integration and Development System, 1 May 2007.
DoDD 5000.1, Defense Acquisition, 12 May 2003.

Recommended Reading
CJCSM 3170.01C, Operation of the Joint Capabilities Integration and Development System, 1 May 2007.

Concepts
Fatigue mitigation strategies
Human systems integration (HSI)
Human Factors Analysis and Classification System (HFACS)

Vocabulary
High performance teams (HPTs)
Human factors engineering (HFE)
Integrated product teams (IPTs)
4.5. Situational Awareness

Andrew D. Woodrow, Lt Col, USAF (Ret), BSC

So the crew fly on with no thought that they are in motion. Like night over the sea, they are very far from the earth, from towns, from trees. The motors fill the lighted chamber with a quiver that changes its substance. The clock ticks on. The dials, the radio lamps, the various hands and needles go through their invisible alchemy. From second to second these mysterious stirrings, a few muffled words, a concentrated tenseness, contribute to the end result. And when the hour is at hand the pilot might glue his forehead to the window with perfect assurance. Out of oblivion the gold has been smelted; there it gleams in the lights of the airport. (Antoine De Saint-Exupery)

4.5.1. Introduction to Situational Awareness

From the earliest cockpits fitted with rudimentary gauges to the 5th generation airframes wired for helmet-mounted displays, the impetus for developing an operational philosophy rooted to situational awareness has been grounded on human capacity to process environmental cues, the limits of which provide a continual challenge for engineers and operators. The concepts encompassing the term “situational awareness” (SA) are broad, but the reader may benefit by the following definition as a starting point in the understanding of SA:

The perception of the elements in the environment within a volume of time and space, the comprehension of their meaning and the projection of their status in the near future (Endsley, 2000).

The context of SA can be further divided into areas of geographical SA, spatial/temporal SA, system SA, environmental SA, and tactical SA; each subarea identifies the conditions and dynamic flow of activities within a period of time. The linkage between the elements is really a series of sampling from long-term memory concerning relative priorities and the frequency with which information changes, again all linked to perception. Safety analysis from the USAF Safety Center and the National Transportation Safety Board (NTSB) consistently shows two major contributing factors in human factors accidents: operational errors and decision/judgment errors. Elements of the errors include pilot skill deficiencies, task errors, bad decisions, and inadequate planning or situational awareness. Spatial disorientation (SD) is a subset of SA related to the flying environment and is discussed in its own section (Section 7.3).

4.5.2. Examples of Loss of SA

One of the most significant accidents investigated in the last decade revolved around the loss of SA between the crew and the breakdown in SA between the controller and the crew.

On December 20, 1995, about 2142 Eastern Standard Time, American Airlines flight 965, a regularly scheduled passenger flight from Miami, Florida, to Cali, Colombia, struck trees and then crashed into the side of a mountain near Buga,
Colombia, in night, visual meteorological conditions, while descending into the Cali area. The airplane crashed 33 miles northeast of the Cali (CLO) very high frequency omni-directional radio range (VOR) navigation aid. The airplane was destroyed, and all but four of the 163 passengers and crew on board were killed. (NTSB, 1996)

This accident demonstrates the need for training that effectively provides pilots with the ability to recognize when they have lost or have failed to obtain situational awareness. As a result, the NTSB forwarded a recommendation to the FAA to include specific guidance on methods to effectively train pilots to recognize cues that indicate that they have not obtained situational awareness and provide effective measures to obtain or regain that awareness (FAA AC 120-51B).

The focus of most analysis during accident investigation is on determining why higher order skills break down. In one typical example of a USAF mishap, a wide-body, long-haul cargo aircraft missed centerline of the intended runway and subsequently experienced extensive damage to the undercarriage due, in part, to loss of SA. During the approach to the airfield there was very little cockpit conversation, and radio traffic was minimal. The pilot stated it was obvious to her the crew was “tired and just wanted to get on the ground.” Joining the crew late in the descent, the jump-seat pilot was cognitively “out of the loop” and made no inputs at all during the mishap. The copilot reached the first segment of the approach 2,800 ft too high and 35 knots too fast. Shortly after the point to configure for landing, the crew failed to extend the flaps. The failure to extend the flaps to the landing configuration constituted a significant breakdown in crew communication and situational awareness. It is likely that due to the copilot’s nonstandard procedure to initially extend his own flaps, the pilot made the assumption that the copilot would also extend the flaps to landing. While the call was made and confirmed by both pilots, neither pilot actuated the flap handle, nor did either pilot confirm movement or full extension of the flaps.

Within the aviation domain, there are several concepts typically used to characterize performance, but most are difficult to define much less measure. For instance, in the military environment the concept of SA is used frequently to describe skills in maintaining an awareness of the tactical situation. Measure of SA, then, is based on outcome of the tactical objectives. The pathway to the tactical objective is lined with opportunities to sustain, lose, or regain SA. Unfortunately, there are no universally accepted tools to measure SA; the challenging domain for developing validated pilot performance measures remains in the performance outcome mode.

Aircraft ground operations can also be subject to loss of SA as demonstrated in the following excerpt from the NTSB:

On September 11, 1999, about 1958 central daylight time, a runway incursion involving United Airlines, a Boeing 767, and Delta Air Lines flight 1211, a Boeing 727, occurred at Chicago O’Hare International Airport (ORD), Chicago, Illinois. UAL2, which was being repositioned on the airport by two UAL mechanics, crossed runway 9L without air traffic control (ATC) clearance. DAL1211, which was departing from runway 9L at the time, passed directly over UAL2 at an altitude of 200 to 300 feet. The incident occurred in darkness under visual meteorological conditions. Neither airplane was damaged, and no injuries were reported. The mechanic who was taxiing UAL2 stated that he was looking for a sign identifying taxiway H. Both crewmembers stated that the area was very
dark and that they did not see any signs or lights identifying taxiway H. Thus, the absence of appropriate signage and markings at the runway 32R/taxiway H intersection apparently contributed to the loss of situational awareness experienced by the UAL2 crew, causing them to miss the turn onto taxiway H. (NTSB, April 24, 2000)

Detection and correction of such problems are important because, especially in unfamiliar situations, flight and ground crews depend on proper signs and surface markings to maintain situational awareness and avoid runway incursions.

The rapid growth of technology in the cockpit and aeronautical systems in general along with broadened operational roles is well known in aviation. Analytical operations by resource-limited operators are strained under the best conditions. It could be argued that even basic flight control under instrument meteorological conditions (IMC) involves analytical processing that will overwhelm situational awareness of the most seasoned aviator. It is well accepted that humans are not reliable monitors. To maintain situational awareness, the human operator needs to have an active role in the control loop. That said, the aim of any systems designer or even trainer should be for an internal (healthy) versus external (unhealthy) locus of control (Jensen, 1989). In addition, according to Arrabito, ensuring such cognitive compatibility as a function of situational awareness in the design of an alerting system may minimize the perceptual demand required for interpretation, thereby reducing the possibility of inappropriate responses under stress (Arrabito et al., 2004). Evaluating the cognitive compatibility of an alerting system as a function of situational awareness in an operational setting may not be practical. Under normal flying conditions, auditory alarms are sounded infrequently, and many flights are completed without an alarm being triggered. Likewise, incorporating the signals in simulators may not elicit the same response as would be expected in actual flight. Incorporating situational awareness in the design of an alerting system is expected to increase warning compliance. Such thoughtful auditory alarm design should elicit the operator’s attention and appropriate response, particularly under conditions of varying cognitive demands on aircrew. Indeed, human intelligence seems to be an essential requirement for successful performance on real-time dynamic problem-solving tasks.

Situational awareness is not a structure or “thing” that people possess. It is a dynamic process that is the result of cues both presented and perceived. Cues are typically channeled through the physiological sensory systems: visual, tactile, aural, olfactory, and even taste receptors. In the flight environment this cueing might consist of overt signals like a flashing warning light accompanied by an aural tone. In other circumstances the cues may be more subtle, a stick-shaker warning of a stall condition for instance. The distance and timeliness of the signal presentation influence the speed and accuracy of response by the operator. In the operational realm of remotely operated vehicles, the pilot may be thousands of miles away from the vehicle being operated with no direct signals perceived. If the only signal presented to support the pilot’s SA is a string of data displayed on one of three monitors, the signal must be compelling enough to gain and hold the attention of the pilot and guide or direct to an action point. As described by Endsley (1988), system sensors collect some subset of all available information from the system’s environment and internal system parameters. Of all the data the system possesses, some portion (determined by the designer) is displayed to the operator via its user interface. Of this subset, the operator perceives and interprets some portion, resulting in situational awareness. A complex process is
further complicated by the condition and motivation of the operator. The introduction of the “glass” cockpit and the third-generation airliner in the 1990s represented a significant change to the flight crew (from three to two) by automating many of the functions formerly managed by the flight engineer. Boeing states that the design has reduced the number of cockpit lights, gauges, and switches from more than 970 in the basic B-747 to only 365 in the B-747-400 (Wells, 2001). Good design and training have relieved pilots from many monotonous tasks of monitoring and freed them up for more cognitive tasks that are still beyond the capacity of computers. But has the reduction in workload increased the level of SA in the cockpit?

Approaches and landings at wrong airports are instances of disorientation and loss of SA on the part of the pilot. Most will agree that a discussion of spatial disorientation cannot exclude situational awareness as a component. Extensive literature exists on spatial disorientation in both clinical and aviation studies but rarely relates to way-finding. In the medical literature, topographical disorientation, a specific instance of spatial disorientation, is a term that relates to way-finding problems and focuses on patients. In aviation, way-finding problems have been termed geographical disorientation. In a study by De Voogt et al. (2007), the analysis of accident and incident reports led to insight into situational awareness problems of pilots. The pilot-navigator has to perform two tasks, known as local guidance or staying on a particular flight path and global awareness or knowing where things are with respect to one’s position and orientation. Landings and approaches to wrong airports refer to the latter. In other words, pilots may be in control of the aircraft and may conduct a well-executed landing or approach, but without being aware of the identity of the airport. Alternatively, distraction from good SA can result in loss of control. On December 29, 1972, an Eastern Airlines Lockheed Tristar crashed into the Everglades, killing 100 of 176 people aboard. In the darkness, the crew, preoccupied with a landing gear problem, failed to notice the autopilot had become disengaged and the plane was losing altitude (Figure 4.5.2-1).

![Figure 4.5.2-1. Loss of Situational Awareness](image)

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4.5.3. Modeling of SA

One of the popular theories underpinning SA is the mapping or modeling a human does when executing a task. The mental model developed in humans when approaching a series of tasks is a popular means of evaluating situational awareness. A well-developed mental model provides knowledge of the relevant parts of an operation and a means of integrating the elements to some level of understanding that leads to the ability to project future states of the system based on a snapshot of the current state and individual understanding of the system dynamics. The leading theory that explains this multitiered SA model is by Endsley (Figure 4.5.3-1).

Figure 4.5.3-1. Model of Situational Awareness Depicting the Levels of SA and Interaction between Environmental and Human Factors (Endsley, 1995).

The dynamic nature of the aviation environment (including air traffic control, aircraft marshalling duties, and systems maintenance) causes variations in the level of SA one possesses in a single period of time. The mental model is often referred to as a bucket of resources that is sampled as necessary. For instance, the air traffic controller must maintain an efficient mental model of the control area of responsibility combined with a rapid retrieval system for applying the knowledge of other important cues when needed. As described by Redding in Aviation Psychology in Practice (Johnston et al., 1997), the mental model provides a framework for more efficient training and learning, forming the basis for teaching knowledge and skills. Based on the mental model, controller procedures should be taught by event type, with training emphasizing the integration of sector aircraft information into sector relevant groupings.

Expectations of the status of a system are crucially important to perception and sustaining SA. The main function of expectation is to allow more efficient processing of information presented. For instance, try to recall word-for-word a conversation you have had with someone in the last 24 hr. Although it will be relatively easy to recount
the general sense of the conversation, the precise words used will likely be forgotten. In aviation, this becomes dangerous when there is a clear expectation as to exactly what should have been transmitted or displayed, but the operator will remember what was expected rather than the actual transmission. Because it is easy to remember the general sense of something, while forgetting the particulars, checklists are the mainstay of flight deck operations. Human memory is most fragile when there are stresses upon the physiological or psychological state of the operator. If one considers all of the information stored in long-term memory relevant to a particular task, it is easy to map out the location of “sector-relevant” information. For instance, consider driving a car on a familiar highway versus through a busy, unfamiliar downtown grid. In the first instance, many of the individual SA resources used for navigation, speed control, and eye-hand coordination can be given up for other, nondriving-related tasks like reviewing the shopping list, talking on the cell phone, or any one of a dozen other tasks. Compare that to the SA resources expended when navigating through a busy network of streets while searching for an unfamiliar address—clearly fewer resources are available for extraneous duties. Any technique for enhancing the amount of relevant information we perceive has to take account of the fact that visual cues are not enough; the driver must systematically look for something specific. This means that there is a link between learning the skill of driving well and learning to apply your vision to the things you need to see. In the driving example, there are fewer categories of things that need to be viewed than in aviation. Nevertheless, the store of knowledge still needs to be vast. Therefore, developing a scanning technique must be linked to the elements of thinking and anticipating effectively. Once basic skills sets are established and the SA “bubble” is expanded from a baseline, learning operations theory and applying skills through practical experience are required for an increase in risk perception. As driver training instructors in the United Kingdom harp to their students—“Mirror, Signal, Maneuver!”—the way to increase the perception of relevant information is to use a scan strategy, not just visually but across the mental model. Developing a systematized way of perceiving information in the environment elicits a regular sampling process without having to consider what to do first. This is particularly true during a single-seat aircraft emergency.

- Fly the airplane.
- Evaluate the proper action/checklist needed.
- Continue to fly the airplane and take the appropriate action/s.

This changes with a multiplace aircraft where the copilot is usually instructed to fly the airplane while the aircraft commander directs the emergency response while cross-checking that the aircraft is really being flown. Too often, the emergency occupies 100% of the pilot’s awareness, the result being loss of aircraft control unrelated to the particular emergency condition.

It could be argued that, with practice, the resources required to maintain control in the most cognitively demanding situations can be titrated efficiently to a successful outcome. The cognitive constructs and processes thought to underpin the SA process have received great attention by organizational and aviation psychology and now human factors engineers.
References

Recommended Readings

Concepts
Situational awareness (SA)

Vocabulary
Instrument meteorological conditions (IMC)
5. AIRCRAFT SYSTEMS EFFECTS

5.1. Pressurization Systems

James T. Webb, Ph.D.

5.1.1. Cabin Pressurization

Cabin pressurization is an increased atmospheric pressure in an aircraft cabin or cockpit. The increased pressure is maintained by pumping pressurized, conditioned air into the cabin from one or more jet engine compressors or an aircraft-powered electronic compressor. In transport and commercial aircraft, cabin pressurization usually keeps the cabin no higher than 8,000 ft. In fighter and most training aircraft, the cockpit pressure is dependent on the aircraft altitude and typically exceeds 10,000 ft, requiring supplemental oxygen.

During WWII, the problems of hypoxia, hypothermia, and altitude decompression sickness (DCS) plagued operators of the unpressurized B-17s and B-24s. The cabin pressurization designed for the B-29 provided a solution to these problems. Since that time, nearly all U.S. military aircraft capable of flight above 20,000 ft have been designed with pressurized cabins for the same reasons. The CV-22 is one notable exception.

5.1.2. Physiologic Requirements of Pressurized Cabins

Unpressurized aircraft potentially expose crew and passengers to an unacceptable level of hypoxia if altitude limitations were not imposed. The Federal Aviation Administration (FAA) and other regulatory authorities limit exposure altitudes for unpressurized aircraft and pressurized aircraft that lose pressurization. Those regulations stipulate that crewmembers must not be exposed to altitudes above 10,000 ft and passengers not above 12,000 ft without supplemental oxygen. The altitude allowed for crewmembers may be lowered at night due to the effects of hypoxia on night vision.

Cabin pressurization allows a “shirt sleeve” environment for crew and passengers of transport and commercial aircraft. Keeping the cabin pressure to less than 10,000 ft, typically 8,000 ft in most military transport aircraft and 6,000 to 8,000 ft in commercial aircraft, eliminates the problem of DCS and minimizes loss of function due to hypoxia. At 8,000 ft (Appendix 1a), the PAO₂ (calculated) of 69 mmHg provides an arterial oxygen saturation of 87% - 95% during rest. This level of oxygenation ensures adequate cognitive performance and reasonable physical capabilities; although some prolongation of reaction times to accomplish previously unlearned tasks has been reported (Denison et al., 1966). These results were obtained with the source of pressurization being atmospheric air (21% oxygen). Crewmembers that are more physically active will experience some reduction in physical and cognitive performance due to the increased oxygen demand associated with mild to moderate exercise at 8,000 ft (Macmillan, 2006).
5.1.3. Physical Limitations of Aircraft Pressurization Systems

When an aircraft is pressurized, the pressure inside the aircraft pushing out exceeds the atmospheric pressure pushing in from the outside (Figure 5.1.3-1). This pressure differential requires that the aircraft be capable of maintaining its structural integrity. For aircraft that utilize a high differential pressurization system (transport/cargo, commercial aircraft), the aircraft skin and structure must be stronger. This adds weight to the aircraft and increases the stress on the fuselage during repeated applications of the differential pressure. In addition to more bleed air or compressed air required to supply the cabin, which further reduces aircraft engine efficiency, maintaining a higher differential represents a physical limitation on aircraft design.

![Fig 5.1.3-1. Cabin Pressure Differential](image)

5.1.4. Types of Aircraft Pressurization Systems

As shown in Figures 5.1.4-1 and 5.1.4-2, hot, pressurized air from the engine compressor or an air intake port is allowed to decompress and cool (adiabatic cooling and refrigeration) to the desired cabin inlet pressure and temperature to maintain the desired cabin environment. An outflow valve controls the pressure by modulating the rate of porting air overboard and is, in turn, controlled in the cockpit by the crew. The engineer (crewmember) of transport and commercial aircraft normally sets automatic controls, which pressurize the cabin to the desired cabin pressure, typically 6,000 to 8,000 ft. This ensures a smooth cabin depressurization to that altitude, and then maintaining whatever differential pressure with the ambient (outside, atmospheric) pressure is necessary. On descent, the same cabin altitude controller adds pressure to the cabin to pressurize it to the destination field pressure altitude. Fighter and most dual-place trainer aircraft usually follow an automatic pressurization schedule such as shown in Figure 5.1.5-1.
The pressurization sometimes felt at ground level when the doors of a commercial aircraft are closed is the cabin air conditioning system adding a small amount of pressurization. The cabin air conditioning system is part of the cabin pressurization system (environmental control system, ECS) and derives its air source from the aircraft engines or a ground air cart. When the doors close, the air supplied does not have as many openings to depart the aircraft, hence a slight increase in pressure. As a commercial or military transport aircraft takes off, two processes are occurring at the same time. The aircraft is being decompressed by ascending to altitude just as your car would be if you drove up to the top of Pikes Peak, only faster. At the same time, the cabin altitude controller is attempting to keep the aircraft cabin from ascending above about 8,000 ft (typical cabin altitude during flight above 8,000 ft) as the aircraft passes that altitude. Your car doesn’t have that option. Therefore, during ascent the cabin altitude controller begins pumping air into the cabin. The amount of air pumped in is regulated by an outflow valve. During descent from cruise altitude, e.g. 35,000 ft, the cabin altitude controller adds more air to the cabin to bring it down from 8,000 ft to sea level (assume you are landing at Honolulu International) in a smooth, slow recompression. The cabin altitude controller actually starts to recompress the cabin well above 8,000 ft so that the descent and repressurization of the cabin occurs more slowly than if it waited until the aircraft passes 8,000 ft during descent, making it easier for passengers to clear their ears.

Figure 5.1.4-1. Aircraft Pressurization System – Engine Source
**5.1.5. Aircraft Pressurization Schedules**

A pressurization schedule is defined as the relationship between cabin altitude and aircraft altitude. Transport aircraft that routinely carry passengers have pressurization schedules that create a high differential pressure between the cabin and aircraft altitude to maintain passenger comfort. With the high differential pressure, the cabin can be maintained below 10,000 ft where supplemental oxygen (oxygen mask) is not required. Most military transport aircraft and commercial aircraft maintain a cabin altitude of 6,000 or 8,000 ft, called an isobaric pressurization system because it maintains a constant cabin altitude despite changes in aircraft altitude (Figure 5.1.5-1). They can manually depressurize and repressurize to accommodate the mission scenario (e.g. air drop). These aircraft cabins decompress during the climb from ground level to 8,000 ft and then supply pressure to maintain 8,000 ft during further climb.
Fighter and some other military aircraft are not pressurized until they reach 8,000 ft, where the pressurization system maintains 8,000 ft isobaric pressure until the aircraft reaches about 23,000 ft (Figure 5.1.5-2). At that altitude, a 5.0 psid (differential pressure) exists between the cabin and ambient (outside) air. This type of pressurization system is therefore called an isobaric differential pressurization system.

The 5 psid with ambient pressure is maintained by the system during any remaining climb as shown in Figure 5.1.5-1. For aircraft with a 5-psid pressurization system capable of cruise at altitudes greater than 50,000 ft, the cockpit altitude will exceed 20,000 ft. Oxygen masks are worn at all times in such aircraft, and their oxygen regulators provide adequate supplemental oxygen to keep the pilots from experiencing hypoxia. At 70,000 ft aircraft altitude, the cabin of an aircraft with a 5-psid pressurization system would be at nearly 25,000 ft. This represents an exposure consistent with development of DCS during more than 5% of the exposures even with no physical activity if the time at 70,000 ft exceeds about 1 hour.

![Figure 5.1.5-2. Isobaric Differential Cockpit Pressurization Schedule](image)

5.1.6. **Advantages and Disadvantages of Pressurized Cabins**

There are several advantages of a transport aircraft pressurized to 8,000 ft.

1. Hypoxia and DCS are avoided without the use of supplemental oxygen and/or pressure garments.

2. Avoidance of hypoxia and DCS allows better utilization of crew and improved crew safety, leading to higher rates of successful mission completion.

3. Functional temperature control associated with cabin pressurization provides a shirt-sleeve environment for passengers and crew to perform their duties, thus enhancing performance and mobility while reducing fatigue.
Pressurized fighter aircraft cockpits are not sufficiently pressurized to prevent hypoxia while at aircraft altitudes exceeding about 25,000 ft without supplemental oxygen. Routine use of a helmet and mask connected to the oxygen regulator is required in these aircraft to prevent hypoxia.

Some transport aircraft, such as the C-5, maintain atmospheric pressure (14.7 psia at sea level) during the climb to 14,000 ft. At 14,000 ft and above, the C-5 maintains an 8.7 psid between internal cabin and external atmospheric pressure. This ensures that even at an aircraft altitude of 40,000 ft, the C-5 cabin would be below 7,000 ft pressure equivalent (8.7 psid + 2.7 psi at 40,000 ft = 11.4 psi, 6,900 ft).

The disadvantages of pressurized cabins are:

1. Most aircraft pressurization systems use bleed air from the engine compressor as a source of compressed air. This reduces engine efficiency. Compressors used on other aircraft require an energy source, which also reduces overall efficiency since electricity or hydraulics to run the compressors ultimately derive their power from the engines. The weight of these systems is added to the aircraft weight, which further reduces efficiency.

2. The differential pressure between the ambient aircraft altitude and the cabin altitude creates a stress on aircraft structures that reduces the life of the airframe, in particular, the aircraft skin covering the fuselage.

3. The initial cost of cabin pressurization systems is also a disadvantage, as it raises the total cost of the aircraft.

4. Loss of cabin pressure in a pressurized cabin creates a need for reduction in aircraft altitude to avoid hypoxic conditions and usually involves thermal stress for the occupants.

5. The reduction in altitude reduces efficiency in converting fuel to miles traveled.

6. A potential loss of efficiency makes maintenance of pressurization systems a priority, which uses time and materials (money) in addition to the initial cost of the systems. Loss of cabin pressure in U.S. military aircraft was reviewed by Files et al. (2005), providing a summary of the many possible physiologic outcomes.

5.1.7. Factors Affecting the Rate and Severity of a Decompression

The rate of a decompression refers to the speed at which the decompression occurs. It is determined by a number of factors. Some of these factors are determined by the reason for the decompression. Is it a mechanical failure of the pressurization system, in which rate of decompression is limited to the rate of altitude change of the aircraft? Is it an aircraft structural failure (hole in the aircraft skin or loss of a hatch, window, or canopy), in which rate of decompression is dependent on the size of the failed area? Or is it an operator error? An error in operation of the pressurization system can usually be corrected before complete decompression occurs. However, if the error involves inadvertent actuation of an irreversible decompression control,
decompression can be very rapid. If the depressurization is very slow, the cabin pressurization system may be able to make up for the rate of loss of air. The following factors apply to determining rate of decompression:

1. How big is the cabin (total cabin volume)? Usually, a large cabin will take longer to depressurize than a small one.

2. How big is the aperture (orifice area) causing the decompression (see Figure 5.1.7-1)? The bigger the hole, the faster the decompression.

3. What is the pressure ratio between the cabin and the ambient (outside) pressure before the decompression begins? The larger the ratio, the longer the decompression duration.

4. What was the pressure differential prior to the decompression? The severity of the decompression is determined by the differential pressure, and a higher differential pressure results in a faster initial rate of decompression.

5. What is the altitude at the time of decompression? The altitude at which the decompression occurs will affect the pressure ratio and the pressure differential. This is important due to the rate of onset of physiologic problems associated with decompression, the most important of which is hypoxia.

Equations (e.g. Haber-Clamann formula) exist to determine the rate of a decompression, taking these factors into consideration.

![Aloha Airlines Decompression Disaster](image)

**Figure 5.1.7-1. Aloha Airlines Decompression Disaster**

**5.1.8. Physical Indications of a Rapid Decompression**

A rate of decompression described as rapid (2 – 15 s) will be accompanied by several physical indications. It usually coincides with a loud noise, closely followed by a drop in cabin temperature. The temperature drops due to adiabatic cooling associated with expanding air, as well as equilibration with the ambient temperature at the aircraft altitude. The actual pressure drop may also be felt as trapped gas in the body expands. Light debris may accompany the rush of air moving toward the source of the decompression, and objects not secured and near the aperture may depart the aircraft.
Based on the pressure and temperature drop, the relative humidity may reach 100%, resulting in fog, another physical indication of a rapid decompression.

5.1.9. Physiologic Effects of Decompression

Any decompression carries some risk of trapped gas expansion; the degree is a function of initial and final pressures. Decompression to above 10,000 ft altitude carries an additional risk of hypoxia, although proper use of oxygen equipment will usually eliminate the effects until appropriate repressurization/descent can be accomplished. The exception is rapid decompression to an altitude where onset of hypoxia is rapid and severe, as discussed in the section on hypoxia. A slow decompression, once recognized, carries less risk of hypoxia due to the ability to take corrective actions, including donning of oxygen masks. However, recognition of the decompression may be a factor depending on the type and function of warning devices in the aircraft and their interpretation by aircrew. Hypoxia symptoms develop insidiously in a slow decompression, progressively degrading the crewmembers’ ability to recognize the decompression and respond accordingly, hence providing a more dangerous situation.

DCS is usually not a factor if an immediate descent is accomplished to below 10,000 ft. If a higher altitude level off is necessary or descent is delayed, DCS must be considered an additional risk, and appropriate and available DCS treatment must be considered when determining where to land.

References

Recommended Reading

Concepts
Cabin pressurization
Pressurization schedule
Rate of decompression

Vocabulary
Rapid decompression (RD)
5.2. Oxygen Systems

Lt Col Steven W. Dawson, USAF, BSC and Mr. George Miller

5.2.1. Introduction

Aircraft oxygen systems are fundamental components to most manned aircraft. Even in the most advanced aircraft, slight flaws in oxygen systems design can result in catastrophic mishaps. Careful consideration is necessary to cover the full envelope of today's advance aircraft capabilities, to include normal flight operations as well as emergencies. In addition to aircraft design considerations, aircrew members must have a thorough understanding of the oxygen systems necessary to operate safely at altitude.

In the early days of aviation, oxygen systems provided very limited and inefficient protection. For instance, the "pipe stem" oxygen system required the aviator to maintain the pipe stem between his teeth. This approach and other early systems used a continuous flow design, which wasted oxygen.

As aircraft became more complex, so did oxygen systems. Designers began to recognize that physiological requirements were based upon a wide variety of factors, including workload, altitude, acceleration, temperature, and physiological stresses. In addition, designers recognized the need for more efficient delivery systems.

Advancements in oxygen systems are based upon those physiological factors. These systems enable higher altitude flights, more efficient oxygen utilization, and more efficient storage. "Demand" oxygen systems, for instance, not only provided for protection against exposure to higher altitudes, but also more efficient oxygen utilization. Further development of "pressure-demand" systems led to automatic capabilities for altitude protection: pressure breathing for altitude (PBA) and acceleration protection pressure breathing for G (PBG). In terms of oxygen delivery, the ability to generate oxygen on board the aircraft provided for extended mission duration, reduced logistical burden, and reduced deployment footprint.

Even today, challenges exist in designing and updating aircraft oxygen systems to cover advanced capabilities while considering the myriad of logistical concerns associated with aircraft engineering constraints.

5.2.2. Standard Components of Oxygen Systems

Aircraft oxygen systems generally consist of four core components:

1. Oxygen storage system or onboard oxygen generating system (OBOGS) (also called a molecular sieve oxygen generating system, or MSOGS).
2. Tubing to flow oxygen from the supply source to a regulator(s).
3. Regulator for controlling oxygen flow and pressure delivered to the user and, in some cases, the oxygen concentration delivered.
4. Oxygen mask and connector to provide oxygen to the lungs.
5.2.3. Oxygen Storage Systems

With any oxygen system, safety guidelines are needed to protect the user from the increased fire hazards when using 100% oxygen. The use of high-pressure systems also increases the explosion and fire risks. Safety procedures for handling and storage of the various types of oxygen equipment and supplies are found in T.O. 15X-1-1.

5.2.3.1. Gaseous Oxygen. Aviator’s breathing gaseous oxygen is designated Type I per military specification MIL-PRF-27210, AVIATOR’S BREATHING, LIQUID AND GAS. Gaseous oxygen shall contain not less than 99.5% oxygen by volume. The remainder, except for moisture and minor constituents specified in MIL-PRF-27210, shall be argon and nitrogen. It must be odorless and free of toxic contaminants. Moisture shall not exceed 7 ppm of water vapor or a maximum dew point of -82°F. Aviator’s oxygen is different from other types of breathing oxygen because it contains very low amounts of water vapor. Although it tends to dehydrate the aircrew member, low moisture content in aviator’s oxygen is necessary. At altitude, excessive water vapor could freeze and restrict the oxygen breathing gas. Aircraft gaseous oxygen cylinders are made from shatterproof materials to minimize injury and damage if the cylinder is struck by a ballistic round.

5.2.3.1.1. Oxygen Gas – Low Pressure. Aviator’s breathing oxygen is stored in yellow, lightweight, non-shatterable cylinders (Figure 5.2.3.1.1-1). These cylinders may be integrated within the aircraft or can be portable, such as “walk-around” bottles. Aircraft integrated cylinders carry a maximum charge of 450 psi and the “walk-around” bottles are typically recharged on-aircraft to a pressure of about 300 psi. The use of low-pressure system reduces the risk of explosions, but limits the amount of oxygen available. Low pressure oxygen cylinders are primarily used during aircraft emergencies.

Figure 5.2.3.1.1-1. Low-Pressure Oxygen Bottles

"Walk-around" bottle cylinders’ duration depends on several factors, including initial charge pressure, altitude, and user breathing characteristics (see Table 4.1 of T.O. 15X-1-1). The limited volume mandates on-aircraft recharge or immediate descent. A system that drops below 50 psi must be filled within 2 hours to prevent water vapor entry and condensation; otherwise, it must be purged to eliminate moisture (per T.O. 15X-1-1).
5.2.3.1.2. Oxygen Gas – High Pressure. Some aircraft are equipped with high-pressure cylinders. Most fighters, bombers, and trainers are equipped with high-pressure cylinders used during aircraft emergencies, such as ejection, and are generally mounted on ejection seats. These cylinders are green and are normally filled to a pressure of 1,800 – 2,200 psi. The main advantage of the high-pressure cylinder is the large amount of oxygen stored in a small volume.

5.2.3.2. Liquid Oxygen (LOX). Aviator’s breathing oxygen in liquid form is designated Type II per military specification MIL-PRF-27210, AVIATOR’S BREATHING, LIQUID AND GAS. Aircraft liquid oxygen systems convert liquid oxygen to gaseous oxygen by allowing the surrounding atmosphere to passively warm the liquid oxygen as it passes through a heat exchanger. When LOX is converted to its gaseous state, it expands to about 860 times its original volume (1 L LOX = 860 L of oxygen gas). The expansion ratio of liquid to gaseous oxygen makes it ideal for aircraft use as long as there is a readily available means for LOX refilling. Once in gaseous form, the oxygen shall contain not less than 99.5% oxygen by volume. The remainder, except for moisture and minor constituents specified in MIL-PRF-27210, is argon and nitrogen. The oxygen is free from contaminants of known toxicity. Like Type I Oxygen, it must be odorless, and moisture does not exceed 7 ppm of water vapor or a maximum dew point of -82°F.

The heart of the liquid oxygen system is a double-walled vacuum insulated container called a LOX converter (Figure 5.2.3.2-1). Connections leading to the inner shell of the container are surrounded by a vacuum space to minimize heat leaks. In filling the system, the pressure buildup and vent valve (a two-position valve) is placed in the VENT position. This allows the flow of liquid into the container and vents container gas pressure to the atmosphere. The filler valve is connected to the liquid oxygen storage tank through an insulated, flexible hose. Pressure in the servicing tank forces liquid oxygen into the aircraft liquid oxygen converter.

![Liquid Oxygen Converter](image)

Figure 5.2.3.2-1. Liquid Oxygen Converter

5.2.3.3. Onboard Oxygen Generating System (OBOGS). Onboard oxygen generating systems (OBOGS) eliminate the need for a stored oxygen supply by generating oxygen from aircraft engine bleed air or environmental control system air.

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2 LOX molar density at 1 atm is 35.65 (mol/L) (Perry & Chilton, 1973). Application of the Ideal Gas Law using 1 atm and 70°F results in 1 liter of liquid LOX converting to 860.7 liters of gaseous oxygen. T.O. 15-1-1-1 states the conversion is 862:1.
Various types of OBOGS technologies have been considered, such as permeable membrane and chemical generation; however, the only current viable system uses pressure swing adsorption technology with a molecular sieve adsorbent. Molecular sieves are synthetically produced zeolites (naturally occurring aluminosilicate minerals), and are characterized by pores and internal cavities of extremely uniform dimensions. These crystalline materials have three-dimensional structures based on silicon oxide (SiO₂) and aluminum oxide (AlO₄) polyhedra. The polyhedra are linked by their corners to produce an open structure with internal cavities in which molecules can be adsorbed. These materials are engineered so that access to the internal cavities is through uniform and molecularly sized pores. The molecular sieve preferentially adsorbs and separates nitrogen from oxygen due to the nitrogen molecule’s slight polarity. Oxygen and argon are non-polar. Oxygen concentrates as nitrogen is adsorbed and removed from the gas phase. As long as sufficient air pressure is available, a continual supply of breathing oxygen can be produced. The oxygen concentration is monitored by an oxygen sensor to ensure it meets the minimum physiological requirements based on the cabin altitude. Most systems use a zirconia oxygen sensor. The gas produced is typically in the range of 40% - 93% oxygen (Miller, 1994; Miller et al., 1998) depending on operating conditions (i.e., cabin altitude, demand flow, inlet air pressure, etc.). Under ideal operating conditions, the maximum oxygen concentration possible is 95.1%. The remainder is mostly argon.

OBOGS is dependent on engine bleed air pressure, temperature, and quality. OBOGS inlet air pressure is the most critical parameter for OBOGS performance. Therefore, proper integration of the OBOGS and the engine bleed air system or aircraft environmental control system is important. Further, emergency oxygen systems are used more frequently on OBOGS-equipped aircraft because engine bleed air pressure reductions or stoppages can lead to OBOGS performance degradation. Hence, emergency oxygen systems must be properly sized and designed for potential frequent use.

Typically, OBOGS is composed of two or three beds or canisters of molecular sieve adsorbent, valving, a purge orifice, a controller, and an oxygen sensor/monitor. The adsorbent beds are alternately cycled through steps of adsorption and desorption. During adsorption, air at moderate pressure (30 – 40 psig) enters the adsorbent bed, whereupon nitrogen is preferentially adsorbed and enriched oxygen is recovered from the system’s product port. The adsorption step is followed by desorption, or venting, of the adsorbed nitrogen to ambient pressure. Ambient pressure varies with aircraft altitude, and this reduction in pressure at altitude is used to desorb the nitrogen from the molecular sieve. At ambient pressure, the molecular sieve desorbs nitrogen. Also, during the desorption step, a small portion of the product oxygen flows into the depressurized bed to purge residual nitrogen. This phase of the process prepares the molecular sieve canister for the next pressurization step. Cycles of adsorption and desorption are repeated every 5 to 15 seconds, resulting in a steady flow of enriched oxygen at the outlet of the OBOGS.

OBOGS is currently installed in several types of aircraft. A brief description of some of the aircraft and configurations is presented below.

**5.2.3.3.1. F-16 Fighting Falcon.** Although most F-16’s use LOX, some use OBOGS (Figure 5.2.3.3.1). The F-16 OBOGS is designed to run at the highest purity possible based on operating conditions, typically 93%. Oxygen purity is monitored by a zirconia oxygen sensor. The CRU-98, aircrew dilution regulator, dilutes

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the oxygen with cabin air prior to delivery to the pilot’s mask. The system has a regulated emergency oxygen system (REOS) with a miniaturized oxygen regulator located within the integrated terminal block (ITB). A molecular sieve filled plenum is installed between the OBOGS and the aircrew regulator. The plenum provides a passive source of oxygen during engine bleed air low pressure transients.

Figure 5.2.3.3-1. F-16 Oxygen Concentrator

5.2.3.3.2. B-1B Lancer. The B-1B OBOGS is composed of a concentrator assembly, a release valve, two purge valves, and four breathing regulators (Figure 5.2.3.3-2). The OBOGS is designed to run at the highest purity possible based on operating conditions. The breathing regulators are non-dilution, pressure-demand regulators, qualified to an altitude of 42,000 ft. For this system, automatic pressure breathing begins at 30,000 ft (cabin altitude), which increases to 15 – 19 mmHg at 42,000 ft. A backup oxygen system (BOS) is automatically activated if cabin altitude exceeds 27,000 (± 2,000) feet (T.O. 1B-1B-1-1). The BOS may also be manually activated. The OBOGS does not have an oxygen sensor, but instead uses pressure sensors to ensure proper operation. The B-1B has the first USAF production OBOGS.

Figure 5.2.3.3-2. B-1B Oxygen Concentrator

5.2.3.3.3. F-15E Strike Eagle. The F-15E OBOGS consists of an oxygen concentrator with an integral self-charging backup oxygen system, zirconia oxygen sensor/monitor, and two CRU-98 breathing regulators (Figure 5.2.3.3-3). Again, the OBOGS is designed to run at the highest purity possible based on operating conditions.
The CRU-98 regulator dilutes the oxygen to that required for the specific cabin altitude. The F-15E OBOGS was qualified to 50,000 ft and approved for use with the Combined Advanced Technology Enhanced G Ensemble (COMBAT EDGE). The F-15E OBOGS has a self-charging 94% backup oxygen system that only charges when the oxygen concentrator produces ≥ 43% oxygen. The system provides about 10 minutes of breathing gas for the two-man aircrew on the ground and a greater duration at altitude.

Figure 5.2.3.3-3. F-15E Oxygen Concentrator

5.2.3.3.4. F-22 Raptor. The F-22 OBOGS uses an oxygen concentrator that has three molecular sieve canisters loaded with molecular sieve adsorbent. A zirconia oxygen sensor monitors oxygen concentration of the product gas. The OBOGS controls the oxygen concentration of the product gas based on the cabin altitude. The control scheme uses variation of the molecular sieve canister pressurization and depressurization frequency to control the oxygen content of the OBOGS product gas. The F-22 uses a non-dilution breathing regulator. This system has a seat-mounted, manually activated regulated emergency oxygen system (REOS). If the cabin altitude reaches 25,000 ft, an automatic warning signal is issued, requiring the pilot to manually activate the REOS. The REOS is automatically activated during the ejection sequence.

5.2.4. Oxygen System Plumbing and Fittings

The plumbing of an oxygen system consists of valves, tubing, and fittings.

5.2.4.1. Valves. Valves regulate the flow, pressure, or direction of oxygen. Valves can be activated manually, electronically, or pneumatically. Check valves are installed to permit flow of oxygen in one direction only. Valves can prevent loss of the oxygen supply in the event a component develops a leak. Various styles of single, dual, and triple check valves are available. The direction of oxygen flow is indicated by an arrow that is molded or stamped on the casing.

Pressure-regulating valves are installed in aircraft and altitude chambers to maintain oxygen pressure at a prescribed level. Oxygen breathing regulators designed to operate with LOX-based systems typically operate at an inlet pressure of 70 psi compared to OBOGS breathing regulators, which typically operate at lower inlet pressure around 10 to 30 psi. On-off valves are installed to control the flow of oxygen.
5.2.4.2. **Tubing.** Tubing refers to the interconnecting piping between the components of the system. Distribution lines are different for high-pressure and low-pressure lines. High-pressure lines are typically stainless steel or an aluminum alloy. Low-pressure lines can be copper, stainless steel, or aluminum alloy.

Oxygen tubing and equipment should be designed to ensure that at least a 2-in. clearance exists between oxygen system components and control cables or other moving parts of the aircraft. To maintain this clearance, oxygen tubing will be bent or rerouted. It is desirable to maintain a 6-in. clearance between oxygen tubing and electrical wires and aircraft fluid lines (fuel, hydraulic fluid, etc.).

These separation requirements don’t apply where barriers such as frames, ribs, or permanent partitions exist between oxygen tubing and electrical wiring or where electrical wiring is leading into oxygen equipment.

In routing the tubing, the general policy is to keep the total length to a minimum. A shallow bend or dip should be placed in each tubing length to allow for expansion and contraction, vibration, and fittings.

5.2.4.3. **Fittings.** Types of fittings include connectors, nipples, elbows, and couplings necessary to connect the tubing to the components of the system. Fittings used on oxygen systems are usually aluminum alloy, stainless steel, or copper alloy. Straight thread, gasket, or O-ring seal fittings are not generally used because of the possibility of leakage. Cast brass fittings aren’t used because of their porosity. Fittings in aircraft and altitude chamber systems typically have flared end fittings. When pipe thread fittings are used, Teflon tape is applied to seal the fittings. The tape must not extend beyond the first thread to avoid the risk of tape entering the fitting. Teflon tape is not used on flare fittings.

5.2.5. **Regulators**

Regulators govern the flow and pressure of oxygen to the aircrew member. Oxygen regulators have advanced significantly over the years. Early oxygen regulators provided a continuous flow of oxygen. These types of regulators were not very efficient because oxygen was wasted. In addition, they were insufficient for some flight parameters. The next generation of regulators, diluter demand, offered the capability of oxygen dilution to improve the efficiency of oxygen use. Pressure-demand regulators added a pressure breathing capability, allowing aircrews to reach even greater altitudes.

5.2.5.1. **Continuous Flow Regulators.** As the name suggests, continuous flow regulators provide a continuous flow of oxygen to the oxygen mask. Continuous flow regulators are used on civilian transport type aircraft and on some military aircraft for decompression emergencies. These systems provide a get-me-down capability up to a maximum emergency altitude of 40,000 ft. Oxygen is normally supplied via storage containers in high- or low-pressure gaseous form. These systems may be stationary or portable and may be automatically or manually activated.

5.2.5.2. **Diluter-Demand Regulators.** These regulators supply oxygen upon demand (inhalation). Inhalation develops a slight negative pressure on the mask side of the regulator and allows oxygen to enter. The regulator dilutes oxygen with ambient air at altitudes up to 32,000 feet (MIL-PRF-83178C). The system delivers enough oxygen for altitudes up to 40,000 feet.
5.2.5.3. Pressure-Demand Regulators. With a pressure-demand system, 100% oxygen can be delivered at pressures higher than the ambient pressure, thereby increasing the partial pressure of oxygen in the lungs. Pressure breathing provides sufficient oxygen breathing pressures during emergencies at high cabin altitudes. Various types of pressure-demand regulators may be found in today’s aircraft. The Regulator provides enough oxygen and delivery pressure for altitudes up to 50,000 feet.

5.2.5.3.1. CRU-73/A Oxygen Regulator and CRU-92/A Oxygen Regulator. The CRU-73/A is a narrow, panel-mounted regulator often used in altitude chambers, as well as various fighter, cargo, and bomber aircraft (Figure 5.2.5.3-1). The inlet pressure of the CRU-73/A regulator is typically about 70 psi. The CRU-92/A is similar to the CRU-73/A but has a night vision light plate.

The following controls and indicators are located on the front panel of the regulator and are typical of most oxygen pressure-demand regulators (Figure 5.2.5.3.1-1):

![Figure 5.2.5.3.1-1. CRU-73/A Regulator](image-url)

**Gauges/Indicators:**

1. **Pressure Gauge:** The pressure gauge is found on the upper right of the panel and indicates inlet pressure to the regulator. The gauge displays the amount of oxygen pressure present in the system in pounds per square inch (psi).

2. **Flow Indicator:** The window area on the left side of the panel marked FLOW indicates the flow of gas through the regulator by a visible blinking action. The flow indicator blinks or shows white during inhalation. It does not indicate what type of gas is flowing through the regulator.

**Control Levers** – The regulator has three control levers:

1. The **SUPPLY** (green) control lever, located on the lower right corner, controls the supply of oxygen.

2. The **DILUTER** (white) control lever, located on the lower center of the panel, has two positions: 100% and NORMAL. In the NORMAL setting, 100% oxygen is mixed with ambient air to gradually increase the percentage of inspired oxygen. The regulator senses cabin altitude and...
increases the levels of oxygen concentration, ultimately achieving 100% oxygen at about 32,000 ft.

3. The **EMERGENCY PRESSURE** (red) control lever, located on the lower left of the panel, has three positions: EMERGENCY, NORMAL, and TEST MASK. In the EMERGENCY position, the regulator delivers a slight positive pressure (at altitudes where positive pressure is not automatically delivered). In the NORMAL position, the regulator operates by providing flow when demanded by the user. In the TEST MASK position, oxygen is delivered to the mask under increased pressure and may be used for checking the seal of the mask.

### 5.2.5.3.2. CRU-68/A Oxygen Regulator

The CRU-68/A oxygen regulator is a narrow, panel-mounted regulator and, though mostly obsolete, may still be found in USAF aircraft (Figure 5.2.5.3.2-1). It does not incorporate an "off" warning for the oxygen supply, which could result in aircrew members inadvertently flying without an oxygen source.

![Figure 5.2.5.2-1. CRU-68/A Regulator](image)

### 5.2.5.3.3. T-6 Regulator

The T-6 oxygen regulator (Figure 5.2.5.3.3-1) is installed on the right side console of each seat (the T-6A has two seats). Each regulator has a supply lever (green), a concentration lever (white), a pressure lever (red), a built-in test (BIT) button (push to test), a flow indicator (blinker), and a maximum concentration flow light. Each regulator panel controls OBOGS electrical power and oxygen flow for the respective cockpit. When the concentration lever is in the NORMAL position, the oxygen concentration in the breathing gas will range from 25% to 70% for altitudes from sea level to 15,000 feet MSL, and from 45% to 95% for altitudes from 15,000 feet MSL to 31,000 feet MSL. These percentages are designed to ensure adequate oxygen supplies to prevent hypoxia. When the concentration lever is set to MAX, OBOGS supplies the highest possible oxygen concentration. The maximum concentration light illuminates when the lever is in "MAX" position. The BIT button activates the Initiated OBOGS BIT (I-BIT) any time after engine start and the three minute warm-up. The I-BIT provides verification that the OBOGS sensor and “OBOGS FAIL” annunciator are operating properly.
5.2.5.3.4. CRU-93/A and CRU-98/A Oxygen Regulators. The CRU-93/A regulator is used in F-16 LOX-equipped aircraft (Figure 5.2.5.3.4-1), and the CRU-98/A regulator is used in F-15E and F-16 OBOGS-equipped aircraft (Figure 5.2.5.3.4-2). These regulators provide automatic positive pressure breathing (PPB) as a function of both altitude (PBA) and G (PBG). At acceleration loads above +4 G₀, the regulator delivers additional pressures to the mask, a mask tensioning bladder in the rear of the helmet, and the counter-pressure vest (if worn). With each G, an additional 12 mmHg of mask pressure is added to improve G-protection, to a maximum of 60 mmHg pressure at +9 G. The system receives a pressure input signal from a remotely located G-valve to provide the PBG.
5.2.5.3.5. F-22. The F-22 uses an integrated Breathing Regulator and Anti-G (BRAG) Valve that controls flow and pressure to the mask and pressure garments (Figure 5.2.5.3.5-1). The BRAG valve combines the functions of both breathing regulator and anti-G valve in one package. The BRAG valve receives breathing gas from the OBOGS and conditioned Environment Control System (ECS) air for the anti-G suit. The regulator portion of the BRAG valve (Figure 5.2.5.3-6) is mechanical, delivering non-diluted OBOGS product gas on demand. The valve regulates the supply and pressure of the breathing gas to the pilot for both acceleration and altitude. For acceleration protection, the BRAG valve requires G-suit inflation before breathing pressurization occurs to prevent loss of consciousness. During G maneuvers, the BRAG valve limits mask pressure to 60 mmHg. PBA is automatically provided by sensing cabin altitude through an internal aneroid port. For altitude, pressure breathing is initiated at about 39,000 ft cockpit altitude (T.O. 1F-22A-1) and reaches a maximum of 70 mmHg at 53,000 ft.

![F-22 BRAG Valve Panel](image)

Figure 5.2.5.3.5-1. F-22 BRAG Valve Panel

The following controls and indicators are located on the front panel of the regulator:

1. **OBOGS (green):** This controls OBOGS operation. Positioning the switch to ON provides electrical power to the OBOGS and opens the OBOGS inlet valves allowing ECS air to operate the OBOGS.

2. **Air Supply (red):** The air supply switch has two positions: NORMAL and BYPASS. Positioning the switch to NORMAL allows OBOGS breathing gas to be supplied from the BRAG valve to the oxygen mask with a constant safety pressure of about 1 inH₂O. In the original design, positioning the switch to BYPASS supplies ECS conditioned (pressure, temperature, and humidity), Chemical/Biological filtered (if filter is installed), regulated air (21% oxygen concentration) at a slightly higher safety pressure. The OBOGS and air supply switches are interlocked to prevent ON and BYPASS from being selected at the same time. Presently, the current system has the BYPASS switch deactivated to ensure air does not inadvertently enter the breathing gas.

3. **Mixture Switch (toggle):** Allows control of the OBOGS oxygen concentration. In AUTO, O₂ mixture is automatically controlled as a function of cockpit pressure up to a cabin altitude of 11,000 feet. Above 11,000 ft, the system automatically switches to MAX mode. At cockpit altitudes up to 11,000 ft, approximately 60% O₂ is provided to the pilot;
above 11,000 ft, the concentration is 90 – 94% (T.O. 1F-22A-1). In MAX, the OBOGS produces the maximum O₂ concentration possible at all altitudes.

4. **Test Button (press to test):** The TEST button activates pressure breathing and inflates the counter-pressure vest and G-suit. The amount of pressure is dependent upon how far the button is depressed. The maximum pressure (60 mmHg) is equivalent to pressure breathing at +9.0 G and 60 mmHg.

5. **Flow Indicator:** Standard, white indicates flow, black indicates no flow.

### 5.2.6. Oxygen Masks and Connectors

#### 5.2.6.1. Quick Donning Oxygen Masks

Quick donning oxygen masks are designed for quick donning with one hand. In the event of a loss of cabin pressure or exposure to smoke and fumes, aircrew members must have access to maximum concentration oxygen immediately.

- **5.2.6.1.1. MBU-10/P Quick-Don Mask.** The MBU-10/P Quick Donning Oxygen Mask is designed to deliver oxygen from a regulator and provide protection from smoke, carbon monoxide, and other incapacitating gases. These masks are used aboard C-130, E-6A, and P-3C aircraft. The MBU-10/P consists of a suspension assembly and an oxygen mask assembly. The hanging suspension holder is mounted in the aircraft to facilitate stowage.

- **5.2.6.1.2. 358-1506V Quick-Don Mask.** The 358-1506V is designed for quick donning with one hand (Figure 5.2.6.1.2-1). The vented anti-smoke goggles are worn with the quick-don assembly. It features one universal-size mask molded from silicone. The mask incorporates a manually operated vent valve to purge the goggles. The assembly provides adequate protection to 43,000 ft when used with a compatible pressure-demand regulator. The mask suspension assembly provides automatic switching from the headset microphone to the mask-mounted microphone when unfolded. The 359 series quick-don mask (Figure 5.2.6.1.2-2) is a variant of the 358, featuring a mask-mounted regulator.

![Figure 5.2.6.1.2-1. 358 Series Quick-Don Mask](image-url)
5.2.6.2. **Pressure-Demand Oxygen Masks.** Pressure-demand oxygen masks are qualified to hold positive pressure delivered from the regulator. The mask forms a seal around the face to get the maximum benefit from the regulator.

5.2.6.2.1. **MBU-5/P Mask.** The MBU-5/P mask is a two-piece mask (hard shell and facepiece) that provides even distribution of sealing force around the mouth and nose (Figure 5.2.6.2.1-1). It is available in four sizes: short narrow, regular narrow, regular wide, and long narrow. It incorporates a combination inhalation/exhalation valve, which requires 1 mmHg greater pressure than mask cavity pressure to exhale. MBU-5/P masks were phased out many years ago with the introduction of the MBU-12/P mask, but they still may be found in some units because they sometimes offer a better leak-proof seal.

5.2.6.2.2. **MBU-12/P Mask.** The MBU-12/P mask is designed to be worn over the face forming a seal on the cheeks, over the bridge of the nose, and under the chin (Figure 5.2.6.2.2-1). The mask provides minimal facial protection from projectiles and fire, is qualified for depths up to 16 ft under water, and permits utilization of the Valsalva maneuver to equalize pressure in the middle ear and sinuses during descent. The basic MBU-12/P subassembly is a lightweight, low-profile oxygen mask. The mask features an integrated face form and hard shell. The mask has a combination inhalation-exhalation valve and a flexible silicone hose. The hose length may be adjusted via the anti-stretch cord inside the hose. The typical mask assembly contains offset bayonets for attaching the mask to the helmet, a three-pin bayonet connector to attach the mask to an oxygen connector, the appropriate cables to connect the mask to the aircraft intercommunications system, and a microphone.
5.2.6.2.3. MBU-20/P Mask. The MBU-20/P mask was designed for use in PBG- and PBA-equipped high-performance fighter/attack aircraft (Figure 5.2.6.2.3-1). However, it is also used as a replacement for the MBU-12/P in non-PBG/PBA applications, for example cargo aircraft. The MBU-20/P oxygen mask is available in five sizes. The MBU-20/P was developed for PBG/PBA breathing schedules up to 60,000 ft and features an oxygen supply hose designed to reduce flow resistance. The lightweight, low-profile mask contour provides optimal mask/face sealing capabilities while minimizing visibility restrictions. It also features separate inhalation and exhalation valves that minimize breathing resistance and reduce aircrew fatigue. The oxygen supply hose incorporates a fitting for a helmet bladder supply hose (although some hoses may be ordered without this fitting). The inflating helmet bladder pulls the helmet back, therefore tightening the mask to maintain a leak proof seal. This helmet bladder supply hose and male quick-disconnect connector interfaces with the female connector attached to the helmet.

5.2.6.3. Oxygen Connectors. Oxygen connectors generally serve the purpose of connecting the aircraft oxygen supply hose from panel-mounted oxygen regulators to the breathing mask and, when required, to the chest counterpressure vest (CSU-17/P). The USAF no longer requires the use of a counterpressure vest on aircraft with a maximum ceiling of 50,000 feet and below.

5.2.6.3.1. CRU-60/P Oxygen Connector. The CRU-60/P is designed to ensure positive locking and prevent flailing during an ejection (Figure 5.2.6.3.1-1). The CRU-60/P is normally secured to a dovetail mounting plate on the pilot’s chest and attached to the parachute harness. It connects to a standard three-pin bayonet connector and incorporates an omni-directional elbow to ensure proper alignment of disconnect forces during an ejection. This fitting also has an anti-suffocation valve that induces increased breathing resistance if a disconnection at the inlet occurs.
5.2.6.3.2. CRU-94/P Oxygen Connector. An integral component of the COMBAT EDGE system, the CRU-94/P Integrated Terminal Block (ITB) provides PBG capability to tactical aircrew and provides an additional port for a counterpressure vest (Figure 5.2.6.3.2-1). The ITB distributes pressurized breathing gas from the aircraft-mounted regulator to the pilot’s oxygen mask and counterpressure vest (when worn). When the counterpressure vest hose is not connected, the fitting is equipped to relieve pressure within the ITB and vents at 32 – 39 mmHg. This configuration might be used on a ferry flight where G protection is not needed. In order to maintain the normal PBG schedule and ultimately achieve 60 mmHg, a port plug must be used when the vest is not connected. Normal COMBAT EDGE pressure schedule reaches 60 mmHg at +9 G, so when the port is not plugged, the pilot will receive less PBG. This may be significant, as studies show that PBG at 60 mmHg produced higher G protection than at lower pressures (Balldin et al., 2005). When the vest is worn, the vest port connects to the vest supply hose using a four-pin connector. Like the CRU-60/P, the CRU-94/P has a standard emergency oxygen inlet port. When the counterpressure vest is not used, the CRU-60/P may be used in place of the CRU-94/P.

5.2.6.3.3. CRU-120/P Oxygen Connector. This connector combines the features of the CRU-94/P with a CRU-79 regulator (the CRU-79 is a small profile, lightweight personnel mounted regulator used primarily by US Navy non-OBOGS systems) (Figure 5.2.6.3.3-1). It allows the COMBAT EDGE ensemble to interface with a regulated emergency oxygen system. This connector is used as part of an emergency oxygen backup system. It is currently installed on USAF OBOGS-equipped F-16s. The CRU-120/P has an integrated pressure demand oxygen regulator which extends emergency oxygen duration by accurately regulating oxygen delivered during emergency conditions.
5.2.6.3.4. CRU-122/P Oxygen Connector. Similar to the CRU-120/P, this connector combines the features of the CRU-94/P with a CRU-79 regulator (Figure 5.2.6.3.4-1). It allows the COMBAT EDGE ensemble to interface with a regulated emergency oxygen system. This connector is used as part of an emergency oxygen backup system. It is currently installed on USAF F-22 aircraft. The CRU-122/P extends emergency oxygen duration by accurately regulating oxygen delivered during emergency conditions. Whereas the CRU-120/P has an altitude ceiling of 50,000 ft, the CRU-122/P is rated to an altitude of 66,000 ft.

Figure 5.2.6.3.4-1. CRU-122/P

5.2.7. Emergency Oxygen Cylinder Assemblies

5.2.7.1. High-Pressure Oxygen. Emergency oxygen assemblies may be found mounted on ejection seats, inside parachutes, or inside a passenger oxygen kit (POK). These assemblies are used for emergency oxygen during descent from ejection or bailout, aircraft oxygen system failure, or cabin pressure failure. When aircraft fly at or above 25,000 ft, emergency oxygen must be available for all aircrew and passengers.

Emergency oxygen cylinder assemblies provide 10 min worth of oxygen, generally enough for a descent from 50,000 ft to 14,000 ft. The cylinder delivers an initial pressure to the oxygen mask of about 16 inH₂O and an initial flow rate of 10 – 12 standard liters/minute. The flow rate decreases as pressure decreases, resulting in a flow rate of about 1 standard liter/minute during the 10th minute.
Emergency oxygen assemblies consist of a high-pressure oxygen cylinder, valve assembly, valve-to-mask tube, connector assembly, and pressure gauge. There are different configurations with slight variances, as shown in Table 5.2.7.1-1.

<table>
<thead>
<tr>
<th>Emergency Oxygen Cylinder</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>H-2 Assembly</td>
<td>Is used in certain aircraft for passengers. The H-2 may be thigh-mounted or used in a carrying bag.</td>
</tr>
<tr>
<td>MD-1 Assembly</td>
<td>Is placed in all back-style parachutes. The hose and connector assembly lengths is 28 inches total. The exposed cable length is 11.56 inches.</td>
</tr>
<tr>
<td>REDAR-K46-01</td>
<td>Is used with the Advanced Concept Ejection Seat (ACES) II seat. The major differences to the other types of cylinders are hose length, housing, hose adapter, actuating mechanism, and clamps.</td>
</tr>
</tbody>
</table>

5.2.7.2. Low Pressure Oxygen. Portable oxygen assemblies are conveniently available inside cargo aircraft. The assemblies allow aircrew members to move about the aircraft to perform duties during unpressurized operations, loss of cabin pressure operations, or smoke and fumes. These assemblies utilize Type A-21 regulators with no dilution. The duration of the oxygen flow is variable, lasting 4 to 30 minutes, depending on activity level.

5.2.7.3. Emergency Passenger Oxygen System. The Emergency Passenger Oxygen System (EPOS) is a vacuum-sealed, self-contained protective breathing device typically used by aircrew members and/or passengers during emergencies (Figure 5.2.7.3-1). The hood provides heat and flame resistance to 1,832°F (1,000°C), 360° visibility, and is able to be donned in approximately 15 seconds. The EPOS provides aviator grade oxygen during aircraft decompression and when smoke and fumes are present. The system consists of five major components: hood, oxygen cylinder, carbon dioxide controller (lithium hydroxide scrubber), neck seal, and storage pouch.

The assembly functions as a modified continuous flow system with a reservoir bag. Donning and activation should take less than 15 seconds. From activation, the EPOS hood will begin to inflate. The sound of oxygen will be heard flowing into the hood for about 5 minutes, fully inflating the hood. Oxygen is supplied to the reservoir bag from a continuous supply source. When the EPOS is worn, the temperature inside the hood will increase and condensation will appear. During inflation, the user breathes from the reservoir bag through the inhalation flapper. If the oxygen has been entirely withdrawn from the reservoir bag, the user received ambient air through the exhalation flapper. During the exhalation cycle, the reservoir bag stores incoming oxygen for the next inspiratory cycle.

The duration of oxygen flow depends upon the user’s body weight and activity level, with a maximum duration rating of 60 minutes (at rest) and approximately 5 minutes under moderate to heavy workload. It can be used up to an altitude of 41,000 ft (AFI 11-2AEV3ADDENDA-A).
5.2.7.4. **Protective Breathing Equipment.** Protective Breathing Equipment (PBE) consists of a lightweight Emergency Escape Breathing Device (EEBD) or protective breathing equipment that is designed to provide respiratory and eye protection to the user (Figure 5.2.7.4-1). The system consists of four major components: a solid state oxygen supply source, a chemical scrubber for carbon dioxide and water vapor, a loose-fitting hood with a head harness and neck seal to enclose the head and provide the breathing environment, and a venturi “pumping” arrangement powered by the oxygen generator, which recirculates the breathing gas within the hood. The rated duration of this device is 15 minutes. It should be periodically inspected every 30 days. To determine serviceable condition, check the window on the side of the storage case for a blue or pink color. Blue indicates a serviceable condition while pink indicates an unserviceable condition and the equipment should be removed from the aircraft and replaced.
References

Recommended Readings

Internet Resources; See Appendix 8.

Concepts
Oxygen systems
Pressure demand regulator
Continuous flow regulators
Diluter demand
High-pressure oxygen
Low-pressure oxygen
Pressure demand regulators

Vocabulary
Pressure breathing for altitude (PBA)
Pressure breathing for G (PBG)
Gaseous oxygen
Oxygen gas: low pressure
Oxygen gas: high pressure
Liquid oxygen
Onboard oxygen generating system (OBOGS)
Molecular sieve oxygen generating systems (MSOGS)
Combined advanced technology enhanced G ensemble (COMBAT EDGE)
Regulated emergency oxygen system (REOS)
6. PERSONAL EQUIPMENT EFFECTS

Success of the human weapons system in the hostile environment of flight requires unique equipment and training. The physiological capacity of aircrew is amplified through the use of technology designed into aircrew flight equipment. T.O. 14-1-1, USAF Aircrew Life Support Equipment and Ensemble Configurations, provides listings of all USAF aircraft and the personal equipment qualified for use in each. USAF aircrew personal equipment is designed to provide protection and enhance performance of crewmembers performing USAF missions in various aircraft. Benefits achieved by use of this equipment typically come at a price. Just as wearing a coat in winter to be more comfortable restricts movement, the design and function of aircrew personal equipment may cause some discomfort or restriction in movement. However, wearing an oxygen mask, anti-G suit, night vision goggles, or other aircrew personal equipment provides the ability to function effectively in environments where performance would be significantly or completely negated without such equipment.

6.1. Night Vision Devices (NVDs) and Helmet Mounted Devices (HMDs)

6.1.1. Night Vision Devices

Maj Eric G. Chase, USAF, BSC; Mark White, MS; and Lt Col Steve Dawson, USAF, BSC

6.1.1.1. Introduction. The battlefields of prior generations saw action most often during periods of daylight when visual acuity was best. Indeed, vision was a limiting factor concerning the most effective time to launch an assault. Only the most important covert missions would be carried out at night, when the element of surprise was best facilitated by darkness and suboptimal enemy performance could be anticipated in response. However, while enemy performance was degraded as a result of poor vision, the offensive force could expect a comparable reduction in its own performance due to this same limitation. Today, technology has advanced to such a degree that human visual limitations have been significantly mitigated. Nighttime battlefield operations are now carried out regularly and with much greater success.

Night vision devices (NVDs) have been instrumental in overcoming the human limitations to nighttime operations. NVDs encompass all technologies used to enhance night vision, and they have been used for decades, dating back to World War II. These older generation devices used an infrared (IR) illuminator, which emitted a beam of IR light. This IR light, invisible to the unaided human eye, would reflect off objects in the viewing scene and bounce back to the device. The emitted IR beam can be thought of as a flashlight; albeit a flashlight the naked human eye cannot detect. The device was sensitive to the reflected IR light, and it would “reconstruct” the scene using wavelengths of light the human eye could perceive. This technology was revolutionary but produced a poor image that was often distorted. The devices had many limitations, including a short life span. In addition, enemy nations quickly found ways to use their own night vision devices to observe the IR beam emitted from these NVDs. Now the technology effectively made the user a target. As such, this technology quickly became obsolete. Almost immediately, work began on developing a passive technique, one
which increased nighttime visual acuity using only available light. The image intensifier (called I² technology) filled this need.

Still in use today, I² technology continues to represent the crux of night vision goggle (NVG) function. Unlike the older technology detailed above, image intensifiers are completely passive in operation and are based on the principle of light amplification. The devices greatly amplify the ambient light produced by the moon and stars for the human eye to more easily see a poorly illuminated scene. These devices do not require a projected IR light, thereby taking away the enemy’s ability to observe IR illuminators. Because they amplify ambient light, it is of paramount importance to note that image intensifiers (and, thus, NVGs) cannot function in complete darkness. There must be some minimum level of light for the image intensifier to amplify. Amplification of this light is what enhances scene detail and subsequent visual acuity. While NVGs intensify ambient light levels within the visible spectrum (400 – 700 nm), it is important to note that they also amplify light in the near infrared range. Thus, unfiltered NVGs intensify light in the range of 570 – 930 nm.

6.1.1.2. Image Intensification. Stating that “NVGs amplify light” is a phrase subject to subtle but important semantics. Strictly speaking from the standpoint of physics, photons (and, therefore, light) cannot be amplified. As such, light must be converted to a signal that can be amplified (Figure 6.1.1.2-1). Electrons, which are readily amplified, are the perfect intermediaries. In the simplest of terms, ambient photons entering the image intensifiers of NVGs are converted to electrons. These electrons are then amplified in number up to 1,000-fold. After this amplification step, the resulting electrons are converted back to photons once again, such that the NVG user now perceives an enhanced scene with much greater illumination and visual acuity.

The complete story of the evolution of image intensification and the physics by which it is governed is astonishingly complex. However, a fundamental description of all the components and their contributions to the enhanced NVG image is possible with a basic understanding of chemistry and physics. This figure represents an exploded view of the image intensifier tube and its components.

![Image Intensification Diagram](image.png)

Figure 6.1.1.2-1. Image Intensification

The image intensifier tube, with a diameter less than that of a quarter, is housed within the NVG and consists of three core components: the photocathode, microchannel plate, and phosphor screen. For our purposes, the photocathode can be thought of as
little more than a glass disk coated on one side with a substance called gallium arsenide (GaAs). GaAs coats the side of the photocathode adjacent to the microchannel plate.

When photons enter the objective lens of the NVG, they first encounter the photocathode. This light interacts with the GaAs coating on the opposite end of the photocathode. Inherent properties of the GaAs are such that incident photons cause oxidation, e.g., an electron is liberated from the GaAs (these liberated electrons are depicted as arrows in the above diagram). This step represents the conversion of a light signal to an electrical signal.

As shown in Figure 6.1.1.2-2, the electrons liberated by the GaAs at the photocathode travel to the next component of the image intensifier tube, the microchannel plate (MCP). This is arguably the most fascinating component of the entire system. The MCP is a wafer-thin disk of glass that contains 10-11 million microchannels within it. Electrons liberated at the photocathode find their way into the MCP via one of the microchannels. Once inside, electrons strike the inner walls of the microchannels. To ensure collisions along their walls, all microchannels within the MCP are oriented at an 8° angle to the horizontal. The distal end of the MCP maintains a positive charge to ensure that a current can be maintained that keeps “pushing” the negatively charged electrons downstream through the microchannels. Each time an electron strikes the inner wall of a microchannel, an additional electron is liberated from that microchannel. This results in an exponential increase in the number of electrons travelling through the MCP. The first collision an electron makes yields 2 electrons, those two yield 4, then 8, 16, and so on down the length of the microchannel. Ultimately, for every one electron that enters one of the millions of microchannels, up to 1,000 are produced. This is the amplification phase of image intensification.

As the electrons exit the MCP, they next encounter the phosphor screen. This component of the image intensifier is responsible for converting the electrical signal back to a light signal. The amplified electrons strike the phosphor screen, causing it to release photons with a characteristic green color. The light emerging from the phosphor screen is exactly proportional to the number and location of the electrons striking it at each point. An intensified image results and is viewed through the eyepiece lens.

Of note are two key features regarding the phosphor screen. First, an inherent property of optics dictates that an image be inverted when light passes through a lens (as illustrated above at the photocathode). Thus, prior to viewing the light emitted by the phosphor screen, a fiber optic “twist” reverts the image exactly 180°. Next, it should be appreciated that the characteristic green color of NVGs is not happenstance. The human eye is most sensitive to green light, so NVG engineers ensured selection of a phosphor that emits light of that color.

6.1.1.3. Performance Factors. This commentary will not concentrate on the specifics or mechanisms of focus and adjustment. However, it must be made clear that properly focused NVGs will not result in the user developing a headache. If
NVGs are not focused properly, it is not uncommon for the user’s eyes to accommodate for a short period of time. Over an extended period, however, the eye muscles will become fatigued and unable to maintain focus. This results in a gradual loss of visual acuity and often causes severe eyestrain, headache, or both.

It is critically important to understand that even under the most ideal conditions, visual acuity will be degraded while using NVGs. Military specifications for currently used aviation NVGs can consistently yield visual acuities of only 20/26.5. In addition, the NVG user is essentially viewing a scene from two tiny television screens. As such, true depth perception is entirely ablated. This makes distance estimation and subsequent closure rates particularly difficult to determine. Moreover, contrast, a vital component to scene detail, is compromised in that the viewed images will all be varying shades of green. For these reasons, aircrew must understand that a preflight assessment of the NVG image is of paramount importance.

Assessing the performance of NVGs is not unlike assessing unaided visual acuity. In fact, it requires a similar approach. During an eye exam, the Snellen chart provides the examiner with an objective means by which to measure visual acuity. The size of characters a subject can see on the chart from a known distance dictates the degree of his/her visual acuity. NVG performance is assessed using a chart especially designed for NVGs using similar theory. Of important note is that this assessment cannot be accomplished while in the aircraft, so it is incumbent on the aircrew to perform the procedures prior to flight.

Unfortunately, NVGs provide an incredible amount of information even if they are not adjusted properly. This fact can lull aircrew into the false belief that it is unnecessary to adjust the goggles prior to each flight. The only means by which to determine if the settings are correctly positioned is to assess performance using a resolution chart (e.g., eye lane or Hoffman box/ANV-20/20). Figure 6.1.1.3-1 represents data from a study that underscores the importance of employing a means by which visual acuity can be assessed.

![Figure 6.1.1.3-1. NVG Adjustment Training](image)

Visual acuity is depicted on the Y-axis. Bear in mind, the better visual acuity, the shorter the column will be. Three different conditions were examined. First, a group of experienced NVG users were asked to adjust their goggles via whatever methods they normally used (baseline). This group of users had received no prior training, nor were they assisted with NVG focusing procedures. The NVGs were then assessed for visual...
acuity performance by having the subjects read from a resolution chart specifically designed for NVG use. As shown, without training or any kind of chart by which to measure visual acuity, aircrew were routinely “focusing” their goggles to acuities ranging from 20/35 to 20/100, with the average visual acuity being 20/50. Next, the subjects were given a resolution chart (but still no training) to aid them in their focusing procedures (chart only). Visual acuity improved significantly, with a range from 20/35 to 20/70, and an average of 20/45. Lastly, the group was given the resolution chart along with instruction on the proper use of the same (i.e., chart and training). Visual acuity continued to improve, with a much smaller range of 20/30 to 20/50, and an average of 20/35.

The study above illustrates that aircrew can routinely focus their NVGs to suboptimal settings and go on to fly sorties with a dramatically decreased visual acuity. Training significantly enhances the visual acuity aircrew can expect. Additionally, these data show that training ensures all aircrew approach the same performance capability, which can be very important when different aircrew gather information in the same environment (e.g., observers estimate distance to trees). The importance to mission effectiveness and safety should be obvious.

Figure 6.1.1.3-2 was developed to make a point relative to NVG experience levels. It was constructed utilizing the same data as those in Figure 6.1.1.3-1 above, but the data here were separated into the different sites where they were collected.

![Figure 6.1.1.3-2. NVG Adjustment Training at Three Sites](image)

At first glance, it would appear that aircrew at Site B were the most experienced since they had the best baseline visual acuity findings when compared to the other two sites. However, the aircrew at Site A were actually the most experienced NVG users. It appears that the most experienced aircrew are more tolerant of poor vision. The crucial point demonstrated by these data is that aircrew must guard against complacency as NVG experience builds. Further, as instructors, we must ensure that we train aircrew regarding correct focusing and adjusting of NVGs.

6.1.1.4. Field of View. Another component of training for NVGs requires the recognition of field of view (FOV) limitation. With the normal, unaided binocular vision, a standard FOV is approximately 120° vertically and 200° horizontally.
The aircrew outfitted with NVGs might expect to have a FOV of $30^\circ$ to $40^\circ$, depending upon the NVG in use. Eye relief and eye positioning also change FOV when using NVGs. Eye relief is defined as the distance from the back surface of the NVG ocular lens to the front surface of the eye; if the distance is greater than 20 mm, then the FOV decreases significantly. Factors affecting eye relief and ultimately FOV performance are user-head anthropometrics, use of additional life support equipment, protective gear, and the wearing of corrective lenses. Cognitively, a decrease in FOV may negatively affect human performance through an increase in task management because of the constant scanning required by aircrew to perform for them to create a complete FOV of the surrounding environment. Simply enlarging the FOV on NVGs is not feasible because as FOV increases the resolution of the image decreases; thus, the aviator’s role in the task-dependent duties of flight are not yet fully without limitations (Miller and Tredici, 1992).

6.1.1.5. **Depth Perception.** Even assuming focus and adjustment procedures have been carried out flawlessly and under the most ideal conditions, depth perception presents one of the most significant challenges to aircrew flying on NVGs. The ability to perceive depth via NVGs is severely degraded. In fact, one could say that this ability is effectively absent. True depth perception involves the visual acquisition of an object in three-dimensional space (rather than the “TV screens” of NVGs). Each unaided eye has a somewhat dissimilar retinal image of an object as a result of a slightly different angle of view from each eye. The retinal images fuse, allowing the brain to compute something of a triangulation, thereby providing depth perception at distances up to approximately 600 ft.

The above process is termed binocular vision, as it requires the use of both eyes, and binocular cues refer to the mechanisms by which aircrew assess depth and distance. Binocular cues are assessed subconsciously and allow for very precise distance estimation, but only at relatively short distances. While this binocular ability is lacking for NVG users, it is still possible to estimate depth using monocular cues. Monocular cues are fundamentally different from binocular cues. While not the “true” depth perception that binocular vision provides, monocular depth perception is learned. Over time, we become familiar with the sizes and shapes of objects in our environment. We are then able to take these learned lessons and apply them to our activities. For example, there are plenty of people who live with very poor depth perception, but the cues they learn throughout life allow them to successfully drive vehicles. Among other things, they have learned that when driving along a road, nearer objects appear to be moving faster than distant objects. The same such information is used to help us determine how far away objects are when they are beyond the useful range of binocular depth perception. More pertinent to the nighttime environment, these cues are drawn upon regularly for the NVG user. Nevertheless, it is important that the NVG user understands that although monocular depth and distance information is still available and usable, it is never as good with NVGs as with unaided vision during daytime.

It is a simple fact that reduced visual acuity, field of view, and depth perception while using NVGs can easily result in illusions and misperceptions that could induce spatial disorientation. Thorough training is a crucial means by which to negate these human factor challenges inherent to NVGs. As detailed above, proper focus and adjustment training is essential to mitigate the degraded visual acuity intrinsic to NVG use. Preparing for the lack of good depth perception and field of view cues can be
accomplished via training and proper planning. Only through such training can aircrew safely obtain information that may keep them from overestimating their visual acuity, field of view, and depth perception capabilities.

6.1.1.6. Human Factors: Operational Issues. In the NVG Academic Instructor Course offered at Randolph Air Force Base, we often say that NVG training courses should not teach students what NVGs can see. What NVGs see is obvious and warrants no prolonged discussion. Rather, courses should center on what the NVGs cannot see or do. These aspects must be learned, as many are counterintuitive. Without such training, it is easy to fall victim to a variety of human factors traps.

6.1.1.7. Fatigue. As noted in the 1992 U.S. Air Force published report, Night Vision Manual for the Flight Surgeon (Miller and Tredici, 1992), fatigue is one of the most serious aeromedical concerns to aircrew in the operational environment. Fatigue caused by sleep disruption due to combat operations can be the most common disruptor. Activities that predispose aircrew to fatigue consist of consistent work shift changes, cumulative sleep loss, and circadian rhythm interruption. As a compounding variable, fatigue increases spatial disorientation logarithmically. Refer to the Human Performance, Fatigue, and Fatigue Countermeasures sections of this handbook for further details concerning fatigue, sleep, and circadian rhythms.

NVGs are a compounding variable to fatigue when considering the risk assessment of night operations with the possibilities of disrupted circadian rhythms, work shift reversals, and cumulative sleep debt. NVGs provide operators with a false sense of security by enabling them to function in an environment that would not be possible without the use of NVGs. Unless preventive measures are taken to identify symptoms of fatigue, such as easy distractibility, inattention to detail, slowed reaction time, poor judgment, irritability, and coordination problems, aircrew members might assume they are capable of peak performance at the least opportunistic time, thus creating a situation for a mishap. Fatigue induced by sleep disruption can be counteracted by a simple and strictly enforced sleep policy; refer to the Human Performance, Fatigue, and Fatigue Countermeasures section of this handbook for further details concerning fatigue, sleep, and circadian rhythms.

Additional types of fatigue an aircrew might incur with the use of NVGs are physical, psychological, and visual fatigue. The physical aspect is in reference to the additional weight of the NVGs (≈ 0.7 kg) to the neck, while psychological fatigue refers to the arduous, task-dependent aspects of operations at night and the associated stresses, all of which can be prevented through exercise (e.g., specific isometric neck strengthening exercises) and acquisition of job-duty knowledge, skills, and ability, respectively. Visual fatigue, also known as asthenopia, can be reduced by wearing of properly prescribed eyeglasses, training, thorough NVG maintenance habits of adjustment, and controlling interfering light sources (Miller and Tredici, 1992).

6.1.1.8. Life Support Integration. The integration of NVGs to the operational aspect of aircrew life support equipment is another factor that may give rise to problems. Aircrew may incur problems with their chemical defense gear, helmet-mounted devices, crash and ejection procedures, and any additional eyewear. Problems might include, but are not limited to, reduction in field of view, increased risk of neck injury due to helmet weight, increased risk of injury due to the additional helmet weight and its effect on crash tolerance under G-forces, and diminished eye protection.
because of improper compatibility with dust/wind goggles. The life support issues of concern are preventable through proper education of aircrew on all individual equipment and their functional integration into the operational environment (Miller and Tredici, 1992).

6.1.1.9. Cockpit Compatibility. The incompatibility of NVGs to the operational environment of the cockpit was a potentially debilitating issue. Any small amount of light that gleams from within or into the cockpit can render NVG technology dysfunctional, i.e., too bright. One resolution to the issue of cockpit lighting was to develop a blue-green filter for NVGs and design cockpit lighting based upon the blue-green light spectra. The benefits are three-fold: first, the short wavelength of the blue-green lights will make it easier for the aircrew to see instrumentation; second, the Purkinje shift is less marked due to the ease of sight for the blue-green light when compared to a red light; and third, blue-green lights are less detectable by the enemy using NVGs. Military specification lighting is available when considering the use of NVG and is required under specific circumstances, for internal and external cockpit lighting applications, when in operational use (Miller and Tredici, 1992).

6.1.1.10. Environment Compatibility. As stated earlier, modern NVG intensifier tubes are capable of harvesting light in the near IR wavelengths. Trees are very readily seen when viewed at night under NVGs. The chlorophyll present in leaves emits light very strongly in the near IR region, exactly where NVGs are sensitive. During certain times of the year, aircrew may become accustomed to seeing a given field of trees clearly as a result of the chlorophyll-containing leaves. However, trees without leaves (and therefore without chlorophyll) can be very difficult to see. In fact, at times when trees have shed their leaves, the entire tree can seem to have disappeared. The normal human reaction to such a situation would be to get closer such that more of the scene can be resolved. As a rule of thumb, whenever aircrew attempt to make something appear at night the way it appears during the day, they are too close! Entire fields of leafless trees provide a particularly dangerous setting for low-flying aircrew. Situations can arise in which a low-flying aircraft collides with a single leafless tree that was just a bit taller than all of its neighbors. In fact, this is such a well-known and dangerous trap that these tall, hard-to-see leafless trees have been given names in the NVG community. They are very commonly called snags or, more foreboding, widow-makers.

One of the most dangerous situations that can be experienced during NVG operations is flight into undetected meteorological conditions. Generally, aircrew flying fixed wing aircraft will not be susceptible to inadvertent weather entry due to the flight altitude at which most missions are flown and the distances at which weather systems are avoided. However, certain missions (e.g., low levels) and flight conditions in certain areas (e.g., fog formation in coastal and mountainous areas) may make fixed wing aircrew more susceptible to this hazard. As discussed above, NVGs can harvest portions of the near infrared energy spectrum. These wavelengths tend to pass through light moisture more easily than visible wavelengths. As a result, what may be a visible area of light fog or rain during daytime may be virtually invisible to aircrew viewing the same scene at night with NVGs. Added to the dilemma is the fact that the thinner areas of moisture may mask areas that have significant moisture content. This can result in a gradual loss of scene detail as the weather system is penetrated, ending in a situation where there is virtually no visual information.
Perhaps even less intuitive than the above scenario is the potential danger posed by new obstruction lighting. In 2008, a Flight Safety Flash was issued by the Canadian Air Force’s Directorate of Flight Safety that identified some red obstruction lighting systems that were clearly visible to the naked eye but not visible to NVGs. Surely, the idea that the naked eye can see something that NVGs cannot is a profoundly counterintuitive notion. Therein lies the human factors trap: it is frighteningly easy to become overreliant on the visual information NVGs provide (or, in this case, do not provide), even when that information is grossly incorrect.

New obstruction lighting systems employ light-emitting diodes (LEDs) instead of traditional incandescent sources. Aviation Red light ranges from about 610 to 700 nm, and military grade aviation NVGs with the corresponding filter sets are mostly sensitive to energy ranging from 665 to 930 nm. Because LEDs have a relatively narrow emission band and do not emit infrared energy like incandescent lights, it is possible for them to meet FAA requirements for Aviation Red but be below the range in which aviation NVGs are most sensitive.

On 6 March 2009, the FAA published a Safety Alert for Operators (SAFO) addressing this issue (FAA, 2009). They recommended action that precisely parallels the NVG Academic Instructor Course philosophy of training in terms of educating flyers regarding what NVGs cannot do:

“Pilots, directors of operations, chief pilots, training program managers, and training centers either using or providing training for NVGs should advise pilots of the limitations outlined in this SAFO and ensure such information is incorporated into the pilot NVG training program.”

As aerospace and operational physiologists, it is our duty to understand NVG limitations and pass them on to our students to enhance mission effectiveness and safety. Again, what the NVGs can do is patently obvious, but the NVG limitations detailed in this commentary must be taught.

References

Recommended Readings
AETC NVG Course Instructor Guide

Vocabulary
Night vision devices (NVDs)
6.1.2. Helmet Mounted Displays

James Barnaba, M.S.

6.1.2.1. Background. A Head Mounted Display, or more commonly a Helmet Mounted Display (HMD), is a device used in many modern aircraft, especially combat aircraft, that projects information, in a manner similar to a Head-Up Display (HUD), onto the aircrew’s visor or a near to eye reticle. An HMD is typically coupled with a head position tracking system and can provide the aircrew with the ability to cue sensors and weapons; while receiving target, weapon and flight data; at high off-boresight angles while maintaining visual contact with the target or targets. When coupled with a night vision device, the HMD can provide a similar capability at night. The overall goal of HMDs is to effectively interface the aviator/aircrew with the aircraft and associated systems, which allows them to acquire and maintain situational awareness. An HMD differs from an Helmet Mounted Sight (HMS) in that the provided symbols are visually coupled to the users line of sight (also called a visually coupled system) and provide relevant symbols for piloting, navigation and weapon cueing; as opposed to a HMS where the symbols may just be an array of light emitting diodes that cue the pilot in a particular direction.

6.1.2.2. Components. In general, helmet mounted displays consist of the following key components:

6.1.2.2.1. Image Source. The image source is a display device where the projected display is produced. This source can be a miniature cathode ray tube either driven in stroke mode, where symbols only are “drawn” by manipulating the cathode to excite phosphor on the tube’s screen in lines or curves, or raster mode where imagery is scanned onto the phosphor screen in a double pass like a cathode ray tube (CRT) television set. This source can also be a flat panel display like a non-emissive liquid crystal display (LCD) or an emissive organic light emitting diode (OLED). If a non-emissive display (one that does not emit light on its own) is used, such as an LCD, then a backlight is needed.

6.1.2.2.2. Drive Electronics. The drive electronics associated with the image source are required to power the display and provide its graphics interface to the aviation electronics (avionics). Included in the drive electronics is the power supply, display driver (usually a semiconductor integrated circuit), and the software interface or graphics processor.

The choice of image source, drive electronics and avionics interfaces determines the capability of the HMD. Whether it projects aircraft state information (air speed, altitude, bank angle, angle of attack), weapons information (mode selected, weapons active, stores status), situational awareness (location of wingmen, friends, foes, threats, other aircrew’s line of sight), navigation data (waypoints, heading, flight path), and/or sensor data (radar, forward looking infrared (FLIR) images, targeting pod images, night vision) all depends on the sophistication of the image source, the drive electronics and the electronics interface with the aircraft systems.

6.1.2.2.3. Collimating Optics. The collimating optics serve to direct the display image from the image source to the eye in a collimated (parallel, non-dispersing...
light rays) fashion. This is typically accomplished with a series of glass and plastic prisms contained within a plastic optics housing that direct the light from the location of the image source to ultimately direct it into the pupil of the eye. For a visor display the light is bounced off of the inner surface of the visor into the eye. For a reticle display it is directed using a glass or plastic reticle mounted in front of the eye. A unique type of reticle is called a waveguide. In a waveguide, the image source is adjacent to the reticle and basically bounces the light image through the glass optic until it is redirected by saw-tooth reflectors inside the glass reticle into the eye.

Another unique type of mounted display technology is called a virtual retinal display. In this case, an eye safe light source basically draws the desired image right onto the retina of the eye. There is no image plane; such as a CRT faceplate or an LCD or LED array involved; just the computer driven light source and the retina.

Depending on the sophistication of the display source, drive electronics and collimating optics, HMDs can be monochrome (one color, green or other), more than one color or a full palate of colors and imagery. By doubling the optics, a dual eye HMD can be created, or hypothetically by creating one large display that is viewed by both eyes.

6.1.2.2.4. Head Tracker. The head tracker provides the capability to tell the computer where the pilot or aircrew are looking. Typically using the boresight (centerline) of the aircraft as a zero reference point, the head tracker's job is to notice changes in pitch, roll, yaw and fore/aft location of the pilot's head. Many different techniques are employed to accomplish this. Magnetic fields can be transmitted into the cockpit from a cockpit mounted source and then received by a magnetic receiver in the helmet. A cockpit mounted camera can view the helmet, or light sources built into the helmet, and observe how they move. A helmet mounted camera can view reference points in the cockpit and observe how they move. A miniature inertial sensor can be placed on the helmet and can sense pitch, roll and yaw (but not fore/aft). Sound waves have even been used. The accuracy at which the head must be tracked depends on how the HMD will be used; and with what weapons or tactics; and the choice of tracking methods can vary based on this accuracy requirement and the cockpit/crew station in which the HMD is to be employed. Whatever the method employed, there must be accompanying electronics that compute the head position so that the weapon or sensor can be cued to that location, the pilot's line of sight. With a head tracked system, the line of sight is typically the center of the HMD field of view.

If an eye tracking system were also added to the helmet mounted display, the aircraft's weapons or sensors could be directed more accurately to where the pilot's eye is pointing as opposed the where their head (nose) is pointing.

6.1.2.2.5. Head Mounted Platform. The display needs to be fixed to the pilot or aircrew's head in a consistent and repeatable manner. This is typically accomplished by "mounting" the display and tracking system onto the helmet, but is also sometimes accomplished by building a helmet that contains the display and tracking system (an integrated design). Strap based systems can also be employed for specific applications (training for example); thus the term head mounted display vs helmet mounted display could apply. The flight helmet has many jobs in addition to being the platform for the HMD. It has to protect the aircrew from impacts and penetration of sharp objects; it has to protect from bullets, and rocks and debris in a rotary wing application, with some amount of ballistic protection; it has to provide communication.
through earcups or ear plugs and a microphone; it has to attenuate aircraft noise and provide intelligible speech through its communication equipment; it may have to provide an oxygen mask; it has to provide eye protection (sunlight, laser threats); it has to protect the pilot if they are ejecting from the aircraft or bailing out; it may need to be low observable or it may need to be highly observable for rescue aid; it likely needs to support night vision goggles (maybe binoculars as well); needs to be compatible with chemical/biological protection hoods; needs to be compatible with aircrew flight equipment; and many more requirements.

A key challenge of HMDs is head supported weight. So much technology and protection requirements are built into the HMD, the helmet and the night vision technologies, but it all has to be supported by the pilot’s neck. Fatiguing the pilot is to be avoided and maintaining an acceptable mass property (both weight and center of gravity) is essential for a limiting this fatigue and for safe ejection in an ejection seat equipped aircraft.

6.1.2.2.6. Helmet to Vehicle Interface. Until such time as a complete wireless solution is possible, the HMD must be interfaced with the aircraft system. This is accomplished with a cable or cables containing a bundle of wires (or fiber optics), each carrying a necessary signal or data component to and from the HMD. For safe aircraft egress this cable must have disconnects built in that permit routine and emergency separation of the cable itself.

6.1.2.2.7. Other equipment. To turn an HMD into a helmet mounted cueing system, there are many other equipment items necessary. Aircrew controls must be provided (brightness, contrast, symbology de-clutter), items that sense the position of the aircrew’s seat may be needed, a means of boresighting the HMD to the aircraft may be needed, a characterization of the magnetic fields in the cockpit to enable magnetic tracking may be needed, support equipment (testers, fitting tools), technical manuals, training curriculum; the list goes on.

6.1.2.3. History. The modern HMD is not a new concept. Its invention has been attributed to Gordon Nash, a British researcher who explored alternative methods of providing additional information to the aviator in the 1950s. Early US operational systems included the Helmet Sight System (HSS), an HMS used in the Army’s AH-1 Cobra attack helicopter in the 1970s. The U.S. Navy’s Visual Target Acquisition System (VTAS), developed in the 1960s, was the first fully operational visually coupled sighting system. The Army’s Integrated Helmet and Display Sighting System (IHADSS) was the first integrated HMD (fielded since 1985 on the Apache). The U.S. Air Force has been testing and developing HMS/HMD technology since the 1970s in what is now called the Air Force Research Laboratory. As examples, the Visually Coupled System (VCS) was tried on the F-4 in the 1970s, the Integrated Night Imaging Goggle Head Tracking System (I-NIGHTS) produced three prototypes in the 1980s that were basically too heavy to be flown safely. In the 1990s the Vista Sabre program finally led to the day Visually Coupled Acquisition and Targeting System (VCATS) and the night Strike Helmet 21 which were flown in test and laid the groundwork for the USAF’s first operational HMD; the Joint Helmet-Mounted Cueing System (JHMCS).
6.1.2.3.1. Joint Helmet Mounted Cueing System. Realizing the USNs Crusader project (F/A-18, F-14 and AV-8B), the VCATS program (F-15) and Lockheed Martin’s “Look and Shoot” demo (F-16) were all headed down similar paths, the DoD decided that a joint (USN/USAF) solution was the fiscally appropriate way to proceed. Based on threat concerns from a Russian MiG 21 seen armed with an AA-11 Archer off-boresight capable weapon and a HMS to cue it, the USAF/USN Joint Helmet Mounted Cueing System (JHMCS) program office was tasked, and awarded a contract to Boeing and an Associate Contractor Agreement (ACA) with Lockheed, to develop a High Off-Boresight System (HOBS) (Figure 6.1.2.3.1-1).

![Figure 6.1.2.3.1-1. The JHMCS High Off-Boresight Targeting System](image)

This HOBS would be required to integrate an HMD with the developing AIM-9X high off-boresight capable Sidewinder missile and integrate into the F/A-18 Hornet, the F-15 Eagle and, via the ACA with Lockheed, the F-16 Falcon and the F/A-22 Raptor.

An early decision in the JHMCS/HOBS program was that the A-10, AV-8B and F-14 avionics architectures were too old to support the communication speeds necessary to keep the symbol presentations accurate and timely with pilot head movement (a.k.a. system “accuracy” and system “latency”). Amongst the HMD bidders, Boeing chose a Kaiser Electronics and Elbit Systems of Israel team, this team formed a Limited Liability Corporation called Vision Systems International (VSI LLC). VSI leveraged the Elbit Display and Sight Helmet (DASH), with Gen 1 HMS and Gen 3 HMD being tested and flown in their home country Israel, and the VCATS, in which Kaiser was a prime developer, to come up with the JHMCS design.

The JHMCS utilizes a high brightness miniature cathode ray tube as an image source, leverages existing head-up display symbology sets in the aircraft types, provides an Electronic Unit (EU) and Cockpit Unit (CU) as drive electronics, improves and integrates Elbit’s magnetic head tracking system, has a removable Display Unit (DU) that situates on a modified HGU-55/P aviators flight helmet, contains all the needed optics/electronics and a video camera to record the mission, offers several visor choices that each reflect the symbols at the needed brightness, has a Helmet to
Vehicle Interface (HVI), and many other items of aircraft interface and support equipment (Figure 6.1.2.3.1-2).

![Figure 6.1.2.3.1-2. JHMCS Components](image)

The CRT, optics, and visor provide a 20 degree circular monochrome display to the pilot’s right eye. The EU is the brains of the system providing a Graphics Processor and Display Driver (GP/DD) capability (two GP/DDs for specific F/A-18 and F-15 two seat models), interfaces with aircraft operational flight programs (and the weapons and sensors) and aircraft power supplies, a line of sight calculation capability and a low voltage power supply. The CU converts the low voltage power to high voltage to drive the CRT and also provides a point of aircraft interface for the HVI. The tracker includes a cockpit mounted Magnetic Transmitter Unit (MTU) and a helmet mounted Magnetic Receiver Unit (MRU) where magnetic field fluctuations are captured and calculated by the EU to equate to head movement in any axis. The DU has an array of male pins that mate into a helmet mounted Universal Connector (UC) (Figure 6.1.2.3.1-3). This concept, developed under VCATS, allows for future modules to be developed and adapted to the JHMCS system through this UC. The HVI runs from the UC to the helmet nape as a ribbon cable and then as a cable bundle down to the CU, attached to the pilot at two locations on the parachute harness. The HVI has three connectors; a Helmet Release Connector (HRC) that separates if the helmet is lost during an ejection windblast, a Quick Disconnect Connector (QDC) that is exercised in every cockpit ingress and egress and also provides a positive electrical signal if it is properly snapped into the harness, and the In-line Release Connector (IRC) that is a back-up to the QDC for safety/redundancy in case of ejection. Other key peripherals include a seat position sensor to inform the magnetic tracker of the location of seat metal, a go/no-go test set for ready room use, and a cockpit mapping device for magnetic field characterization of each JHMCS cockpit.
The JHMCS was originally intended as an air-to-air dogfighting system. It provides full symbology, equivalent to HUD aircraft state data, but most importantly it provides weapon seeker location symbols as well as “leader lines” (arrows) to cue the pilot to threat locations or other points of interest (e.g. threats, waypoints or designated targets). It even provides two Light Emitting Diode (LED) targeting crosses high on the visor (one high-left, one high-right) to give an HMS capability in a dogfight beyond the pilots ability to move their head and neck (the pilot selects “uplook” and then moves their eyes from the cross in the middle of the JHMCS 20 degree circle up to the appropriate, EU selected, uplook reticle a.k.a. “pupper” (pop-up pipper)). Since its fielding in 1999 and beyond, it has found many uses as an air to ground tool as well as providing a means to share point of interest data and cues between fore and aft crew stations and between fighters. Design challenges were many and included achieving high accuracy for the location of weapon line-of-sight symbols to pilot line-of-sight, low latency to symbol movement with pilot head movement, low total head supported weight (4 pounds met a compromise at 4.3 pounds) and good center of gravity balanced against module/visor retention for facial protection in an ejection event (600 Knots Equivalent Airspeed (KEAS) met a compromise at 450 KEAS).

6.1.2.3.2. Night Vision Cueing and Display Module. The first JHMCS module to be developed was the night vision cueing and display (NVCD). Originally part of the Insight Technologies Panoramic Night Vision Goggle (AN/AVS-10) family of systems, but ultimately a VSI design in production, the NVCD brought the JHMCS capability to night pilotage. The VSI Quadeye™ NVCD offers either a wide field of view (WFOV) night vision image (~100° w x 40° h) (Figure 6.1.2.3.2-1) or a standard field of view (~40° circular). NVCD components include the Night Vision Device (NVD), the Night Display Adaptor (NDA) and a stepped-in visor.
The NVD utilizes image intensifier tubes that are smaller and lighter than the AN/AVS-9 NVG (Figure 6.1.2.3.2-2), utilizes a prism in one tube to both inject JHMCS symbology using an Organic Light Emitting Diode (OLED) image source from below, and records that night image and symbols with a camera from above. The NDA mounts to the JHMCS UC, supports the NVD and contains an MRU, display electronics and a back-up battery for night vision. The stepped-in visor provides facial protection from the NVD and windblast in case of ejection. The USN fielded the Standard FOV NVCD, but the USAF has not chosen to field NVCD.

A significant challenge with combining HMD technologies with night vision technologies is the combined weight of both and the fact that the majority of this weight is hung on the front of the helmet, thus causing a forward center of gravity and equivalent moment of inertia on the neck in an ejection. For the NVCD, this head supported weight is as much as 5.5 pounds for the WFOV version. This, in terms of safety risk in ejection, is not considered ejectable weight as the acceleration of the seat out of the aircraft is sufficient energy to dislodge the NVD from the NDA if the pilot does not remove it themselves before ejecting.

6.1.2.3.3. Helmet Mounted Integrated Targeting. In 2010 a contract was competed to integrate an HMD into the A-10C and Air National Guard F-16s (Block 30s). Gentex Visionix’s “Scorpion” HMD was chosen. The image source for the Scorpion Helmet Mounted Integrated Targeting (HMIT) is a Liquid Crystal on Silicon
(LCOS) color flat panel display projected to the eye through a novel 32° diagonal FOV waveguide optic (Figure 6.1.2.3.3-1).

A key characteristic of the HMIT, compared to the JHMCS/NVCD, is affordability; the HMIT allows a single display solution to be converted in-flight to a night solution by changing visors and adding existing AN/AVS-9 Night Vision Goggles (or the 100° wide AN/AVS-10 Panoramic Night Vision Goggles). It was designed to cue the A-10 targeting pod in wide field of view mode for both ground and air targets (thus having a less stringent accuracy requirement than the JHMCS/NVCD), and display ground stabilized symbols accurately enough to allow correlation with target. Like the JHMCS/NVCD, it provides symbology cues to lead the pilot to tagged data link symbols or aircraft sensor points of interest. Unique to the HMIT is the ability to display this symbology in 24 bit color and the ability to display targeting pod raster video in a “picture-in-picture” type presentation. The HMIT also requires significantly less real estate for its aircraft mounted avionics, offering a cockpit mounted electronics unit to accomplish what the avionics bay or behind the seat mounted JHMCS EU provides.

The HMIT waveguide, or lightguide, optical element replaces traditional angled combiners with a thin optical element (Figure 6.1.2.3.3-2). A very attractive aspect of this technology is what is termed its eye box. For traditional optics there is a specific “sweet spot” to the light bundle entering the pupil that requires relatively accurate helmet fitting to optimize this and limited movement of the HMD in flight is required to maintain it. If the helmet and optics shift relative to the pupil then vignetting occurs; portions of the display are no longer visible to the pilot. The HMIT display design provides a larger eyebox than these traditional optics approaches.
Like the JHMCS/NVCD, HMIT utilizes a magnetic tracker, although significantly lighter and smaller, with a transmitter that mounts on the aircraft canopy and a receiver in the HMD. To reduce the burden of characterizing the cockpit magnetic field effects (required for using a magnetic based tracking system), the HMIT (now under the ownership of Thales Visionix) is proposing a Hybrid Optical Inertial Tracker (HObIT).

Also like JHMCS, the HMIT incorporates an HVI with similar connector requirements, although the HVI is simplified in that high voltage current to the display is not needed to drive the LCOS source; as compared to the JHMCS CRT where high voltage is required.

Regarding ejection safety, the HMIT also has to be safe in this environment. The structural integrity of the HMD and the visors need to resist breakage to at least 450 KEAS and it must be safe regarding neck loading. A downside to the fact that HMIT was a selection between off the shelf designs is that funding was not applied toward weight reduction. The HMIT is a ½ pound heavier than JHMCS.

6.1.2.4. New HMDs. The value of HMDs is proven and the trend continues to find new applications as well as upgrading older aircraft with this technology.

6.1.2.4.1. F-35 Helmet Mounted Display System. The F-35 Lightning II (Joint Strike Fighter) is being fielded to ultimately replace the A-10, AV-8B, F-14, F-15, F-16, and other aircraft used in multiple foreign partner nations. It utilizes three versions; conventional take-off and landing, vertical take-off and landing, and carrier version. Awarded by Lockheed Martin Tactical Aircraft Systems (LMTAS) to Vision Systems International (VSI), the F-35’s Helmet Mounted Display System (HMDS) has gone through several major design evolutions. Originally designed as a 50° wide, dual eye presented display (Figure 6.1.2.4.1-1), the now dubbed Generation (Gen) I HMDS had a line of bifurcation in the visor where the curved projection surfaces for the left and right projectors met (the right projector shown in Figure 6.1.2.4.1-1 bounced off the left curved visor side and the left projector off the right). This bifurcation line originally thought to not be a visual issue to the pilot was deemed by LMTAS test pilots to be so.
The F-35 HMDS does all that JHMCS does, but in a larger display area. The VSI HMDS designs utilize Active Matrix Liquid Crystal Display (AMLCD) display sources backlit with a high brightness green LED arrays. This Gen I design also incorporated a 16mm image intensifier based night camera; not for the purpose of night vision but, as well as providing a post flight debriefing capability, would be compared with images captured by an aircraft glareshield mounted camera to provide head tracker updates. By adding a second magnetic receiver in the HMD and utilizing this image capture scheme, unlike the JHMCS, the HMDS would not require every aircraft to be mapped magnetically at any regular interval nor would it require the pilot to boresight their HMD to the aircraft centerline. In the JHMCS aircraft, this boresighting is accomplished every flight by placing the JHMCS targeting cross visually over an equivalent cross presented on the HUD and marking with a switch click. The F-35 however, for the first time in fifty years of fighter design, does not have a HUD. The HMDS operates in two modes depending on pilot head position. When looking in the forward direction, the HMDS presents a virtual HUD (vHUD) with critical flight and weapons symbology. As the pilot’s head moves, the HMD vHUD is aircraft stabilized above the glareshield and appears as a fixed display in that location in the cockpit. When the viewing angle changes towards off-boresight, the vHUD symbology is replaced by off-axis symbology that includes both aircraft state and weapons symbology. In the off-axis mode, symbology becomes helmet stabilized and shifts with pilot head movement.

Also absolutely unique to the F-35 approach is the night vision capability. Unlike HMDs to date, the F-35 HMDS would receive its night vision capability through externally mounted aircraft cameras called the Distributed Aperture System (DAS). These six wide field of view (WFOV) cameras (stare up, down, left, right, fore and aft), when fused together provide a NVG equivalent wavelength view of the night in a full sphere around the aircraft. The HMDS avionics computes the pilot’s line of sight and choses which one, two, or three DAS camera outputs to fuse together to give this WFOV night vision presentation to the HMDS. In the F-35, the pilot can actually look down and see what is below the floor of the aircraft using their HMDS.

A compromise was reached and the Gen II version would be a reduced display size (40° wide and 30° high), but would have a symmetrical curve to its visor. It also received some improvements to the head tracking subsystem. Another major change was that VSI introduced a digital night vision capability into the helmet and glareshield. The Intevac Silicon Imaging Engine (ISIE) utilizes an Electronic Bombardment Active Pixel Sensor (EBAPS) technology and provides a digital sensor and read-out integrated circuit (ROIC) in an NVG equivalent wavelength. Vision Systems International’s (VSI, now Rockwell Collins/Elbit Systems of America Vision Systems (RCESAVS)) Gen II HMDS for the F-35 is shown in Figure 6.1.2.4.1-2.

Figure 6.1.2.4.1-2. VSI’s Gen II F-35 HMDS
The Gen II HMDS has been flown in the F-35 for most of the development and initial training and operational phase of the F-35 program. The Gen II HMDS has limited night vision camera capability and exhibits some display issues during dark night operations. In addition, it was discovered that the canopy bow structure in the aircraft partially blocks the helmet mounted night vision camera, hindering the ability of the pilot to view some objects in the external scene. For some specific operational reasons, it was later decided that the HMDS night vision camera should be an option to the DAS as a night vision imagery source to the HMDS display to the pilot. VSI, with the Rockwell Collins headquarters leading, was tasked to modify the Gen II to accomplish this. This involved upgrading the EBAPS to a higher resolution version and upgrading the relay optics. This version, dubbed the Gen III is likely the final design (Figure 6.1.2.4.1-3).

These VSI HMDSs have many similar design challenges to the JHMCS and HMIT HMDs. They too have to be safe for ejection but, in the case of the F-35, they need to be retained and protect the face and eyes to 550 KEAS (as opposed to 450 KEAS for JHMCS and HMIT). This calls for a significantly stronger HMD structure. Due to the F-35 canopy hinging in the front vs the back, it must be explosively fractured in an ejection. This places additional impact, penetration and noise protection requirements on the HMDs.

The helmet includes high performance optics and displays, a high resolution night vision camera, a day camera for debrief purposes and a hybrid tracker system incorporating magnetic, optical and inertial tracking technologies to track the position of the helmet in all operational environments. Two Active Matrix Liquid Crystal Display (AMLCD) channels present symbology and video to the pilot with a 40° w x 30° h FOV with the two channels nearly 100% overlapped – meaning that both eyes are viewing essentially the same information. The displays are required to meet the focus and alignment requirements necessary for the system to accurately display most symbology to both eyes. This type of display where a single sensor presents data to two eyes is called biocular as opposed to the AN/AVS-9 which is binocular (two image intensifier sensors viewed by both eyes).

The hybrid tracker combines three technologies together to maintain a high level of accuracy for pilotage and tactical operations. The magnetic tracker is used at all times and provides the basic accuracy required for operation. The optical tracker provides the highest accuracy when the pilot’s line-of-sight is in the forward direction and also functions as a means to maintain and improve alignment between the HMDS
to the aircraft. The inertial tracker primarily operates in high vibration and other
environments when head position is moving rapidly due to external forces.

Near Infrared (IR) night vision is provided by two high-resolution ISIE-11 Night
Vision Cameras (NVC). The Helmet Night Vision Camera (HCAM) is located on the
helmet and the Fixed Night Vision Camera (FCAM) is mounted atop the glareshield.
Together these two cameras provide Near IR night vision imagery for the pilots. The
function of the FCAM is to effectively remove the canopy bow that blocks a portion of
the HCAM field of view when the pilot is looking in the forward direction. The FCAM is
fixed in the forward viewing area and whenever there is an overlap between the HCAM
and FCAM imagery, the imagery in the overlap area is provided to the pilot by the
FCAM. This has the effect of removing a majority of the area of the HCAM that is
obscured by the canopy bow.

As mentioned above, the F-35 HMDS not only needs to provide head-up
symbology and night vision capability, but it also needs to perform traditional helmet
functions such as pilot protection. The F-35 aircraft is required to provide safe escape
for aircrew weighing as little as 103 pounds and as much as 245 pounds. The F-35
HMDS affects neck loads during ejection and therefore minimizing the head supported
weight of the HMDS is essential. The final head supported weight for the Gen III
HMDS can exceed 5.0 pounds, which can contribute to neck load exceedances for
lighter weight aircrew. Therefore, a weight reduction program will be executed in order
to provide a fully functioning helmet that can meet the safe escape requirements for the
entire F-35 anthropometric range.

The F-35 Gen III HMDS architecture shown in Figure 6.1.2.4.1-4 consists of both
helmet and aircraft mounted hardware. The aircraft mounted hardware consists of the
Display Management Computer/Helmet (DMCH), the Helmet Tracker Unit, and the
Fixed Camera that includes optical tracker components, a high resolution night vision
camera, and a boresight reticle unit (BRU) for alignment checks and adjustments.

Figure 6.1.2.4.1-4. F-35 Gen III HMDS Architecture

6.1.2.4.2. JHMCS II. Vision Systems Internation (VSI, now Rockwell
Collins/Elbit Systems of America Vision Systems (RCESAVS) with Rockwell Collins
having taken ownership of the Kaiser Electronics portion of the VSI LLC) has
progressed the JHMCS from a CRT to a flat panel display by leveraging a new design they had been marketing worldwide called Targo.

The simplified low-voltage HVI is part of this new design as well (Figure 6.1.2.4.2-1). There is a night vision capability to the JHMCS II that involves mounting a color eye piece to one tube of the AN/AVS-9 NVG. New head-tracking technology is easier to maintain while requiring less support equipment than previous trackers. Electronics enhancements enable all processing to be done within the helmet, eliminating most aircraft-mounted equipment, which also contributes to a system cost savings.

The JHMCS II is planned to be implemented on AC-130 aircraft in use by U.S. Air Force Special Operations Command, in the pilot’s position only.

![Figure 6.1.2.4.2-1. JHMCS II](image)

6.1.2.4.3. 3-D Audio. Head tracking technology enables a non-visual type of helmet mounted display, 3-dimensional audio. Using sound to help the pilot localize the position of threats, wingmen, or aircraft issues and state information, 3-D audio can drive the pilot to look in a very small section (4 degrees) of their sphere of coverage in a matter of a second. Coupled with a visual HMD, this technology can drastically reduce target acquisition times. This technology has been in development for decades and is technologically ready, but no large scale application has yet occurred.

6.1.2.5. Future Helmet Mounted Displays. The next generation HMD will likely be completely digital, from the sensor to the display. Image Intensifier tubes may be replaced with sensor on a chip/ROIC technologies with on helmet computing/fusion with aircraft mounted sensors to provide a high-definition synthetic day/night vision display. This digital presentation could be shared via data-link day or night. The future HMD could also be capable of monitoring the pilot’s state (O₂ saturation, heart rate, stress, alertness). Tracking technologies could include eye-tracking. Display technologies could even take the waveguide concept into a whole visor display concept.

6.1.2.6. HMD Advantages. In operational settings where displayed information is critical on a second-to-second basis, such as an aircraft flying close to the ground, the operational environment changes so rapidly that even the brief time it takes
the pilot to glance down at one or more displays to obtain aircraft flight status information may severely degrade their situational awareness.

6.1.2.6.1. Field of Regard. While HUDs also provide this advantage, they are limited to the forward quadrant and not as useful when the pilot also has to scan around the aircraft for threats. The key advantage of HMDs is the ability to look at a target anywhere around the aircraft and select it for attack. Using an HMD in air-to-air or air-to-ground engagements, the pilot can quickly select the enemy target, launch the weapon, and then quickly turn to the next target. The concept of multiple kills per pass is possible with an HMD integrated.

6.1.2.6.2. Situational Awareness. Helmet mounted displays are said to contribute strongly to pilot situational awareness (SA). Situational awareness has been defined as the total information available, used to create an accurate picture of a battle theater, including spatial position and orientation of the aircraft, the surrounding areas and any aircraft relevant information (Rash et al., 2009). Another operational definition is offered by Geiselman and Osgood: “A pilot’s (or aircrew’s) continuous perception of self and aircraft in relation to the dynamic of flights, threats, and mission, and the capability to forecast, then execute tasks based on the perception” (Geiselm ann and Osgood, 1994).

6.1.2.7. Potential Problems. Their design and implementation, however, are not without problems and limitations. Virtually every HMD, concept or fielded system, suffers from one or more deficiencies, such as high head-supported weight, center of mass (CM) off-sets, inadequate exit pupil, limited field of view (FOV), low brightness, low contrast, limited resolution, fitting problems, and low user acceptance. Of the potential problems with HMDs, none are more troublesome than those associated with the interfacing of the system with the human user. The wide variation in head and facial anthropometry makes this a formidable task, requiring HMD designs rich in flexibility and user adjustments.

An HMD designer must develop a system that is capable of satisfying a large number of widely different and often conflicting requirements in a single system. Such design goals may include, but are not limited to, the following (McLean, 1997):

- Maximum impact protection
- Maximum acoustical protection
- Maximum speech intelligibility
- Minimum head-supported weight
- Minimum bulk
- Minimum CM offset
- Optimum head aiming/tracking accuracy
- Maximum comfort and user acceptance
- Maximum freedom of movement
- Wide FOV
- Minimum obstructions in visual field
- Full color imagery
- Maximum resolution
- High brightness and contrast
- No induced sensory illusions
- Hazard free
- Maximum crashworthiness
- 24 hour, all-weather operation
- Minimum training requirements
- Low maintenance
- Low design cost and minimum schedule
6.1.2.7.1. **Display Configuration.** One challenge with HMDs is the configuration of data presented in such a way to maintain SA. Since configurable displays promote information saliency, well-mapped information for comprehension of task goals, and ability to see trends in systems states, all of which represent the three levels of SA as proposed by Endsley (1995), the use of configurable displays in integration tasks should, in turn, provide for SA development. The theory of situational awareness provides a framework for what must take place for SA to be established and developed (i.e., information should be highly salient and related to operator goals), but SA theory does not provide a specific means by which this can be achieved (Jenkins, 2008).

Depending on the amount of data presented, the way in which it is presented, the state of the aircraft and other distractions, the process of cognitively digesting incoming data to produce and update an accurate SA model taxes the pilot and breaks down when the capacity to process the information exceeds their resources. In other words, “In the complex and dynamic aviation environment, information overload, task complexity, and multiple tasks can quickly exceed the aircrew’s limited attention capacity. The resulting lack of SA can result in poor decisions, leading to human error” (Endsley, 1995).

6.1.2.7.2. **Mass Properties.** Beyond the display configurations, the issue of neck strain and injury is another consideration when discussing HMDs. Loading weight to the head in the form of an HMD may have significant effects on the musculoskeletal system of the head and neck complex. The head’s center of gravity (cg) is located at the top of the clivus, a position that corresponds to a location that can be measured as 46.6% to the vertex of the head and 53.6% to the chin-neck intersect for a line drawn connecting the vertex of the head to the chin-neck intersect (Clauser, 1965). In a static load condition (neutral), the effort required of the extensor muscles is increased as the cg is shifted forward or the weight is increased in the HMD. It can be hypothesized that this increase of neck extensor muscle activity is necessary to generate an increased extensor moment to counteract the increased flexor moment of the added weight. During a flexing activity (e.g., during flight or while scanning), the horizontal distance of the center of mass of the head is increased from its axis of rotation. In doing this, the flexor torque, against which the extensor muscles of the neck must work to maintain the head in a static posture, increases. This explains the increase of neck extensor electromyography (EMG) due to a flexed head position.

One engineering practice that has been used to reduce this loading is the use of counterbalance weights on the helmet. The idea behind adding a counterbalance to a frontal load is to cancel out the increased flexor moment of the frontal load. By doing this, the amount of neck muscle force required to hold the head in a static position should decrease. What has been found through EMG analysis with counterbalance devices is, on the whole, mean muscle activity increased by a greater degree due to a counterbalanced load than a frontal load (Knight, 2004). One possible reason for the increased muscle activity could be head and neck stability. In the study, using a 10-muscle, 3-joint model of the head and neck, it was found that the head and neck were inherently unstable, especially around the neighborhood of an upright posture, and suggested that maintenance of head stability requires considerable co-contraction of antagonists. Although adding a load to the rear of the head to some extent counteracts the load at the front of the head, in terms of balancing moments, it may have an effect of increasing head instability.
Operationally, the strain on the neck while scanning (e.g., door scanner in a MH-60 helicopter) a counterbalanced head load could be particularly counterproductive due to the added load at the rear of the head; with the head flexed there is a net extensor moment generated by head load, which must be counteracted by a flexor muscle force. Current systems and materials have been designed to reduce or eliminate the need for counterbalancing weights, but the concern for head stability while wearing HMD is still an operational reality for crewmembers.

The unique challenges of designing HMD systems for a tactical fighter, as opposed to the rotary wing user, includes the potential for high g loads which serves as a multiplier to any head supported weight in maneuvering and escape. This head supported weight serves as a lever arm against neck muscles and vertebrae due to seat acceleration and parachute opening shock. For these reasons, the USAF has adopted criteria based upon ejection forces of the Advanced Concept Ejection Seat II (ACES II) used in its front-line fighters that, due to the involvement of AFRL researcher Dr. Theodore Knox, has been termed the “Knox-Box”. This two axis cg plot and limit is based on an assumed max head supported weight of 5.0 pounds (believed to be the limit of pilot acceptability in this environment) and provides acceptability limits for deviation of mass properties from the occipital condyles (the pivot of the head about the C1 cervical vertebra). HMDs used in USAF ACES II equipped fighters are measured against this criterion in order to assess the injury risks they carry (Albery and Kaleps, 1997).


6.1.2.8.1. Fixation. Not unlike HUDs, HMDs are also prone to fixation issues where the pilot becomes too fixed on looking at the HMD and fails to notice events in their field of vision or outside the aircraft. This well-known HUD fixation issue was clearly seen with early JHMCS test pilots in Boeing simulators. McCann et al. have questioned if pilots can actually simultaneously process this HUD/HMD and real world data (McCann et al., 1993).

6.1.2.8.2. Conformal Symbols. If symbols are not conformal (overlaid in a one-to-one relationship to match shapes and features in the real world) they are perceived as different form the outside world and will drive the pilot to shift their attention back and forth between the symbols and the outside view instead of merging them as one. This can add to the aforementioned HMD fixation issue (Wickens and Long, 1994).

6.1.2.8.3. Clutter. HMD symbols can obscure critical objects in the outside scene or too many symbols and/or too many in motion simultaneously can overwhelm distract or again add to the issue of HMD fixation. Pilots have to be able to know when it is time to use typically available de-clutter modes in order to reduce this issue before it becomes problematic.

6.1.2.8.4. Latency. Latency in the system must be minimized. The pilot expects and needs the head driven symbology to track with their head movement. When the targeting cross is moved and placed on a target (moving or fixed target), the pilot needs to have the weapon’s seeker symbol to track to that targeting cross. If it doesn’t, the pilot will try to look past the target to get the weapon or sensor symbol to
catch up. This may cause the seeker symbol to overshoot and then the pilot will try to undershoot to pull it back. This pilot induced oscillation effect is unacceptable and can be prevented with low latency avionics.

6.1.2.8.5. Accuracy. Accuracy is key in providing confidence in employing weapons. If the weapon seeker symbol does not accurately overlay the target/targeting symbol, then the pilot lacks confidence in the system and may delay releasing the weapon based on this lack of confidence. High accuracy can prevent fratricide and loss of non-military targets in vicinity of military targets. As a challenge, however, there are many contributors to system accuracy that must be addressed by HOBS designers. These contributions include but are not limited to:

- The accuracy of the symbology presentation to the eye by the HMD computer (probably the smallest contributor to accuracy error)
- The accuracy that the pilot can place the targeting symbol over the target in a dynamic environment
- The accuracy that the pilot can boresight or zero the HMD to the aircraft centerline
- Compensation in symbology location for the distortion in vision caused by the aircraft canopy; and more specifically where in the canopy the pilot is looking, as the distortion varies based upon this
- The accuracy of the head tracking system and how well it corrects for and adapts to environmental conditions that affect it; such as magnetic field distortions (magnetic), sunlight (optical), drift (inertial), noise (audio)
- The accuracy of the weapon seeker or sensor
- The accuracy by which the weapon or sensor is mounted to the aircraft
- The amount that this mounting of weapon or sensor moves in flight (wings lift and twist)

6.1.2.8.6. Display Quality. For a good quality display, symbols must be free of bumps, gaps, dots or other discontinuities. They must be consistent in brightness throughout the symbol and across the full field of view of the display. They need to retain their shape and size as they move dynamically. They must be presented with adequate contrast against the background. They should not flicker or jitter or present other similar "noise."

6.1.2.9. Summary. Helmet mounted displays are becoming more common and more obtainable as off-the-shelf solutions for USAF aircraft. Their inherent capability to take much that keeps the pilot looking down into the cockpit and present it head-out is very attractive and can contribute to increased situational awareness and weapon employment capability. There are challenges to the acquirers, however, in determining the adequacy and the safety of using these devices in the
proposed environment and in assuring that the design and integration of the HMD is accomplished in a thorough and appropriate manner. There is so much head mountable technology available that can be of significant value to the pilot in executing their mission, but this has to be balanced against the head supported weight and the other aspects of what a helmet’s purpose is in the cockpit. The appropriate balance must be achieved between inserting technology that is available as-is and spending the time and money needed to refine the technology for the application (to include weight reduction, accuracy increases, latency reduction, design of symbology and methods of presentation to the pilot). While adding significant capability to, and thus survivability of the platform and its occupants; without taking these physiological issues into account, unnecessary safety risk will be added for these aircrew.

References

Concepts
Helmet mounted display (HMD)
Head up display (HUD)

Vocabulary
Joint helmet mounted cueing system (JHMCS)
Night vision device (NVD)
Night vision cueing and display (NVCD)
Helmet mounted integrated targeting (HMIT)
6.2. Anti-G Suits/Ensembles

William Albery, Ph.D.

The major objective of the anti-G suit (AGS) is to help protect against the detrimental effects of $+G_z$ exposure, especially $G$-induced loss of consciousness or GLOC. As AGSs are developed, the trend is toward integrated aircrew ensembles (IAEs) that include the AGS as part of a zipped garment that doubles as a flight suit. IAEs are being developed that also provide water immersion protection; thermal considerations; and even chemical, biological, radiological, nuclear, and some explosive protection. One of the complaints from USAF pilots is that current flight suit ensembles have too many layers, which can lead to thermal problems, especially in temperate environments such as the Middle East. The IAE is currently being pursued as a replacement to the current AGS and its additional layers of garments.

6.2.1. Types and Features of Anti-G Suits

Extensive research has been done in designing mechanisms to support the cardiovascular system during high-performance flight. Different techniques have been used, varying from merely taping the lower part of the body and limbs to complicated pulsating pneumatic systems.

The basic theory of the anti-G garment is to supply external pressure to the lower part of the body. Pressure on the abdomen prevents descent of the diaphragm and maintains the heart-to-eye distance, while compression of the legs prevents pooling of blood and assists in maintaining cardiac output.

6.2.1.1. Standard CSU-13 B/P Anti-G Suit. The Air Force anti-G garment as shown in Figure 6.2.1-1 consists of five interconnected pneumatic bladders that support the calves, thighs, and abdomen. This "standard" anti-G suit (AGS), as it is called, was developed by the David M. Clark Company in close association with Drs. Wood and Baldes at the Mayo Clinic in the early 1940s. The suit is still the most common anti-G suit used in modern air forces. The system is controlled by a valve, which varies the amount of air entering the bladders. The higher the level of $G$ encountered, the greater the pressures exerted on the body by the bladders. The standard AGS gives about $+1.0$ to $+1.5$ G increased protection above the normal relaxed threshold for $+G_z$ acceleration. Bladders over the thighs, calves, and abdomen are inflated and pressure increases as a function of increasing $G$ and provide effective lower torso counterpressure.

The anti-G suit (Figure 6.2.1.1-1), combined with the L-1 maneuver, is the best way of increasing $+G_z$ tolerance of crewmembers in high-performance aircraft, offering 3 to 5 G elevation in tolerance. USAF pilots who transition into high-performance fighter aircraft have to demonstrate they can tolerate $9$ G$_z$ for 15 seconds on the Holloman AFB human centrifuge. Many trained centrifuge subjects and pilots can sustain $9$ G$_z$ for 1 minute or more when protected with the standard AGS and performing a good straining maneuver. The uninflated AGS alone gives approximately $+0.4$ G protection, while the inflated AGS gives approximately $+1.0$ to $+1.5$ G protection over a 15-second duration. The human tolerance range for subjects wearing an inflated AGS and performing a good L-1 maneuver varies from $+7.5$ to $+9.0$ G.
6.2.1.2. **ATAGS G Suit.** The Advanced Technology Anti-G Suit (ATAGS) is considered a “full coverage” anti-G suit (Figure 6.2.1-1). The ATAGS covers the entire legs with a continuous bladder for uniform pressurization. The “pressure socks” are no longer a part of the ensemble. The ATAGS covers 95% of the lower torso of the pilot and has been shown to provide a 60% improvement in G-time tolerance on the AFRL centrifuge (Krutz et al., 1990). The ATAGS is currently flown as the anti-G suit trousers in the F/A-22.

6.2.1.3. **Integrated Aircrew Ensemble.** The Integrated Aircrew Ensemble (IAE) is currently under development by the Aeronautical Systems Group (77th AESG) at Brooks City-Base in cooperation with industry. The IAE will incorporate the AGS and other personal equipment pilots currently wear once it is fielded.

6.2.2. **Degrees of Protection, Protective Techniques, and Devices**

Any action or device that would increase the pressures in the hydrostatic column during increased $+G_z$ loads should increase the level of G, and time at G, that the human operator may tolerate. This concept is the underlying basis for two commonly used G-protection principles, the active straining maneuver and the AGS. There are two types of straining maneuvers in use, the M-1 and the L-1 maneuvers (Leverett and Whinnery, 1985). The M-1 ($M = Mayo Clinic$) consists of pulling the head down between the shoulders, slowly and forcefully exhaling through a partially closed glottis and...
simultaneously tensing all skeletal muscles. The L-1 (L = Leverett) maneuver is similar to the M-1 except the exhalation is forcefully applied to a completely closed glottis. When properly executed (about every third second), the exhalation phase of these maneuvers can result in an increase of intrathoracic pressure of approximately 50 to 100 mmHg, which raises arterial pressure at eye level. Adding +1.5 G to +5.0 G of G-tolerance to the straining pilot can raise G-tolerance from approximately +5 G to +7 – 10 G. The M-1 and L-1 maneuvers, while quite effective, are physically taxing and can interfere with speech and other communication modalities. When the maneuvers are performed for long periods (1 to 3 minutes), pilots can become severely fatigued. When this happens, the maneuver obviously loses its effectiveness, which, in turn, impacts on mission performance.

The standard AGS consists of bladders inserted into a full- or partial-body coverall (Figure 6.2.1.1-1). The bladders inflate during the onset of acceleration. The inflation pressures usually start around +2 G and increase as G increases to a maximum pressure at +9 G of approximately 570 mmHg, or 11 psi, in the bladders. The external increase in pressure afforded by the G suit reduces body deformation and blood pooling in the extremities and mechanically increases internal blood pressure. The relaxed subject wearing a G suit has an increased G-tolerance of 1 – 2 G (Wood, 1988; Davis et al., 2008). When combined with a straining maneuver, G-tolerance can increase up to +3 to +5 G, allowing the human to tolerate +9 G without losing consciousness (Burton et al., 1974).

### 6.2.3. COMBAT EDGE (PPB and APPB)

Another method of increasing G-tolerance is by applying positive pressure breathing (PPB) or assisted positive pressure breathing (APPB). PPB or, more appropriately, pressure breathing during G (PBG) refers to the application of pressure by a regulator to the breathing gas throughout the respiratory cycle (Prior, 1995). PBG was adapted to G protection from altitude protection. In the late 1980s, the USAF developed a system called COMBAT EDGE (combined advanced technology enhanced design G ensemble). COMBAT EDGE combines PPB with the standard AGS to provide additional G protection, or a protection edge, for pilots of high-performance aircraft. COMBAT EDGE includes a counterpressure vest to help maintain intrathoracic pressure and helmet-tensioning bladder to help tighten the oxygen mask seal on the pilot’s face during PBG. Assisted PPB, or APBG, is applied as the pilot inhales or takes a breath while straining. Pressures of 45 to 70 mmHg have been shown to increase G-tolerance time by increasing intrathoracic pressures, reducing the mechanical effects of increased G on respiration and reducing the effort needed to perform straining maneuvers. APBG systems for G protection are now being implemented in U.S. Air Force F-16, F-15, F-22, and F-35 aircraft with mask pressures up to 60 mmHg at +9 G.

The pressurized breathing gas results in an elevation of intrapulmonary pressure that is transmitted to the left ventricle and intrathoracic vessels, which results in an increase in systemic arterial blood pressure (Balldin and Wranne, 1980; Prior, 1995). Increased intrathoracic pressure can impair venous return unless lower body counterpressure via an AGS is applied. Breathing against PBG tends to reduce the inspiratory reserve volume of the lungs, although total lung capacity is increased under PBG. To prevent overdistension of the lungs when PBG exceeds 30 mmHg, chest counterpressure is recommended. Until recently, USAF pilots wore the counterpressure vest for COMBAT EDGE, but because of thermal complaints and the reduced threat for
high G operations, the requirement to fly with the vest has been dropped for F-16 pilots. It is currently (2009) in use by F-22 pilots where it may be needed for high-altitude protection during a possible rapid decompression above 50,000 ft. Some pilots are able to tolerate +9 G_z without having to perform a straining maneuver while protected with a properly fitted COMBAT EDGE ensemble. Other countries including Great Britain, France, Canada, Spain, Italy, and Germany are also fielding COMBAT EDGE-like systems for their high-performance aircraft. Sweden and Finland use assisted pressure breathing during G in combination with a full coverage anti-G suit. G-tolerance experiments on the Brooks City-Base, TX, centrifuge demonstrated that subjects protected with APBG in addition to the standard AGS had superior endurance on a +5 – 9 G simulated aerial combat maneuver (SACM) than those subjects protected with the standard AGS alone (Burns and Balldin, 1988). Also, in human performance studies involving subjects in the Wright-Patterson AFB (WPAFB) dynamic environment simulator (DES) centrifuge, those subjects protected with APBG systems were able to perform a simulated flying task to peaks of 9 G nearly twice as long as those subjects protected without APBG (Albery and Chelette, 1998). APBG raises the intrathoracic pressure of the subject/pilot, thus increasing eye level blood pressure and making it less fatiguing to breathe and to perform the straining maneuver (L-1) under high G for extended maneuvers.

In high-intensity wartime scenarios, pilots may be required to fly multiple, strenuous missions during the same day. A study was undertaken to investigate if up to 80 peaks at +9 G_z and 80 peaks at +8 G_z in five simulated sorties with four engagements in each sortie within a 4-hr period were feasible (Balldin et al., 2003). Nine well-trained centrifuge subjects were exposed to the above-mentioned rapid onset simulated aerial combat maneuver G-profiles in the centrifuge using the extended coverage anti-G suit with assisted pressure breathing during G. Seven of the nine subjects could endure all five sorties, even if some had extensive visual performance loss, maximal reported effort level, heart rates up to 173 bpm, and blood oxygen saturation down to 75%. Two subjects experienced GLOCs, and four cases of almost loss of consciousness occurred. Performance deteriorated during all G-exposures as measured with a tracking task, and neck muscle contraction was impaired. The conclusion was that it is possible to train subjects to withstand a large number of G-exposures.

For future high-performance aircraft design it is important to know the upper limit of various protective equipment and techniques. In a study by Burns et al. (2001), the G-tolerance up to +12 G_z was studied in six centrifuge subjects with different anti-G suits and seat configurations. The results showed that all six subjects were able to achieve +12 G_z with various combinations of +G_z-protective equipment, seat-back angle, and various amounts of straining. The data confirmed that effortless protection to +9 G_z was available using an extended-coverage anti-G suit and assisted pressure breathing with both 13° and 30° seat-back angle and to +10.5 G_z with 55° seat-back angle. With an extended-coverage anti-G suit, assisted pressure breathing, and a moderate straining, +12 G_z was definitely achievable at 55°, even with reduced anti-G suit pressure. With additional straining, +12 G_z was also achievable at the 13° and 30° seat-back angles.
6.2.4. Thermal or Other Considerations

The standard AGS has been a popular G protection ensemble because it is relatively simple, comes in numerous sizes, and is fairly universal. Because it does not cover the entire lower torso as does some other AGS, it is relatively comfortable in temperate climates. The rubber bladders that reside over the thighs, claves, and abdomen can become hot, however, and pilots complain about “hot spots” with the standard AGS. The USAF Air Combat Command (ACC) complained about the thermal issue of the COMBAT EDGE (CE) counterpressure vest several years ago. The pilots felt the vest led to overheating, thus reducing pilot effectiveness in temperate climates. A study was conducted with pilot-subjects wearing COMBAT EDGE as well as the standard AGS (STD) (Balldin et al., 2002). The subjects were heated in a controlled temperature thermal chamber at Brooks City-Base and then exposed to multiple high-G exposures on the AFRL centrifuge. Following heat stress, no significant differences were found between CE and STD with regard to core and skin temperature or dehydration level in contrast to the anecdotal information from the pilots. Use of CE did produce a significantly higher relaxed, gradual onset G-tolerance both before and after heat stress. Despite these findings, ACC requested a study to determine if the counterpressure vest impaired the acceleration tolerance up to +9 G (Balldin et al., 2005). The study report concluded that the elimination of counterpressure vest during use of PBG did not hinder an individual’s ability to reach +9 Gz or complete a short-duration simulated aerial combat maneuver G-exposure (Balldin et al., 2005). As a result of this research, ACC decided to make wearing the COMBAT EDGE vest optional for F-16 pilots but currently not for F-22 pilots as described above in 6.2.3.

Several years ago, a German manufacturer presented a novel AGS that incorporated water channels as the activating device for the suit rather than inflated air bladders. This AGS, called the Libelle (or Dragonfly), was evaluated by ACC as well as AFRL. The concept was not theoretically sound, and the AGS was not successful in either the ACC or AFRL evaluation. The concept was that garden-hose-sized water channels that ran the full length of the suit would tend to swell under G as water would be naturally drawn down into the lower torso. As these channels swelled they became elliptical, thus tightening the fabric adjacent to the channels. This tightening conferred counterpressure, and the counterpressure conferred increased arterial pressure to the pilot/subject. The Libelle was evaluated both in-flight in the F-16 and under controlled conditions on the Brooks and WPAFB centrifuges. Although the suit had some advantages (no requirement for hook-up to the jet), it did not protect as well as COMBAT EDGE with a full coverage AGS. A preliminary comparison showed that a G suit with full coverage in combination with pressure breathing during G gave a G-intensity tolerance of +9 Gz. With the Libelle it was only +6.3 Gz (Eiken et al., 2002). It was concluded that, under the conditions tested, the G protection afforded by the Libelle suit was not adequate for use in a +9 G aircraft.
References


Recommended Readings


Balldin, U and Burns, J. +Gz protection with assisted positive pressure breathing (PPB), Aviat Space Environ Med 1988; 59:225-233.


Burns JW. Re-evaluation of a tilt-back seat as a means of increasing acceleration tolerance.. Aviat Space Environ Med 1975; 46:55-63.


Burton RR. Guidelines for a research and development (RandD) program for high sustained G.. Physiologist 1993; Feb 36 (1 Suppl.):S94-7.


Concepts
Simulated aerial combat maneuver (SACM)

Vocabulary
Advanced technology anti-G suit (ATAGS)
COMBAT EDGE
G-induced loss of consciousness (GLOC)
L-1
M-1
7. MISSION-IMPOSED EFFECTS

I was anxiously waiting for my clean (no bombs, no bomb racks, no external fuel tanks) F-4D to accelerate past its transient limit of Mach 2.2 for a personal record. At sea level, the wind resistance in the relatively thick air and higher temperature would have kept us from exceeding about Mach 1.1. We were rapidly climbing through 48,000 ft after taking off from Phu Cat AB, Vietnam on this functional check flight in 1970, closing in on that record at about 1125 knots (over 1290 mph, Mach 1.96). The sky above was turning black because we were above more than 85% of earth’s atmosphere. While focusing on the altimeter and Mach indicator, the engine inlet ramps closing to avoid effectively choking the engines with the now-compressed air, I felt a shudder and the jet began to wobble from side to side a bit. My quest for Mach 2.3 would have to wait. I slowly pulled the throttles back to avoid compressor stalls, started a turn home to lose altitude and airspeed and called it a day. While taking off my harness and LPU, I mentioned to my back seat pilot that I sure was glad we didn’t have to eject up there. He replied “Why?” I just looked at him and said “Do you think we would have made it out alive?” At nearly 50,000 ft while wearing a summer flight suit and no jacket, leaving a nearly Mach 2 aircraft into –55°C air would almost assuredly have shattered the thin visors in our helmets, impaling our eyes with plastic shards, probably ripped off our helmets, mangled both arms, and then frozen them solid with most of the rest of our battered bodies before we reached an altitude where thawing would take place. Some of the guys said I should have just pressed on for the record. I think not.

7.1. Physiologic Effects of Acceleration

Andrew D. Woodrow, Lt Col, USAF (Ret), BSC; Robert M. Shaffstall, Col, USAF (Ret); William B. Albery, Ph.D.; and Lt Col Ryan W. Maresh, USAF, BSC, Ph.D.

1990 The USAF School of Aerospace Medicine laboratory functions including the centrifuge and chamber research facilities at Brooks AFB became the Armstrong Laboratory (AFSC Historical Publication).

7.1.1. Introduction

Aeromedical concern about the effects of acceleration has a long history. Concern was first stimulated as early as World War I when pilots complained of a loss of vision and consciousness during pullouts from dives in aerial combat. Interest in this area has continued until the present, where the effects of sustained acceleration have become a major limiting factor in the operation of frontline fighters and advanced trainer aircraft. Because of the higher thrust-to-weight ratio and structural strength, aircraft like the F-16, F-15, F-18, F-22, and F-35 are able to routinely fly in the +7 to +9 Gz range for sustained periods. This chapter aims to highlight the human responses to acceleration in terms of physiology, tolerance, and protective measures against the adverse effects of acceleration.
7.1.2. Basic Principles

Speed is the linear distance covered per unit of time and can be quantified as meters per second (m/s), feet per second (ft/s), miles per hour (mph), or in aeronautical terms as nautical miles per hour (knots, kts). Velocity is speed in a specified direction. When velocity changes due to a change in speed or direction, acceleration occurs. Acceleration is the rate of change of velocity per unit of time and can be quantified as feet per second per second (ft/s²). Common equations for calculation are:

- **Speed** = \( \frac{\text{distance}}{\text{time}} \)

- **Acceleration** = \( \frac{\Delta v}{\Delta t} \) or \( \frac{v^2}{r} \), where \( v = \text{velocity}; r = \text{radius} \)

- **\( G \)** = \( \frac{\text{applied acceleration}}{\text{acceleration of gravity}} = \frac{a}{g} = \frac{v^2}{gr} \)

- **Jolt** = \( \text{rate of increase in acceleration} = \frac{G}{s} \)

The units of acceleration are defined relative to the force of gravity on Earth. Near the surface the force of gravity exerts an acceleration of 32.2 ft/s², or 9.81 m/s². That force would cause a free-falling object to attain a velocity of over 100 mph in a vacuum within 3 seconds. The resistance of air results in a terminal velocity of about 120 mph for parachute jumpers at relatively low altitudes. The opening of their parachute results in a deceleration (opening shock), reducing that 120 mph to less than 15 mph in less than a second.

The unit \( G \) is a dimensionless ratio of both force and acceleration. It is often confused with \( g \), with \( g \) is the acceleration of gravity. \( G \) represents the total reactive force (mass \( \times \) acceleration) divided by the body weight and resisted gravitational acceleration (mass \( \times \) gravity) \( (G = \frac{F}{W} = \frac{\text{mass} \times \text{acceleration}}{\text{mass} \times \text{gravity}} = \frac{\text{acceleration}}{\text{gravity}}) \). Under sustained acceleration conditions, one can determine the amount of force by multiplying \( G \) by the weight (W) of the body.

Figure 7.1.2-1 shows the relationship between the various equations listed above.

![Figure 7.1.2-1. Relationship between Acceleration and G](image)
Newton’s third law states that for every action or force, there is an equal and opposite reaction. The reaction is the inertial force of the body, which is equal to, and opposite in direction from, the accelerative force. For example, the inertial force presses a crewmember back into the seat of an aircraft during the forward acceleration on takeoff and pushes him/her forward during deceleration on landing.

The force of deceleration during parachute opening shock is from head to foot or, in the aircraft vernacular, eyeballs down (Table 7.1.2-1 and Figure 7.1.2-2). Instead of eyeballs moving in a direction opposite the acceleration force, you could think of where your head would tend to go under each linear motion.

**Table 7.1.2-1. Forces in Response to Aircraft Acceleration**

<table>
<thead>
<tr>
<th>Linear Motion</th>
<th>Acceleration Description</th>
<th>Physiologic Standard</th>
<th>Aircraft Vernacular</th>
</tr>
</thead>
<tbody>
<tr>
<td>Backward</td>
<td>Backward acceleration</td>
<td>-G_x</td>
<td>Eyeballs-Out</td>
</tr>
<tr>
<td>Forward</td>
<td>Forward acceleration</td>
<td>+G_x</td>
<td>Eyeballs-In</td>
</tr>
<tr>
<td>Upward</td>
<td>Headward acceleration</td>
<td>+G_z</td>
<td>Eyeballs-Down</td>
</tr>
<tr>
<td>Downward</td>
<td>Footward acceleration</td>
<td>-G_z</td>
<td>Eyeballs-Up</td>
</tr>
<tr>
<td>To the Right</td>
<td>Right lateral acceleration</td>
<td>+G_y</td>
<td>Eyeballs-Left</td>
</tr>
<tr>
<td>To the Left</td>
<td>Left lateral acceleration</td>
<td>-G_y</td>
<td>Eyeballs-Right</td>
</tr>
</tbody>
</table>

The direction of the application of force is important in describing and understanding the physiological effects. Generally, we are mostly concerned with the direction of the inertial force on the hydrostatic column, or the direction the blood is being pulled, with respect to the long axis (head-to-toe) of the body. It is the inertial forces that produce the physiological effects of concern. As shown in Table 7.1.2-1 and Figure 7.1.2-2, each of the 3 axes can be referenced in either a positive or negative direction.
- **+G<sub>z</sub>** acceleration (positive G) occurs when inertial force acts toward the feet and the body is, therefore, forced toward the feet or aircraft seat. Examples are an abrupt recovery from a dive, turning maneuvers, or parachute opening shock. An example known to all pilots is the increase in +G<sub>z</sub> during a level turn as shown in Figure 7.1.2-3.

- **-G<sub>z</sub>** acceleration (negative G) occurs when the inertial force acts toward the head and the body is lifted up out of the seat. This occurs during abrupt dives or flying an outside loop.

- **+G<sub>x</sub>** acceleration (forward transverse G) occurs when the accelerative force acts across the body at right angles to the long axis in a back-to-chest direction. The inertial force would be in a chest-to-back direction and the body would be forced back into the seat. A flyer experiences +G<sub>x</sub> acceleration on takeoff.

- **-G<sub>x</sub>** acceleration (backward transverse G) occurs when the accelerative force acts across the body at right angles to the long axis in a chest-to-back direction. The inertial force would occur from back-to-chest and the body would be pressed forward away from the seat. This form of acceleration occurs during arrested, or “gear up,” landings. The F-35 pilot experiences -G<sub>x</sub> acceleration when the speed brakes are applied and tends to be thrust toward the instrument panel.

- **±G<sub>y</sub>** acceleration (right or left lateral G) occurs when the accelerative force acts across the body at a right angle to the long axis in a side-to-side direction. While not typically a concern in most aircraft, may be more relevant in vectored thrust aircraft.

![Figure 7.1.2-3. G-Force vs. Bank Angle in a Level Turn](http://www.rainierflightservice.com/blog/maneuvering-speed/)
Because a 154 pound (70 kg) body weighs 616 pounds (280 kg) at +4 G₂, mobility is grossly restricted during acceleration. During tight turning maneuvers in a high-performance aircraft, for example, pilots are pushed down into the seat, their arms and legs feel heavy, their cheeks sag, and they become incapable of free whole body movement. Above +2 to +3 G₂, escape from a disabled aircraft would be impossible without ejection devices.

Increased gravitational fields increase the weight of body parts (von Gierke et al., 1991). Body parts can become elongated or compressed under the G vector; this can affect the shape and function of the soft internal organs including the heart, lungs, kidneys, liver, etc. Higher muscle forces are required to keep the head, torso, and limbs in desired positions. At G forces of approximately +2 G₂, there is increased pressure on the buttocks, drooping of the face, and noticeably increased weight of all body parts; at this level of G force it is difficult to raise oneself, and at +3 to +4 G₂ it is nearly impossible. Experiments were conducted in the Wright-Patterson AFB (WPAFB) dynamic environment simulator (DES) centrifuge where subjects arose from a seated position and performed a whole body jump at G₂ levels up to +1.8 G₂. Although most subjects had no problem standing up at 1.8 G₂, jumping and leaving one's feet was very difficult (Constable and Carpenter, 1995; Chelett et al., 1995). Above +3 to +4 G₂, controlled motions require greater effort, accommodation, and learning to offset loss of fine motor control. One typically cannot raise the arm at greater than +8 G₂ or legs at greater than +3 G₂. Head pitching is difficult at greater than +4 G₂, and some individuals who get their heads pitched forward at high G₂ (> 6 G) are unable to right themselves in the seat until the acceleration is unloaded. The hand can be raised slightly at +25 G₂. Speech is severely affected yet possible up to +9 G₂ if the operator is utilizing protective techniques properly. Sensory inputs such as vision affected through eyeball deformation, vestibular orientation through the semicircular canals and otoliths, and force/weight judgments in manual dexterity tasks can be affected. Acceleration protective equipment, as discussed in Section 6.2, can either improve or limit/degrade performance through mechanical interference.

The higher the jolt or rate of increase in acceleration, from any direction, the greater the physiological effects on the body. The total force applied and the time it takes for the force to be applied must be considered when determining the effects on the body.

The greater the surface area over which a force is applied, the less the final effect on the body. Consider the consequences if an equal force is applied to a nail and to a block of wood held against the body. Since the wood distributes the force to a large area of the body, a much larger force would be required on the block of wood to produce injury. An application of this principle in flying is the shoulder and parachute harness, both designed to distribute abrupt deceleration forces over larger areas of the body.

A pilot in a high-performance aircraft may be exposed to lateral buffeting, which results in ±G₂ acceleration. Spins in aircraft such as the T-6 expose the crewmembers to lateral G, but the effects are negligible.
7.1.3. Basic Physiological Effects of Acceleration

In general, the greater the magnitude of the accelerative force, the greater the effect on the body. Generally, the structural system of the body can withstand short duration forces up to approximately +25 G, with little or no permanent damage depending on the axis of exposure.

The body normally functions under a condition of 1 G because of gravity. Any increase in G-force causes a proportional increase in the weight of the body. At G levels two to three times normal, it becomes impossible to move freely because of the increased body weight. Consequently, it has been necessary to install ejection seats in high-performance aircraft to enable the crewmembers to escape if necessary. Most high-performance aircraft are designed to withstand +9 G with a 50% safety factor built in before structural damage is imparted to the airframe; +9 G plus 50% overload equals approximately +13.5 G maximum load. Within this range of accelerative forces, the primary physiological effect is on the cardiovascular system, since other body structures can easily withstand these forces.

The time duration a force is applied will determine the effect of acceleration on the body. These effects will vary depending on whether the body is exposed to impact acceleration or prolonged acceleration. It has been generally accepted that acceleration for a time greater than a 1 second exposure should be called prolonged acceleration, and an exposure duration less than 1 second should be called impact acceleration. For example, jumping from a table 3 ft (0.9 m) high would produce an impact G-force of 12 – 15 positive G, or +Gz, for a fraction of a second with no ill effects. If a subject is exposed to 12 – 15 positive Gz for 2 seconds or longer, severe, physiological changes are produced.

The physiological effects of acceleration can be viewed from several perspectives. This section will take the systems approach. Different body systems will be reviewed, and the effects of acceleration on each system will be discussed. Prolonged acceleration affects the body in five principal ways: by restricting mobility, by impairing respiration, by affecting the cardiovascular system, by reducing visual acuity, and by stimulating the vestibular apparatus in the inner ear.

7.1.4. Cardiovascular System Effects

The cardiovascular system is man’s limiting factor when exposed to prolonged +Gz accelerations. The hydrostatic pressures produced by +Gz passively dilate the blood vessels below the heart and result in pooling of blood within these vessels. High hydrostatic pressures cause circulating blood volume to be further decreased due to an increased flow of serum through the capillaries, causing edema formation. Both edema formation and blood pooling reduce the amount of venous blood returning to the heart, limit the heart’s output, and reduce the circulating volume of blood.

If the accelerative force remains constant at a visual blackout level for 4 to 6 seconds or longer, vision may spontaneously return. This occurs because of compensatory mechanisms, which increase the blood pressure to the point where blood flow to the eye resumes. The primary mechanisms in this reflex are the pressure sensitive nerve endings in the carotid sinuses and in the arch of the aorta.

When blood pressure drops in the aortic and carotid arteries, specialized receptors relay this information to the vasomotor center in the medulla. The vasomotor center responds by sending impulses to the arterioles throughout the body, causing
them to constrict. This vasoconstriction increases peripheral vascular resistance and, along with an increased heart rate, causes arterial blood pressure to increase. The increase in blood pressure brought about by this reflex may be enough to overcome the intraocular pressure; blood flow returns to the retina, and vision returns. A time lag of about 4 to 6 seconds between blackout and return of vision is required by this reflex mechanism.

Centrifuge studies have shown that the effects of ±$G_x$ on the cardiovascular system are diminished when compared to ±$G_z$. During transverse acceleration, subjects have withstood +15 $G_x$ without blackout, but performance capabilities are impaired at levels as low as +5 $G_z$, when it becomes impossible to raise the head or the arms against the high $G$ force. Some limb movement can be achieved between 5 to 9 $G$, but it is restricted—it may be impossible for a pilot to reach forward and activate a switch located on the instrument panel at this level of $G$. Minimal movement using wrists and fingers is possible and can be accomplished if the arm is not required to be moved from the armrest.

The adverse effects of the increased weight on the body components are most clearly shown by the cardiovascular system. The blood stream is supported by elastic blood vessels and depends upon specific pressures for normal function. Excessive acceleration causes gross disturbances in the distribution of pressures and volumes within the system.

These effects are especially pronounced when a subject is exposed to prolonged +$G_z$ and -$G_z$ accelerations and can be explained by the effects of gravity on the body as observed in Figure 7.1.4-1 (Burns, 1995). Here, the hydrostatic effects on the arterial blood pressure of a person in an upright and supine position undergoing an accelerative force of +5 $G_z$ are shown.

In this example, a 6 ft (182 cm) man standing upright exposes a 57 inch (145 cm) column of blood from the heart to the feet to the effects of +1 $G_z$. Because of its weight, this column of blood exerts a hydrostatic pressure of 113 mmHg in addition to the normal arterial pressure in the feet. For a man with a mean blood pressure of 100 mmHg at heart level, the mean arterial blood pressure in the feet would be 213 mmHg. When the subject is in the upright position, this hydrostatic pressure passively distends the blood vessels of the legs and results in an additional 0.53 quart (0.5 L) of blood being pooled within the feet and legs. Each additional $G_z$ unit increases the blood pressure at foot level another 113 mmHg, resulting in more blood being pooled in the lower extremities. This redistribution of blood within the body reduces the volume of circulating blood available to supply the body's requirements.
In Figure 7.1.4-1, blood pressure above the heart is reduced by hydrostatic pressure. Under conditions of +1 Gz, the 12 inch (30 cm) fluid column from the heart to the brain exerts a hydrostatic pressure of 23.4 mmHg (the model and corresponding math is discussed in more detail below). Therefore, if the mean blood pressure at the heart is 100 mmHg, the pressure at the level of the brain will be about 77 mmHg. For each additional +Gz, the blood pressure at brain level would be reduced another 23.4 mmHg (average range is 22 – 25 mmHg/G) (Burns, 1995), eventually producing stagnant hypoxia. In the upright man under +5 Gz, mean arterial pressure at eye level is therefore approximately -17 mmHg.

The easiest way to understand many of the basic cardiovascular effects of G is to model human circulation as a simple hydrostatic column. The important parameters are the height of the column, the pressures within the column, and the density of the fluid affected. For all practical purposes, this model is a good representation of the body’s response to rapid onset, short duration +Gz stress.

Assuming this model, the hydrostatic pressure at any point in the circulation can be predicted using the following formula:

\[ P_H = h \times d \times G \]

where:
- \( P_H \) = the hydrostatic pressure (mmHg)
- \( h \) = the height of the column (mm)
- \( d \) = the specific density of blood relative to mercury \( \left( \frac{1.06}{13.6} \right) \) (Trudnowski and Rico, 1974; Banks et al., 2008)
- \( G \) = the accelerative force in G
The hydrostatic column that is of most interest is between the heart and the brain. The brain is approximately 300 mm above the heart. Therefore, at 1 G there is a hydrostatic pressure gradient \( P_H \) of:

\[
P_H = 300 \text{ mm} \times \frac{1.06}{13.6} \times 1 = 23.4 \text{ mmHg}
\]

The hydrostatic pressure represents the downward force exerted due to the weight of the blood in response to gravity. The result is that the blood pressure above the heart is reduced. This decreased pressure at the eye and/or brain level \( P_{eye/brain} \) can be determined by subtracting the \( P_H \) from the heart level systolic blood pressure. This is represented by:

\[
P_{eye/brain} = P_{systolic} - P_H
\]

If one assumes an average heart level systolic blood pressure of 120 mmHg, then the \( P_{eye/brain} \) is 120 – 23 = 97 mmHg. At just over +5 G, the \( P_H \) will exceed the average \( P_{systolic} \) of 120 mmHg, and the lack of blood will cause unconsciousness. To understand the phenomenon of visual “blackout,” one needs to remember that the average intraocular pressure is 20 mmHg. This intraocular pressure is the pressure the retinal artery must overcome to supply oxygenated blood to the retina. “Grayout” occurs as the retinal artery pressure in the periphery can no longer overcome the intraocular pressure and the blood flow to the peripheral retina ceases. Blackout is explained by the complete lack of blood flow to the eye, which causes it to cease to function before the brain shuts down. Blackout will precede unconsciousness by about +0.87 Gz (20 mmHg/23 mmHg).

The model outlined here does not adequately explain the cardiovascular response to gradual onset +Gz because the cardiovascular reflexes can compensate for the changes caused by +Gz stress. These reflexes will also play a role in the response to rapid onset G after 6 to 10 seconds of exposure. The reflexes, which are mediated by the carotid artery and aortic arch baroreceptors, result in increased sympathetic discharge and a resulting increase in cardiac rate, vasoconstriction, and venoconstriction and an increase in cardiac contractile forces. In general, forces lasting less than 2 seconds have little effect on the cardiovascular system.

An increase in heart rate has been one of the generally observed responses to +Gz. The response has been highly variable due to individual variation, physiological stress, and the amount of muscular straining being performed by the subject. The amount of absolute increase in heart rate is affected by the maximum G level reached and the rate at which the G was applied. Occasionally, individuals have been observed to have paradoxical bradycardia at high G levels. This finding is thought to be a sign of cardiac decompensation and is grounds for halting the G exposure. The heart-rate response to G has not been shown to be predictive of G-tolerance. Cardiac output has been noted to transiently increase under +Gz. The measurement of cardiac output under increased G load is difficult. It has been shown that there is a decrease in venous return under high G loads, which decreases the preload to the heart. So, despite the increase in heart rate, most authors believe that cardiac output is, at best, maintained under high G and that the output probably decreases in most cases.

Numerous cardiovascular symptoms have been noted under G stress. These symptoms include grayout, blackout, loss of consciousness with accompanying
seizures, convulsions, amnesia and confusion, cardiac dysrhythmias (tachycardia and bradycardia), heart blocks, and stress cardiomyopathy. The rhythm disturbances seen under +G\(_z\) have been of great aeromedical interest. There has been and continues to be debate on the significance of these observations. In asymptomatic and otherwise healthy individuals, these dysrhythmias are probably benign. Three specific dysrhythmias cause particular concern because of the potential for sudden incapacitation. The dysrhythmias are sinoatrial (SA), atrioventricular (AV) dissociation, and ventricular tachycardia. Table 7.1.4-1 summarizes a 3 year history of acceleration-related dysrhythmias observed in healthy subjects on the USAFSAM centrifuge (Leverett and Whinnery, 1985).

<table>
<thead>
<tr>
<th>Rank</th>
<th>Occurrences</th>
<th>Dysrhythmia Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1,566</td>
<td>Sinus arrhythmia (rating varying &gt;25 bpm)</td>
</tr>
<tr>
<td>2</td>
<td>1,073</td>
<td>Premature ventricular contractions (PVCs)</td>
</tr>
<tr>
<td>3</td>
<td>768</td>
<td>Premature arterial contractions</td>
</tr>
<tr>
<td>4</td>
<td>546</td>
<td>Sinus bradycardia (rate &lt;60 bpm)</td>
</tr>
<tr>
<td>5</td>
<td>372</td>
<td>Ectopic atrial rhythm</td>
</tr>
<tr>
<td>6</td>
<td>272</td>
<td>Premature junctional contractions</td>
</tr>
<tr>
<td>7</td>
<td>171</td>
<td>PVCs with bigeminy/trigeminy</td>
</tr>
<tr>
<td>8</td>
<td>126</td>
<td>Multiform PVCs</td>
</tr>
<tr>
<td>9</td>
<td>104</td>
<td>AV dissociation</td>
</tr>
<tr>
<td>10</td>
<td>104</td>
<td>Paired PVCs</td>
</tr>
</tbody>
</table>

*Based on the exposure of 544 different individuals exposed to 9,831 +G\(_z\) runs (Leverett and Whinnery, 1985).

Figure F.1.4-2 shows an example of an EKG tracing of a preventricular contraction (PVC) from a volunteer during a centrifuge run.

Many of the foregoing observations on cardiovascular response to +G\(_z\) were based on observations of response to one-time exposures to +G\(_z\) on the centrifuge. Recent research work has been concentrated on the response to multiple +G\(_z\) exposures and the effects of fatigue on this response. Generally, heart-rate response is...
dependent on $+G_z$ level, but it also appears to be related to the blood lactate level, which is an indication of the amount of anaerobic work involved in resisting the effects of $+G_z$ and the performance of the anti-$G$ straining maneuver (AGSM) (Burton et al., 1987). This ability to perform anaerobic work may explain the observed effects of physical training regimens on aerial combat maneuvering $G$-tolerance (Epperson et al., 1982; Epperson et al., 1985; Burton, 1986).

7.1.5. Respiratory Effects

During $+G_z$, the increased weight of the viscera pulls the diaphragm down, increases the thoracic volume, and disturbs the mechanics of respiration. The major physiologic effects of $+G_z$ on pulmonary function can be summarized as: 1) altered ventilation/perfusion ratios resulting in hypoxemia, 2) airway closure, and 3) atelectasis (Banks et al., 2008). There is also concern that exposures above $+9 G_z$ may result in pathophysiologic changes such as a compromise of chest wall mechanics, pulmonary edema, and disruption of anatomical integrity of the lung (Banks et al., 2008).

Observations of pulmonary function during increased $+G_z$ have shown an increased respiratory rate, an increased tidal volume (limited at the upper $+G_z$ by the $G$-force and the compression on the $G$ suit), and an increased physiologic dead space. As a result, the $P_{a}CO_2$ changes very little with increasing $G$ stress (Banks et al., 2008).

Through a reflex mechanism, the respiratory rate is increased to almost double its resting value when $G$ loads as low as $+3 G_z$ are encountered. As the $G$ loads are increased, fast, shallow breathing occurs. This leads to ineffective respiration and can result in hypoxic hypoxia. Increased $+G_z$ loads also produce an increased ventilation/perfusion ratio in the upper portions of the lung. In the lower portions of the lungs, this ratio is decreased. The net result is an impaired ability of the lung to oxygenate blood.

The hydrostatic theory just explained would predict that at a higher $+G_z$ there would be less perfusion of the upper areas of the lung and that at higher $-G_z$ there would be more. This theory has been nicely demonstrated in a study of perfusion scans of the lung at various $G_z$ levels (Whinnery, 1980). Similar ventilation-perfusion inequalities would be expected from $G$ exposure along the axes, and this has been observed (Banks et al., 2008; Popplow et al., 1983).

Positive pressure breathing systems, straining maneuvers, and inflated anti-$G$ garments also influence the breathing patterns in man during flight. With $+G_x$ forces above $+8 G_x$, respiration is mechanically impeded due to a number of factors: the pressure of the viscera on the diaphragm, attempting to inspire against the increased chest weight, and the gross distortion of the nose and mouth. Above $+8 G_x$, respiration becomes increasingly difficult because the rib cage becomes more and more fixed in the expiratory position, and diaphragmatic breathing is required to maintain sufficient air exchange. Some subjects have been able to withstand transverse acceleration of $+26 G_x$ for several seconds with complete post-run recovery. Some subjects, immersed in water and protected with positive pressure breathing, have endured exposures of over $+30 G_x$. Painful respiration is usually the limiting factor in human tolerance to $\pm G_x$, and time tolerance at various levels of acceleration is usually defined by this difficulty in breathing. During $\pm G_x$, embarrassment of the respiratory system appears to be the most important limiting factor.

Acceleration atelectasis is a collapse of alveoli in the dependent lung caused by absorption of the alveolar gas. It has been associated with symptoms of cough, chest
pain, and dyspnea. Acceleration atelectasis has been observed to be exacerbated by breathing 100% oxygen and by the use of the anti-G suit (Tacker et al., 1987). It has been shown that the absorption atelectasis produced by +Gz exposure can be reduced progressively with the addition of an inert gas (N2) into the breathing mixture until it is almost entirely prevented with a mixture of 40% N2 by the use of unassisted positive pressure breathing (PPB) and the anti-G straining maneuver (AGSM) (Tacker et al., 1987). Acceleration atelectasis has been one of the reasons diluter demand regulators have been favored by the USAF. The use of an onboard oxygen generating system (OBOGS), or more appropriately a molecular sieve oxygen generating system (MSOGS), in new-generation fighter aircraft that provides 95% oxygen on a continuous basis has prompted concern that acceleration atelectasis may become an operational problem of some significance (Tacker et al., 1987).

7.1.6. Renal System Effects

Decreases in renal blood flow observed when humans stand upright and when they exercise make it reasonable to predict that +Gz stress would decrease renal blood flow. Although this prediction has not been investigated in man, animal models confirm this prediction (Banks et al., 2008). Oliguria has been noted in humans, and increased levels of plasma rennin have been measured at +2 and +2.5 Gz (Banks et al., 2008). Further work on this area may be useful, as even small deficits of water and sodium balance have been associated with decreased +Gz tolerance.

7.1.7. Musculoskeletal Effects

Back, neck, and limb problems are the most frequently reported musculoskeletal problems. There are reported cases of intervertebral disk ruptures under high +Gz and many complaints of sore necks after the centrifuge rides and flights in high-G aircraft. Permanent injury is rare enough to warrant a case report.

7.1.8. Central Nervous System Effects

The observed central nervous system (CNS) effects are explainable by the effect of G on the cerebral circulation.

7.1.9. Visual Effects

Impairment of vision is one of the consequences of exposure to +Gz. Because of hydrostatic pressure, there is a decrease in the blood pressure in areas above the heart when +Gz is applied. The driving power or systolic pressure supplying blood to the brain is reduced approximately 23 mmHg for each +Gz exerted. The first effect of this decreased blood pressure is impairment of vision. For a subject in a relaxed condition and unprotected by an anti-G suit, loss of peripheral vision and reduction of visual acuity (grayout) occur at levels of +3 to +4 Gz. When G-forces reach +3.5 to +4.5 Gz, vision is lost and blackout occurs. At this point, the subject is still conscious and can hear and respond to questions. Blackout occurs before unconsciousness because the intraocular pressure of 16 to 20 mmHg reduces the driving power of the blood to the eye.

Human centrifuge experiments have proven visual acuity decreases progressively as the magnitude of the acceleration increases. The changes in acuity
cannot be completely accounted for in terms of reduced circulation to the eye and brain. One possible explanation is that these changes result from displacement of the crystalline lens of the eye in the direction of the G-force.

Another visual effect of acceleration is the decreased response to the retina to the same illumination level of light. The adverse effect of acceleration on the ability of the pilot to read the instruments in a night-lighted cockpit may be so pronounced that it results in reading errors.

Col (Dr.) Rick Allnutt discovered changes to the human color vision system under sustained G while riding in the dynamic environment simulator (DES) centrifuge at Wright-Patterson AFB in the late 1990s. Allnutt found centrifuge subjects experienced a shift in hue from light blue to white and green to yellow as they observed a color chart at or near central light loss of vision under G (Allnutt and Tripp, 1998). Dr. Tamara Chelette (2000) investigated colorimetric factors under sustained acceleration including luminance, contrast ratio, saturation, wavelength, display techniques, and individual variability. Balldin (2000) found hue shifts in subjects exposed to 7 to 9 G\textsubscript{z}. Balldin observed yellow hues shifting to yellow-red, red hues shifting to red-yellow, and green shifting towards yellow. These results indicate the ability to correctly identify the basic colors at high G\textsubscript{z} may be impaired. The physiological basis of this phenomenon has never been confirmed. It is apparently due to a hypoxic effect at the eye-brain level. The implications on cockpit color display design in high-performance aircraft are obvious.

During lateral acceleration experiments in the DES centrifuge, it was observed that subjects lost vision in one eye relative to the other when exposed to both increasing +G\textsubscript{z} as well as lateral G\textsubscript{y}. It was noticed subjects were reporting the loss of vision in one eye before the other during an agile aircraft experiment on the DES. This is the first report of this phenomenon, since most combined axes experiments in the past did not have loss of eye level blood pressure with increasing +G\textsubscript{z} as the end point. This phenomenon, called temporary monocular vision, can occur in aircraft with thrust-vectoring capability or canards that allow the aircraft to slip laterally while turning and pulling G (Allnutt, 2000).

Visual disturbances, such as blurring and excessive flow of tears, have been noted above +12 G\textsubscript{x}. The decrease in vision above +12 G\textsubscript{x} may, in part, be attributed to eyeball distortion caused by the large force acting on the anterior of the eye.

7.1.10. Vestibular Effects

The vestibular apparatus of the inner ear plays an important role in spatial orientation and balance. The otolith organs are stimulated by gravity and linear accelerative forces to give the flyer a sense of direction; the semicircular canals respond to angular accelerations to give the flyer another sense of direction. Accelerative forces in flight may influence the vestibular apparatus and induce disorientation. There are several illusions that can be generated when G > 1 (see Section 7.3).

The somatogravic illusion is the classic "pitch up" or "pitch down" illusion. When positive or negative G\textsubscript{x} is generated during flight, the otolith organs of the vestibular system can be stimulated; since we normally sense this stimulation and relate it to head tilts at 1 G, a pilot can falsely perceive a +G\textsubscript{x} exposure in the absence of good visual cues as a head tilt upward, or pitch up (Figure 7.1.10-1). Pilots may incorrectly pitch their aircraft down based on this false perception. When decelerating or experiencing -G\textsubscript{x}, the opposite effect can be perceived.
The G-excess illusion is similar to the somatogravic illusion but typically occurs when \( G > 1 \) and the pilot is looking out of the cockpit, such as during formation flight. If \( G > 1 \) and the pilot’s head is tilted up, the otoliths will slide further aft than when \( G = 1 \). This “excess” slide can be interpreted by the brain as a false pitch up; to correct this perception, pilots may pitch the nose of the aircraft down and drop the wing. These maneuvers can result in a mishap.

Figure 7.1.10-1. Accelerative Effects on Otolith Stimulation

7.1.11. G-Tolerance

The human tolerance to acceleration has been the subject of much research. This research has traditionally looked at the relaxed tolerance of subjects to single exposures of \( +G_z \) and the absolute level of \( +G_z \) the subject is able to withstand. This research has defined the limits of G-tolerance in the z axis very well. The limits of G-tolerance in the other axes are not so well defined. More recently there had been increasing interest in repeated exposures to multiple \( +G_z \) levels for varying time courses. The purpose of this research would be to define the other parameter in G-tolerance, namely, that of durations of tolerance and the various endpoints that have been used by researchers in their experiments.

The accelerative forces acting at right angles to the long axis of the body, chest-to-back or back-to-chest direction, are annotated by the symbol \( \pm G_x \). The transverse position of the body shown in Figure 7.1.4-1 provides the greatest tolerance to acceleration because of the limited hydrostatic effect on a blood pressure.

Research studies have been directed toward increasing human tolerance to accelerative forces. The main thrust of these investigations has been to develop methods to maintain an adequate eye-level blood pressure.

7.1.11.1. Blood Pressure. No correlation has been found between chronic high blood pressure and high G-tolerance, although higher blood systolic and/or diastolic blood pressure appears to aid G-tolerance (Webb et al., 1991). When fear or excitement cause blood pressure and heart rate to be elevated, improved G-tolerance
results. The reason is that a greater G force is necessary to reduce the blood pressure to the point where cerebral blood flow ceases.

7.1.11.2. Body Position and Seat Back Angles. Since man can tolerate much higher G-forces when applied in the transverse direction, attempts have been made to alter pilot and crew stations to tilt back positions in the aircraft. The simplest and most effective means of increasing tolerance to $+G_z$ acceleration is to change the position of the body’s long axis relative to the inertial force. The intent is to reduce the vertical height of the hydrostatic column of blood extending from the heart to the eyes. In Figure 7.1.4-1, the heart-eye distance in the standing and supine posture is compared with other body positions. Short of full prone or full supine positions, the semi-kneeling position would offer the most protection with no gross symptoms to approximately $+10\ G_z$.

To calculate the $P_H$ for a given seat angle, the heart-eye distance is decreased by the cosine of the seat angle as represented below and in Figure 7.1.11.2-1:

$$P_H = h \cos \theta \cdot d \cdot G$$

where:

$\theta$ = seat tilt back angle

A backward tilt of 30° exists in the F-16 aircraft, while the F-15 seat is tilted 12.5° from the vertical, the F/A-22 is tilted 14.5°, and the F-35 is adjustable from 12 – 17.5° from vertical. A backward tilt of about 45° is required to significantly increase the blackout threshold. To obtain maximum tolerance to $+G_z$, it is necessary to tilt the subject to 60° to bring the eyes level with the heart. The following calculations show the $P_H$ reductions for varying seat tilt back angles (0 – 60°) and Figure 7.1.11.2-2 shows it graphically:
We can use the equations above to predict the G level at which an individual will experience blackout and loss of consciousness occurs when no countermeasures, such as the anti-G suit, AGSM, or cardiovascular reflexes, are used. Starting with

\[ P_{\text{eye/brain}} = P_{\text{systolic}} - (h \cos \theta) \times \left( \frac{1.06}{13.6} \right) \times G \]

and rearranging for G, we get:

\[ G = \frac{P_{\text{systolic}} - P_{\text{eye/brain}}}{(h \cos \theta) \times \left( \frac{1.06}{13.6} \right)} \]

Using the same heart-brain distance (300 mm) and systolic pressure (120 mmHg) as before, we can predict the G level at which an individual will blackout (P_{\text{eye}} < 7-16)
20 mmHg) and lose consciousness ($P_{\text{brain}} = 0$ mmHg). Keep in mind that the pilot will only experience the full benefit of the seat angle if they are leaning back with their head against the headrest. In operational settings, most pilots lean forward to fly and fight the aircraft, and therefore diminish the benefit from a backward tilting seat. Table 7.1.11.2-1 summarizes the calculations.

<table>
<thead>
<tr>
<th>Seat Angle (degrees)</th>
<th>G at Blackout</th>
<th>G at LOC</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>4.28</td>
<td>5.1</td>
</tr>
<tr>
<td>12.5 (F-15)</td>
<td>4.38</td>
<td>5.26</td>
</tr>
<tr>
<td>14.5 (F-22)</td>
<td>4.42</td>
<td>5.3</td>
</tr>
<tr>
<td>17.5 (F-35)</td>
<td>4.48</td>
<td>5.38</td>
</tr>
<tr>
<td>30 (F-16)</td>
<td>4.94</td>
<td>5.9</td>
</tr>
<tr>
<td>45</td>
<td>6.05</td>
<td>7.26</td>
</tr>
<tr>
<td>60</td>
<td>8.55</td>
<td>10.26</td>
</tr>
</tbody>
</table>

7.1.11.3. M-1 Maneuver. The M-1 maneuver is a straining technique that improves G-tolerance. It was named M after the Mayo Clinic, where it was developed by Dr. Earl Wood. Pilots refer to the M-1 maneuver as the “grunt” maneuver since it approximates straining to lift a heavy weight. The M-1 maneuver consists of leaning forward at the hips, slowly and forcefully exhaling through a partially closed glottis, and, simultaneously, forcefully contracting all skeletal muscles. Leaning forward gives some degree of circulatory protection, the pressure within the chest is increased by strong muscular contraction against a partially closed glottis, and the contraction of voluntary muscles externally compresses and reduces blood pooling. For the best results, slow exhalation must be repeated every 4 to 5 seconds.

Properly executed, the M-1 maneuver results in a fluctuating intrathoracic pressure of 50 to 100 mmHg, which raises the arterial blood pressure and increases $+G_z$ tolerance about 2 G. Human centrifuge subjects wearing anti-G suits, who have been instructed in the correct performance of the M-1 maneuver, have been able to withstand $+9 \, G_z$ for 60 seconds and longer without visual symptoms.

An aircrew member may obtain equal protection from either the M-1 maneuver or by attempting to exhale against a closed glottis if he/she also simultaneously contracts all skeletal muscles. This variation is called the L-1 maneuver.

7.1.11.4. L-1 Maneuver. This G straining maneuver was developed by Dr. Sidney Leverett, thus the L nomenclature. Equivalent protection is provided by each of the maneuvers both with and without the anti-G suit. The L-1 maneuver is preferred by most aircrew today as it is easier on the throat and larynx. Forcefully exhaling against a closed glottis without vigorous skeletal muscle tensing can lead to an episode of unconsciousness at relatively low G levels. Therefore, instruction on the proper method of performing the straining maneuver is essential. Note: Anti-G suits are discussed in Section 6.2.
7.1.11.5. **Individual Variations in Tolerance to $+G_z$.** In discussing the physiological effects of G-forces, a range of $+G_z$ acceleration was expressed for a particular effect. It was stated that grayout occurs at an average value of +3 to +4 $G_z$. Because of individual differences in heart-to-head distance, blood pressure, muscle tone, and venous return, the true range might be 2.2 to 7.1 $G_z$.

7.1.11.6. **Factors Decreasing Tolerance to $+G_z$.** Any factor that reduces the overall efficiency of the body, especially if it reduces the reserve of the circulatory system, causes a marked reduction in man’s tolerance to G. Although not correlated with G-induced loss of consciousness (GLOC) or predictability of G-tolerance, the following anthropometric/physiologic parameters were associated with higher G-tolerance during pilot training on the USAFSAM centrifuge at Brooks AFB, TX (Webb et al., 1991):

- Greater age
- Lower height
- Lower height/weight ratio
- Higher systolic and/or diastolic blood pressure

An example of self-imposed stress is the lowered tolerance caused by an alcohol hangover. Several abnormalities exist that would preclude the individual from being exposed to greater than $+1 G_z$: varicose veins, hemorrhoids, hernia, and eye disorders, especially glaucoma.

Early research in $+G_z$ tolerance attempted to define the point of unconsciousness. As the understanding of the response to G became more refined, “blackout” or visual loss was used as the endpoint in human research. The modern concept defines $+G_z$ in terms of the rate of acceleration, whether it be rapid (>0.33 G/s) onset or gradual onset, and the point at which there is peripheral light loss or the point of central light loss.

7.1.11.7. **G On-Set Rate and G-Time Tolerance.** Research at USAFSAM (Brooks City-Base) has used three kinds of centrifuge runs to evaluate G-tolerance. The gradual on-set run (GOR) is conducted at 0.1 G/s, the rapid on-set run (ROR) at 1 G/s, and very high onset G (VHOG) at 6 G/s. The GOR evaluates the body’s baroreceptor response to G, as the cardiovascular responses have time to be effective. The RORs and the VHOGs are more representative of the type of G-onset profiles that aircrew are likely to experience in the F-15, F-16, and F-22 generation of aircraft. This kind of research on normal unprotected subjects has resulted in the data found in Figure 7.1.11.7-1.
The G-Time Tolerance Curve (Stoll, 1956) (Figure 7.1.11.7-1) models the response of a human with no previous G exposure. Modifications in the curve occur if a negative G exposure is followed by a +Gz exposure resulting in what has been termed the push-pull effect (Banks et al., 2008). The push-pull effect has been implicated in a number of GLOC mishaps (Banks, 1994). During repeated exposure to +Gz in a high-performance aircraft, it is common practice to push negative Gz while regaining aircraft energy prior to another high G turn. During this period of negative G, which may last only 1 or 2 seconds, there is a rapid development of bradycardia, peripheral vasodilation, and the likelihood of cardiac arrhythmias. The push-pull effect occurs when the pilot pulls positive G giving rise to profound changes in cerebral perfusion and a marked reduction in +Gz tolerance (Prior, 1995). A good anti-G straining maneuver and an operating anti-G suit reduce the magnitude of the push-pull effect, but a significant lowering of G-tolerance remains.

As discussed in the section on cardiovascular effects, the simulated aerial combat maneuver has been used to evaluate longer duration tolerance to +Gz stress and to evaluate the fatigability of G-tolerance. This research shows that the work of being at high +Gz is anaerobic and that understanding human performance and endurance at high +Gz is best related to isometric exercise physiology and to anaerobic metabolism (Burton et al., 1987).

7.1.12. G-Induced Loss of Consciousness

Unconsciousness occurs at G levels slightly higher than those that produce blackout and is produced in most unprotected individuals when +4.5 to +6 Gz is exerted for more than 6 seconds. It occurs when the effective blood pressure at the level of the brain is reduced to the point where inadequate blood flow and unoxygenated blood from impaired respiration cause both stagnant and hypoxic hypoxia.
This combination is probably the reason for the long recovery period, which may take up to 1 minute following the end of the acceleration. A period of disorientation may also be present for some time after consciousness is regained. During this period of disorientation, the individual is unaware of his/her surroundings—a poor situation for a pilot during low level flight or in an air combat environment.

G-induced loss of consciousness (GLOC) has become a major issue of research and operational interest. The USAFSAM definition of GLOC is “a state of altered perception wherein (one’s) awareness of reality is absent as a result of sudden, critical reduction of cerebral blood circulation caused by increased G forces” (Burton, 1988). Although centrifuge subjects apparently recovered from the total incapacitation period of GLOC in less than 30 seconds, Tripp et al. (2006) found that their cognitive performance scores on a tracking and math task never returned to pre-GLOC levels for an additional 30 seconds after recovery.

The loss of aircraft and aircrew are, of course, a major concern, but every GLOC episode does not result in a loss of an aircraft. Surveys by the USAF and the U.S. Navy indicate that the pilot population in the fighter-attack-trainer is reporting about a 12% - 14% incidence of GLOC (Johanson and Pheeny, 1988). Full function of higher levels of cognitive processing may not be restored for several minutes or more. The brain is not an on-or-off organ and, in fact, comes on in stages. This is most significant to fighter pilots in combat, since statistics show that a majority of the GLOCs occur during defensive maneuvering against airborne threats. These higher cognitive levels are used when the fighter pilot processes information in basic flight maneuvers (or dog fighting) and defensive maneuvers against surface-to-air missiles and anti-aircraft artillery. If a fighter pilot were engaged in actual combat and experienced GLOC, it is quite probable that he/she would not survive, as he/she would be a non-maneuvering target for 12 – 24 seconds on the average and then not a “full up” fighter pilot for possibly several more minutes—very easy target to kill. The work of Whinnery on the occurrence of amnesia in GLOC suggests that at least half of the pilots will not recall an incident of GLOC, so the incidence may be as high as 24%, with occurrence rates on aircraft such as the F-18 of 9.3 incidents per 10,000 flying hours (Johanson and Pheeny, 1988; Whinnery, 1988). This is obviously an issue of major operational concern.

Research on the degree of incapacitation caused by GLOC has indicated that there is an average total (unconsciousness) time of 15 seconds followed by a period of relative incapacitation (confusion and disorientation) of 12 to 15 seconds, resulting in a total time of incapacitation of between 24 and 37 seconds (Whinnery, 1988).

Several studies indicate that most GLOCs are accompanied by involuntary movements (funky chicken, etc.). Although not thoroughly studied, the origin of these movements is thought to be the brain-stem reticular formation. As blood flow to the brain returns after the G load is reduced, differential reperfusion allows the reticular formation to become tonically active, causing involuntary movements of many of the skeletal muscles throughout the body. These involuntary movements last for an average of 4 seconds and cease at the end of the absolute incapacitation period. Involuntary movements generally occur during “deeper” GLOC episodes and can cause unintentional actuation of switches and controls by the pilot. Involuntary movements during GLOC have been responsible for inadvertent throttle reduction and gear lowering at high speeds.

One final point concerning the GLOC phenomenon: one might assume that because the eyes tend to fail at lower G levels than does the brain, aircrew should
receive sufficient warning (problems with vision first) and could avoid GLOC. However, such is not always the case. If the pilot rapidly increases and maintains the applied aircraft G forces above his tolerance level for longer than oxygen reserve in the brain can supply the eyes and functional areas of the brain (the oxygen reserve is the same for both), simultaneous failure of vision and consciousness can occur; the progression from grayout to blackout to GLOC would effectively be a single event. Thus, there will not be any visual warning of the impending GLOC.

Several protective strategies to increase G-tolerance and to prevent GLOC have been explored. These strategies include centrifuge training, weight training, new G suits and G valves, altering the seat back angle in the aircraft, and the use of positive pressure breathing both assisted (with counter-pressure) and unassisted.

Centrifuge training has been received with enthusiasm by all those who have experienced it. Most major North Atlantic Treaty Organization (NATO) air forces, including the USAF, either have centrifuge training programs or are developing them (Gillingham and Fosdick, 1988). These programs usually consist of 1 day of lectures on the physiology of G, a GOR run in the centrifuge, followed by several ROR to a maximum of +9 Gz (Gillingham and Fosdick, 1988).

The work on G-valves for inflating G suits has revolved around the need for faster inflation rates with high flow valves, variable inflation rates that match the G-onset profile, and the use of “smart” microprocessor-controlled systems that may pulse the pressure in the suit to “milk” the venous return from the legs (Van Patten, 1988). The first workable anti-G suit was developed by Franks in Canada during World War II (Gell, 1961). The suit was not acceptable operationally because it was water filled, but it laid the groundwork for what was to follow. The current USAF anti-G suit is the CSU 13-B/P, which has calf, thigh, and abdominal air bladders that can be inflated to a maximum of 10.0 psi. This suit must be individually fitted and provides about +1 Gz of protection. Most of the protection seems to be provided by the abdominal pressure bladder, or the combination of all the bladders, as inflation of the leg bladder alone only provides a 0.2 increase in +Gz tolerance (Banks et al., 2008).

The use of reclined seats to increase G-tolerance has been incorporated into the F-16, which has an inclined seat of about 30°. Most research would suggest that there is no significant increase in G-tolerance until the seat is inclined 45°. Experimental work has shown great potential for this technique, but at present the practical problems of incorporating supinating seats into the cockpit have not yet been solved (Burns, 1988).

Positive pressure breathing with chest counter-pressure has been shown to increase G-tolerance only about +0.5 Gz, but it has had significant fatigue reduction effects (Burns, 1988). This technique, using 60 mmHg of breathing pressure above ambient pressure, has been incorporated into the combined advanced technology enhanced G-ensemble (COMBAT EDGE), which is a combination G suit, chest counter-pressure jerkin, and higher pressure mask.

One other area of “GLOC protection” is the development of an auto recovery system. The current generation of fly-by-wire aircraft can be flown by the mission computers without pilot input. If systems can be developed to correctly identify that a pilot has lost consciousness, the aircraft’s computer can be programmed to recover the aircraft to straight and level flight. Problems with this concept at present are the unequivocal identification of the loss of consciousness (LOC), the provision of appropriate pilot override capability, and pilot acceptance of the machine doing the flying. While the problem of GLOC has not been entirely solved, the technology to solve many of the problems and the training to use technology are at hand.
7.1.13. -G<sub>2</sub> Effects

When you stand on your head, a force of -1 G<sub>2</sub> is experienced, and it becomes uncomfortable after several seconds. During -G<sub>2</sub>, the blood is forced toward the head, causing congestion and swelling of the tissues above the level of the heart. Forces of -2 to -3 G<sub>2</sub> cause extreme congestion in the tissues of the head and neck and produce the sensation that the eyes are bulging. The threshold limit of -G<sub>2</sub> is in the range of -2.5 to -3.0 G for 7 to 10 seconds. Severe headaches and confusion may follow exposure to -G<sub>2</sub> forces, which are of low magnitude and short in duration (several minutes).

Some aircrew members experience red-out under the influence of -G<sub>2</sub> forces. This phenomenon has never been observed in the centrifuge and is difficult to explain physiologically. Red-out may be caused by light shining through the lower eyelid, which is sometimes forced up over the eye by -G<sub>2</sub>. Since the blood vessels within the eyelids would be engorged with blood, light could be seen through the blood-filled eyelids, giving the impression a red curtain had been drawn over the eye. Some researchers believe that the red appearance may be due to increased pressure applied around the retinal area and optic nerve. In early acceleration literature, it was believed retinal damage would occur during -G<sub>2</sub> maneuvers and would lead to blindness. However, aircrew members experiencing red-out during -G<sub>2</sub> acceleration have had no retinal damage.

The increased blood pressures that occur in the head and neck during -G<sub>2</sub> frequently rupture the small, thin-walled venules and capillaries in these areas. The eye is particularly prone to develop hemorrhages, which usually occur between the conjunctiva and the sclera. Vessels within the skull are not subject to ruptures since the cerebrospinal fluid pressure and venous pressure in the cranium are increased proportionately with the arterial blood pressure. This counter-pressure protects the cerebral vessels from rupture.

As the pressure in the vessels of the neck increases during -G<sub>2</sub>, the baroreceptor reflex causes a slowing of the heart and a dilation of the arterioles. This is an attempt to bring the arterial pressure back to normal and is the likely reason for the push-pull effect if followed by a +G<sub>2</sub> exposure. If the G-force is severe, the heart may stop for 10 to 15 seconds. The reduced heart rate causes the arterial pressure to approach the venous pressure. As the pressure gradient across the capillaries declines, cerebral blood flow becomes slower. This may result in cerebral hypoxia when acceleration is prolonged.

No satisfactory methods have been devised for overcoming or reducing the effects of -G<sub>2</sub>. The limit of human tolerance remains in the range of -2.5 to -3.0 G<sub>2</sub> for 7 to 10 seconds. Normally, the endpoint for tolerance is the onset of a severe headache, and recovery follows within 24 hours after exposure. Flyers protect themselves from high -G<sub>2</sub> acceleration by avoiding maneuvers that impose these forces.
References

**Recommended Readings**

**Concepts**
- G-Tolerance
- G-Time Tolerance Curve
- Law of Action and Reaction

**Vocabulary**
- Acceleration
- Acceleration atelectasis
- +G\(_x\) acceleration (forward transverse G)
- -G\(_x\) acceleration (backward transverse G)
- ±G\(_y\) acceleration (right or left lateral G)
- -G\(_z\) acceleration (negative G)
- +G\(_z\) acceleration (positive G)
- L-1
- M-1
7.2. Physiologic Effects of Vibration

Henning E. von Gierke, D.Eng. and William B. Albery, Ph.D.

7.2.1. Definitions

7.2.1.1. Vibration. Vibration is generally defined as the motion of objects relative to a reference position, which is usually the object at rest (von Gierke et al., 1996). Vibration is a series of oscillations of velocity, an action that necessarily involves displacement and acceleration. Vibration is described relative to its effects on man in terms of frequency, intensity (amplitude), direction (with respect to the anatomic axes), and duration of exposure.

7.2.1.2. Amplitude. Amplitude is the maximum displacement from a position of rest. Nonperiodic or random motions are usually expressed in terms of octave band or third-octave band frequency analyses, and the energy in the bands is expressed in terms of root-mean-square (rms) values; peak values or the crest factor (peak/rms) can be important variables for random vibration. For sinusoidal vibration, the rms value is 0.707 the maximum peak value. Vibration amplitudes can be referred to as displacement (in m) or velocity (m/s) but are usually expressed in terms of acceleration (m/s² or G, where 1 G = 9.81 m/s²).

7.2.1.3. Direction. In addition to the three linear vectors outlined below, vibration can also have three rotational degrees-of-freedom known as pitch (rotation around the y axis), roll (rotation around the x axis), and yaw (rotation around the z axis).

7.2.2. Operational Vibration Exposures

Powered automation in helicopters, automobiles, trucks, tanks, and motorcycles exposes operators to increasing acceleration magnitudes with a frequency range extending up to 100 Hz, depending on the roughness of the air/road/terrain and the vehicle speed. Similarly, ship-at-sea motions can extend from extremely low frequencies produced by ocean waves (below 0.1 Hz) to high frequencies in high-speed surface attack ships. Military exposures can extend over several hours per day. Flying, particularly military flying in high-performance combat aircraft, can increase sustained acceleration exposures up to +9 G during combat maneuvering. Exposure times range from a few seconds to up to 1 minute. Potentially higher G levels are limited only by human tolerance considerations. Air combat maneuvers modulate these loads and change their direction with respect to the aircrew (Figure 7.2.2-1). In addition, air turbulence can superimpose vibration and buffeting on these sustained accelerations. Low-altitude, high-speed flight in military operations (as well as air turbulence in commercial and general aviation) causes the most severe vibration exposures. In rotary wing aircraft, vibration frequencies are associated with revolution rates of rotors, gearboxes, and other engine or mechanical parts. The largest amplitude vibrations occur at main rotor blade frequencies and increase as speed loading increases and with increased power of the helicopter propeller system.
As human beings, we are accustomed to living 24 hours a day in the Earth’s gravitational field (defined as a sustained 1 G acceleration). Upon this sustained gravitational field, our motions and activities superimpose accelerations of various amplitudes, frequencies, and durations; walking, running, and other physical activity expose the body to acceleration fluctuations with frequency components ranging from a fraction of a second to several cycles per second (Hz).

Operators and passengers of all types of transportation vehicles, in the air, space, on the ground, or underwater, are exposed to some kind of vibration during some phases of the operation. The oscillations of the vehicle motions around a reference stationary state, at rest, or during constant velocity and/or acceleration are transmitted to the occupants through the supporting seat and floor or through wall vibrations or vibrating handles. This transmission results in motions of the whole human body or body parts. In studying biochemical interaction, it is somewhat artificial to separate body motions into sustained, transient, rotational, or impact acceleration and linear oscillations, although this is driven, in part, by our analytic, experimental, and laboratory stimulation tools. In taking this conventional approach, it is important to keep its limitations in mind and not to forget that vibrations are only a small part of the total mechanical force or motion spectrum. The physical, physiologic, and performance effects to be discussed for the vibration spectrum of interest, from 0.5 Hz to a few hundred hertz, often occur simultaneous with and are modified by the effects of sustained and/or transient accelerations. Low-altitude, high-speed flight in military operations and storm and clear-air turbulence in commercial and general aviation cause the most severe vibration exposures of concern. Their severity depends on the input
gust velocities and acceleration spectra, as well as on the aerodynamic properties and flexibility of the aircraft. In military aircraft with manual or automatic terrain-following control systems, maneuvering loads with maxima between 0.01 and 0.1 Hz are superimposed on the gust-response spectra of the aircraft and crew.

Vibration environments are studied in the laboratory through the use of different types of vibration, or “shake,” tables. These types of acceleration and vibration laboratory simulations are limited with respect to emulating real-world six-degrees-of-freedom motion. There are few real-world operational investigations reported in the literature.

7.2.3. Pathophysiologic and Physiologic Effects of Vibration

There is no specific target area or organ for low-frequency, whole-body vibration, and the mechanical stresses imposed can potentially lead to interference with bodily functions and tissue damage in practically all parts of the body (Dupuis et al., 1986). Fortunately, operational stresses are almost never that high, and vibration exposures remain below injury and interference levels. Severe buffeting in one military aircraft led to a few oscillations best described as repetitive impacts that resulted in spinal fractures. Based on scanty human evidence and animal studies, damage to renal functions and pulmonary hemorrhages are suspected of being the first sign of injury from acute overexposure in the frequency range of maximum abdominal response (4 to 8 Hz). Whole-body vibration of intensities voluntarily tolerated by human subjects up to the limit of severe discomfort or pain has not resulted in demonstrable harm or injury (Lippert, 1963). Minor kidney injuries in truck and tractor drivers have been suspected to be due to vibration exposure of long duration at levels that produce no apparent acute effects, but epidemiologic studies have yet to prove any clear correlation. Similarly, higher incidents of back pain in helicopter pilots and tractor operators have been assumed to be related to the vibration produced by the vehicles; in spite of several studies and plausible arguments, clear dose-response relationships are lacking and difficult to obtain. Modern exposure limits for health and safety reasons are, therefore, primarily based on voluntary tolerance limits, pain thresholds, and experiences with occupational exposures assumed to be safe. Most physiologic effects in the 2 to 12 Hz frequency range are associated with the resonance of the thoracoabdominal viscera. It has been shown to be responsible for the pain occurring in the 1 – 2 Gz (peak) and 2 – 3 Gx ranges, suspected to be caused by the stretching of the perichondrium and periosteum at the chondrosternal and interchondral joint capsules and ligaments. Movement of abdominal viscera in and out of the thoracic cage, in both x- and z-axis excitation, is responsible for the interference of vibration with respiration. It causes the involuntary oscillation of a significant volume of air in and out of the lungs, leading to an increase in minute volume, alveolar ventilation, and oxygen consumption. In some experimental exposures to Gz vibrations, PCO₂ decreased, and clinical signs of hypocapnia were observed, suggesting hyperventilation. Dyspnea results from short exposures to high amplitudes.

Cardiovascular functions change similarly in response to x-axis, and y-axis, and z-axis excitation (Kent et al., 1986). In general, the combined cardiopulmonary response to vibration in the 2 to 12 Hz range resembles the response to exercise. Although the increased muscular effort of bracing against the vibration and psychologic factors may account for some of the response, observance of the same general pattern in anesthetized animals speaks for the stimulation of various mechanoreceptors.
The resonance of the abdominal viscera, with its resulting distortions and stretching, is also responsible for the epigastric or periumbilical discomfort and testicular pain reported at high amplitudes. Headache is frequently associated with exposure to frequencies above 10 Hz. Vibrations that are transmitted to the head directly from the headrest can lead to extremely uncomfortable and disturbing impacts of the head against the headrest. Raising the head away from the headrest and attempting to counteract the forces lead to neck muscle strain, spasm, and soreness. Restraining the head to follow the motion can result in disorientation during the exposure. Rubbing the body surfaces against the seat, backrest, or restraint straps (e.g., "back scrub" in some tractor or vehicle arrangements) can lead to discomfort and skin injuries.

The severe vibration responses and injuries observed in animal experiments have not been reported in humans due to appropriate safety criteria (ANSI, 1979; ANSI, 1983; ISO, 1985). In interpreting animal experiments, it is of utmost importance to consider appropriate scaling laws due to changed body dimensions and resonances; therefore, maximum-effects frequencies are considerably higher in small animals.

7.2.4. The Vibration Syndrome

The only specific vibration-induced disease with well-supported etiologic data is the vibration disease, or "white finger syndrome," caused by habitual occupational exposure over months and years to the vibration of machinery and certain hand tools such as chain saws, chipping hammers, and other pneumatic tools (Taylor et al., 1975).

7.2.5. Effects on Vision

Difficulties in reading instruments and performing visual searches occur when vibrations introduce relative movement of the eye with respect to the target (Benson and Barnes, 1978; Griffin et al., 1978; Figure 7.2.5-1). Although persons and instruments might be excited by the same structural vibrations, their response is completely different, causing different displacements in different frequency ranges. A complex relationship exists between all of the relevant parameters, such as vibration frequency, amplitude and direction, viewing distance, illumination, contrast, and the shape of the viewed object (Griffin et al., 1978). Large effects on the resolution of visual detail occur under z-axis, whole-body vibration and for y-axis and z-axis vibration of viewed objects. The main difference between the object versus the subject vibration is the compensating ocular reflexes mediated by the vestibular system (vestibulo-ocular reflex), which enable the eye to compensate for body and head motions, thereby fixating the gaze on the target (Griffin et al., 1978). Although effectiveness of the vestibulo-ocular reflex drops off about 1 Hz, the reflex has been shown to affect results up to 8 Hz. Analysis and prediction of visual capability are further complicated by the fact that translational body motion results not only in translational but also rotational head movements. The latter influences passive eye movement as well as vestibular feedback. The same compensatory reflexes have been shown to remarkably degrade visibility on head-mounted or helmet-mounted displays under vibration when the display moves with the head (Griffin et al., 1978). Although mechanical eye resonances have been investigated in several studies up to 90 Hz, their influence on vision is apparently of secondary importance, and no sharp resonance phenomena as a function of frequency have been observed.
Unfortunately, the large number of test results cannot yet be presented in uniform curves allowing the prediction of visual decrement. The large number of variables prevents generalization of the results. These variables include, for example, small changes in subject posture and restraint, affecting translational and rotational head responses, and large intersubject intervariability among others.

For $G_x \pm G_y$, $G_y$, and $G_z$ vibrations and for $G_x \pm G_x$ vibrations, the largest effects were found in the 11 to 15 Hz range. Decoupling the head from the headrest improved capability in this frequency range, whereas head restraints generally reduced reading errors at 6 Hz and below. The type of helmet and restraint, however, is crucial for these experiments. All of these results underline the previously stated conclusion that because of the complexity and large number of variables, important vehicle performance requirements should be tested for each specific configuration in realistic simulations.

### 7.2.6. Vibrations Producing Motion Sickness

Although the symptoms and causes of and therapeutic measures for motion sickness are discussed in another chapter, the frequency and amplitude range of vibrations producing the discomfort or acute distress associated with motion sickness will be mentioned briefly in this chapter. The frequency range for vertical ($z$-axis) vibration leading to this disability extends downwards from 1 Hz on, i.e., it starts right below the frequency range so far discussed. Because vibration-caused motion sickness can occur in most transportation systems, and controlling vibrations in one frequency range can easily magnify the amplitudes in another frequency range, design guidance with respect to motion sickness will be mentioned here briefly. The absolute levels for severe discomfort after 30-minute, 2-hour, and 8-hour exposures are very tentative and open to many variables. The boundaries as presented apply to infrequent, inexperienced travelers and are assumed to cover approximately 95% of such a
population (5% probably never adapt to motion below 1 Hz). Civil and military vehicle operators and many travelers clearly have much higher discomfort and tolerance thresholds due to habituation and selection.

7.2.7. **Sustained Acceleration Combined with Vibration**

Limited experimental evidence suggests that accelerations and vibrations are not synergistic. On the contrary, it appears as if vibration tolerance at 11 Hz $+3 \, \text{G}_x$ was increased by the simultaneous application of $+3.8 \, \text{G}_x$. This finding can be theoretically explained by the inertial preload effect of the sustained acceleration, which at the same time has a static preload or restraining effect on the subject. On the other hand, it can be argued that the vibrations partially alleviate or counteract the circulatory and respiratory manifestations of sustained acceleration.

Whole body vibration exposure can result in various pathologic and physiologic effects located in different body regions, which are determined by the exposure frequency. For most operational exposures, however, effects on task performance and interference with activities were found to be of primary concern. The only well-documented vibration-induced disease is the “white finger syndrome” caused by habitual exposure to vibrating hand tools. The standards for safety, performance capability, and comfort for whole-body vibration (1 to 80 Hz) and for risk assessment of hand-tool vibration (8 to 1000 Hz) were recommended as practical guidelines for the assessment of operational vibration exposure. The guidance for the evaluation of vibration in air, spacecraft, and other transportation vehicles was supplemented by weighting curves (0.1 to 0.63 Hz) to estimate the incidence of motion sickness in vertical vibrating motions. The information and references presented should be adequate for the evaluation of existing and future aerospace vibration problems (CHABA, 1987).

Impact forces of less than a second’s duration that occur under crash or emergency aircraft escape conditions are not considered in this chapter. Sustained acceleration environments are experimentally obtained on human centrifuges, where human performance during simulated aerospace missions has been studied extensively.

To fully capture the influence of acceleration or vibration on mission effectiveness, it is essential to simulate the real-world environment as closely as possible and to know the task requirements, the time of day during exposure, duration of exposure, repetition rates, and timing with respect to other stressors. These factors should be kept in mind during the discussion that follows.
References

Recommended Readings

Vocabulary
Vibration
Amplitude
7.3. Spatial Disorientation

William Ercoline, Lt Col, USAF (Ret); Eric Geiselman; and Andrew McKinley

7.3.1. Introduction

Despite training and ongoing research efforts, spatial disorientation (SD or SDO) remains a significant factor in the death of aircrew and military members. Information presented in the following chapter provides an operational overview of one of the most serious but often ignored causes of human error associated with aircraft flight.

The failure of the pilot to correctly perceive the spatial orientation of his or her aircraft has always been a problem; unfortunately, it was never realized as such until 1927 (Ocker and Crane, 1932). Although more than 80 years have passed since first being recognized as a pilot killer, the problem has not been eliminated. At the annual Aerospace Medical Association (AsMA) conference, accident rates are presented each year by the military branches in attendance. The statistics are alarming, and SD continues to account for several of the major Class A mishaps each and every year.

There are several known countermeasures, but the one most promising is the instrument crosscheck. And although several technologies exist that are capable of making the instrument crosscheck more efficient for the pilot, such as audio and tactile feedback, few have been employed in modern military aircraft beyond the use of an auditory alerting system. The most recent technology to be employed in military aircraft is an automatic ground collision avoidance system or Auto GCAS. Some consider this a countermeasure for SD. This system is being installed in some F-16s and is planned for other fighter aircraft. It prevents the aircraft from flying into the ground (when the wheels are retracted). This will no doubt prevent the unexpected Controlled Flight Into Terrain (CFIT) accident, which often results from SD, but it will not prevent the pilot from experiencing SD. Because this technology is now being installed on USAF fighter aircraft, and because a couple aircraft have already been considered as being saved because of Auto GCAS, we believe it is instructional to know the technology exists and it is being used. Even though it may not prevent the pilot from experiencing SD, it will prevent the end result of an unexpected collision with the ground. The outcome will be a reduction in SD related Class A mishaps (a good thing).

Before beginning a detailed analysis of SD causes and consequences, the following paragraph penned by Dr. Kent Gillingham, a distinguished USAF SD scientist and researcher, is provided that discusses the physiologic origin of human SD in aviation.

The evolution of humans saw us develop over millions of years as an aquatic, terrestrial, and even arboreal creature, but never an aerial one. During this development humans were subjected to many different varieties of transient motions, but not to relatively sustained linear and angular accelerations commonly experienced in aviation. As a result, humans acquired sensory systems well suited to maneuvering under our own power on the surface of the Earth but poorly suited for flying. Even birds, whose primary mode of locomotion is flying, are unable to maintain spatial orientation and safe flight when deprived of vision by fog or clouds. Only bats seem to have developed the ability to fly without vision by replacing vision with auditory echolocation. Considering our phylogenetic heritage, it should come as no surprise that our sudden entry into the aerial
environment resulted in a mismatch between the orientational demands of the new environment and our innate ability to orient. The manifestation of this mismatch is spatial disorientation (Gillingham, 1993).

While Dr. Gillingham’s rationale for spatial disorientation causes should not be surprising, the SD phenomenon involves a complex relationship between human physiology and perception (both visual and somatosensory) that can be challenging to fully alleviate and to fully appreciate. However, it is important for all individuals in aerospace careers to gain an appreciation of the definitions, illusions, and known countermeasures of SD. Such knowledge contributes to future SD countermeasures, the identification of SD risks, and the development of habits that can aid in the prevention of SD. This chapter is primarily devoted to the causes of SD and the various SD illusions that have been reported over the many years of man’s attempt to conquer his aerial environment. It is recommended that should the reader want more insight into the physiology and psychology associated with SD, he or she should read any of the aerospace medicine books currently on the market or the unique text titled “Spatial Disorientation in Aviation” published by the American Institute of Aeronautics and Astronautics (Previc and Ercoline, 2004).

7.3.2. Definitions

Let us take a stepwise approach to the more complex term of SD. We’ll start with the fundamental definition used throughout the chapter and then develop the more commonly known definition used throughout the research community.

7.3.2.1. Illusion. An illusion is a false impression or misconception with respect to actual conditions, better known as reality. Visual illusions are perhaps the most prominent and deceptive causes of SD. Think of all the times you have seen something that looked to be true but upon closer inspection it turned out to be false, e.g., a mirage, a magic trick, the perception of motion. Amusement parks are loaded with these types of illusions, and artists try to capture many visual illusions in their works of art. Figure 7.3.2.1-1 displays a visual illusion where subtle shadows actually mask a potential hill. Just by changing the angle of incident light the depiction changes to one of more contrast. In tile A little contrast is seen, but with the light coming in from the side as in tile B, the hills are now visible.

![Figure 7.3.2.1-1. Visual Illusion of a Hill Hidden by the Angle of Incident Light](image)

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Such visual illusions abound in the world and create significant problems during flight. Subtle slopes of cloud decks can create an illusion of an aircraft bank; narrow runways may appear longer due to the size constancy illusion, etc. It is important to note these types of illusions contribute to SD events. Visual ones are not the only ones. We use this type of illusion for the introduction to the world of SD since it is by far the most common. Our mind often struggles to correctly interpret the things we sense, and interpretation can be influenced by our prior experiences and our sensory inadequacies. Some researchers have further defined subcategories of these illusions, but for the sake of simplicity, we’ll limit our presentation to the ones that primarily apply to aviation.

### 7.3.2.2. Orientation

To get a little closer to the accepted definition of SD, we must define one more term—spatial orientation in aviation. Spatial orientation in the worldly environment is simply the ability of the pilot to correctly perceive the position of an object and the direction in which it aligns with respect to a standard coordinate plane—in this case the surface of the Earth. For example, let's consider a parked car. You might recognize characteristic features such as the taillights, bumper, etc. indicating that the vehicle is facing away from you, and you are oriented ‘behind’ the car. On the other hand, perception of self-orientation often involves both visual and somatosensory input. The visual system detects the orientation of surrounding objects, which, when combined with experience (memory), presents a constructed representation of the body's orientation with respect to the object's frame of reference. Hence, self-orientation is referenced to other objects in the surrounding environment, and if vision was the only mechanism to determine spatial orientation, it would be a simple interpretation. However, it gets complicated because the brain also receives proprioception and vestibular cues, which inform the individual if he/she is standing, lying, inverted, etc. These cues can be misleading in the complex and dynamic flight environment, and even more so in a visually degraded environment. Our ability to orient in space is not a trivial task. With this simplistic and basic concept of spatial orientation, we are now ready to deal with the definition of SD.

### 7.3.2.3. Spatial Disorientation

If orientation is about knowing one’s relationship to a particular place in space, then SD deals with misperceptions of spatial cues leading to a loss of orientation in that given space. In the aviation environment, self-orientation actually refers to the orientation of the aircraft due to the fact that the pilot and aircraft are coupled. Often, the Earth’s surface is used as a stable coordinate reference. However, other objects can be substituted, such as another aircraft, when terrestrial references are unavailable. This is how military pilots orient themselves while flying other than lead in a formation. For a more precise definition, we quote the definition penned by Allen Benson in 1974 when he wrote the following: “Spatial disorientation is a state characterized by an erroneous sense of one's position, motion and/or attitude with respect to the plane of the Earth's surface (or other object).”

Although this definition first remained controversial for many years, it is now accepted throughout the military community as the best definition. It provides a foundation to study the topic and to objectively define the degree of SD being experienced. Essentially, it encompasses all the possible positions, velocities, and accelerations both in translation and rotation for an aircraft within a given Cartesian coordinate system (an orthogonal, three-axis coordinate system) using the Earth’s surface as a method of measuring the vertical displacement. It also allows for an understanding of the causes of SD from a sensory perspective.
It is important to note before going further that SD involves not only correct alignment with a standard coordinate system but also spatial location within the geographical boundaries of that three-dimensional (3-D) system. Geographic disorientation, on the other hand, involves only the misperception of sensory cues (usually visual) in the 2-D sense. When a person loses his or her position while on land, it is often referred to as "being lost." It is a state characterized by an erroneous locational percept but only in the two coordinates that are used to define the surface of the Earth and not the vertical component. This is known as geographic disorientation, and it is analogous to being lost when driving a car or on a hike.

7.3.2.4. Vertigo. Vertigo is usually considered a clinical term used to describe a false sense of motion or, in the more classic case, spinning. There are several types of vertigo. This chapter will mention only one—pilot vertigo or pilot’s vertigo. Pilot’s vertigo is an illusion originally defined as a sensation of rotation occurring during flight. Most flyers refer to all forms of spatial disorientation, with or without subjective rotation, as pilot’s vertigo. Pilot’s vertigo and SD were for many years considered synonymous. It wasn’t until approximately 1990 when the term SD was broadened to include those situations described by pilots as “misorientation” or “unorientation.” Getting rid of these terms and developing a commonly held definition of SD helped focus research in the emerging science, which is now known as SD countermeasures. It also permitted critical issues associated with the high workloads in military cockpits to be addressed—the loss of situational awareness. Perhaps most important was physiologic research leading to a comprehensive understanding of human equilibrium and orientation perception.

7.3.3. The Organs of Equilibrium

Three sensory systems are especially important in helping the human maintain his or her equilibrium, orientation, and balance: the visual system, the vestibular system of the inner ear, and the proprioceptive system as shown in Figure 7.3.3-1. Equilibrium and spatial orientation are normally maintained by the combined functioning of these senses. For most normal physical activities on the ground, these three organs of equilibrium are effective, and the brain interprets their input accurately. However, when man is subjected to the complex motions and forces involved in flight, the organs of equilibrium become less effective in providing meaningful input to the brain. Such errors or misinterpretations are the foundation upon which illusions occur, and in a more global sense, spatial disorientation is often the result.
Figure 7.3.3-1. Integration of the Sensory Systems Predominantly Used for Spatial Orientation

Of the three sensory systems, vision is by far the most important and reliable in flying. The vestibular and the proprioceptive senses are susceptible to producing erroneous or incorrect cues to the pilot due to accelerations varying in both magnitude and direction. However, this was not always common knowledge. It took many years and numerous lives lost to SD to recognize the shortcomings of these two senses in the flight environment. Because the cues from the vestibular and proprioceptive systems can be misleading, it is paramount for pilots to understand the underlying physiology and function. With such knowledge, pilots are more capable of recognizing errors caused by these systems, thereby leading to fewer incidents and accidents.

NOTE: Individual systems are discussed in more detail in previous chapters.

7.3.3.1. The Visual System. Vision is the most critical of the three orientation senses (See Section 1.4 for a complete discussion of the principles and problems of vision). However, the visual system has significant limitations. At higher altitudes, even in clear visibility conditions, textural density in the environment is not sufficient for the human visual system to be relied upon for accurate judgment of absolute values or small changes of basic orientation dimensions such as altitude, airspeed, or heading. Proper orientation to these dimensions can only be maintained by reference to the appropriate flight instruments. In instrument meteorological conditions (IMC), known as being "in the weather," visual reference of the environment outside the aircraft is further obscured. Features such as the natural horizon and terrain, which are otherwise used to maintain overall spatial orientation, are not likely available. When the pilot cannot see these features, the pilot must look inside the cockpit and transition to the flight instruments to maintain awareness of the aircraft’s state. These instruments, as mentioned earlier, are the only known countermeasure for SD. Be aware, it is extremely difficult to trust the readings on the instruments when it is already “believed” that the visual information from outside the aircraft is accurate. Reliance on instrument
provided information rather than input of the other senses demands discipline and practice. Use of flight instruments to maintain spatial orientation requires training, proficiency, and currency.

In flight, the integration of the eyes and the flight instruments is absolutely critical for safe flight. The eyes must constantly scan the instruments for any unexpected changes that might occur because of turbulence, distraction, inattention—all causes of spatial disorientation. This integration is known to pilots as the “instrument crosscheck.” ANYTHING that interferes with the pilot’s instrument crosscheck can lead the pilot to SD.

7.3.3.2. **The Vestibular Apparatus.** The inner ear contains the vestibular apparatus, the motion-and-gravity-detecting sense organ. It is located in the temporal bone on each side of the head. Each vestibular apparatus consists of two distinct structures, vestibule proper and the semicircular canals (Figure 7.3.3.2-1). Although these organs provide important cues for basic orientation on the ground, they often provide misleading information, or an entire lapse of critical information, during flight. It behooves the pilot to have a basic understanding of how this occurs (See Section 1.5 for a complete discussion of vestibular physiology).

![Figure 7.3.3.2-1. Details of the Inner Ear and the Vestibular System](image)

**7.3.3.2.1. The Otolith Organs.** The otolith organs, the utricle and saccule, are small sacs located in the vestibule. Lining both the bottom of the utricle and the medial wall of the saccule is a patch of cells called a macula. Sensory hairs project from each macula into overlying gelatinous membrane containing chalk-like crystals called otoliths. The otolith organs respond to changes in motion known as accelerations. Changes in head position augment the direction of the acceleration due to the Earth’s gravitational pull. This causes the otolithic membrane to shift position on the macula. Because the sensory hairs in the macula are coupled to this membrane, the shift causes the hairs to bend and send signals to the brain indicating the change in head position. The signal is in the form of the cellular action potential firing rate. When
the head is upright in a static environment, the cells are firing at a nominal “resting” frequency. As the macular hairs bend, the firing rate changes to indicate the new situation (see Figure 7.3.3.2.1-1). It is this same shearing response that is so predictable on the ground by alerting a person of head position relative to the gravitational vector, yet the same basic mechanism can be very troubling when it happens in flight. Shearing of the otolith in flight is often the result of a net vector containing both the Earth’s gravitational pull and the acceleration generated by the motion of the aircraft.

![Displacement of the Cilia](image)

**Figure 7.3.3.2.1-1. Displacement of the Cilia**

Linear accelerations also stimulate the otolith organs, since inertial forces resulting from linear accelerations cannot be distinguished physically from the force of gravity. A forward acceleration, for example, results in backward displacement of the otolithic membranes, which can create an illusion of backward tilt. The misperception is often enhanced if adequate visual references are not available to correct the mental image generated by the vestibular input.

7.3.3.2.2. **The Semicircular Canals.** The semicircular canals, like the otoliths, respond to accelerations. The difference is that the semicircular canals respond primarily to angular accelerations rather than linear accelerations. There are three semicircular canals structured as shown in Figure 7.3.3.2.2-1 and are situated in three roughly mutual perpendicular planes. They are filled with a fluid called endolymph, which is put into motion by the inertial torque resulting from angular acceleration in the plane of the canal. Motion of the fluid exerts a force upon a gelatinous structure called the cupula, located in the ampulla of the canal. The translation of the fluid causes hair cells situated beneath the cupula to bend, thereby stimulating the vestibular nerve. The impulses are transmitted to the brain, where they are interpreted as rotation of the head.
Since each canal lies in a different plane, they can sense rotation in three dimensions. However, because of gyroscopic physical principles, the resultant signal may produce an unsettling sensation in a dynamic acceleration environment. This interaction can result in a vestibular illusion or, in some cases, a response known as motion sickness (See Section 7.4 for more information on Motion Sickness).

Let’s go through a simple example of how the misperception is generated. When a semicircular canal is put into motion; for example as one rapidly generates angular acceleration to the left, the fluid within the semicircular canal lags behind the accelerated canal walls and bends the hairs in an opposite direction, i.e., to the right. Because the brain has habituated to simple positional changes that occur in a static environment, it tends to interpret the movement of the hairs to the right as angular displacement to the left. If the turn continues at a constant rate for several seconds or longer, the motion of the fluid in the canals catches up with the canal walls and the hairs are no longer bent. The brain receives the completely false impression that turning has stopped although rotation to the left continues. If the visual information is not available to correct the misperception, then the stage is now set for a dramatic and sometimes frightening illusion.

With a sudden stop, the canal walls arrest rotation, although the canal fluid continues to flow for a short period of time due to its inertia forcing the hairs to now bend to the left. This gives the brain a false impression of movement to the right (opposite to the original direction). Given the fact that the illusion can be profound in the absence of other information to correct the misperception, the pilot is naturally driven to counteract the illusion by rotating the aircraft in the original direction—back to the left. Without an instrument crosscheck or visual information to correct the situation, the pilot would most likely continue the left rotation. Historically, once this illusion was understood it wasn’t long before researchers called the in-flight consequence the “graveyard spin.” The pilot would recover from the spin but soon reenter the spin in the
original direction, while attempting to negate the erroneous spin sensation. The first word in the illusion name happened to reflect the usual end point of the SD event.

This demonstration was first discovered in 1927 by Capt Bill Ocker and Capt (Dr.) David Meyers. It remains today as the best demonstration of the inadequacies associated with the vestibular system and SD. There are other systems such as the proprioceptive system that come into play, but the basic understanding remains within this simple demonstration. This finding is arguably the most significant scientific discovery related to aviation in the 20th century.

### 7.3.3.3. The Proprioceptive System

Proprioception includes the vestibular, subcutaneous, and kinesthetic sensors, which enable an individual to determine body position and its movement in space. The subcutaneous pressure receptors and kinesthetic muscle activity sensors are important inputs to the perception of body orientation (Fig. 7.3.3.3-1).

**Figure 7.3.3.3-1. Proprioceptive Receptors Found in Various Parts of the Body**

The kinesthetic sense generally does not orient individuals to their surroundings but informs them of the relative motion and relative position of their body parts. These include feedback of muscle stretch, joint position, tendon tension, etc. to the cerebellum. The subcutaneous pressure receptors are capable of informing individuals of their position in relation to the Earth, if they are in contact with some earthbound object. These receptors are stimulated by the pressures created on the buttocks when sitting, on the feet when standing, or on the back when lying down. These sensations provide the “seat-of-the-pants” sense often referred to in flying. While early aviators believed they could determine their aircraft’s position by seat-of-the-pants sensations, they were often fatally mistaken. Inertial forces caused by multi-axis accelerations encountered in the flying environment can cause sensations that have little correlation with the true orientation of the aircraft. For example, rapid linear accelerations may provide a
sensation of aircraft pitch up. The bottom line is that the vestibular and proprioceptive cues that provide stable and reliable information on the ground become erratic and misleading in flight. The pilot must learn to suppress these natural cues and trust the instruments displayed in the cockpit.

As you may have surmised, within the broad term of SD, there are two major kinds of illusions—vestibular and visual. Of course nothing is ever definitive, so there are several illusions that result from a mix of both the visual and vestibular sensory responses. The following sections touch upon many, but not all, of the known SD illusions.

7.3.4. Vestibular Illusions

The human vestibular system, which has evolved to work in ground-based environments, is prone to providing the brain with misleading information during flight. This misinformation produces illusionary perceptions. As described previously, the response of the semicircular canals may be inappropriate during angular acceleration prevalent in flight. The otolith organs cannot distinguish between the force of gravity and linear accelerations. Some vestibular illusions are commonly experienced, while the most dangerous are less frequent. These vestibular responses can be further subdivided into illusions related to semicircular canal stimulation and illusions related to otolith organ stimulation.

7.3.4.1. Somatogyral Illusions. Somatogyral illusions are vestibular-induced illusions generated from rotations of the body. These rotations stimulate the semicircular canals due to either angular velocity or angular acceleration, and the result can be one of several well-documented SD illusions.

7.3.4.2. Nystagmus. Angular motion may cause the eyes to pulsate in the direction of rotation in what is known as the vestibular-ocular-reflex (VOR). Nystagmus is the term used to describe a sweeping motion of the eyes in a direction that is opposite of an imposed angular acceleration and followed by a quick return of the eyes to the center position. This occurs with oscillatory repetition of the sweep and return, resulting in an apparent jerking of the eyes in the direction of the angular motion. Nystagmus may appear when rotational eye movement exceeds 3 – 5 deg/second. The pilot’s head movement coupled with the aircraft movement can greatly exacerbate this situation. Natural head movements are of high frequency; however, the visual system, encumbered by relatively slow retinal processing, cannot produce eye movements that stabilize the retinal image. When such eye motion occurs upon cessation of rotary motion, it is called post-rotatory nystagmus. Nystagmus can be horizontal or vertical depending on the plane in which the angular acceleration acts and the particular canals stimulated.

7.3.4.3. The Leans. The leans illusion is the most common vestibular illusion derived from stimulation of the semicircular canals. These organs do not perceive rotary motion below a certain threshold. The threshold, under ideal conditions, is described by the product of angular acceleration and the time in which the acceleration occurs. This product must be equal to, or greater than, approximately 2 deg/s, a constant known as Mulner’s Constant. The leans results from unperceived, subthreshold angular acceleration followed by an abrupt and perceptible angular
acceleration in the opposite direction. For example, a slow, inadvertent rate of roll may be introduced during flight that is less than the vestibular threshold, thereby making it impossible for the pilot to sense without paying careful attention to his/her instruments. Once the bank error is observed on the attitude indicator, the pilot often initiates a corrective roll in the opposite direction that has an acceleration exceeding the threshold for vestibular stimulation. Because the original roll was not perceived, the pilot feels that he has rolled into a bank in the opposite direction of the original unnoticed roll, even though the instruments indicate straight and level. Pilots in this situation will do one of two things: 1) respond inappropriately to the spatial disorientation by rolling the aircraft in the direction of the original roll, until their vestibular perception indicates the aircraft is straight and level, or 2) trust the attitude indicator and regain control of aircraft attitude, even though they may retain their false vestibular perception of bank. This common illusion is presented in Figure 7.3.4.3-1. Note: This post-rotatory roll illusion has been called the Gillingham Illusions in honor of the USAF’s most noted SD researcher.

![Diagram showing motion that could lead to the “Leans” illusion](image)

Figure 7.3.4.3-1. Motion that Could Lead to the “Leans”

There is also a visual analogue of the leans illusion. In this case the pilot is responding to the visual information and not necessarily to his or her vestibular signal. Visual leans are most commonly associated with false horizons, such as a sloping cloud deck, sloping terrain, or a shoreline at night. In these situations the pilot tends to orient his or her body and/or aircraft to align with the perceived horizon.

7.3.4.4. Graveyard Spin. The somatogyral vestibular illusion, known as the graveyard spin and described earlier, is appropriately named as it is particularly profound and difficult to eliminate once initiated. The illusion often begins after a pilot enters and remains in a spin for several seconds. During the sustained spin, the semicircular canals equilibrate to the rotary motion, thereby eliminating the perception of
continual motion. As the pilot recovers from the spin, he/she undergoes an angular deceleration that is sensed by the semicircular canals. The central nervous system interprets this sensation as a spin in the opposite direction. Although the pilot’s instruments indicate a recovery from the spin, the vestibular system continues to produce a strong sensation of being in a spin that is difficult to ignore. If deprived of external visual references, the pilot is often tempted to make control corrections against the falsely perceived spin. Upon doing this, he or she will reenter a spin in the same direction as the original angular motion. The graveyard spin is illustrated in Figure 7.3.4.4-1. A proper cross-reference of flight instruments and proficiency in instrument flying will often prevent this situation.

Figure 7.3.4.4-1. The Real Motion and Perceived Motion (shown by dashes) of the Classic Graveyard Spin

7.3.4.5. **Graveyard Spiral.** The graveyard spiral may sound like it is very similar to the graveyard spin, but the two are completely different illusions. The former is purely a false sense of rotation (yaw), while the latter is the result of a misperception of the aircraft’s bank brought about by a variety of variables, some of which may not be related directly to the vestibular system. Rather than a spin, the illusion is brought about by a slow, prolonged turn (Fig. 7.3.4.5-1). Even if the pilot senses the initial bank of the aircraft, after a period of several seconds, the pilot loses the sensation of being in a turn and believes the aircraft to be level. When reestablishing a new bank, the pilot places the aircraft in a greater than expected bank,
does not compensate with increased back pressure on the stick, and continues a spiraling motion toward the ground. It has been shown that the pilot may easily fail to detect bank even if the bank has reached a magnitude of 90 degrees or more. In addition, if not properly trained on an instrument crosscheck, the novice pilot may increase back pressure on the stick to compensate for the loss of lift but fail to do so adequately and not realize the aircraft is descending. Especially during a steep bank, this kind of stick input has been shown to tighten the descending turn, and the result is a descending corkscrew flight path.

**Figure 7.3.4.5-1. An Illustration of the Actual Path and the Perceived Path of the Graveyard Spiral**

The direct cause of the loss of turning sensation remains unknown, although it is likely the result of the habituation of the semicircular canals to the continual turn. Another possibility is that the pilot is sufficiently distracted such that he/she fails to attend to the sensation of turning. Alternatively, the triggering mechanism may result from purely visual illusions such as a slanted cloud bank creating a false horizon (hence a false sense of bank). Regardless, if an inexperienced pilot neglects to crosscheck the attitude indicator and fails to realize the true bank of the aircraft, it is likely that he/she may induce a bank to make the aircraft feel level and begin an unrecognized descent. Should this go unchecked, it can quickly develop into a dangerous condition while the pilot remains unaware of the new spatial orientation of the aircraft. This spiraling descent can result in ground impact, hence the name graveyard spiral, just like its kissing cousin the graveyard spin. An adequate visual crosscheck of the instruments and proficiency in recognizing this SD illusion may likely prevent this situation from deteriorating into such a mishap.

**7.3.4.6. Coriolis Illusion.** The Coriolis illusion (sometimes called cross coupling) is probably the most dangerous of the vestibular illusions because it can be both subtle and overwhelming to the pilot (Fig. 7.3.4.6-1). It is particularly dangerous at low altitudes and occurs most frequently when a pilot is engaged in a constant-rate turn, such as a penetration turn, holding pattern, or overhead traffic pattern at night. In
such situations, the fluid within the semicircular canals will become stable and in line with the constant angular rotation (assuming the pilot’s head does not turn significantly). Any subsequent quick, large amplitude head movements in any plane other than the turn of the aircraft cause the semicircular canal to be aligned with a different coordinate plane. This causes the pilot to experience the illusion of moving in a plane of rotation in which no real angular motion exists. In attempting to correct the new perceived rotation, the pilot could lose control of his/her aircraft, potentially leading to a serious mishap. However, turns capable of causing Coriolis are not common in most fixed wing aircraft and are traditionally more prevalent in rotary wing aircraft or aircraft with vectored thrust.

![Figure 7.3.4-1. The Motion and Cross Coupling Associated with the Coriolis Illusion](image)

To prevent this illusion from materializing, pilots should avoid sudden, extreme head movements, especially while making turns. To avoid succumbing to the effects of the illusion, pilots should consult their attitude indicators prior to any reflexive “corrective” responses.

It should be noted that the extreme case was explained above; and although this is not common, the more subtle case may always be there. The pilot must beware of conditions leading to Coriolis. Even if the sensation is subtle, the end result may be an unnoticed stick input, which could lead to a necessary power and/or pitch adjustment.

7.3.4.7. **Oculogyral Illusion.** The term “oculogyral illusion” is used to describe the apparent relative motion of an object in an individual’s foveal field-of-view while both the person and object are subjected to angular acceleration. Oculogyral illusions can be observed in the cockpit during Coriolis stimulation and spins. The oculogyral illusion is caused by semicircular canal stimulation and results in an apparent motion of the instrument panel. The apparent motion may be described as follows.

If a pilot were to stare at an instrument while being rotated to the left, the oculogyral illusion makes the instrument appear to move rapidly to the left. It gradually becomes motionless and then may appear to slowly move to the right, although it may appear motionless with a prolonged constant angular velocity. If the left rotation is suddenly stopped, the instrument appears to move rapidly toward the right and may not come to rest for 30 – 40 seconds.
The direction of the apparent movement of the target is typically in the direction of the angular acceleration in line with the semicircular canals. The magnitude of an oculogyral illusion varies based on the rate of angular acceleration, position of the head, illumination of the target and background, acoustic noise, and the experience of the individual.

In daylight, the apparent motion of a target is seen only after a relatively high rate of angular acceleration. Strong illusions can be initiated with small angular accelerations in the darkness. Therefore, the pilot would be expected to experience the oculogyral illusion as a result of the small angular accelerations experienced while flying at night. Care must be exercised and a constant update of the instrument crosscheck is necessary to prevent this illusion from becoming overwhelming.

7.3.4.8. Somatogravic Illusions. Somatogravic illusions, like their somatogyral counterparts, are generated due to accelerations. However, these illusions are caused by linear accelerations rather than rotational. The otolith organs are stimulated by the net gravito-inertial force, which is the vector summation of linear accelerations due to inertial reactions and gravity itself. For example, with the head upright, the gravitational force of +1 G_z is acting on the gelatinous otolith containing membranes in the utricles and saccules. The resulting pattern of nervous impulses causes the brain to perceive the upright position of the head. If the person is now accelerated such that the forward acceleration is +1 G_x, the net force acting on the head (and thus the otolith) is +1.414 G, acting at a 45 degree angle to the vertical. Because the resulting acceleration vector can be produced by a multitude of vector pairs, the source of the shearing force on the otolith can be ambiguous, especially when exterior visual cues are absent and instrument crosschecks have been omitted.

The absolute thresholds of otolith organ function are known, although there is considerable variation among individuals. A change of only 1.5 degree gravitational force vector acting on the otoliths can be perceived under ideal conditions. However, a change in the magnitude of gravito-inertial force of +0.01 G has been perceived by experimental subjects. False sensations arise from the stimulation of the otolith organs primarily because these organs are unable to distinguish between the Earth's gravity and superimposed inertial forces resulting from linear accelerations.

7.3.4.9. Oculogravic Illusion. The oculogravic illusion can be described as the apparent movement of an object in the visual field resulting from the linear accelerations placed upon the body. It is thought to result from the inertial response of the eyeball to these forces. The term "oculogravic illusion" has, through erroneous usage, come to mean the false sensation of change of body position that occurs when an inertial force combines with the force of gravity to produce a resultant force. The oculogravic illusion should not be confused with the somatogravic illusion. The former is the result of forces on the eye and the subsequent change in the visual field, while the latter is the result of forces on the vestibular system and the sensations produced from those forces.

7.3.4.10. Pitch-Up or Pitch-Down Illusion. This illusion is synonymous with the somatogravic illusion. However, to keep the generic name of a certain group of illusions different from its parts, this particular illusion is expressed as either a false sense aircraft pitch (up or down, depending upon the net gravito-inertial force). Forward acceleration without the ability to see external visual cues and an
inadequate instrument crosscheck would result in a sensation that the aircraft is pitching up significantly. The intuitive (although wrong) corrective action is to pitch down, which becomes a severe problem during low-visibility takeoffs. It is suspected that a number of pilots have been lost because they experienced the pitch-up illusion shortly after takeoff at night over unlighted terrain or water and on missed approaches. One-tenth of one G of acceleration is capable of producing a sensation of about a 6 degree of false pitch. Figure 7.3.4.10-1 illustrates this phenomenon.

Figure 7.3.4.10-1. An Illustration of the Pitch-Up Illusion Resulting from Acceleration (the perceived orientation is shown in the bubble)

Furthermore, the illusion of a nose-down attitude occurs during decelerations caused by extending speed brakes or reducing forward velocity such as rapid reduction of the throttles. This is often reported as a climb sensation during sudden changes in airspeed during low-level flights.

7.3.4.11. Inversion Illusion. Another variation of the somatogravic illusion is called the inversion illusion. It occurs during an abrupt pushover following a climb. Under these circumstances, the sudden aircraft attitude change and ensuing decrease in gravitational force acting downward on the otolith organs and seat-of-the-pants cause a sensation of the aircraft pitching upward. Instinct causes the pilot to try to correct for this illusory attitude by pushing the nose of the aircraft downward, which only intensifies the sensation and the illusion. After several seconds of this stick input, the pilot may perceive an inverted spatial orientation.

7.3.4.12. Elevator Illusion. The elevator illusion is caused by a change in acceleration acting in line with Earth’s gravity, which stimulates the otolith organs. The sensation is analogous to the increase in acceleration experienced when riding an elevator from a lower floor to a higher floor (e.g. a compressional force). Essentially, the inertial force is detected from below initially, followed by a sudden decrease when arriving at the selected floor. In an aircraft, the feel of an upward linear acceleration can
occur while the aircraft is actually level, as in a sudden level off. Hence, the pilot will often sense the resultant force as a climb. If the altimeter is not crosschecked to confirm the actual orientation of the airplane, the pilot is likely to allow the nose to lower while attempting to maintain level flight from the misperception of a climb. The opposite occurs when making a sudden level off from a climb, which can result in an unexpected continuation of the climb. This level-off situation of the Elevator Illusion has been the culprit of several major aircraft accidents.

7.3.5. Visual Illusions and Problems

Humans are visually dominant creatures that rely heavily on visual stimuli to describe the surrounding environment. This reliance on vision has evolved because our vision often provides the most reliable information. This is not meant to imply that other sensory information is unimportant. The other senses often simply support, reinforce, and/or refocus the visual system on stimuli that are of interest. Due to various motions and accelerations along with unique visual perspectives and occasional low-visibility conditions encountered in flight, a pilot cannot be expected to always perceive correctly his or her geographic position, attitude, heading, altitude, and airspeed with natural environmental stimuli. Artificial cues such as the instrument panels within the aircraft have been implemented to assist the pilot to maintain awareness of the aircraft state. Nevertheless, human vision remains prone to perceiving illusions. An additional reason certain visual illusions are more prevalent in flight is due to the increased freedom of motion that allows for visual perceptions that would not normally be experienced on Earth. Some are almost never encountered on Earth (other than with some who have experienced brain damage) because our feet are firmly planted on the ground. We rarely if ever experience a brief absence or reversal of our normal 1 G force field. However, fighter pilots can maneuver their aircraft through any bank attitude of 360 degrees and experience reversals of the 1 G field. In situations where a pilot’s vision is totally focused on a lead aircraft (such as during in-flight refueling), a pilot can easily begin to feel inverted. Reports of such false sensations are commonly discussed among pilots. Many pilots report this sensation as having the “leans.”

7.3.5.1. Confusion Regarding Lights. A common problem associated with night flying is the confusion of ground lights with stars. Many incidents have been recorded where pilots have put their aircraft into very unusual attitudes to keep some ground lights above because they thought the lights were stars. Sometimes pilots have mistaken certain geometric patterns of ground lights, such as freeway lights, with runway and approach lights, or assumed a line of ground lights as the true horizon. Such illusions are illustrated in Figure 7.3.5-1a and Figure 7.3.5-1b.
7.3.5.2. False Vertical and Horizontal Cues. As seen in Figure 7.3.5.2-1, cloud formations may be confused with the horizon or ground. Momentary confusion may result when the individual looks up after prolonged attention to a task in the cockpit. When uniformly sloped, a cloud or horizon line may appear to be level. This will entice the pilot to bank the aircraft to align with the slope while believing the aircraft is perfectly level. Likewise, the false horizon may be experienced at night, even though night flying techniques should place less reliance upon outside references. Isolated ground lights from buildings and other structures may appear as stars. Furthermore, it is possible for ground areas devoid of lights to blend with an overcast sky. As a result, the pilot is susceptible to nosing the aircraft down or completely losing perception of a stable horizon. All perceived horizons must be crosschecked by the aircraft’s true reading artificial horizon. When in doubt always choose to follow the artificial horizon unless an instrument malfunction can be confirmed. Pilots often refer
to this experience as the Leans, although it is more correct to call it a false horizon. Regardless, remember the three A’s for preventing SD—attitude, altitude, airspeed!

![Figure 7.3.5.2-1. An Illustration of a Sloping Cloud Deck](image)

7.3.5.3. **Relative Motion or Linear Vection.** This illusion is typically caused by peripheral visual cues that are moving relative to the pilot's aircraft. The moving objects can easily be interpreted as self-motion and lead to erroneous aircraft commands to correct the apparent and unanticipated motion. An example of this type of illusion can be found in normal automotive driving conditions. The neighboring automobile creeping forward at a stoplight can give the illusion that one’s own vehicle is creeping backwards. And vice-versa is also true. You may misperceive a neighboring vehicle as moving only to find out you were the one actually doing the moving. One may instinctively apply the brakes and be surprised of the sensory feedback. In the flight environment, this illusion is relatively common during formation flying due to the close proximity of the aircraft to one another. Relatively small changes in velocity can produce an intense illusion.

7.3.5.4. **Autokinesis.** Although not common, the autokinesis illusion manifests only in dark environments (e.g., night) absent of many external cues such as stars and other lights. It begins with a small, singular, and stationary light source within the pilot’s field of view. After focusing on the light for 6 – 12 seconds, the light appears to move around and can become confusing (Figure 7.3.5.4-1). Autokinesis usually ceases when the pilot increases his or her visual scan. For this reason, aircraft have several staggered formation lights rather than a single light source.
7.3.5.5. Glare at High Altitudes. Glare is actually a visual hazard rather than a true visual illusion. Nevertheless, the effect may lead one to experience a more serious SD episode and is mentioned here just to make the reader aware of any cause that may lead to SD. It has been reported that a pilot who flies at high altitudes may encounter the problem of glare from the cloud layer below the aircraft. The facial contour is not formed to protect from glare emanating below the eyes; the sun’s reflected rays cause the pilot to develop a visual haziness. The cause of this subjective haze is probably the persistence of a positive afterimage of the bright cloud floor. Other causes, such as fluorescence of the crystalline lens of the eye caused by the greater intensity of ultraviolet light at high altitude and intraocular scattering of light, have been suggested and investigated. The glare from below and the sides, in combination with the lack of light scatter in the environment at high altitudes, may cause a relative shadow on the instrument panel. Since the external environment is bright and a relatively small amount of light diffuses into the cockpit, the instrument panel may appear to be in a shadow when the pilot turns his/her attention from outside the aircraft to the instruments. The solution to this problem is the use of white light in the instrument panel. The brightness of the panel can then be equalized with environmental lighting using a rheostat-controlled panel light intensity.

7.3.5.6. Haze and Fog. Dust, smoke, and haze caused by water vapor in the atmosphere tend to blur the outlines and reduce the colors and intensities of distant objects. These atmospheric conditions result in apparent increases or decreases in one’s perception of distance, depending on the filter and the particular situation (Figure 7.3.5.6-1). Excessive haze or fog would result in the appearance of the runway being farther away than its actual location.
Figure 7.3.5.6-1. Haze and Fog on Approach

Aerial perspective may play a role in low-level flying, but it does not play a great role in distance judgments during approach and landing. If the atmosphere is excessively hazy or foggy, the pilot often perceives the object to be further away than actual, as darker objects are usually perceived as further away (Figure 7.3.5.6-2).

Figure 7.3.5.6-2. Aerial Perspective

When using certain vision-enhancing devices to view the same situations, the pilot’s judgment may be just the opposite. Care must be taken when using enhancements to the visual system such as night vision goggles or laser eye protection.
7.3.5.7. **Space Myopia or Empty Visual Field.** At high altitudes, or during extended overwater flights, pilots may develop physiological myopia due to the normal ciliary muscle tone when the eye is at rest. Under these conditions, one may not have a distant object on which to fixate. In such an empty visual field, a reflex accommodation occurs, creating a varying degree of relative nearsightedness.

Theoretically, individuals with normal vision would be incapable of detecting a target at their normal far point. For example, a pilot with normal visual acuity of 20/20 is able to discern an aircraft having a fuselage diameter of 7 feet (2.1 m) at a distance of 4.5 miles (8.3 km). The same individual acclimated to an empty visual field would not be able to detect the same aircraft at a distance greater than 3 miles (5.6 km).

7.3.6. **Approach and Runway Problems.**

Approaches to the runway and landing maneuvers are perhaps the most challenging segments of flight. Hence, when further complicated with compelling illusions, the pilot must be especially vigilant in performing frequent instrument crosschecks and to integrate into the crosscheck any useful external visual aids to avoid potentially fatal SD consequences. Although there are many visual illusions relevant to approaches and landings, this section focuses on those that are especially common.

7.3.6.1. **Night Landing.** The reduced visual cues available during night and inclement weather landings can obviously contribute to a loss of situational awareness and provide increased risk of certain SD visual illusions. Specifically, there is always the danger of confusing approach and runway lights, misjudging the approach path, and being unfamiliar with the specific runway and its markings. When a double row of approach lights joins with the boundary lights of the runway, pilots have reported confusion in determining where approach lights terminate and runway lights begin.

The first problem can manifest from the runway lighting coupled with inclement weather. Although it is mandatory to clearly mark the front edge of the runway with runway threshold lights, it is not uncommon for them to be missing. Additionally, approach lighting systems can inadvertently give illusory or false information. When such situations include dense fog, the result from the decreased brightness can be the illusion of a false climb. Not surprisingly, the pilot is inclined to abruptly pitch down to compensate, which can lead to an impact with the ground or runway.

Most airports have tower-controlled runway lights, while some are controlled by the pilot. That is, the intensity of the runway lighting may be adjusted when the pilot approaches the airfield. If the tower has set the lights too bright, and especially during low-visibility conditions, it will give pilots the illusion that they are closer to the surface than they really are. Likewise, with light intensity that is too dim, pilots may perceive they are further from the surface than is actually the case (Figure 7.3.6.1-1).
Approach lights have also been known to create a false sensation of bank. This illusion is caused by one row of runway lights being brighter than the other. The brain interprets the brighter row to be closer than the dim row, thereby indicating a bank. Likewise, under certain conditions, approach lights can make the aircraft seem higher when it is in a bank than when its wings are level.

The fact that different airfields utilize different systems certainly complicates the pilot’s task of making height and distance judgments during approach and landing. Instrument approach systems, combined with a standardized improved approach lighting and glide-slope system, could eliminate or drastically reduce the false or illusory information received by the pilot. However, such changes are quite costly and therefore and unlikely to be installed. Remember, airfield lighting is not standard and should be reviewed prior to flight into a strange airfield, and to further learn about potential hazards, talk to a pilot who has recently flown into the airfield.

7.3.6.2. Runway Illusions (Day or Night). Pilot perspective and expected object dimensions can play a major role in the perception of distance to the object and its orientation/slope. Specifically, runways that are narrower or wider than expected can produce compelling illusions of increased/decreased distance from the aircraft. Likewise, varying runway slopes can provide similar illusions of height. Additional detail can be found in the following sections.
7.3.6.2.1. **Width.** A runway that is narrower than normally experienced during routine operations often appears to be further from the pilot’s aircraft than actual. Consequently, the pilot may establish a dangerously low visual approach, thereby producing the tendency to undershoot or land short of the runway (Figure 7.3.6.2.1-1). The compensated flight path is an attempt to maintain a certain visual picture, i.e., the same runway size, at a given point on the approach. This is called size constancy. Wide runways produce the opposite effect. Therefore, the apparent height is greater than the actual height and often results in a high approach and the tendency to overshoot, land further down the runway, an unplanned go-around, or an unexpected drop to the runway.

![Normal Approach Picture](image1)

![Approach Picture with A Wide Runway](image2)

![Approach Picture with A Narrow Runway](image3)

**Figure 7.3.6.2.1-1. Runway Width (Size Constancy)**

7.3.6.2.2. **Runway and Terrain Slope.** Most runways are level, but those that have some degree of slope can provide deceptive illusions to the pilot. Those with an upslope tend to make pilots feel they are at a greater height above the terrain, causing a normal glide path to seem too steep (Figure 7.3.6.2.2-1). Establishing a compensatory glide path that seems more normal could result in an approach and landing that is short of the runway. The slope of the runway should be checked during the pilot’s preflight.
A similar situation may occur when the runway is level but the approach terrain is sloped downward away from, or below, the runway elevation. Such terrain provides an illusion that the aircraft is higher than desired (Figure 7.3.6.2.2-2, upper pane). To compensate, the pilot may fly a revised steeper glide path that will result in collision with the terrain short of the runway. Conversely, an upslope of the approach terrain away from the runway creates the opposite visual illusory effect (e.g., pilot feels aircraft is too low) (Figure 7.3.6.2.2-2, lower pane). Inclement weather and poor ambient luminance generally enhance the effect of these illusions.
Figure 7.3.6.2.2-3 illustrates real-world examples of runways that with varying approach terrain and runway slope.

The “duck under” or “black hole approach” is another well-known visual illusion (Figure 7.3.6.2.2-4). There are many explanations but in reality we do not understand exactly why it happens. However, we do know it happens. If a pilot makes a visual approach at night into an area of low cultural lighting and does not use glide slope guidance, inevitably, the pilot will fly the approach below the normal approach angle and often find himself quite a bit short of the threshold or overrun. This fact has been repeatedly demonstrated to be true, yet the exact reason is not well understood. Some have attributed it to a visual angle seen by the pilot that causes the pilot to maintain the visual angle, resulting in an undershoot. Others have shown how the lack of cultural
lighting causes the pilot to inadvertently descend prior to safe minimum altitudes. Regardless of the reason, pilots should know that if they make a black hole approach and do not use glide path assistance, such as VASI or PAPI, they will land short of the runway. You can avoid it! If the reader is inclined to know more about this illusion, the authors recommend looking at some of the published literature regarding the black hole illusion.

![Figure 7.3.6.2-4. Black Hole Approach](image)

When doubts exist concerning the runway and approach terrain slopes, pilots should elect to perform an instrument approach while remaining mindful of the potential runway illusions. Due to the fact that these illusions are often complex (multiple illusions simultaneously), it is advisable to make the first approach and landing into a strange airfield using an instrument-aided approach with visual aids as a backup when possible (see discussion on approach systems below).

### 7.3.6.3. Other Landing Problems.

#### 7.3.6.3.1. Water.

Landing over water reduces visual cues to a minimum (e.g., visual flow, peripheral structures), and pilots not accustomed to this environment tend to land either too low or short of the runway. External objects on the approach path serve to provide the information concerning the height of the aircraft. Often, this information is in the form of size constancy. Pilots know the relative size of houses, towers, trees, etc. and can therefore make inferences of height based on the relative size of such objects when peering out the windscreen. Hence, if a pilot was accustomed to landing with an approach over large evergreen or spruce trees and was required to land in the Aleutian Islands, he/she might misjudge height and distance from
the runway because the spruce trees in the Aleutians are small and scrubby, resulting in the pilot flying closer to the environment than expected.

One of the best means of eliminating or reducing aircraft accidents from these types of visual illusory cues is careful briefing of the aircraft crew prior to the flight. A pilot must be aware that distance and height judgments can be affected by biases created from experience and prior knowledge, and he or she should fly an instrument approach if available. If not available, a low approach should be executed before making a landing. The combination of good briefings, aircrew proficiency, and discipline is required for safe approach and landing procedures over all types of terrain and landing field configurations.

7.3.6.3.2. Flicker Vertigo. Light flicker is defined as a rapid fluctuation in the brightness of a light source or the appearance that it is being turned on and off repeatedly. When presented to individuals who are sensitive to light strobe effects, the result can be a seizure or disorientation. Typically such “flicker vertigo” occurs when the light fluctuation is low frequency. Other reactions may include nausea, dizziness, headaches, grogginess, and unconsciousness or confusion, uneasiness, nervousness, hypnosis, gastrointestinal discomfort, and a feeling of severe panic.

Flicker vertigo is most frequently encountered in rotary wing and single engine propeller aircraft. The spinning rotors obstruct a light source (such as the sun) each time they pass through an individual’s field of view. Hence, if looking up through the rotors, the sun will appear to flicker.

Fatigue and frustration tend to increase the annoying quality of flicker vertigo and make the manifestations more pronounced. Low G-tolerance, hypoxia, hyperventilation, and hypoglycemia seem to suggest a greater susceptibility to the effects of light flicker. Protecting aircrews from the effects of flicker vertigo involves avoidance and reduction of factors that increase susceptibility.

7.3.7. Approach Systems

7.3.7.1. Visual Approach Slope Indicator. The Visual Approach Slope Indicator (VASI) system consists of a series of four lights designed to give pilots information concerning their glideslope during landing approaches (Figure 7.3.7.1-1). When all lights are white, the aircraft is high, whereas a change to all red lights indicates the aircraft is low. To inform the pilot that he/she is on the optimal glideslope, the far row of lights remains red while the near row of lights becomes white. By communicating glideslope state information in a simplistic fashion, these lights have reduced the possibility of landing illusions.
7.3.7.2. **Precision Approach Path Indicator.** The Precision Approach Path Indicator (PAPI) consists of four light boxes similar to the VASI, except all four are arranged horizontally. Two red and two white lights are an “on glide-path” indication with corresponding color changes the same as the VASI (Figure 7.3.7.2-1).

7.3.7.3. **Fresnel Lens Optical Landing System.** This electro-optical landing aid was designed for carrier landings but is also installed at most Naval air stations. The Fresnel Lens Optical Landing System (FLOLS) appears as two sets of green (datum) lights arranged horizontally on either side of a large yellow light (the meatball) (Figure 7.3.7.3-1). When above the glide-path, the meatball appears above the green datum lights, and the meatball will appear below these lights when low on the glide-path. If more than 0.75 (¾) degree from the glide-path, the meatball will disappear.
7.3.8. Proprioceptive Problems

The seat-of-the-pants sense is unreliable as an aircraft attitude indicator. Although every pilot is taught this understanding from day one of instrument flight training, many pilots forget the basics and often rely on their seat for orientation information. When coupled with the vestibular signals and a lack of clear visual information, the pilot is ready to experience an SD episode. Hopefully the pilot will recognize the illusion before it progresses too far to recover.

7.3.9. Factors Influencing Spatial Disorientation

A particular set of linear and/or angular accelerations or misleading visual cues will not always produce illusory phenomena. When adequate external visual references are available, spatial disorientation may or may not occur, which is one of the reasons SD is such a problem. It is also important to note that the absence of outside visual cues only serves to increase the risk of SD. Therefore, the same complex vestibular cues can be presented to a pilot on several different occasions with the illusory phenomena only being present in a fraction of the trials. Additionally, mental and physical stress and fatigue reduce the pilot's ability to resist SD illusions. As a result, the pilot should always be aware of the potential for SD regardless of the outside visual conditions. Proficiency in instrument or formation flying remains the only proven countermeasure to SD. Additional factors influencing spatial disorientation are included in the following sections.

7.3.9.1. Cockpit Configuration. Head movements necessary to use some of the cockpit instruments, gauges, switches, and radios during the critical phases of takeoff or approach to landing may cause the Coriolis illusion. Aircraft manufacturers are very aware of this problem, and modern aircraft are generally designed to place radios and other related instruments in positions where extreme head movements are not required.
In the past, poor lighting conditions in cockpits have caused difficulty in reading instruments and performing cockpit tasks under normal environmental conditions. Therefore, poor lighting hampered recovery from the symptoms of spatial disorientation. Today, most aircraft have adequate lighting, but all aircrew personnel are not aware of proper lighting techniques. Differences in the intensity of lighting of various instruments and aircraft compartments may cause serious visual problems and compound the symptoms of spatial disorientation.

7.3.9.2. Visual Flight Rules-Instrument Flight Rules (VFR-IFR) Transition. The experienced, proficient pilot has no difficulty in flying either in visual meteorological conditions (VMC) or instrument meteorological conditions (IMC). When the pilot is making a transition from external visual reference to instruments, or when the pilot is under stress, his or her instrument crosscheck is less than adequate. This is also a physically and mentally demanding task. Consequently, transitioning back and forth repeatedly from IMC to VMC can develop into a dangerous situation with high risk of SD illusions.

7.3.9.3. Disorientation Produced by Pressure Change in the Middle Ear. A relatively rare form of disorientation can be observed in some aircrew members when flying with upper respiratory infections. These individuals complain of spinning sensations and motion sickness during ascent, descent, or while performing a Valsalva maneuver. The mechanism contributing to this vertigo is not known, although it may be caused by a blocked Eustachian tube that suddenly opens. This would allow the rapid dissipation of the pressure differential that develops gradually during ascent. The result is a mechanical stimulation of the organs of the inner ear that may produce the sensation of spinning (positional vertigo).

7.3.9.4. Stressors. Any factors that adversely affect the judgment or cognitive state of the pilot will decrease his/her chances of survival in a spatial disorientation situation. Physiological stresses such as hypoxia, high G acceleration, and thermal stress can negatively affect the pilot’s ability to prevent or recover from a spatial disorientation event. Similarly, self-imposed stresses, such as alcohol and certain medications, will predispose a crewmember to spatial disorientation and make SD incidents more severe and the successful recovery from SD less likely.

Psychological problems and stresses also predispose a crewmember to spatial disorientation. One such condition is fascination or target fixation, which results when a pilot ignores orientation cues while his/her attention is focused on some other object or task. A missed radio call is often considered a sign of fascination or attention narrowing. This is often called task saturation. Although all factors contributing to fascination are not well documented, it is generally accepted that hypoxia, fatigue, drugs, and basic personality are usually influential factors.
7.3.10. Conditions Most Conducive to Spatial Disorientation

A few of the conditions most conducive to SD are listed below. A complete list is beyond the scope of this chapter.

- Weather
- Lack of visual references
- Poor instrument crosscheck
- Loss of situational awareness
- Distraction
- Personality
- Health

7.3.11. Incidence of Spatial Disorientation

It has been repeatedly observed that spatial disorientation is now experienced by almost all pilots on many sorties. This was not always the case. Until recently most pilots would not admit to experiencing SD. The cause is most likely due to a poor understanding of the causes of SD. Perhaps an increased knowledge base of SD definitions and causes has led to greater recognition of SD events. Nevertheless, unrecognized SD will, of course, remain unreported, thereby skewing the results and providing only a portion of the true number of SD events. The danger presented by SD is evidenced in the significant number of disorientation accidents resulting in fatalities.

Fighters and jet trainers accounted for 84% of the spatial disorientation accidents; however, such accidents were also found to occur in multi-crew bombers, cargo-utility aircraft, and helicopters. Nineteen percent of disorientation accidents occurred during the takeoff departure phase of flight, 18% during the approach-landing phase, and 63% during the in-flight phase. The majority of accidents occurring in-flight involved formation flight, aerial refueling, gunnery, or aerobatic maneuvers.

These statistics do not include undetermined accidents, a category that involves approximately 10% to 12% of the total annual number of Air Force aircraft accidents. It is possible, based on the phase of flight or type of flight activity, that a number of these accidents were caused by, or related to, SD. The large numbers of accidents coupled with a significant loss of life and aircraft necessitates a concentrated and continuing program of prevention and training. To be effective, such training must include academic presentations, ground level simulations, and in-flight training.

7.3.12. Preventing Spatial Disorientation

Training, experience, and professional knowledge are the keys to preventing accidents caused by spatial disorientation. There are only two accepted countermeasures for SD—awareness of it and a good instrument crosscheck. This chapter provides the reader with a beginning to awareness. It is important to develop a habit of continual SD education and refresher training throughout the lifetime of the pilot. The pilot must constantly practice his or her instrument crosscheck during flight training. The following sections examine these objectives in more detail.
7.3.12.1. **Training.** Indoctrination of pilots is the first important step to take in the fight against spatial disorientation accidents. Lectures, demonstrations, and movies discussing sensory functions and the conditions in which they become inadequate must be given to pilots by physiological training officers and flight surgeons. Updating and improvement of training aids should be accomplished frequently to ensure adequate dissemination of pertinent knowledge to the pilot population. Summaries of accidents, statistics, and trends should be used in such training programs.

7.3.12.2. **Experience.** While experience alone, as measured in terms of flying hours, does not preclude occurrence of spatial disorientation, several experience factors are involved. Experience gained in ground-level simulations using the Barany chair, new SD training devices like modified flight simulators, and basic flight simulators themselves, if used properly, will reinforce the fact that sensory illusions are a definite threat to the pilot’s ability to maintain orientation. Ideally, ground trainers would allow the pilot to fly the simulator into an environment that produces an illusion or disorientation. Next he/she would be required to recover using instruments that display actual orientation. Such a trainer would reinforce the reliance on visual input from the instruments and prove that disorientation can be corrected. Such SD trainers or “simulations” are just now being installed in undergraduate training bases. They will be used for SD illusion demonstrations and they will be used to reinforce the need for a constant instrument crosscheck.

For now, experience with SD illusions while at the flight controls has been obtained only in-flight. This is now beginning to change. Experience in actual instrument flying and the proficiency gained while using these SD trainers will contribute to prevention of spatial disorientation accidents (See Section 8.7 for more on USAF SD training). However, it is important to note that maintaining minimum currency requirements for instrument flying may not be sufficient since all IFR-rated pilots involved in spatial disorientation accidents can be presumed to have met these requirements. The difference between the mandated “currency” and being “proficient” can often be of great consequence. Don’t forget to use the basic three words which form the foundation of spatial orientation—attitude, altitude,airspeed. Fly safe!

7.3.12.3. **Knowledge.** Cumulative knowledge gained from studies of spatial disorientation accident trends has resulted in alterations to the art and science of flying. Aerospace manufacturers are now aware of cockpit configuration issues, instrument design, and instrument placement. These critical design corrections have consistently contributed to the reduction of SD accidents.

Both basic and applied research in vestibular physiology and illusions continues to provide knowledge that will reduce the risk of SD. Aircraft accident investigation teams must compile data relative to runway conditions, width, length, lighting slope and surrounding terrain, and human factors that can reveal factors responsible for certain types of accidents—all relevant to illusions or disorientation.
7.3.13. Overcoming Spatial Disorientation

While prevention of SD is of prime importance, recommendations regarding actions a pilot should take if he/she becomes disoriented must be included in any discussion of spatial disorientation. The following sections describe standard recommendations for overcoming disorientation.

7.3.13.1. Get on the Instruments. The visual input from the instruments is fundamental to the recovery from the effects of spatial disorientation. Do not transition back and forth between instruments and visual references. Establish and maintain a good instrument crosscheck.

7.3.13.2. Believe the Instrument Indications. The pilot must learn to ignore, overcome, or control the urge to believe false sensations perceived from the supporting body senses. Concentrate on the instruments to shorten the effects of the symptoms of spatial disorientation.

7.3.13.3. Place the Head Back into the Headrest. Minimizing head movements and establishing a constant head position with respect to the neck will tend to minimize effects of SD.

7.3.13.4. Fly Straight and Level. Once the pilot attains straight and level flight from information provided by the instruments, he/she should avoid further maneuvers until full orientation is attained and sensory illusions are minimized.

7.3.13.5. Turn the Controls Over to the Other Pilot in Multi-piloted Aircraft. After turning over the controls, the pilot must get on instruments to regain orientation and resist the tendency to resume or take control until full orientation is attained. If the aircraft is equipped with an auto-pilot, it should be utilized.

7.3.13.6. Egress. If the pilot cannot achieve orientation, particularly in situations involving low altitude, ejection may be the only chance for survival. In high-performance aircraft, the decision to eject must be made quickly. Pilots must know before flight what their course of action will be under such critical situations.


7.3.14.1. Helmet Mounted Displays. Helmet-Mounted Displays (HMDs) pose a somewhat unique and relatively unknown threat. The technology is quite new and, in some significant ways, still under development. Simply put, there are aspects of HMD use that may help reduce the risk of SD but there is also reason to be cautious. After a brief description of the technology itself, this section is intended to present applications where operators should be educated about the intended benefit of the technology as well as potential unintended consequences.

7.3.14.2. HMD Concept and Technology. Similar to the development of the Head-Up Display (HUD), the conceptual origin of the HMD is as a component of an enhanced weapon system. Simply put, the primary object of the HMD is to afford the operator a large field-of-regard (FOR) transparent aiming device with which symbology
can be overlaid on the outside world. The display hardware incorporates optical
techniques for displaying collimated symbology and imagery. Since the graphics on the
display are focused at optical infinity, the operator is able to simultaneously view the
distant real-world scene through the display combiner and the information drawn to the
display (symbology and imagery). Collimation allows this to happen without an
associated change of observer visual accommodation (lens or vergence). The HMD
shares a similar functionality with the HUD with several significant differences. First, in
order for the operator to “slew” the HUD drawing surface over a point of interest in the
outside world, the aircraft must be maneuvered. On the other hand, use of an HMD
allows the same function via operator head movement and corresponding change in
line-of-sight (LOS). Of course the HMD-based ability for the operator to superimpose
the display over points of interest can happen independent and regardless of aircraft
maneuvering. This dynamic enables significant agility and higher rates of display
“slewing” versus aircraft maneuvering rates. Depending on the acceleration
environment and other factors, human head movement rates can approach 400
degrees per second. Also, operators can move LOS independent of head location
within the limitations of the human visual system.

Also much different than a HUD, some of the HMD functionality is dependent on
tracking technology in order to translate approximate LOS azimuth and elevation back
to the aircraft system so that the relative location and orientation of system produced
representations are properly registered (or stabilized) to outside world (earth fixed),
objects in the outside world (target fixed), to the aircraft (ownship fixed), or to the helmet
display itself (head fixed). These disparate frames-of-reference enable the relatively
small HMD field-of-view (FOV) (typically around 20 degrees subtended visual angle) to
represent an unlimited FOR (head-movement limited). While this large and quickly
accessible HMD-based FOR is one of the main operational advantages of the system—and
it also generates potentially negative effects. The operator is not able to “look
around” HMD drawn information in the same way as with a physical HUD. This fact,
and the fact that the HMD FOV is small drive a design trade-off where it is understood
that symbology on the HMD surface should be sparse in order to minimize clutter.
Thus, only critical information (symbology and imagery) should be worthy of HMD
inclusion. Given the objective of the HMD as an offensive and defensive tactical
technology, the natural disposition exists for targeting information to take a higher
priority compared to ownership status information. This was true of the early versions of
the HUD as well. The consequence this represents for spatial orientation is still not well
understood. While the targeting information may be very compelling and attention
demanding, the lack of ownership status information may not be comparatively supportive
of orientation maintenance—in an environment where aircraft acceleration vectors and
head movements create a complex vestibular dynamic.

The ownership status information typically provided via the HMD consistently
include airspeed and altitude readout but other information that is standard in terms of
HUD portrayal is either missing or is displayed is quite varied. For example, aircraft
attitude information (roll, pitch, climb/dive angle, and flight path) displayed as a
graphical representation of the horizon superimposed over the real-world horizon is
either absent from the HMD at lines-of-sight off the aircraft longitudinal axis or is
displayed much differently. This is direct result of the expanded envelope of frames-of-
reference available with a head-tracked HMD system. Attitude information can be
referenced to the longitudinal axis of the aircraft (helmet fixed and forward referenced)
or relative to the appearance the natural horizon would take for a given observer line-of-

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sight (earth fixed line-of-sight referenced). Intensified imagery such as that provided by Night Vision Goggles (NVGs) is an example of the latter configuration. Imagery of the real-world through NVGs is intuitively interpretable by an observer. This is the case for the limited FOV of the HMD as well but the observer must be looking near horizon line to see it. With the dynamic combination of head and aircraft movement during tactical targeting tasks, this is potentially a spatial orientation concern.

7.3.14.3. Virtual HUD Mechanization. The most recent generation of HMD for fixed-wing tactical aircraft is intended to replace the HUD hardware versus being used in conjunction with a HUD. While there are a compelling number of reasons for this design decision, operators should be aware that this too poses SD concerns. The HUD replacement approach includes ownership status symbology that is aircraft fixed and forward referenced—to mimic the presentation of information on a HUD combiner. This virtual HUD symbology is only visible when the HMD FOV is located forward near the aircraft longitudinal axis. Although the HUD metaphor and its respective interpretation is somewhat maintained by this design approach, there are significant differences. Because of the limited HMD FOV, unless the operator is looking at the center of the virtual HUD coordinates, some part of the information will be clipped and not available to the operator (symbology will be off the edge of the HMD drawing surface). With a physical HUD, the operator need only make small eye movements to see HUD presented information at significant head angles off the HUD centroid. The same eye movements in the HMD application results in the operator looking away from the display exit pupil where information is available. Information gathering scan behavior with an HMD only transparent combiner approach is likely quite different from that required with more conventional HUD only or physical HUD and HMD combined applications.

7.3.14.4. Frames-of Reference and Head Tracking. The different frames-of-reference and information fixing options available with HMD employment are mostly made possible through the use of head tracking. Only information that is head fixed is possible on an HMD system without tracking. For this reason, the head tracker is a critical component of the system. Tracker performance in terms of update rate, lag, and stability define how well HMD information is stabilized and registered relative to direct viewing of real-world points of interest through the transparent combiner. Less than very fast and accurate tracker performance results in symbology that may not correctly overlay the real-world by registration (offset), by trailing behind due to lag, and/or appearing to jitter relative to the object the symbols or imagery is intended to overlay. To the extent they exist, these tracker induced artifacts affect all ownership status information that is not fixed to the helmet regardless of its FOR. Again, the implications of this for spatial orientation are not fully understood but the existence of these effects needs to be appreciated.

7.3.14.5. Distributed Aperture System and Head-Based Sensors. Sensors mounted externally to the aircraft as a distributed aperture generate imagery that is graphically stitched together in a way that visual information is available to the operator. It appears as if the operator can see “through” the aircraft. This imagery is combined with the symbology previously discussed on the HMD combiner. The distributed aperture system (DAS) generated imagery is subject to the same tracker induced artifacts as all other than HMD fixed symbology/imagery. Additionally, because
the sensors are located at locations on the aircraft that are different from the operator’s actual eye location, some natural parallax distortion exists in the imagery versus its registration with the real-world scene as perceived directly though the transparent HMD combiner. The severity of the distortion is somewhat mitigated by range and lack of resolution of the DAS imagery. The SD implications of the ability to effectively see through the aircraft in the manner afforded by a DAS approach are not well understood. Access to this information may be quite helpful for orientation maintenance but degraded aircraft secondary spatial cue reference, such as to the canopy rails and the top of the glare shield, has been shown to cause performance decrements during flight tasks. In actuality, use of the DAS imagery via transparent combiners allows the operator to simultaneous see imagery of the real-world as well as some of the aircraft interior/exterior. Perhaps this is enough secondary spatial reference to suffice.

Head-based sensors (cameras mounted to the helmet), such as an image intensifier, provide information to the HMD combiners as well. Parallax exists here, but in this case the sensor is typically fairly close to the operator’s eye location. Unlike the mechanization of DAS, the head-based sensor is not subject to artifacts introduced by the tracker system. For this reason, simultaneous use of a head-based sensor and DAS can present differing effects of lag, update rate, mis-registration, and jitter. Other known concerns and potential SD issues include image resolution, monocular and binocular views, combiner optical imperfections, and luminance.

7.3.15. Summary

Man possesses unique physiological systems that provide information pertaining to orientation, body position, and motion. Visual, vestibular, and proprioceptive systems are influenced by flight maneuvers and can produce motion sickness and spatial disorientation. And with the advent of new technology such as HMD, there is an increasing concern regarding the potential for spatial disorientation accidents. The severity of sensory illusions depends in part on how the pilot decides to utilize sensory information to control the aircraft. A correct decision results in adequate control of the aircraft, but an incorrect decision may prove fatal. Reduction in external visual cueing caused by low ambient light or inclement weather and physiological, emotional, or self-imposed stresses will greatly enhance a pilot’s susceptibility to illusions. Special training and instrument flying proficiency are paramount to ensuring safe flying and recovery from SD. Bottom line: Maintain a proper instrument crosscheck and always be aware of your ATTITUDE, ALTITUDE, and AIRSPEED! Safe flying.

References

Recommended Readings

Concept
Spatial disorientation

Vocabulary
Illusion
Orientation
Otolith organs
Proprioceptive system
Semicircular canals
Vertigo
7.4. Motion Sickness

Lee Diekmann, MSgt, USAF (Ret); Lt Col Quentin Bagby, USAF, BSC; and Lt Col Ryan Maresh, USAF, BSC, Ph.D.

7.4.1. Prevalence of Motion Sickness during Flight

Motion sickness is a normal response to real or apparent motion to which an individual is not adapted and can occur in modes of transportation (Dobie and May, 1994; Oosterveld, 1995). Motion sickness has been well known for thousands of years. Ancient seafaring nations were very familiar with this malady. In fact, the term “nausea” is derived from the Greek word naus (ship) (Simmons et al., 2008). Motion sickness has become increasingly prevalent with development of the many forms of vehicular travel, amusement park rides, and ever more dizzying visual stimuli. Various names give an indication of this ailment’s many causes: seasickness, airsickness, car sickness, amusement-park-ride sickness, motion-picture sickness, microscope sickness, flight-simulator sickness, and space-motion sickness. While at first glance these may appear to all be the same thing, and often share many of the same symptoms, in reality there are subtle, yet distinct differences between many of them. Surveys have found that car sickness occurs in 58% of children under the age of 12 years (Griffin, 1990), space motion sickness occurred in roughly 70% of shuttle astronauts during their first space mission (Lackner and DiZio, 2006), and incapacitating airsickness occurs in 29% of airline pilots (James and Green, 1991). Of those airline pilots experiencing airsickness, 48% stated that it actually or potentially influenced flight safety (James and Green, 1991). Up to 100% of ship passengers become seasick under rough conditions, especially in small vessels (Benson, 2002).

It has been reported that 11% - 39% of military student pilots experience motion sickness, but after implementing an airsickness management program, only a 1% washout rate has been noted (Dobie, 1974; Hemmingway and Green, 1945; Tucker et al., 1965). Once a student has overcome motion sickness, it is rare for the symptoms to return, at least to the extremes found before starting the program.

7.4.2. Types and Causes of Motion Sickness

7.4.2.1. Airsickness. Airsickness is one aspect of the motion sickness syndrome. Airsickness is generally accepted as resulting from the complex interaction of a person’s sensitivity to motion, sympathetic nervous system arousal, performance anxiety or nervousness, and even motivation. The conflicting sensory impressions can occur due to purely visual stimuli, as well as via vestibular involvement or a combination of both. Another potential evolutionary relationship between vestibular disturbances and an emetic response may be in the effect of poisons on the vestibular system (Money and Cheung, 1983). It is well known that flying (or seafaring) while under the influence of alcohol or other mood altering drugs can induce more severe symptoms of airsickness and motion sickness. Jackson (1994) proposed a multimodal conceptual model of airsickness onset involving behavior, affect, sensation, imagery, cognition, interpersonal, and drugs/biology. However, a fully adequate explanation of motion sickness does not currently exist (Lackner, 2014). While several theories have been proposed to explain motion sickness, the most widely accepted one is the sensory conflict theory of motion sickness.
The sensory conflict theory of motion sickness describes the mismatch between sensation and expectation or a mismatch between different sensory channels (Rubin, 1942; Graybiel, 1969; Ernsting et al., 1999). In humans, movement through the environment is inferred by two principal sensory systems: the visual sense and the two components of the vestibular system of the inner ear. The vestibular system includes the semicircular canals, which detect angular acceleration, and the otolith organs, which sense linear acceleration (other proprioceptive sensations have a minor contribution to motion sickness). Earlier theories that motion sickness is produced by vestibular over-stimulation have been discounted. It is now fairly widely accepted that motion sickness is caused by conflicting inputs between the visual and vestibular systems (vestibular mismatch) or between the two vestibular systems and comparison of those inputs with the individual’s expectations derived from previous experience.

7.4.2.2. Simulator Sickness. Depending on the cues received from the visual or vestibular systems and how they are reconciled leads to different types of motion sickness (Benson 1978; Benson 1984). Aircrew can experience motion sickness when using full-motion simulators due to the neural mismatch and the resulting condition is referred to as simulator sickness (O’Hare and Roscoe, 1990). It has been reported that between 20% - 40% of military pilots experience at least one motion sickness symptom following the use of a simulator (Kolasinski, 1995), with others reporting the incidence as high as 60% (Kennedy et al., 1989). Some of these symptoms can persist for hours after exposure (Baltzley et al., 1989).

While simulator sickness and motion sickness have similar symptomology, they are not the same thing due to different causes, although sickness induced in full-motion simulators may be argued to be classical motion sickness (Kolasinski, 1995). Non-motion simulators can lead to symptoms induced by the vision-only cues and some researchers argue that simulator sickness is primarily induced by visual stimuli (Kennedy et al., 1992; Kolasinski, 1995). The use of head mounted displays (HMD) while in a simulator may also influence sickness susceptibility, as well as overall performance, and is an area for further research (Mollenhauer, 2004). Due to the cause of sickness, simulator sickness shares many similarities with cybersickness, which is presented in more detail below.

7.4.2.3. Cybersickness. While the cause of motion sickness is generally considered to be a mismatch of vestibular and visual sensations, actual movement of the body is not necessary to produce symptoms. Purely visual stimuli (i.e. virtual environments), such as those from flight simulators, video games, panoramic movies, or even the movement of slides under a microscope, can produce symptoms more effectively than actual physical motion. This form of sickness is referred to as cybersickness and is distinct from the classical form of motion sickness, which requires vestibular stimulation to induce sickness (La Viola, 2000). Unlike classical motion sickness, with cybersickness the user is stationary but experiences a compelling sense of self-motion due to moving visual stimuli. The degree of sickness appears to be directly related to how well the visual stimulus simulates motion. With the increased use of unmanned aerial systems for both military and civilian operations, more research on user-interface is necessary to quantify the design considerations and potential for inducing cybersickness in operators. Some studies have indicated that the use of helmet mounted displays (versus a conventional computer monitor and joystick) to
perform an unmanned aerial vehicle sensor operator target search task lead to increases in self-reported nausea, disorientation, and eye strain (Morphe et al., 2004).

In susceptible individuals, the negative effects of cybersickness can persist for hours, even days, after exposure to the virtual environment (La Viola, 2000). Like with classical motion sickness (i.e. airsickness, car sickness, sea sickness), the most commonly accepted explanation for the cause of cybersickness is the sensory conflict theory, although there are a number of problems with it in this application and more research is needed to fully understand the mechanism.

A few of the factors that have been shown to contribute to cybersickness that do not fit perfectly with the sensory conflict theory are presented below (La Viola, 2000):

**7.4.2.3.1. Display and Technology Issues**

**7.4.2.3.1.1. Position Tracking Error.** The ability to track the user’s head and body in physical space plays an important role in providing an accurate representation in the virtual environment. If the tracking equipment is not fluid and provides unstable, jerky information, the visual image that the user sees can be perceived as an uncontrollable movement even through their body is stationary. This can lead to dizziness and lack of concentration.

**7.4.2.3.1.2. Lag.** Lag is the time between the user initiating a command (i.e. turning the head) and the action actually occurring (i.e. the virtual image catching up to where the user is looking).

**7.4.2.3.1.3. Flicker.** The perception of flicker varies between individuals and increases as the image moves into the peripheral visual field. A refresh rate of 30 Hz is usually sufficient to removed perceived flicker for foveal, or central, vision. However, a higher refresh rate is necessary to remove perceived flicker from peripheral vision. Flicker is distracting, causes eye strain, can be disorienting, and lead to cybersickness.

**7.4.2.3.2. Individual Factors.** As with airsickness and other forms of motion sickness, there are large variations in individual susceptibility.

**7.4.2.3.2.1. Gender.** Women appear to be more susceptible than men, most likely due to their wider field of view than men (see above).

**7.4.2.3.2.2. Age.** Susceptibility to cybersickness appears to be greatest between the ages of 2 and 12 years and then decreases rapidly up to age 21, when susceptibility slows.

**7.4.2.3.2.3. Illness.** As with other forms of motion sickness, illness increases susceptibility to cybersickness.

**7.4.2.3.2.4. Position in the Simulator.** Sitting appears to reduce susceptibility to cybersickness, most likely because this position reduces the demands placed on maintaining postural control and body position. Being in control also reduces susceptibility, just as drivers are less likely to get car sickness than passengers.
7.4.3. Signs and Symptoms

Characteristically, motion sickness begins with epigastric discomfort often described as "stomach awareness," which is usually accompanied by increased salivation, belching, and a feeling of bodily warmth. The most commonly reported signs of motion sickness are pallor, cold sweating, and vomiting, while the most frequent symptom is nausea (Money, 1970). Anxiety may be linked to a higher incidence of airsickness and may be related to motivation factors (Tucker and Reinhardt, 1967; Ryback et al., 1970).

Some researchers suggest that there is another distinct syndrome of motion sickness that lacks these gastrointestinal complaints and is instead characterized by drowsiness, headache, apathy, depression, lack of initiative, personality changes, and generalized discomfort. An individual may experience profound drowsiness and persistent fatigue following a brief exposure to high-intensity stimulation, such as acrobatic flying, or after sustained exposure to low-intensity motion stimulation (Lackner, 2014). These behavioral responses are known as the sopite syndrome and may not be recognized as resulting from mild motion sickness. Yawning has also been shown to be a potential behavioral marker for the onset of mild motion sickness and a reduction in multitasking cognitive performance (Matsangas and McCauley, 2014). The sopite syndrome may result when an individual does not experience nausea, or after it has subsided (Lackner, 2014). It is quite likely that many people experience both epigastric and sopite syndromes to varying degrees.

As mentioned above, even mild motion sickness without nausea or vomiting can result in significant decreases in motivation and performance. In the flying environment, especially during pilot training, these effects may not be recognized as motion sickness but rather may be attributed to other causes, such as fatigue, lack of situational awareness, or lack of flying skill. Changes in behavior and performance, such as decreased spontaneity, inactivity, or being quiet or subdued may be misperceived as fatigue. Decreased muscular coordination and eye-hand coordination may be confused with spatial disorientation. Decreased squeezing force in the hand may be associated with fatigue or even decompression sickness. Decreased ability to estimate time and decreased performance of arithmetic computation may be confused with a loss of situational awareness or other cognitive management issues.

7.4.4. Susceptibility

Motion sickness is not unique to humans and has been documented in numerous species. While nearly everyone is susceptible to motion sickness, their susceptibility is influenced by the relative level of stimulation, individual sensitivity and variability, and prior experience. It has even been shown that blind individuals are susceptible when exposed to provocative physical motion (Lackner, 2014; Graybiel, 1970). Only those individuals with total loss of vestibular function appear to be immune (Lackner, 2014), although there is some evidence that even they may be susceptible to visual stimuli designed to induce the sensation of self-motion (Golding, 2006).

Susceptibility to motion sickness varies as we age. Children younger than 2 years of age are rarely affected, but susceptibility rapidly increases with age, peaking around 12 years and then gradually declining until age 21 (Oosterveld, 1995). A further decline in susceptibility is seen with age, probably as a result of declining vestibular afferent information, and is rare in civil aviation after age 50 (Oosterveld, 1995).
As mentioned previously in the discussion on cybersickness, women tend to be more susceptible to motion sickness than men, with a higher susceptibility near the beginning of menstruation and pregnancy (Oosterveld, 1995; La Viola, 2000). In men, increased aerobic fitness appears to increase susceptibility (Oosterveld, 1995).

An individual’s location within the moving vehicle, be it a car or an aircraft, has been shown to have a large influence on susceptibility, as has an individual’s body position relative to the motion (Money, 1970). Susceptibility is typically decreased when in the supine (i.e., face up) position compared to sitting upright, or even lying in the prone (i.e., face down) position. Keeping the body, especially the head, aligned with the direction of motion may also help to decrease susceptibility by reducing the conflicting visual, vestibular, and somatosensory signals.

While many susceptible individuals desire cooler temperatures, ambient air temperature has not been shown to influence susceptibility (Money, 1970).

Diet and other self-imposed stressors also influence an individual’s susceptibility to motion sickness. Recent ingestion of food, particularly dairy products and foods high in sodium, protein, or calories, has been associated with increased susceptibility, as has sleep deprivation (Lindseth and Lindseth, 1995; Lackner, 2014).

7.4.5. Treatment Methodologies

Treatment methodologies are tailored to the specific type of mismatch, e.g., acclimation training reduces the mismatch between sensation and expectation. Acclimation implies adaptation, which has been described (Benson, 1984) as an essential feature of motion sickness treatment. Over the years, a variety of approaches have been used to treat motion sickness. Starting with simple education and training, it has been estimated that one hour of classroom awareness training on the basics of motion sickness (normalization and identification of symptoms, basic vestibular physiology), along with rudimentary methods of prevention (foods, dietary schedule, stress management, etc.) can reduce airsickness incidence by as much as 30% (Jones et al., 2000).

7.4.5.1. Non-pharmacologic Therapy. Alternative medicine remedies are becoming increasingly popular, and many have been recommended for treatment of motion sickness. Regardless of the source of information or validity of claims, it is important for aviators to remember that all over-the-counter drugs and herbal remedies (including supplements) must be cleared through Flight Medicine before being used in the aircraft. The most popular herbal preparation for nausea is ginger root given in candied form, powdered in capsules, or as a tea. Although there is much anecdotal evidence that ginger is beneficial, scientific studies have yielded varying conclusions as to its true antiemetic potential. There is some evidence that at doses of 1 g/day, ginger does help to reduce nausea and gastric activity (Ernst and Pittler, 2000; Lien et al., 2003). While the mechanism of action is still not fully understood, evidence suggests that its effects are more peripheral, rather than central, and this may explain the lack of central side effects such as drowsiness seen with the use of other anti-motion sickness medications (Lien et al., 2003).

Acupressure has generated a great deal of interest as a non-pharmacologic means of preventing motion sickness. The P6 acupuncture point, located on the inside of the forearm, about 2 inches above the wrist, is the common site for treating nausea and vomiting (Hu et al., 1995). Stimulation of this site via acupressure has been used...
as a non-invasive substitute to acupuncture. Initial studies involving a popular acupressure wristband found no evidence that the band prevented motion sickness, compared with a placebo (Lentz, 1982; Bruce et al., 1990). The authors of both studies cited potential misalignment of the band or insufficient wrist movement leading to a lack of constant stimulation as possible reasons for failure. However, Hu et al. showed significantly reduced symptoms of visually-induced motion sickness and gastric tachyarrhythmia as a result of P6 stimulation (Hu et al., 1995). Any perceived benefit from mechanical devices such as acupressure bands are usually associated with psychological effects and should be approved through Flight Safety. Acupressure has thus been deemed impractical in the flying environment.

7.4.5.2. Pharmacological Therapy. The preferred medication to suppress motion sickness symptoms is a combination of scopolamine HBr (0.5 mg) and dextroamphetamine sulfate (5 mg) tablets or patches (Scop/Dex). Scopolamine is the anti-motion sickness medication but requires a stimulant to combat its depressive effects. This pharmacological combination should be ingested 2 hours before flight. While the combination has proven benefits to reducing or preventing motion sickness, the use may slow or interfere with an individual’s adaptation to motion (Parrott, 1985). The use of scopolamine, alone or in combination with dextroamphetamine, is only authorized for U.S. Air Force aircrew use during the early phases of formal training programs.

7.4.5.2.1. Scopolamine. Scopolamine is a long-lasting antiemetic that was first approved by the Food and Drug Administration for preventing motion sickness in 1979. It is a muscarinic antagonist structurally similar to the neurotransmitter acetylcholine and is a high affinity selective competitive antagonist of the G protein-coupled muscarinic receptor for acetylcholine and has both central and peripheral effects (Antor et al., 2014). Scopolamine acts on the central nervous system by blocking acetylcholine-mediated neural signals from the vestibular nuclei to higher central nervous system centers, as well as from the reticular formation to the vomiting center in the medulla (Antor et al., 2014; Parrott, 1989) (See also Section 1.5, Vestibular Physiology).

As an anti-cholinergic agent, scopolamine also acts on other autonomic nervous system functions. Potential effects include reduced visual accommodation, enlarged pupil diameter, blurred vision, decreased salivation leading to dry mouth, decreased sweating, and increased heart rate due to decreased vagal input (Parrott, 1989; Schmedtje et al., 1988). Central nervous system functions involved with committing new information to memory, sustained attention, and alertness may also be reduced (Parrott, 1989; Schmedtje et al., 1988). Drowsiness is a common side effect of scopolamine use.

The effects of scopolamine peak approximately 1 – 2 hours after oral administration, with levels returning to baseline 5 – 6 hours later (Parrott, 1989). For longer protection, scopolamine can be administered via a transdermal patch applied behind the ear. Applied in this manner, the patch administers scopolamine in a slow, continuous manner for up to 72 hours, with detectable levels in the plasma within 4 hours and a peak around 24 hours after administration (Antor, et al., 2014). With this in mind, if the transdermal patch is used for treatment of motion sickness, it must be applied several hours before exposure, with some studies recommending up to 8 hours needed for significant protection (Levy and Rapaport, 1985). After removal of the patch,
plasma scopolamine levels gradually decrease with a half-life of about 9.5 hours (Antor, et al., 2014).

7.4.5.2.2. Dextroamphetamine. To balance the depressive effects of scopolamine, the amphetamine dextroamphetamine is commonly prescribed in conjunction with scopolamine. It is a sympathomimetic and central nervous system stimulant that stimulates the release of norepinephrine from central adrenergic receptors (DrugBank). Peripheral effects include the release of noradrenaline by acting on the adrenergic nerve terminals and alpha- and beta-receptors (DrugBank).

Dextroamphetamine has stronger central nervous system effects and weaker peripheral action than the amphetamine itself (TUSOM). The increase in noradrenaline counters the activity of acetylcholine-sensitive neurons stimulated by activation of the vestibular system (Crampton, 1990). In addition, dextroamphetamine counters some of the side effects, such as drowsiness, of scopolamine.

Similar to scopolamine, dextroamphetamine is rapidly absorbed and peak plasma levels occur within 1 to 3 hours, with complete absorption approximately 4 – 6 hours after administration (TOXNET).

Dextroamphetamine is also prescribed as an anti-fatigue countermeasure, more commonly known as the “Go-Pill.”

7.4.5.2.3. Antihistamines. A few antihistamines have proven effective in the treatment of motion sickness. While not commonly used in military aviation, one of the most common commercially available antihistamines used for the prevention of motion sickness is dimenhydrinate, which is the active ingredient in the over-the-counter anti-motion sickness product Dramamine®. A typical adult dose is 50 mg, which can lead to drowsiness and minor dizziness (Crampton, 1990). It is primarily an H1-antagonist, but also possesses an anti-muscarinic effect (DrugBank). To prevent motion sickness, the first dose should be taken 30 minutes to 1 hour before you travel or begin motion activity (MedlinePlus). Another common over-the-counter product is Bonine®, which contains the active ingredient meclizine. It is also the active ingredient in the Dramamine® Less Drowsy formula.

7.4.5.3. Alternative Therapy. Alternative therapies have been thoroughly researched and time tested to prove their reliability. These include rest, hydration, diet, physical fitness, biofeedback training, progressive relaxation training, diaphragmatic breathing, and motion acclimation therapy using the Bárány chair. A brief discussion of a few techniques is presented in more detail here.

7.4.5.3.1. Diet. Poor eating habits and poor nutrition have been recognized to negatively affect pilot performance and compromise pilot alertness, motivation, and safety (Copp and Green, 1991). The importance of a good diet cannot be stressed enough, not only for physiological reasons, but also because it has been shown that the “correct” diet will also reduce stomach awareness during flight. It must be emphasized that an empty stomach will not eliminate airsickness symptoms and is counterproductive. Students should eat something at least 2 hours prior to flight but minimize or eliminate dairy products and greasy, acidic, and high fat content foods that require increased stomach acids to digest. Dairy products, especially milk and yogurt, are often higher in fat and take longer to digest, resulting in a tendency to exacerbate stomach awareness symptoms and should be avoided. Foods containing complex
carbohydrates appear to work best (Lindseth and Lindseth, 1995). Students should alter their preflight diet to reduce fats and salt as much as possible and moderate their total intake to reduce stomach awareness during flight. Table 7.4.5.3.1-1 provides some examples of complex carbohydrates that may help to reduce airsickness symptoms.

Table 7.4.5.3.1-1. Complex Carbohydrates to Help Reduce Airsickness

<table>
<thead>
<tr>
<th>Whole grain cereals, rolls, breads muffins, bagels</th>
<th>Cornbread, corn tortillas</th>
<th>Pita bread</th>
<th>Pancakes, waffles, French toast (no syrup)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pasta (no sauces)</td>
<td>Crackers, popcorn, pretzels</td>
<td>Grits, oat cereals</td>
<td>Brown rice, wild rice</td>
</tr>
<tr>
<td>Barley, bulgur wheat, millet, grains</td>
<td>All vegetables, especially: peas, beans, lentils, corn, lima beans, potatoes, sweet potatoes, squash (unless they cause gas)</td>
<td>Raisins, figs</td>
<td>Juices (non-acidic)</td>
</tr>
</tbody>
</table>

7.4.5.3.2. Head Movements and Visual Field. Minimizing excessive head movements will reduce cross coupling in the vestibular system. When semicircular canals are stimulated in a particular plane of rotation, head movements can disrupt this motion and/or introduce a new rotation, creating cross coupling. If head movements are required, leading with the eyes first and then slowly rotating the head can help minimize the conflict between visual and vestibular cues. Keeping the head back against a headrest, especially one that restricts head movement, has been shown to effectively reduce motion sickness when visual cues are present (Money, 1970).

Ensuring the visual field has minimal obstructions will help reduce the effects of this cross coupling (Shupak and Gordon, 2006). When possible, viewing the horizon is recommended to provide a stable visual reference point and aid in reducing motion sickness (Money, 1970). The more of the visual field used (i.e., the peripheral visual field as well as the central visual field) the more effective the technique. If the only visual field available is moving, or if below decks on a ship or in the back of an aircraft, it is better to close the eyes, restrict head movements, and if possible, lay down in a supine position (Money, 1970).

7.4.5.3.3. Biofeedback. Some motion sickness programs have the added capability of using biofeedback (Levy, 1981; Dobie and May, 1994). This training is usually used after all other avenues listed above have been tried and the student is still experiencing debilitating motion sickness. But, it can be helpful at any time in the program if the flight surgeon recommends it. There is no set number of biofeedback training sessions required. The sessions needed are determined on a case-by-case basis. No set Air Force wide profile has been issued; however, experience shows that helping students concentrate on rhythmic/relaxation breathing and muscle relaxation will help them to lower their arousal level. To gain the students' confidence, the session may show the students how breathing can be entrained with heart rate variability, giving them the idea that they can execute a physiological change in their body, something they might not have had any idea they could accomplish. Another physiological indication would be an increase in skin temperature, which can be related to a progressive state of relaxation. Depending upon where the electromyogram sensors have been placed, the instructor can show the student any progressive relaxation that has taken place during the training session. Profile for subsequent sessions can
concentrate on tensing and relaxing muscle groups to simulate the stresses of flying and learning new flight maneuvers.

7.4.5.3.4. Cognitive Behavior Therapy. As evaluated by Cowings and Toscano (1982), autogenic-feedback training appeared to be a successful training tool in overcoming airsickness. Jackson (1994) proposed a multimodal conceptual model of airsickness treatment involving behavior, affect, sensation, imagery, cognition, interpersonal, and drugs/biology.

7.4.5.4. Other Therapies Include Precautionary Instruction To:

1. Ensure seat height is adequate to provide sufficient visual cues to minimize mismatch between visual and vestibular cues

2. Ensure adequate hydration

3. Maximize airflow to head area

4. Minimize restrictive clothing

5. Emphasize controlling rate and depth of breathing (Fleur et al., 2003)

6. Actively fly the aircraft versus being a passenger

7. Replace negative thoughts of motion sickness with task-oriented thoughts like “crosscheck”

8. Discuss interpersonal factors to include changing instructor pilot (IP).

7.4.6. Aircrew Rotational Training Program

Motion sickness programs have in years past have been primarily used during Specialized Undergraduate Flight Training (SUFT). More recently, others have been using variations of the time-tested program to address motion sickness issues in other crew positions. The primary objective of this program is to diagnose and prevent the physiological, psychological, and behavioral phenomena whose interactions often lead to some form of airsickness. This is done through a three day regimen of combined jet flying, spinning in Bárány chair, relaxation training for stress reduction, and diaphragmatic breathing techniques. Aerospace and Operational Physiology Teams (AOPT) have been using the training profiles to assist helicopter door gunners, airborne warning and control system (AWACS) mission crew, and high altitude airdrop mission support personnel overcome debilitating airsickness.

The purpose of the US Air Force’s Aircrew Rotational Training (ART) program, one component of the overall Airsickness Management Program (AMP), is to prevent airsickness during SUPT through the timely, coordinated efforts between the flight surgeon, mental health provider, aerospace physiologist, and flying supervisors (AETCI 48-102). This can be done using a very structured ART protocol involving the use of desensitization and biofeedback. If airsickness does occur during SUPT, the student is enrolled in Phases I – III, which has proven prevention methods to rapidly stop any
further airsickness episodes. Utilization of ART is based on the principle that airsickness detracts from the ability of the student pilot to learn to fly, impairs the student’s capability to demonstrate his or her flying skill, and interferes with an instructor pilot’s (IP) ability to decide if a student has the capability to fly. It is designed to be a confidence training method that saves valuable flight training dollars and time by preventing airsickness. It is not designed to get the “weak” or “unmotivated” student through SUPT.

Aircrew Rotational Training is divided into four phases (0 – III) and is the updated version of the original Airsickness Prevention Training (APT) program designed and implemented by Giles and Lochridge in 1980 at the Euro NATO Joint Jet Pilot Training (ENJJPT) program at Sheppard Air Force Base, TX. The original APT was divided into three phases. In Phase I, a flight surgeon interviewed students having one to two episodes of airsickness and gave them strategies on how to prevent airsickness. No medication was ever administered. In Phase II, flight surgeons interviewed the students with recurrent episodes of airsickness and if the students were motivated to fly, they were sent to a psychologist for relaxation and biofeedback training. Phase III was named Behavioral Airsickness Management (BAM) and was reserved for motivated students who continued to have airsickness episodes. Flight surgeons and psychologists exposed these students to a variety of Bárány chair spin profiles during three consecutive days. During these three phases, the students were never grounded. Giles and Lochridge reported that 95% of the airsick students who entered their BAM program continued to fly in the ENJJPT program without any recurrence of airsickness (Giles and Lochridge, 1985). The majority of the remaining 5% had manifestations of apprehension. Of interest, a separate group of flight surgeons, physiologists, and psychologists used the basic BAM format designed by Giles and Lochridge on 15 more student pilots with the same success rate.

The success of the U.S. Air Force airsickness management program has been validated by other agencies that also experience motion sickness and conduct similar type programs. Since 1984, NASA has stressed the use of desensitization and biofeedback in their overall anti-space sickness prevention protocols. In addition, the British Royal Air Force (RAF) and Royal Canadian Air Force (RCAF) have incorporated airsickness desensitization into their flight training and have had excellent success. Interestingly, NASA and other organizations spend prolonged periods of time, use expensive equipment, and expend valuable flying hours in their programs. In contrast, the U.S. Air Force’s AMP requires minimal time commitment, uses inexpensive equipment, and requires no grounding or lost flying time.

**7.4.6.1. Phases of Aircrew Rotational Program.** AETCI 48-102, *Medical Management of Undergraduate Flying Training Students* and AFI 11-403, *Aerospace Physiology Training Program*, include a description in very broad terms of the four-phase program. Table 7.4.6.1-1 summarizes the four-phase program, with details on each individual phase below.
Table 7.4.6.1-1. Aircrew Rotational Program Phases

<table>
<thead>
<tr>
<th>Phase</th>
<th>Enrollment Criteria</th>
<th>Program Time</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>JSUPT Student (syllabus training)</td>
<td>1 hour</td>
</tr>
<tr>
<td>I</td>
<td>1 active or 1 passive episode in flight</td>
<td>15 – 30 minutes</td>
</tr>
<tr>
<td>II</td>
<td>2 active or 2+ passive episodes in 5 flights</td>
<td>30 min/day for 5 consecutive days</td>
</tr>
<tr>
<td>III</td>
<td>3 active episodes within 5 flights or 1 post-solo episode**</td>
<td>1 hr/day for 3 consecutive days</td>
</tr>
</tbody>
</table>

*The student must be seen and referred by a flight surgeon in order to enter Phases II and III.

** According to AETCI 36-2205v4, para 5.2.9, the SQ/CC will assess the student’s potential to complete training. If the student’s potential to graduate is high, the SQ/CC will make an entry on the AF Form 4293 (Student Activity Record) to continue the student in training. If the student’s potential to graduate is low, the commander’s review process will determine whether the student is retained in or eliminated from training.

7.4.6.1.1. Phase 0. Phase 0 constitutes an initial introduction to ART. Prior to any flying, all JSUPT students are given a briefing and literature by Flight Medicine or Aerospace and Operational Physiology personnel on the prevention of airsickness. This may include education of causes, symptoms, and prevention of airsickness in the flight environment, stress management, progressive relaxation, diaphragmatic breathing, and nutrition guidance. This training is incorporated into the physiological training syllabus and does not require any additional training hours.

7.4.6.1.2. Phase I—Initial Airsickness. Individuals experiencing their first episode of airsickness will report to Flight Medicine before their next flight to rule out possible underlying medical issues. Flight Medicine will review the airsickness episode with the individual to determine if preventative measures and techniques outlined during Phase 0 were applied both before and during flight. The student will be questioned by a flight surgeon on what phase of the flight the airsickness episode occurs, symptoms prior to the airsickness, motivation to fly, anxiety, if they had or felt any other sickness prior to the flight, and any previous history of airsickness or motion sickness. If no medical condition is found, the student will be given the anti-airsickness training again should be returned to flight status.

7.4.6.1.3. Phase II—Repeat Airsickness. Treatment for the second episode of airsickness is identical to Phase I; however, it also involves the flying squadron, aviation psychologist/mental health provider, and Aerospace and Operational Physiology personnel. Flight Medicine has the option of treating with Scop/Dex in accordance with AFI 48-123, AETCI 48-102, and AETCI 36-2205. Medications are taken 2 hours before flight. Treatment will be limited to a maximum of three consecutive sorties during training. JSUPT students will stop pharmacological treatment no later than five sorties prior to initial solo.

Flight Medicine will establish communication with the student’s flight commander and/or supervisor to ensure awareness of airsickness problems and allow tracking of the student’s progression.

The student also receives a mandatory referral to Mental Health to evaluate for manifestation of apprehension, review stress management and progressive relaxation techniques, and conduct biofeedback training (if available). Following Mental Health,
the individual will report to Aerospace and Operational Physiology to be interviewed by an ART instructor. This interview is very important because it allows the instructor to thoroughly explain alternative therapies and how they work to counteract airsickness symptoms. The majority of airsickness cases can be alleviated through alternative therapies; therefore, emphasis should be placed on these techniques during the interview.

7.4.6.1.4. Phase III—Continued Airsickness. Individuals experiencing a third episode of airsickness will report to Flight Medicine for medical evaluation. Flight Medicine has the option of treating with Scop/Dex following the same guidance listed under Phase II. The principle behind Scop/Dex is to prevent an aversion to flying based solely on airsickness. Generally, Scop/Dex will not be prescribed for less than three episodes of airsickness or in combination with Bárány chair training. It will also not be prescribed for JSUPT students within five flights of either a check ride or solo flight. If, after three consecutive sorties on Scop/Dex, the individual experiences airsickness, he/she will be enrolled in motion acclimation training at Aerospace Physiology. Following the second and third airsickness episodes, Flight Medicine has the option to either use Scop/Dex or enroll the individual in motion acclimation training.

7.4.6.2. Adaptation Training (Phase IV). Adaptation training is usually conducted by Aerospace and Operational Physiology personnel over the course of three consecutive days, using the Bárány chair as the chief training device. Each day of training or session includes two or three each 10 minute “spins.” The ultimate goal of these training spins is to allow the students to practice using the techniques taught to reduce their nausea in a safe (outside the flying) environment. It is not meant to evoke vomiting, as that would be counterproductive. The instructor must be aware of the student arousal level and capabilities at all times to avoid causing the student to vomit.

7.4.6.3. Refresher Spin. Spinning is employed at some pilot training bases, and may have different names associated with it. Refresher Spinning is different from the 3-day airsickness training in that it is only warranted after the initial 3-day program has been exhausted. The instructor will try to acclimate the student to the specific aircraft maneuvers that have been causing the airsickness. The instructor conducts an interview to nail down these specific aircraft maneuvers and recreate their vestibular sensation in the Bárány chair. This helps students employ all previously taught techniques during a close simulation of the aircraft maneuvers that have been plaguing them. Traditional Refresher Training is one spin session to re-acclimate a motion-sickness-susceptible individual after a break in flying.
References
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AETCI 48-102, Medical Management of Undergraduate Pilot Training Students. 5 November 2013.
AFI 11-403, Aerospace Physiology Training Program. 25 March 2015.
AFI 48-123, Volume 3, Medical Examinations and Standards. 5 June 2006.
Crampton GH. Motion and Space Sickness. CRC Press. 1990.

7-82
Levy RA, Jones DR, and Carlson EH. Biofeedback Rehabilitation of Airsick Aircrew. Aviat Space Environ Med 1(981) 52:118-121.
Recommended Readings

Concepts
Aircrew Rotational Training program
Airsickness management (ASM) program
Motion sickness

Vocabulary
Bárány chair
Neural mismatch
Simulator sickness
Vestibular mismatch
7.5. Impact and Ejection

John R. Buhrman, James W. Brinkley, and Jennifer L. Davis

7.5.1. Introduction

Impact acceleration is experienced in everyday life as a result of slips and falls, industrial accidents, and automotive crashes. In the Air Force these events also occur, but the most relevant exposures to impact accelerations are aircraft crashes and the multiple short-duration impact acceleration exposures experienced during an emergency escape from disabled aircraft.

It is important to consider a few fundamental definitions to more fully understand this subject. Acceleration is defined as a change in the magnitude and/or direction of the velocity of a body. The term “deceleration” simply means an acceleration that reduces an established velocity of a body. The dimension of velocity is ft/s (or m/s) and acceleration is ft/s^2 (or m/s^2). An object dropped in a vacuum at the Earth’s surface will accelerate at 1 g. The value of g is 32.2 ft/s^2 (9.81 m/s^2) by international agreement. Acceleration is often expressed in terms of multiples of the acceleration caused by the force of gravity. For example, an acceleration of 322 ft/s^2 is equal to 10 g. Confusion results from referring to “g” as a unit of force instead of acceleration.

In aviation, pilots, physiologists, and others in the field of aerospace medicine frequently use the term G. This term is the cause of the above-mentioned confusion. The unit G represents the total reactive force (mass x acceleration) divided by the body weight and resisted gravitational acceleration (mass x gravity):

\[ G = \frac{F}{W} = \frac{\text{mass} \times \text{acceleration}}{\text{mass} \times \text{gravity}} = \frac{\text{acceleration}}{\text{gravity}} \]

It is thus a dimensionless ratio of both force and acceleration. It is a vector and therefore has direction. This relationship of force and weight explains why a G meter of an aircraft at rest on a runway will indicate 1 G in the absence of any acceleration. Under sustained acceleration conditions, one can determine the amount of force by multiplying G by the weight W of the body (F = GW).

Impact acceleration is defined as a short-term or transient acceleration that is not sustained long enough to result in a significant constant or steady state component in the mechanical response by an accelerated body. The response in the human body is largely biomechanical, i.e., the accelerated body must be compressed, rearranged, or otherwise mechanically affected by the acceleration (Brinkley and Raddin, 2002). Various parts of the impacted body will experience somewhat different accelerations in response to an impact.

When describing the direction of the acceleration or the G vector, numerous coordinate systems have been used to describe physiological acceleration and the physiological reaction to acceleration, as shown in Figure 7.5.1-1 (Parmet and Gillingham, 2002). The coordinate system used here is a right-hand coordinate system that describes linear and angular acceleration directions with respect to the human body. A +z axis acceleration acts headward and parallel to the spine of an individual. An acceleration acting perpendicular to the z axis, from back to chest, is +x axis acceleration. The +y axis is mutually perpendicular to the x and y axes, with acceleration acting from right to left.
The physiological reaction to acceleration is often described within a gravito-inertial (G) coordinate system, as shown in Figure 7.5.1-1b. This coordinate system uses a backward, inverted, right-hand rule. Notice that the +Gy axis within the gravito-inertial coordinate system is not in agreement with the third law of motion with respect to the linear and angular acceleration coordinate system. This source of potential confusion makes it necessary for authors and speakers to clearly describe the coordinate system they are using.

The directions of the angular displacements, velocities, and accelerations are shown in Figure 7.5.1-1a. Note that the angular reactions in the gravito-inertial coordinate system correspond to the acceleration coordinate system. It is important to be aware that the coordinate systems used to describe acceleration or inertial response vectors usually do not correspond to the coordinate systems used in aerospace systems.

### 7.5.2. Application of Impact Force

The manner in which an impact is applied may be categorized in terms of penetrating impact, blunt impact, and whole-body impact. If the area over which the impact occurs is small and/or if the tissue is fragile, the contact force may cause penetration into the impacted body. Vehicles, seats, and personnel equipment should be designed to eliminate trauma from penetrating impact. Nonpenetrating impact applied to a specific area is referred to as blunt impact. Blunt impact is the common means of force application for restrained occupants of vehicles. The blunt impact forces are typically applied to a vehicle occupant by means of the seat surfaces, restraints, or
some combination of these factors. Localized blunt impact to inadequately restrained body parts may occur when a body part strikes a fixed surface such as an instrument panel or a seat.

Impact injury results from distortion of body structures beyond their recoverable limits. This distortion, termed strain, is a result of stress within the structures. Stress is defined as force per unit area. Strain is equal to force divided by the area of the applied force. There are different kinds of stress that can be applied to a body part. These are compression, tension, bending, shear, and torsion. Shear stress is produced by a nonaligned force-couple, which varies with cross-sectional area (Raddin, 1997). Stress is relatively independent of material characteristics. The material characteristics, however, determine what an object will do when subjected to stress. Any stress will produce some strain. The various types of strain responses to stress are formalized in dimensionless units expressing either a ratio of the change of length or a trigonometric function of the distortion angle associated with bending.

The human body responds to applied forces by a combination of acceleration and strain. Accelerations are force dependent and are determined by the ratio of force to mass. Strains or deformations are stress dependent and are determined by the ratio of force to area and by the viscoelastic properties of the affected body tissues. To avoid injury, impact acceleration must be applied over the appropriate times to produce the required velocity change while minimizing strain. Strain is associated with injury (Brinkley and Raddin, 2002). The apparent adverse effects of strain occur at the points of stress application. These include contusions, lacerations, and joint injuries. Acceleration of the body or its parts may also cause injuries at more distant locations from the point of application of force such as fractures within the spinal column during ejection acceleration.

The factors influencing human tolerance to impact acceleration are multiple. They include acceleration direction, amplitude, rise time or rate of onset, duration, velocity change, area of application of impact force, and limb flail. Human tolerance to impact acceleration has been defined in several ways. First, it may be described in terms of the maximum acceleration or applied force that does not result in significant injury. Second, it may be the point at which a volunteer subject is unwilling to continue to be subjected to higher acceleration. And third, it may be the point at which an experienced physician decides to end further exposure on the basis that measured physiological responses may be indicators of impending injury. However, as suggested by Brinkley and Raddin (2002), a given level of impact may result in no injury for one subject and death for another, so these definitions are vague at best. As mentioned earlier, the most common application of our understanding of impact acceleration is in aircraft escape systems, which will be discussed in the subsequent sections.

7.5.3. Historical Background of Emergency Escape Systems

Prior to our entry into World War II, the Germans anticipated crewmember escape from high-performance aircraft and initiated the development of a ballistic catapult ejection system, which was successfully installed in aircraft as early as 1941 (Henzel, 1967). In 1942, the German test pilot of a Heinkel HE-280V-2 prototype became the first pilot to eject to escape a crash (Tuttle, 2002). By the end of WWII, approximately 69 ejections had taken place from several different German aircraft (Billings and Treadwell, 2000). Concerned about escape from fighter planes, the U.S. Army Air Corps at Wright Field, OH, began investigating German technology such as
the Me-162 seat (Figure 7.5.3-1), and in 1946 the first live ejection in the U.S. took place when First Sgt Lawrence Lambert ejected from a P-61 aircraft at Wright-Patterson Army Air Base in Ohio (Tuttle, 2002). The U.S. Army Air Corps soon began incorporating ejection seats into aircraft such as the XP-84 (1946) and the XB-47 (1947), and in 1951 the B-52 was built to incorporate both upward and downward ejection seats. In those early ejection systems, the pilot had to manually initiate the ejections, including canopy removal, pulling the armrests up, unbuckling the seat belt, and pulling the parachute ripcord (Billings and Treadwell, 2000).

![Early Ejection Seats Included the German Me-162 and United States P-84](image)

**Figure 7.5.3-1. Early Ejection Seats Included the German Me-162 and United States P-84**

During the 1950s, ejection began to be more automated with simplified handle pull and automatic canopy jettison, lap belt release, and parachute deployment. Also during this period, so-called “zero-zero” ejection systems were being developed by Martin-Baker to provide the capability for successful escape at zero airspeed and zero altitude (ground level) (Billings and Treadwell, 2000). In 1955, test pilot George Smith became the first person to survive an ejection at supersonic speed as he abandoned an F-100 at Mach 1.05. During the 1960s, the use of encapsulated ejection seats was investigated and incorporated into the B-58 and XB-70 bombers. During the latter half of the 1960s, the F/FB-111 crew escape module system was also developed. Unfortunately, the lack of substantial low-altitude escape capability was a serious limitation of these systems. In addition, weight growth of crew escape modules due to avionics modifications caused serious parachute landing injuries.

### 7.5.4. Performance of Contemporary Ejection Seats

In the 1970s, the Advanced Concept Ejection Seat (ACES) II, shown in Figure 7.5.4-1, was developed and flight qualified and included improvements such as simplified ejection controls, automatic escape sequencing, lightweight seat structure, and seat stabilization control (Billings and Treadwell, 2000; Billings and Sadler, 2009).

When the ejection initiation controls of the ACES II ejection seat are actuated, a sequence of events is automatically initiated, including powered shoulder harness retraction, aircraft canopy jettisoning, ejection catapult ignition, and recovery system sequence power. As the seat enters the airstream, pitot tubes mounted on each side of
the headrest and parachute container and an internal static pressure barostat sense the aircraft speed and altitude. The recovery sequencer uses these measures to select the mode of operation appropriate for the environmental conditions.

Figure 7.5.4-1. ACES II Ejection Seat

As the seat separates from the ejection rails of the aircraft, a gyro-controlled rocket is ignited to control the pitch attitude of the seat. On some aircraft installations, a trajectory divergence rocket is ignited. The remainder of the recovery system sequence depends upon the mode that has been selected. The modes of ejection are shown in Figure 7.5.4-2 as functions of pressure altitude and knots equivalent airspeed (KEAS).

Figure 7.5.4-2. ACES II Ejection Seat Ejection Modes

In Mode 1, the recovery parachute deployment is initiated 0.2 s after the rocket catapult is initiated. A mortar propels the parachute container away from the seat, parachute reefing line cutters are actuated, and a recovery system pilot chute is deployed. The parachute is deployed and inflated to a reefed condition until reefing line cutters are actuated to permit full inflation. In Mode 2, as the seat approaches the top of
the rails, a drogue parachute is deployed to decelerate the seat. Then after release of
the drogue parachute, the recovery parachute is deployed and seat-occupant
separation occurs. In Mode 3, the sequence is the same as in Mode 2, except the
deployment of the recovery parachute is delayed until the seat descends or decelerates
to the Mode 3 boundary condition (McDonnell Douglas, 1992; Billings and Treadwell,
2000).

The ACES II is the seat used in the majority of USAF fighter aircraft including the
F-15, F-16, F-117, F-22, A-10, B-1, and B-2 and can now accommodate pilots in the
range of 103 – 245 lb (Moore and Hampton, 2001). During the period from 1978 –
2007, there were 431 USAF ACES II ejections, with a survivability rate of 91%. All but
three were from the F-16, F-15, A-10, and B-1B aircraft (Air Force Safety Center, 2007).
The results of ACES II ejections for the period of August 1978 through September 2007
are summarized in Table 7.5.4-1.

<table>
<thead>
<tr>
<th>Type</th>
<th>Total</th>
<th>Survived</th>
<th>Rate (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>F-16</td>
<td>281</td>
<td>261</td>
<td>93</td>
</tr>
<tr>
<td>F-15</td>
<td>75</td>
<td>69</td>
<td>92</td>
</tr>
<tr>
<td>A-10</td>
<td>52</td>
<td>42</td>
<td>81</td>
</tr>
<tr>
<td>B-1B</td>
<td>20</td>
<td>19</td>
<td>95</td>
</tr>
<tr>
<td>F-117</td>
<td>2</td>
<td>2</td>
<td>100</td>
</tr>
<tr>
<td>F-22</td>
<td>1</td>
<td>1</td>
<td>100</td>
</tr>
<tr>
<td>Total</td>
<td>431</td>
<td>394</td>
<td>91</td>
</tr>
</tbody>
</table>

Significant improvements have been made to the ACES II seat over the past two
decades. Additional improvements have been proposed but not yet implemented. The
improvements are based upon advancements in escape system technology developed
through the CREST (Crew Escape Technology) advanced development program, the
Fourth Generation Escape System Technologies Demonstration Program, and the
Cooperative Modification Program (CMP), as well as through other ACES II design
modification efforts.

These improvements have included the digital recovery sequencer, limb restraint,
windblast protection, and expanded weight range accommodation. These
improvements have resulted in an in-envelope survival rate of over 95% (Billings and
Treadwell, 2000) and a fatality rate per 100,000 flight hours that has approached zero in
recent years, as shown in Figure 7.5.4-3 (Air Force Safety Center, 2007).

In particular, the CMP addressed concerns with the ACES II seat due to the large
variation in weight and size among the aircrew population, including the headrest
position, head impact attenuation capability, inertia reel strap angles with respect to the
largest and smallest aircrew, canopy breaker position relative to the helmet on the
largest aircrew, and limb restraint and seat stability for all aircrew. A modular seat now
being tested is expected to replace the current structure with one able to be constructed
in its final design in the aircraft and includes an adjustable headrest with energy
attenuation to prevent head injuries (Ross et al., 2006).
7.5.5. **Accelerations Occurring During Emergency Escape**

An ejection is not a single event but is actually a series of events that are initiated when the crewmember pulls the ejection handle and includes rapid retraction with occupant restraint or “haulback,” ejection catapult launch, rocket acceleration, windblast exposure, parachute opening shock, and ground landing. During all these phases, the crewmember may experience potentially traumatic impact accelerations.

7.5.5.1. **Haulback.** During this phase, the upper torso is repositioned to align the spine with the back of the seat to reduce the potential of spinal injury during ejection and protect against injury caused by windblast. The crewmember is first pulled back into the seat by a powered inertia reel. During haulback into the seat, the head may impact the headrest, particularly if the occupant is wearing a weighted helmet (Pint and Buhrman, 2000). The current USAF standard requires that repositioning before ejection must be completed in less than 300 ms (Air Force MIL-R-8236F, 1995). However, more recent studies have shown that upper torso retraction can be completed safely in about 200 ms, even with 4.5 lb of helmet weight, although females may be more at risk of neck injury under these conditions (Pint, 2003).

7.5.5.2. **Catapult.** The ejection catapult acceleration occurs after the ejection sequence is initiated. It occurs in two phases. During the first phase, a ballistic charge at the base of a telescoping tube fires and propels the seat and its occupant vertically up the guide rails. The second phase occurs as the seat reaches the end of the guide rails, when a rocket is ignited to further accelerate the seat and occupant to a height to adequately clear the vertical stabilizer of the aircraft and, at low altitude, to provide adequate height for safe recovery parachute deployment and occupant descent.

The accelerative force associated with the first phase of the catapult operation acts parallel to the spinal column. An ejecting crewmember may experience anywhere
from +10 to +20 G, depending on the type of ejection seat, the mass of the crewmember, the propellant temperature, and variance in catapult performance (Raddin and Brinkley, 2005). The acceleration vector of the rocket is directed through the center of gravity of the seat and occupant combination by the rocket nozzle.

Human tolerance levels during catapult operation are understood to be just below the point where irreparable damage would occur in the most vulnerable component of the vertebral column. Studies using cadavers have shown that the vertebral endplates and vertebral bodies are the tolerance-limiting components of the axially accelerated spinal column. Cadaver studies have also shown that the thoracic-lumbar region of the spine is most prone to injury during ejection (Henzel, 1967). U.S. Air Force operational experience has supported this finding. The crucial biodynamic factors that determine the severity of loading on the vertebrae during acceleration are the rate of onset of acceleration or rise time to peak acceleration, the maximum acceleration that occurs during the length of time that peak acceleration is in effect, and the time period of the acceleration.

Neck injury due to severe cervical compression or flexion can also occur during the catapult phase of ejection. There are several nonfatal fractures that can be caused by the compression forces encountered during ejection. These fractures have been categorized as fractures of the vertebral body margins, anterior wedge fractures, lateral wedge fractures, and cleavage fractures of the centrum. The most common ejection neck injury is the anterior wedge fracture, which is classified by the collapse of the frontal part of the vertebral body and is normally in the C5-T1 region. This fracture is benign in most instances and total recovery is likely, although there may be considerable pain and discomfort that may result in at least 2 months of disability (White and Panjabi, 1990).

7.5.5.3. Windblast. As an ejection seat and its occupant are accelerated out of the cockpit during escape from a high-speed aircraft, the occupant is exposed to a combination of the ejection catapult acceleration and the aerodynamic pressure of the wind stream surrounding the aircraft, windblast. The resulting aerodynamic forces are defined with respect to the flight path of the seat. The force acting along the flight path to decelerate the seat is called drag. The upward and side forces are called lift. Forces that may cause a change in the angle of the seat and occupant are called moments. The wind stream produces drag, lift, and moments that increase as a function of the aerodynamic pressure \((q)\), which is proportional to the air density and the square of the wind stream velocity, and to a lesser degree other factors such as air viscosity and elasticity. The drag that is produced is determined by the area and shape of the seat and occupant relative to the flight path. If the aerodynamic forces and moments dislodge the arms and legs, they can be injured when the joint strength is surpassed or the long bones are fractured after coming into contact with the seat (Specker and Brinkley, 1983). In most ejection seats, only the torso is restrained, leaving the arms and legs to flail and sustain injury at high airspeeds. This has been rectified in aircraft such as the B-1 bomber and F-22 fighter by the use of arm and leg restraints that are deployed during the initial phase of the ejection sequence. But as with any safety device, the success of the feature is only manifest when the ejection sequence is initiated within the known envelope of operation.

Another potential mode of injury during the windblast phase of ejection can occur when the crewmember ejects while the aircraft is traveling at a high rate of speed, causing the resulting high wind velocity to violently push the head backwards into the
headrest (Figure 7.5.5.3-1). Serious head injuries such as concussions and brain hemorrhages can be the result. This phenomenon is more likely to occur in ACES II seats where there is a relatively large angle between the seat back and rails, such as in the F-16, and can be exacerbated by the crewmember wearing weighted helmets and not bracing the head properly against the headrest.

![Figure 7.5.5.3-1. ACES II Ejection Injury Risk](image)

The majority of USAF ACES II ejections over the past decade took place at airspeeds between 50 – 400 IAS (indicated airspeed). IAS is the airspeed read directly from the airspeed indicator on an aircraft. As shown in Table 7.5.5.3-1, the occurrence of major and fatal injuries increases at airspeeds greater than 300 – 350 IAS, with 100% of ejections at or above 450 IAS resulting in major or fatal injuries (Air Force Safety Center, 2007).

<table>
<thead>
<tr>
<th>Airspeed (IAS)</th>
<th>Total Ejections</th>
<th>Fatal Injuries</th>
<th>Major Injuries</th>
<th>% Major and Fatal Injuries</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 – 49</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>50 – 99</td>
<td>14</td>
<td>2</td>
<td>1</td>
<td>21</td>
</tr>
<tr>
<td>100 – 149</td>
<td>22</td>
<td>0</td>
<td>3</td>
<td>14</td>
</tr>
<tr>
<td>150 – 199</td>
<td>43</td>
<td>0</td>
<td>3</td>
<td>7</td>
</tr>
<tr>
<td>200 – 249</td>
<td>23</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>250 – 299</td>
<td>9</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>300 – 349</td>
<td>7</td>
<td>1</td>
<td>3</td>
<td>57</td>
</tr>
<tr>
<td>350 – 399</td>
<td>9</td>
<td>2</td>
<td>0</td>
<td>22</td>
</tr>
<tr>
<td>400 – 449</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>450 – 499</td>
<td>3</td>
<td>2</td>
<td>1</td>
<td>100</td>
</tr>
<tr>
<td>500 – 549</td>
<td>2</td>
<td>0</td>
<td>2</td>
<td>100</td>
</tr>
<tr>
<td>550 – 599</td>
<td>3</td>
<td>2</td>
<td>1</td>
<td>100</td>
</tr>
<tr>
<td>600+</td>
<td>3</td>
<td>2</td>
<td>1</td>
<td>100</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>142</strong></td>
<td><strong>11</strong></td>
<td><strong>15</strong></td>
<td><strong>18</strong></td>
</tr>
</tbody>
</table>
When the crewmember encounters windblast, extreme lift and side forces and moments acting on the head and neck can cause damage to the cervical spine (Specker and Brinkley, 1983). For example, the airflow over the curved surface of the helmet decreases the pressure above the helmet, producing a lift force as the seat enters the air stream. Air pressure entering under the helmet may add to this lift force. The lift force at very high speeds is often enough to overcome the inertial loading on the helmet due to the ejection acceleration, causing the helmet to lift up on the head and neck. This causes a large force to be applied to the crewmember's neck until the windblast diminishes or the helmet breaks away. The airflow also applies a drag force, pushing the crewmember's head backwards into the seat (Pellettiere, 2003). Recently developed neck injury probability curves predict a 5% probability of neck injury for tensile neck forces of 520 pounds for large crewmembers and 390 pounds for small crewmembers (Carter et al., 2000).

In the past, helmets were supplied with a chinstrap that could, under certain load conditions, permit the helmet to separate from the crewmember during ejection. However, new straps such as the integrated chin-nape strap (ICNS) have been designed to stabilize the helmet and improve retention (Pellettiere et al., 2005). Although the ICNS has the potential to increase helmet retention and stability when additional helmet equipment is added, tests demonstrated that increased neck loading occurred when a helmet using the ICNS was used (Pellettiere, 2003). To reduce the risk of neck injury due to helmet retention, the breakaway integrated chin-nape strap was proposed, which is designed to release the helmet after a certain point to maximize helmet retention while minimizing the risk of neck injury (Pellettiere et al., 2005).

7.5.5.4. Seat Deceleration. After the ejection seat travels vertically up the guide rails and clears the cockpit ejection rails, the seat decelerates horizontally due to aerodynamic drag of the seat and its occupant and opening of the drogue parachute attached to the seat. The drogue parachute is a small, strong parachute that inflates quickly, resulting in an immediate decelerating force (Wittendorfer, 2003). The primary functions of the drogue parachute are stabilization and deceleration under high-speed escape conditions and stabilization during descent from high altitude (McDonnell Douglas, 1992).

The horizontal deceleration of the seat resulting from the overall drag force can be as high as +44 G. In high-speed ejections, the windblast will force the occupant’s head back against the seat headrest, thus mitigating much of the head and neck flexion and neck loading due to the seat deceleration. However, at lower airspeeds, the deceleration of the seat can be sufficient to cause head and neck flexion. If hyperflexion of the neck occurs, it could generate excessive tensile loads in the neck similar to those seen during automobile frontal impacts (Pellettiere, 2003). Flexion injuries are assumed to occur when the head and neck are forced forward past normal limits and include sprains, fractures, and facet dislocations (White and Panjabi, 1990).

7.5.5.5. Parachute Opening Shock. Parachute opening shock (POS) occurs during the phase of ejection when the parachute opens and the ejecting crewmember decelerates suddenly. One of the possible effects of POS is whiplash, as occurs in car crashes. The precise means of injury during whiplash is not definite; however, it is usually considered to involve hyperextension (White and Panjabi, 1990). Symptoms such as pain, torn muscles, ligament damage, joint injuries, and bone damage have been reported during this phase (Mertz and Patrick, 1967). These
injuries are rare in the current ACES II seat, but a number of neck injuries did occur in
the escape seat that was installed on several fighter aircraft prior to their being fitted
with the ACES II seat. The potential for neck injuries also still exists in some older
ejection seats that are still in use, such as those in the B-52, which have high catapult
accelerations. This is of particular concern if the ejecting crewmember is a small female
and/or is wearing a weighted helmet system.

To avoid harmful parachute opening shock injury to the crewmember and major
damage to the personnel parachute canopy, the parachute must not be opened at high
altitude. Contemporary escape systems and parachute opening devices are designed to
prevent opening above 14,000 to 15,000 ft. As more crewmembers are equipped with
helmet-mounted devices (HMDs) that increase the neck-supported weight and alter the
head/helmet center-of-mass, the number of neck injuries due to POS throughout the Air
Force is expected to increase. To minimize the risk of these type injuries, it is
recommended that the weight of helmets with HMDs be kept under 5 pounds with
minimal shift in the center-of-gravity (Doczy et al., 2004).

7.5.5.6. Landing. Approximately 40% of all nonfatal injuries related to
ACES II ejections happen while landing (Air Force Safety Center, 2007). The principal
injuries reported due to landing impact include leg/ankle fractures and spinal fractures.
A review of F/FB-111 aircraft ejections from 1967-1980 shows that 11 out of 23 injuries
that were incurred during successful ejections by 78 crewmembers were due to ground
landing. The mechanism responsible for these injuries was deemed to be axial
compression and flexion (Hearon et al., 1981). Injuries such as severe abrasions and
fractures can also occur when the parachute and parachutist are dragged by the wind
across the terrain (Ejection Seats, 2007). While ankle injuries are the most predominant
landing injury in both civilian and military parachutists, other lower extremity injuries can
also occur and include sprains of various ligaments and muscles as well as foot and leg
bone fractures. Extreme impact forces and moments, mostly due to poor landing
techniques, cause these injuries.

The landing technique used by experienced parachutists is the parachute landing
fall (PLF), which is designed to decrease the impact force and injury by increasing the
amount of time used to absorb the impact and by distributing the impact over a larger
area of the body (Kong et al., 2002). Other factors influencing the likelihood of injury
are the parachutist’s prior ejection experience and the landing attitude with respect to
the direction of horizontal drift (Madson, 1975). Parachute landing impact injuries are
also heavily dependent on aircrew weight and descent rate.

7.5.6. Crew Protection

Other important factors influencing the likelihood of injury during ejection include
the ejection seat configuration, the crew restraint system, and the seat cushion. Before
each takeoff, the crewmember must be firmly strapped into the seat to avoid excess
body movement and seat cushion compression during an ejection. The restraint
harness and other mechanisms of reducing limb flailing must be properly attached and
fastened. The helmet visor must be in the proper position to protect the eyes, and the
helmet must be properly fitted and secured to avoid injury to the eyes, head, and neck
from windblast during ejection (Ernsting et al., 1999). Proper training in ejection
procedures will reduce many potential hazards during ejection, such as:
7.5.5.7. **Seat Configuration.** Aircraft ejection seats are configured with varying offsets between the seat back angle and the catapult rail angle that can either mitigate or increase spinal injury risk during upward ejection. For example, the ACES II seat back has a forward offset of 4° - 7° with respect to the catapult rails, depending on the type of aircraft. With seat back angle inclined forward as little as 5°, injury risk would likely increase slightly due to increased head x axis acceleration, increased head angular acceleration, and increased chest z axis acceleration (Perry et al., 1991). Conversely, studies have shown that aircraft ejection with the seat reclined 20° from the vertical rails would result in lower spinal/neck injury risk compared to the upright position, as demonstrated by lower compressive upper torso and head shear accelerations and lower seat pan force (Brinkley et al., 1981).

Another consideration is the offset of the headrest with respect to the seat back. USAF ACES II headrests are mounted in-line with the seat back, while headrests on most seats used by the Navy are mounted at least 2 inches forward from the seat back to provide pilots with better vision of the cockpit instruments during catapult launches from aircraft carriers. The forward-mounted headrests generate greater neck flexion during upward ejections, especially when the pilots are wearing helmets with forward-mounted devices, and may contribute to greater injury risk due to higher neck loads. This is consistent with laboratory test results that have demonstrated statistically significant increases in maximum horizontal head displacements in human subjects during vertical impact tests with the headrest positioned 2.25 inches forward of the seat back plane (Brinkley et al., 1982). However, a positive benefit of forward-mounted headrests is that they may act to decrease the magnitude of head impact caused by the head accelerating back from the initial neck flexion and striking the headrest. Conversely, rearward offsets of as little as 1 inch in the headrest during vertical impacts may predispose the occupant to cervical extension, possibly causing neck pain (Brinkley et al., 1982). However, Perry (2003) found that positioning the headrest 1 inch rearward while wearing heavy helmet systems allows the occupant to better control head/neck pitch during vertical impact, while not inducing significant rearward head rotation.

7.5.5.8. **Restraint System.** An effective restraint system should minimize the transmission of loads to the occupant as well as control the occupant motion with a minimum of contact stress. The lap belt alone provides a relatively low level of impact protection and may slip over the pelvis and against the abdomen, causing the belt loads to be applied against the lumbar spine. The use of shoulder straps improves injury tolerance by increasing the restraint-bearing area, increasing the load paths into the torso, and reducing the relative motion between body parts (Brinkley and Raddin, 2002). Experiments using dummies and volunteer human subjects have been carried out to assess the effect of the attachment angle of shoulder straps on human dynamic response. Shoulder harness angles of less than 0° relative to the horizontal plane of the aircraft have been determined to be a contributing factor in the spinal injury rate. A negative angle will cause vertical compression loads as a reaction...
to horizontal forces carried by the shoulder straps (Brinkley et al., 1979). A shoulder harness angle of $25^\circ$ has been shown to minimize seat pan loads and head accelerations as compared to an angle of $0^\circ$ (Kuennen et al., 2003).

Another area of concern in restraint system design is the placement of the lap belt tie-down points. Laboratory studies with human subjects have demonstrated an increase in head accelerations and seat pan forces during frontal impacts when the tie-down points are mounted directly below the seat back/seat pan intersection as compared to mounting the tie-down points 3 inches aft of this location. The greater head accelerations and seat pan forces could contribute to an increase in the risk of neck or spinal injury during the main parachute opening phase of ejection. However, this more forward location may have the beneficial effect of decreasing the potential for the occupant’s torso to slip under the lap belt, also known as “submarining” (Kuennen et al., 2003).

7.5.5.9. Seat Cushions. The seat cushion most commonly used in the ACES II ejection seat is composed of two layers: a top layer of rate-dependent foam and a bottom layer of polyethylene foam (Perry et al., 2000). Although seat cushions may provide comfort during long missions, the likelihood of vertebral fracture may increase depending on the rate of cushion deformation. The cushion can magnify the impact reaction by delaying the beginning of acceleration of the individual or by collecting and then releasing energy elastically during recoil. Either way, the individual in the seat encounters a larger change in velocity than the seat itself. Therefore, the benefits of better seat comfort must be weighed against the chance of spinal injury (Hearon and Brinkley, 1986). Vertical impact testing has shown that rate-dependent foam cushions transmit less energy because of their ability to reduce the amplification of impact acceleration as well as supply a comfortable seat cushion. The safety margin of the cushion is dependent upon both the cushion material and the cushion configuration (Cheng and Pellettiere, 2004).

7.5.7. Impact Injury Criteria

7.5.7.1. Spinal Injury Criteria. When the first ejection seats were developed, ejection catapult performance was evaluated in terms of the rate of onset of the acceleration and the maximum acceleration. This was a simple means of preventing spinal injury. However, as new escape systems such as the B-58 capsule and spacecraft such as the Mercury capsule were developed, the complex acceleration-time histories that were produced during the operation of these systems did not lend themselves well to simple characterization in those terms, or in the trapezoidal profiles that were suggested by others. As a result of the combination of this problem, experimental efforts, and an improved understanding of biomechanics, relatively simple mathematical models were proposed (Stech and Payne, 1969). These models involved the use of dynamic mechanical models to simulate the response of the human body to short-duration acceleration and to estimate the probability of injury. The Dynamic Response Index (DRI) model is one such model developed to estimate the probability of compressive fractures of the lumbar and thoracic spinal column due to upward acceleration during ejection. The model is a simple linear, lumped parameter mechanical model consisting of a mass, spring, and viscous damper. The dynamic response of the model is computed using its mathematical analog, a second order, linear differential equation, and then related to the risk of spinal injury.
The DRI model properties, damping coefficient ratio, and undamped natural frequency are based upon vibration and impact tests with volunteer subjects. These calculations are based on engineering formulae and are used in determining injury criteria during investigations of survivable crashes. The probability of spinal fracture was initially based upon the results of compression tests of cadaver vertebrae. The model was then validated by comparing the results of the analyses of ejection catapult accelerations of specific ejection seats to the injury rates experienced operationally by those specific escape systems (Brinkley and Shaffer, 1970). The validation process demonstrated that the operational injury rates were lower than predicted using vertebrae from cadavers. However, the probability distribution from the vertebral tests was found to be reasonable. The model and the resulting relationship between spinal injury and the value of the DRI are shown in Figure 7.5.7.1-1.

![Dynamic Response Index (DRI) Model and Spinal Injury Rate](image)

**Figure 7.5.7.1-1. Dynamic Response Index (DRI) Model and Spinal Injury Rate**

A similar approach was used to develop models for the other primary axes. Estimates for the likelihood of injury were made on the basis of experimentation with volunteers as well as accidental injuries (Brinkley et al., 1990). These models were then used to develop a method to evaluate multiaxial acceleration exposures. Each model was developed to compute a dynamic response (DR) value for each of the primary acceleration vectors (x, y, or z), which could then be compared to preestablished injury risk levels (DR risk limit values) as shown in Table 7.5.7.1-1. The injury risk values were developed for the case where a seat occupant is restrained by a lap and two shoulder straps and a negative-g strap or similar restraint system configuration.

<table>
<thead>
<tr>
<th>+DR₂</th>
<th>-DR₂</th>
<th>+DRₓ</th>
<th>-DRₓ</th>
<th>DRᵧ</th>
<th>Risk Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>15.2</td>
<td>13.4</td>
<td>35</td>
<td>28</td>
<td>14</td>
<td>Low</td>
</tr>
<tr>
<td>18.0</td>
<td>16.5</td>
<td>40</td>
<td>35</td>
<td>17</td>
<td>Moderate</td>
</tr>
<tr>
<td>22.8</td>
<td>20.4</td>
<td>46</td>
<td>46</td>
<td>22</td>
<td>High</td>
</tr>
</tbody>
</table>

Table 7.5.7.1-1. Multiaxial Dynamic Response Injury Risk Values
7.5.7.2. **Multiaxial Dynamic Response Criteria.** The multiaxial whole-body impact exposure limit method that was developed by the Air Force is known as the Multiaxis Dynamic Response Criteria (MDRC). The overall injury risk of multidirectional impact acceleration is obtained by dividing the computed DR values for each orthogonal axis (x, y, and z) by the DR limits, then squaring each result and taking the square root of the sum.

\[
\beta = \left[ \frac{DR_x}{DR_{XL}} \right]^2 + \left[ \frac{DR_y}{DR_{YL}} \right]^2 + \left[ \frac{DR_z}{DR_{ZL}} \right]^2\right]^{1/2}
\]

The multiaxial acceleration of an ejection seat or a spacecraft seat and restraint system is determined to have surpassed the specific injury-risk level if the calculated value of the MDRC is greater than 1.0 (Brinkley et al., 1990). Where injuries are of concern from extremity motion or blunt impact, other methods must be used to evaluate the likelihood of injury. The effects of impact acceleration of the head and neck are of concern. Methods that are used to evaluate the effects of automotive crash should be considered, but not without serious consideration of the differences between the automotive crash environment and that of the aerospace application. These include the impact profile, impact direction, seat, and restraint system. Clearly, there are major differences. Although the criteria cannot be applied in terms of absolute limits, the criteria may be of value when comparing alternative protection systems.

7.5.7.3. **Automotive Crash Neck Injury Criteria.** A method used to calculate automotive neck injury criteria is the Nij method, developed by the National Highway Traffic Safety Administration (NHTSA). The neck injury criteria are composed of tolerance limits for axial loads (tension and compression) and bending moments (flexion and extension) determined in crash tests using dummies. These criteria are referred to as Nij criteria, where the “i” represents either tension or compression and the “j” represents flexion or extension. The Nij value must be less than or equal to 1.0 to have an acceptable load and bending moment. An Nij value of 1.0 compares to about a 30% risk of serious injury (Kleinberger et al., 1998). These criteria are used mainly for evaluating the neck loads experienced by instrumented manikins during frontal automobile crash tests. A version of the Nij method has also been used to evaluate effects of accelerations experienced during tests of aircraft ejection seats. The Air Force typically uses a lower Nij criteria value of 0.5, which corresponds to an injury risk of 5%, when conducting simulated ejection tests.

7.5.7.4. **Head Injury Criteria.** The method used by NHTSA to determine the likelihood of skull fracture from direct impact with structures such as an automobile instrument panel is the Head Injury Criterion (HIC). This form of head injury criterion was first established by Gadd (1966). The weighted-impulse criteria were first used as the head impact Gadd severity index (SI), a means to describe the severity of a head impact with respect to the Wayne State Tolerance Curve (WSTC). The WSTC was developed by dropping cadaver heads onto flat surfaces. It provided a technique to establish a relationship between peak acceleration, pulse duration, and the likelihood of skull fracture and concussion that result from a linear impact. After the initial use of the SI, it was found that a more practical means of computation of the criteria was necessary. This resulted in the development of the HIC. The critical value of HIC is...
1000, computed from accelerations measured within the head of a mid-sized male automotive test dummy over a 36 ms time duration (Kleinberger et al., 1998). This HIC value has been related to a probability of skull fracture of approximately 48% (Hertz, 1993). A plot showing injury risk as a function of HIC values is shown in Figure 7.5.7.4-1.

![Injury Risk Curve for the HIC](image)

**Figure 7.5.7.4-1. Injury Risk Curve for the HIC (Kleinberger et al., 1998)**

### 7.5.8. Additional Injury Risk Factors

#### 7.5.8.1. Gender

The Air Force pilot population now includes both males and females with a weight range of 103 – 245 pounds and height range of 64 – 77 inches (Figure 7.5.8.1-1), leading to concerns about both cockpit accommodation, which includes aspects such as overhead clearance, leg clearance, control stick operation, visual field, etc. (Kennedy and Zehner, 1995), and ejection injury risk to small occupants. Studies conducted at the Air Force Research Laboratory (AFRL) have determined that the biodynamic responses of males and females are not significantly different during either compression (Buhrman and Mosher, 1999) or frontal impact (Buhrman and Perry, 2000) and that the vertebral stress experienced in the thoracic-lumbar region is comparable for both genders during compression. Therefore, the parameters and probability curves used to calculate the Air Force’s DRI injury criteria for lower spinal injury are probably valid for both genders. However, females do appear to be more at risk of neck or cervical spinal injury during impact acceleration events, such as those occurring during aircraft ejections. This is primarily a function of the higher dynamic stresses generated during ejection on their cervical vertebrae, resulting primarily from their smaller vertebral cross-sectional areas (Gallagher et al., 2007). Studies have also shown that females have less neck flexor and extensor strength when compared to males (Foust et al., 1973; Morris and Popper, 1996), which makes them less able to offset neck loading during preimpact bracing and, therefore, less able to withstand shear forces during -Gx impact accelerations (Doczy et al., 2004).
7.5.8.2. Helmet-Mounted Systems (HMS). With the Air Force’s emphasis on improving and developing new warfighting technologies, it is important to consider the effects of HMS during ejection. Helmet-mounted systems can include integrated helmet-mounted cueing systems, helmet-mounted night vision devices, and helmet-mounted aircraft display systems that may provide improved aircraft flight control, target acquisition, and weapon delivery. With the addition of one or more of these HMS, the helmet’s inertial properties (weight, center of gravity, and moment of inertia) may be altered. If these HMS are being used during ejection, the possibility exists that the helmet’s inertial properties will increase the rate of ejection-related neck injuries by increasing the dynamic forces generated in the cervical spine. The HMS may also affect the pilot’s performance by increasing the fatigue of the posterior neck muscles that balance the head and affect the fit and comfort of the helmet (Perry and Buhrman, 1996).

Helmets weighing more than 5 pounds with severe forward center-of-mass are not recommended due to the increased risk of neck injury and neck fatigue (Perry, 1998; Doczy et al., 2004; Caldwell and Gallagher, 2006). Current operational systems include the ANVIS 49/49, the joint helmet-mounted cueing system (JHMCS), and the panoramic night vision goggles (Figure 7.5.8-2). Some of these helmet systems approach or slightly exceed the above recommended limits. AFRL is actively evaluating the safety, comfort, and performance effects of wearing these and other helmet systems, as well as investigating the potential for long-term chronic neck pain and injury in pilots wearing HMS over several months or years.
References


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Concepts
   Haulback
   Restraint system

Vocabulary
   Acceleration
   Advanced concept ejection seat (ACES II)
   Ejection catapult acceleration
   Impact acceleration
   Parachute landing fall (PLF)
   Parachute opening shock
   Windblast
7.6. Crash Survival

Andrew D. Woodrow, Lt Col, USAF (Ret), BSC

“We must never forget that we are working with men of flesh and blood and nerves.” Major General Douglas Haig, 1903.

7.6.1. Aircraft Crashes

If ejection (see Section 7.5. Impact and Ejection) is not an option, occupants are only protected during an aircraft crash by aircraft design criteria and nature of the crash. The overriding requirement for all aircraft design is that of safety. The order of safety required is specified in the Airworthiness Requirements published by the U.S. Federal Aviation Regulations (FAR) and the European Joint Airworthiness Requirements (JAR). Despite all efforts to maintain safety in flight, some aircraft and crews fail, resulting in a crash. No two aircraft crashes are alike; one may be due to a loss of power and result in low velocity impact with ground, while others may be high speed impact with the water. Still others may relate to loss of control and in-flight break-up of the fuselage or a very hard landing resulting in airframe damage and occupant injury. In all cases, concern for protection of the passengers and crew is the primary goal of design and operation of aircraft.

7.6.2. Prior Planning to Reduce Injury during Unaided Escape

During planning for a flight, both crewmembers and passengers should consider some precautions and procedures that would increase their chance of surviving a crash.

- Consider the route of flight as well as the destination and plan clothing choices accordingly.
  - If route is over cold environments, bring a jacket onboard.
  - Avoid non-protective clothing such as nylon, silk, or polyester.
  - Choose cotton, wool, or, even better, Nomex® or other flame-retardant material.
  - Wear only flight-certified boots.

- Listen to the briefing.
  - Know the escape routes, primary and alternate.
  - Keep materials that could cause injury strapped in or as confined as practical.

- Keep seat belts fastened whenever movement about the cabin is not necessary.

7.6.3. Factors that Impede Escape and Survival

Statistical accident analyses have shown that the probability of aircraft crashes has diminished dramatically in the past decade; however, the percentage of persons killed in fire-related accidents has remained constant, at about 15% - 16% over the past
35 years (Taylor, 1989). After the initiation of an open fire in the cabin, heat, smoke, and toxic gases are produced, which are composed of soot and partially burnt hydrocarbons, carbon monoxide, fluorine, and cyanide compounds. Along with respiratory difficulties, the passengers will also face degraded visibility in the cabin; transmission values of 20% - 30% at 1.1 meters above the floor can occur within the first 2 minutes following initiation of the fire (Winterfeld, 1992). Within 3 minutes, interactions are strong enough to incapacitate due to skin burns, loss of consciousness, or visual obstruction. External fires can also impede escape and evacuation. Evidence from aircraft accidents show that people react in a very competitive manner when confronted with a survival situation. Evidence suggests that this may disrupt orderly evacuation, especially in circumstances when visibility is reduced and there are obstructions like seats, bulkheads, cargo/baggage, or other passengers in the way. In military single- or dual-seat aircraft, there is the problem of an unassisted escape from an environment encumbered by additional restraint systems.

Another factor that influences the chance of survival or injury following a crash is the physical damage to the airframe or exit. Once impact is made, the frame of the fuselage may bend or warp, causing a reduced living space for occupants or an exit that cannot be operated. Evidence from wide-body aircraft crashes that resulted in multiple head and neck injuries reveals that the hull of the aircraft can flex or fracture into the livable space, yet spring back to near normal shape at the end of the crash pulse. Exits, whether a hinged door, hatch, or a canopy that raises and lowers, can be made useless by heat or structural damage. In cases of water landings or ditchings, there is often precious little warning time prior to impact. Accident investigation analysis indicates that in upwards of 80% of sea ditching accidents, the aircrew had less than 15 seconds warning. A typical example of short warning time comes from a Canadian Forces Sea King mission:

The Sea King took-off for a water landing training mission. After initial sequences had been demonstrated including a single engine take-off, the student pilot then carried out a single engine landing and attempted a single engine take-off by himself. The take-off had to be aborted due to low rotor rpm and when rotor rpm recovered, the instructor directed the student pilot to continue the take-off. During the second attempt, rotor rpm again dropped before an abort could be executed, the aircraft struck the water nose down at approximately 20 – 25 knots ground speed. The aircraft pitched forward, severing its tail pylon, and came to rest inverted in 20 feet of water.

Because the engine and transmission of a helicopter are on the top of the fuselage, the problem of capsizing compounds the efforts of safe escape. The fact that water, usually cold, rushes into the cabin and creates a darkened environment can lead to disorientation during escape. Additionally, if the occupants are not wearing protective clothing or life vests and are unsecured from harnesses or restraint systems prior to impact, the effectiveness of finding and using an emergency exit or egress point is reduced. In fixed wing examples, like the ditching of the Airbus 340 in the Hudson River in January 2009, the aircraft often floats well, providing more time for passengers and crew to find an exit and safely egress the aircraft. It is interesting to note that few, if any, of the passengers or crew wore a life vest or exited with the seat flotation cushion despite a highly likely egress into water.
In any case of emergency egress from an aircraft, the element that often leads to failed escape is “rule-based behavior.” Every set of events that can be anticipated has been considered and reduced to a set of procedures or rule-based behaviors for the crew or passengers to follow (Green, 1996). These are not motor memory actions but rather a series of preconditioned “rules” formed and stored in long-term memory. Unlike other aircraft emergency situations that may afford time to reference documented procedures, aircrew practice emergency procedures such as egress and escape to minimize the time to respond. One of the most common rule-based behaviors observed during egress of nonaircrew on multiplace aircraft is the use of the primary exit rather than the closest exit, a result of using a single entry point in 99% of all previous flight experiences.

Although the ditching depicted in Figure 7.6.3-1 was highly successful, the physiologic hazards during any aided or unaided egress can be considerable. Injury is likely during any crash, and mobility is decreased accordingly. The presence of water, especially very cold water, reduces the chances of survival even if egress is successful due to hypothermia and drowning. A crash on land can add the physiologic hazards of temperature extremes, terrain altitude producing hypoxia, and an inhospitable environment that does not provide adequate water or access for rescue and treatment of injury.

![Figure 7.6.3-1. US Airways Successfully Ditched in Hudson River, 2009](image)

**Figure 7.6.3-1.** US Airways Successfully Ditched in Hudson River, 2009

### 7.6.4. Human Tolerance to Impact: Proper Positioning During Impact

No discussion on body position during impact can be complete without a review of human tolerance to crash forces. The most common cause of injury during accidents is the very abrupt deceleration that occurs when an aircraft strikes the ground or water. On impact a passenger or crewmember seated in an intact portion of the aircraft is propelled forwards. Any object in the path of the body or limb will become a hazard. For example, if the knees strike the bottom of the seat in front, initial injuries to the knee, tibia, and femur often result. The impact forces are transmitted up the femur, driving it into the pelvis and resulting in pelvic fractures. Any lower limb injury that prevents a person from walking to an exit has a negative influence on successful egress. In terms of the physics of impact forces, the primary concern is centered on biodynamics, the response of the body to the magnitude, duration, and direction of acceleration. The
kinetic energy that passes through the aircraft and internal objects will quickly exceed limits of strength and result in damage or injury.

**MAGNITUDE** is expressed as G, or multiples of the acceleration due to gravity.

**DURATION** (in crash analysis) is measured in fractions of a second.

**DIRECTION** is more complex because of the three axes of force but can be calculated using known conditions (e.g., forward facing, vertical impact).

In some cases the forces that reach the occupant are less than those applied to the airframe. Each aircraft part that collapses takes up some of the force, so peak G is reduced, helping to reduce the chance of injury. If any part of the restraint system allows movement of the occupant (head or limbs), protection from striking hard objects in the immediate environment must be designed. Investigations in the past have led to direct enhancements to the design of helmets, harnesses, and other personal protective equipment and the development of more crashworthy airframes.

The overall objective of designing for crash resistance is to eliminate injuries and fatalities in relatively mild impacts and minimize them in all severe but survivable mishaps. A crash-resistant aircraft will also reduce aircraft crash impact damage. By minimizing personnel and material losses due to crash impact, crash resistance conserves resources, is a positive morale factor, and improves the effectiveness of the fleet both in peacetime and in war. Results from analyses and research have shown that the relatively small cost in dollars and weight of including crash resistance features is a wise investment.

One accident that attracted attention from the National Transportation Safety Board, the Federal Aviation Administration, the Air Accident Investigation Board (UK), the aircraft manufacturer, and the seat manufacturer was the B-737 crash in 1985 at Manchester Airport in the UK (Figure 7.6.4-1). Of the 126 occupants, 47 died as a result of the accident and a further 74 suffered serious injury (Carter, 1992). Although the aircraft sustained an impact lasting 2.2 seconds, the impact was broken into two segments, the first at the tail and the second as the nose impacted an upward embankment, resulting in the fuselage breaking in half and experiencing massive crushing. The causes of mortality and mechanical injury have been identified as crushing within a collapsing airframe, entrapment within the wreckage, being struck with loose objects, and absence or failure of restraint systems.

![Figure 7.6.4-1. Tail Section of British Midland B737 at Manchester Airport](image-url)
To provide as much occupant protection as possible, a systems approach to crash resistance must be followed. The systems approach to crash resistance means that the landing gear, aircraft structure, and occupant seats must all be designed to work together to absorb the aircraft kinetic energy and slow the occupants to rest without injurious loading. In addition, the occupants must all be restrained and a protective structural shell maintained around the occupied areas during a crash to provide a livable volume. Weapon sights, cyclic controls, glare shields, instrument panels, armor panels, and aircraft structure must be delethalized if they lie within the strike envelope of the occupant. Postcrash hazards, such as fire, entrapment, drowning, emergency egress, and rescue, must also be considered in an effective crash-resistant design.

Results of research on tolerance of the human body to impact forces are also discussed in Section 7.5, Impact and Ejection. Although numerous experiments have been conducted and a wealth of information has been collected, very few criteria that may be useful in system design have been developed and validated. Those criteria that are generally accepted for practical application in assessing the crash resistance of an aircraft system were reviewed by Zimmermann and Merritt (1989). As discussed here, these criteria may be used to determine the acceptability of an aircraft or components, such as seats and restraint systems, based on the results of dynamic testing with anthropomorphic dummies or computer simulation.

A crash can involve a wide range of dynamic conditions, from a simple unidirectional impact to a complex combination of rotational and multidirectional impact conditions. Performance requirements under crash impact conditions for Army light fixed- and rotary-wing aircraft were reviewed by Zimmermann and Merritt (1989).

7.6.5. Attaching the Human to the Aircraft

One of the most important aspects of safety during crashes is the manner a passenger is harnessed to the aircraft, generally on a seat secured with a restraint system. Although military aircraft seats are not designed with maximum comfort in mind, it is worth a quick look at the evolution of the aircraft seat (Figure 7.6.5-1).

Figure 7.6.5-1. C-130 Web Seating with Aluminum Supports (Left) and High-Density Foam Seats with Leather Exterior (Right)
As air travel expanded through the 1920s and the commercial airline industry realized that revenue depended on returning customers, amenities driven at comfort took a “front” seat in the overall design of the aircraft. Safety and comfort are part of the design considerations for military aircraft, although comfort may take a “back seat” in many designs. Military aircraft are traditionally designed to a particular set of requirements that maximize payload while enduring rugged environments and conditions (Table 7.6.5-1).

Table 7.6.5-1. Seat User Groups with Physical and Cognitive Needs and Expectations (from Kovarik, 1999)

<table>
<thead>
<tr>
<th>Seat User</th>
<th>Physical Needs and Expectations</th>
<th>Cognitive Needs and Expectations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Passenger</td>
<td>Safety and comfort&lt;br&gt;Protection during survivable crash</td>
<td>Can easily locate seat in aircraft&lt;br&gt;Understand how to operate restraint system</td>
</tr>
<tr>
<td>Crew</td>
<td>Can reach and operate all controls&lt;br&gt;Protection during survivable crash</td>
<td>Know how to make minor adjustments and repairs in flight&lt;br&gt;Know how to operate harnesses and stow seat</td>
</tr>
<tr>
<td>Maintenance</td>
<td>Safely clean and maintain seats&lt;br&gt;Scheduled maintenance and repairs can be performed safely and easily</td>
<td>Procedures to repair easy to understand?&lt;br&gt;Maintenance procedures with standard assembly and tools?</td>
</tr>
</tbody>
</table>

Location of aircraft doors and hatches is a technical issue such as body loads, wing and engine mounts, limitations on escape slides, and configuration of the seats (Figure 7.6.5-2). Regulatory requirements that guide certification authorities and designers are found in a series of Federal Air Regulations (FAR). Mission, certification, and standard requirements for aircraft ensure at least the minimum safety equipment and features are designed into the cabin. For instance, FAR 25.562, Emergency Landing Dynamic Conditions, states:

(a) The seat and restraint system in the airplane must be designed as prescribed in this section to protect each occupant during an emergency landing condition when:

(1) Proper use is made of seats, safety belts, and shoulder harnesses provided for in the design; and

(2) The occupant is exposed to loads resulting from the conditions prescribed in this section.

(b) Each seat type design approved for crew or passenger occupancy during takeoff and landing must successfully complete dynamic tests or be demonstrated by rational analysis based on dynamic tests of a similar type seat, in accordance with each of the following emergency landing conditions. The tests must be conducted with an occupant.
simulated by a 170-pound anthropomorphic test dummy, as defined by 49 CFR Part 572, Subpart B, or its equivalent, sitting in the normal upright position.

Figure 7.6.5-2. Singapore Airlines A-380 Configuration for 471 Crew and Passengers

From a design and configuration standpoint, survival from crashes is highly dependent upon the number of passengers, location and availability of exits (FAR 25.803, Emergency Evacuation; FAR 25.807, Emergency Passenger Exits; and FAR 25.813, Emergency Exit Access). Any design trade-offs for comfort must be considered against the minimum safety and survivability requirements.

7.6.6. Design Principles for Personal Restraint Systems

Restraint harnesses for personnel should provide the restraint necessary to prevent injuries to all aircraft occupants in crash conditions approaching the upper limits of survivability. Appropriate strength analysis and tests (Zimmermann and Merritt, 1989) should be conducted to ensure that a restraint system is acceptable (Figure 7.6.6-1). Qualities that a harness should possess are listed below:

- It should be comfortable and light in weight.
- It should be easy for the occupant to put on and take off even in the dark.
- It should contain a single-point release system, easy to operate with one (either) hand since a debilitated person might have difficulty in releasing more than one buckle with a specific hand. Also, it should be protected from inadvertent release, e.g., caused by the buckle being struck by the cyclic control or by inertial loading.
• It should provide personnel with freedom of movement to operate the aircraft controls. This requirement necessitates the use of an inertial reel in conjunction with the shoulder harness.

• It should provide sufficient restraint in all directions to prevent injury due to decelerative forces in a survivable crash.

• The webbing should provide a maximal area, consistent with the weight and comfort, for force distribution in the upper torso and pelvic regions and should be of low elongation under load to minimize dynamic overshoot.

![Image](image_url)

**Figure 7.6.6-1. Seat Restraint Testing Completed with Crash Dummies or Computer Simulations Prior to Operational Test and Evaluation**

**7.6.7. Escape from Aircraft**

As aircraft speeds and operational altitudes have risen, so, too, has the need to escape from the aircraft using assisted measures. The means for escape must be available at all times and must take into account the forces that may be operating on the aircraft, e.g., aerodynamic, accelerations, and rotations. Most high-performance military and training aircraft have ejection systems engineered into the cockpit (see Section 7.5. Impact and Ejection). These systems use mechanical power for the escapee to leave the aircraft, whereas earlier, unassisted escape systems simply relied on physical strength. Ejection systems must provide sufficient thrust to eject the occupant clear of the aircraft structure at all reasonable operational speeds and attitudes and provide sufficient ground clearance to enable full deployment and inflation of the main parachute before ground impact. Modern systems are typically designed to be fully automatic once the ejection sequence is begun. Data analyses of RAF aircrew have shown a reduction in spinal injury rates compared with a previous RAF study of accidents where nonrocket-assisted ejection seats were used. In the earlier study the observed percentages of aircrew who sustained spinal fractures from RAF nonrocket-assisted seats were between 39% - 69%. The current analysis demonstrates that the modification of the acceleration profile by the addition of the rocket motor has
decreased the number of aircrew who have sustained spinal fractures (Lewis, 2006). There have been some attempts at engineering an automated system that would initiate the sequence if the occupant was unresponsive and dangerously close to the ground, but to date acceptance from the pilot community has been limited. The system should restrain the occupant sufficiently and modulate any forces on the body so that the risk of injury is minimized (Ernsting, 1999).

The initiation mechanism and ejection from the cockpit apply accelerations in excess of 12 G for up to 500 ms with an onset rate up to 300 G/s. The restraint system is of vital importance, as it retains the position of the escapee. A properly fitted restraint system increases the coupling with the seat and should minimize the possibility of “dynamic overshoot.” This is when the person sits on an elastic cushion, and as the seat accelerates the cushion depresses until it is fully compressed. The seat can then impart a sudden high energy force resulting in a very high-amplitude, short-duration impact, which may cause injury. Ideally the seat and occupant should be attached rigidly to each other so that the coupling moves as a single mass. This is impractical, and so, invariably, a well-damped thin foam pad is used between the occupant and the seat (Davis et al., 2008; Ernsting et al., 1999).

Another concern in aircraft escape, aided or unaided, is windblast. Within 0.2 second of the first movement up the rails of the ejection system, the seat and occupant are subjected to the full windblast while still traveling at approximately the same speed as the aircraft. The threshold of injury for blast is probably about 4.5 psi – equivalent airspeed of 440 knots – while serious damage occurs at about 8 to 9 psi – equivalent airspeed of 582 – 620 knots (Ernsting et al., 1999). Theoretically, very high blast pressures could lead to rupture of internal organs and death. The highest speed ejection in recent USAF record was from an F-15E; both the pilot and weapons system officer ejected at over 700 knots, and while the pilot survived with multiple severe injuries, the weapons system officer perished due to flailing injuries.

The precise procedure for bail out or unaided escape varies with the configuration of hatches and type of aircraft, but certain principles must be observed if the escape is to be successful. The first requirement is that the aircrew are correctly fitted with appropriate equipment and understand its operation and use. This is typically going to include a helmet, visor or goggles, parachute, and harness. They must be familiar with the escape sequence and know the location and method of egress from emergency exits, escape hatches, tunnels, or chutes. Unless these drills are practiced, escapees may not only prejudice their own chances of escape but may also impede the progress of others (also see Section 8.2. High Altitude Mission Support) (Davis et al., 2008; Ernsting et al., 1999).

### 7.6.8. Parachutes and Associated Equipment

The design of the parachute harness varies according to its use. Some harnesses are installed as part of the seat, some are fitted as part of the aircrew clothing, and some are a separate bailout system worn throughout the flight or only during an emergency. The actual designs vary, but they all have similar elements. The harnesses are made from webbing straps, usually nylon approximately 50 mm wide, which spread the load. The straps are routed to support the wearer comfortably beneath a deployed parachute with a sling passing under the buttocks and around the top of the thighs. Vertical loops pass up the back and over the shoulders to a central fitting. Some degree of lateral restraint is necessary. The harness is extended upwards.
from the shoulders to suspension strops, which connect to the parachute. Four of these are usually fitted to each harness, two each side of the body (Ernsting et al., 1999).

Parachutes must be fitted correctly, deploy appropriately, and have the function of steering built into the risers for most effective injury reduction. Without steering lines, parachutes are difficult to maneuver. Steerable parachutes allow aircrew to avoid obstacles in the descent path. There have been a number of ejections where aircrew had been unable to or did not attempt to steer their parachute and as a result sustained lower limb fractures. Well-designed equipment, adequate time during an emergency to deploy survival equipment, and the appropriate training to effectively and safely use the equipment are essential to preventing injury during emergency egress. Mishap investigations and data analysis have led to tremendous advances in design modifications and improvements in equipment performance, aircrew procedures, and industry standards aimed at reducing injuries and increasing survivability during egress.

References


7.7. Laser Principles and Awareness

Martin Johnson, BSEE, CLSO and Lt Col Ryan W. Maresh, USAF, BSc, Ph.D.

LOS ANGELES – November 2009. A Southern California man who aimed a laser beam at two airliners as they approached an airport has been sentenced to 2 ½ years in federal prison for disrupting the flights (Associated Press, 2009). The U.S. Attorney’s Office in Los Angeles says Dana Christian Welch of Orange, who was sentenced Monday, was the first person in the U.S. to be convicted at trial of interfering with pilots by aiming lasers at their planes. Authorities say the 37-year-old aimed a handheld laser at two Boeing jets as the passenger planes were about to land at John Wayne Airport on the night of May 21, 2008. The laser beam struck one pilot in the eye, causing “flash blindness,” and interfered with pilots’ ability to land the other plane.

A US News and World Report article from June 2014, revealed the following facts: There were fewer than 300 reports of aircraft being targeted by lasers during 2005. By 2013, that number had risen to nearly 4,000 (Soergel, 2014; Brown, 2014). In response to this alarming trend, the Federal Bureau of Investigation (FBI) instituted a targeted program to combat lasing of aircraft near major airports around the country. The program offered rewards of up to $10,000 for information leading to the arrest of individuals using lasers to illuminate aircraft (FBI Press Release, 2014). The FBI credited this program with the recent sentencing of a Clovis California man to 14 years in prison for repeatedly lasing two helicopters with an extremely powerful handheld laser. As of December 2013, the FBI has confirmed at least 141 arrests, 107 cases forwarded for prosecution, and 84 convictions related to aircraft lasing incidents (Soergel, 2014).

7.7.1. Background

These articles demonstrate not only the alarming rise in the number of aircraft/laser interactions, but they also underline the seriousness with which the Federal Aviation Administration (FAA) and the FBI treat these incidents. Civilian and law enforcement pilots are now being targeted by individuals using handheld lasers in an attempt to disrupt operations, or possibly “just for fun.” Military pilots flying in civilian airspace are experiencing similar encounters with lasers. In addition, military pilots must also be prepared for lasers used on the battlefield. These lasers may be used for targeting in an attempt to damage the aircraft or weapons payload, or as “dazzle lasers” intended to disrupt or degrade flight operations. Lasers are currently used as defensive measures by the US military, and they have even been posited as offensive weapons (Freedberg, 2015). No matter what the intent of the laser/aircraft interaction, flight crews need to understand the threat various types of lasers pose to ensure not only mission effectiveness but crew/aircraft survival.

The importance of the emerging technology of laser weapons and protection stimulated creation of the Directed Energy Task Force by the Air Force with emphasis on protection of airmen and sensors. The yearlong Directed Energy Net Assessment study completed in 2009 assessed threats from a wide variety of sources ranging from near-peer nation military assets to commercial-off-the-shelf (COTS) threats from less technologically sophisticated adversaries (Scott D and Robie DL, 2009). Commercial and military aircrews are regularly reporting laser exposure incidents. According to the FAA, only 16.5% of laser cockpit illuminations occurred below 2,000 feet AGL; however,
these incidents accounted for 31% of all visual effects, 42% of all pain and injuries, and 42% of all operational problems (Nakagawara et al., no date)!

The technology is available to counter these dazzling or lethal systems with aircrew laser eye protection, sensor protection filters, and energy limiters (also known as neutral density, or ND, filters). In addition, the FAA has posted a series of recommendations for aircrew that are illuminated by lasers (Nakagawara et al., no date). These actions can range from changing flight profiles to increasing cockpit illumination. With their effect on aircrew and sensors, lasers can affect the way operators and intelligence organizations respond; hence, they affect the battle plan.

In order to appropriately detect and respond to the growing laser threat, aircrew must be trained and provided with the knowledge necessary to confidently operate in a laser environment. To aid in that purpose, this chapter will:

- Define basic laser concepts and terminology.
- Define laser characteristics and their effects.
- Describe the threats and hazards posed by lasers.
- Provide possible solutions to deal with the laser environment.

7.7.2. Basics

Lasers are prevalent in our daily lives. Checkout scanners use low power visible (red) diode lasers; telephone communication systems utilize invisible (infrared) diodes to transmit information; laser pointers for use as presentation aids come in red, green, and even blue. Industrial lasers cut steel and weld automotive assemblies with high power infrared systems. Scientific lasers used in research labs and universities cover every wavelength region from the far ultraviolet (UV) to the far infrared (IR). Military lasers run the gamut from target designators/illuminators to systems designed to bring down missiles or aircraft.

Lasers can cause damage to materials given sufficient energy and time on target. For some lasers, the time on target to cause damage to the human eye or sensitive optical detection systems can be as short as a nanosecond ($10^{-9}$) or even less! Lasers cause damage to materials through a variety of mechanisms, but the most common are thermal and photoacoustic shock (terms we’ll explain later).

In the past, lasers capable of producing enough visible energy to disrupt flight operations were bulky, expensive, and required an external power source. Now with the advent of higher power diode systems, laser pointers that exceed the safety limits for the human eye can be purchased for less than $200 (in some cases much less than that) and are the size of a small flashlight. While the number of bird strikes reported to the FAA each year outweighs the number of reported laser/aircraft incidents (10,856 bird strikes on civil aircraft in 2013 vs. 3,960 reported laser incidents for the same year) (Dolbeer et al., 2014; Brown, 2014), the rate of laser/aircraft incidents is growing at an alarming rate. In 2006, the FAA reported only 384 laser/aircraft incidents (Brown, 2014). That means the number of laser/aircraft incidents in the civil aircraft population has grown over 10 times in the eight year period covered by the study. In comparison, bird strikes over that same time frame increased by just over 50% (Dolbeer et al., 2014). Laser pointers are increasingly being reported as flight disruptions by civilian, law
enforcement, and military pilots. As the cost of laser pointers has decreased, the number of incidents has risen correspondingly. Now that highly visible green laser pointers have become so inexpensive, green laser incidents lead all other laser types in reported incidents and now make up 91% of reported incidents (Nakagawara et al., no date). Because the human eye is so much more sensitive to green light than to red, the apparent brightness of two laser pointers with identical power outputs can be as much as 30 times brighter for a 532 nm green laser pointer than for the 670 nm red laser pointers common just five years ago (Nakagawara et al., no date). To better understand the hazards lasers pose to aircrew, it is best to start at the beginning and understand some laser fundamentals and terminology.

7.7.3. Laser Fundamentals

The word “Laser” is an acronym for Light Amplification by the Stimulated Emission of Radiation. In its simplest form, a laser has three components: 1) a gain medium, 2) a pump or excitation source, and 3) an optical cavity.

The gain medium is simply the "stuff we want to get light out of." Laser gain media are chosen because of their ability to produce photons of the desired wavelength, or possibly to produce continuous wave (CW) or pulsed laser beams (these will be discussed later). The laser gain medium can be a gas, a liquid, or a solid. Typical gas lasers are Argon Ion (usually blue or green output), Helium-Neon (typically red although other colors are available), and CO₂ (deep infrared, felt as heat). Liquid gain medium lasers are much less common and are typically found in research facilities, except for a special class that is used in adaptive optics systems at observatories. These systems are typically referred to as “dye lasers,” and those used at observatories produce a green-yellow light and are directed parallel to the astronomical telescope’s line of sight. I have personally witnessed these systems in use in the crowded airspace around Washington, D.C.! In this instance, radar was used to detect aircraft and temporarily block the laser beam when aircraft were in close proximity. Solid state lasers are by far the most prevalent systems in use today. Checkout scanners, laser pointers, laser sights used by hunters, law enforcement, and military personnel, military target designators, range finders, and laser measuring systems used by surveyors and construction workers all use solid state lasers. Common solid state lasers are referred to as “diode laser pointers” (usually visible red, green, or blue output and less than 100 mW in power), while higher power systems utilize neodymium-doped yttrium aluminum garnet (Nd:YAG) or neodymium-doped yttrium lithium fluoride (Nd:YLF). Often just called “YAG” or “YL,” the laser media is composed of a transparent crystal doped with neodymium to produce light in the near-infrared region of the spectrum (Nd:YAG, 1,064 nm; Nd:YLF, 1,047 nm and 1,053 nm). These can be extremely damaging as they are most commonly used to produce very short, high intensity pulse trains of laser energy. Knowing what the gain medium is can help determine how to provide appropriate protection from the laser as well as post-exposure treatment.

The pump, or excitation source, is used to add energy to the gain medium. We have all been told that there is no such thing as free lunch and with lasers that is abundantly clear. To actually produce light from a laser, you must first add energy to the system—in fact, because of those pesky conservation of energy laws you must add more energy to the system than you get out of the laser. Gas lasers are usually pumped by electric discharge. Liquid or dye lasers are pumped by another laser, and solid state lasers are pumped either by flowing electric current through the medium or
by optically introducing higher energy light particles (called photons) into the gain medium via another laser or flash lamps. The optical cavity comes into play once the active molecules or atoms in the gain medium have been prodded into a higher energy state by the pump source. The excited atoms or molecules will eventually spontaneously relax to their original state by emitting photons. These photons are reflected back into the gain medium by the optical cavity. The reflected photons travel through the gain medium and stimulate the release of more photons from the gain medium until there is an enormous cascade of photons. This Amplification and Stimulated Emission of more and more photons creates a large number of photons inside the laser gain medium. All of these photons were created in the same process, so they all have the same wavelength (color) and phase (they all vibrate at the same time and frequency). The mirror faces that are used to reflect the emitted photons back into the laser gain medium are aligned parallel to each other, thus creating a highly directional column of photons. In a simple laser, the optical cavity consists of only two reflectors: the high reflector (HR) and the output coupler (OC). Figure 7.7.3-1 shows how spontaneous emission leads to stimulated emission and the formation of a coherent laser beam.

![Diagram of laser operation](image)

**Figure 7.7.3-1. Basic Laser Operation (Weaver, 2012)**

As you can see by the diagram, light from a laser is much different than that from an ordinary incandescent or fluorescent bulb in three very specific ways: directionality, monochromaticity, and coherence. In a laser, directionality describes the fact that photons are emitted in one very well defined direction, as opposed to the photons from a light bulb that are designed to go in all directions and illuminate the entire room. Monochromaticity describes the fact that the photons coming out of a laser are all the same wavelength or color. Light bulbs are designed to produce “white light” which is a combination of as many different colors as possible. Coherence describes the fact that the photons emitted by a laser are in phase with each other, while those photons emitted by a light bulb have random phase.

These three factors mean that light of a specific color (often chosen for maximum absorption by the target) can be directed to a very small spot on the target. Since the photons hitting the target are coherent (in phase with each other) their energy is added together much more efficiently than the random emissions from a light bulb.
explains why a relatively small amount of laser light can be so much more destructive than a corresponding amount of light emitted from even a high power searchlight.

7.7.4. Terminology

So far we have been talking about how the laser is actually produced—the basic physics involved. Now that you are familiar with the basic concepts, let's talk about some of the characteristics of laser light and the terminology used to describe the output of various laser systems.

- **Wavelength.** Usually expressed in units of nanometer (nm) or microns (μm), this describes what color light is emitted from the laser. Different colors have different damage potential depending on the sensor involved or the targeted material.

- **Frequency.** Frequency (f) is inversely proportional to wavelength and is usually used when describing the energy potential involved in different colors of laser light.

- **Energy.** A measure of the destructive capability of a laser beam, usually measured in Joules (J). This is used to describe pulsed laser output.

- **Power or Average Power.** The total energy emitted by a laser each second, usually measured in Watts (W). High power systems will emit many thousands (kW), or even millions of Watts (MW).

- **CW.** Continuous Wave output. This means the laser is constantly emitting a steady stream of photons.

- **Pulsed.** The laser literally produces a train of very short time duration, high energy intensity bursts of light (Figure 7.7.4-1). Because the pulses of light are so brief (sometimes as short as 100 femtosecond (fs, 10^{-15}) or less), the peak power can be extremely damaging even though the average power is relatively low.

- **Peak Power.** This is the energy in a single pulse divided by the pulse duration. Peak powers can reach the gigawatt (GW) range in ultrashort lasers. With such high powers in such short time periods, the damage mechanism is different than that caused by CW lasers.
- **Divergence.** Measured in milliradians (mrads), divergence is the measure of how well collimated the laser beam is. Typical divergence for a commercial-off-the-shelf (COTS) system is 0.5 mrad, which means the laser spot size will become 0.5 mm larger for every meter the beam has traveled. The initial beam diameter, power/energy, and divergence are used to calculate the down-range beam size and intensity.

- **Mode.** Describes how uniform the laser output is. A perfect laser beam is described as having a Gaussian profile. This means the beam is very intense in the center, and falls off gradually towards the edge (Figure 7.7.4-2). The better mode the original beam had, the more energy can be transmitted down range.

![Figure 7.7.4-2. Intensity Plot of an Actual Laser Beam](image)

**7.7.5. Laser Classes**

Lasers are broadly grouped into four output levels called classes. Each increasing level represents an increase in the damage potential and hazards associated with the laser (ANSI Z136.1-2014). Class 1 lasers represent the least risk to personnel because of their low output power, while Class 4 lasers are the most dangerous. The classes are defined as follows in ANSI Z136.1-2014:

**7.7.5.1. Class 1.** Any lasers that are safe under reasonably foreseeable conditions of operation. This means that either the laser is of such low power/intensity that the individual can be exposed for up to 8 hours continuously with no risk, or as is more common, Class 1 lasers are typically contained in an enclosure with no visible light emitted from any aperture under normal use. Because there is no visible laser light emitted from a Class 1 laser enclosure (such as a CD player) (Figure 7.7.5.1-1), you can be around them continuously with no risk.

![Figure 7.7.5.1-1. Class 1 Laser](image)

**7.7.5.2. Class 1M.** Highly divergent or large diameter beams such that only a very small amount can enter the eye. If using optical aids for viewing, the acceptable exposure limit (AEL) is greater than Class 1 but less than Class 3B.

**7.7.5.3. Class 2.** CW lasers emitting light in the visible region of the spectrum (wavelength range from 400 nm to 700 nm). Eye protection is normally
afforded by aversion responses including the blink reflex, which is 0.25 second. Typical Class 2 laser uses would be checkout scanners (Figure 7.7.5.3-1) and some laser levels.

![Class 2 Laser](image)

**Figure 7.7.5.3-1. Class 2 Laser**

7.7.5.4. **Class 2M.** Highly divergent or large diameter beams such that only a very small amount can enter the eye. If using optical aids for viewing, the (AEL) is greater than Class 2 but less than Class 3B.

7.7.5.5. **Class 3R.** Lasers that are safe for viewing with the unaided eye. For lasers emitting in the wavelength range from 400 nm to 700 nm, protection is afforded by aversion responses including the blink reflex. Direct intrabeam viewing of 3R lasers with optical aids (e.g., binoculars, telescopes, microscopes) may be hazardous. These lasers are less than five times the output of Class 1 or Class 2 lasers. Most diode pointers are 3R (Figure 7.7.5.5-1). No matter the class of the laser, never look down the barrel.

![Class 3R Laser](image)

**Figure 7.7.5.5-1. Class 3R Laser**

7.7.5.6. **Class 3B.** Lasers that emit in excess of Class 3R power. These laser emit between 5 mW and 500 mW of laser power. Direct intrabeam viewing of these lasers is always hazardous. These lasers must carry danger labels. Viewing diffuse reflections is normally safe. The Air Commander Pointer (Figure 7.7.5.6-1) is a Class 3B device.

![Class 3B Laser](image)

**Figure 7.7.5.6-1. Class 3B Laser**

7.7.5.7. **Class 4.** Lasers that are also capable of producing hazardous diffuse reflections. They may cause skin injuries and could also constitute a fire hazard.
The LP-1000 laser pointer (1.2 watts; 836 nm) and AN/AAQ-28(V) LITENING Targeting Pod (which has a Class 4 target designator) are shown in Figure 7.7.5.7-1.

Figure 7.7.5.7-1. Class 4 Laser (Left: LP-1000; Right: LITENING Targeting Pod)

7.7.6. Laser Hazards

Lasers pose some unique hazards to the human eye. They are especially dangerous at night when the pupil is fully dilated and there is a greater risk of a damaging laser beam entering the eye. The wavelength or color of light emitted from a laser also has a very important role in determining the hazard potential of a laser beam. A green laser is in the heart of the visible spectrum where it receives the maximum magnification when passing through the cornea of the eye. The cornea and lens combination of the human eye has the capability to focus light by a factor of 100,000 (Sliney and Wolbarsht, 1980). This means that a relatively small amount of laser light can have very damaging effects on the retina.

Laser damage potential for flight crews can be characterized by the following terms:

- **Dazzle.** Imagine you are driving home, heading west into the setting sun, and your pitted windshield is almost impossible to see through.

- **Glare Blindness or Flash Blindness.** Imagine you have momentarily looked at the sun, or possibly Uncle Bill used the flash on his camera right in your face. You will see spots that gradually fade away, but for several seconds or even tens of seconds your vision is impaired. Figure 7.7.6-1 shows the effects of glare when a laser strikes a cockpit window or canopy.

Figure 7.7.6-1. Glare from a Laser Exposure

- **Retinal Burns.** At this point you have a permanent blind spot. The laser has impacted the retina with sufficient energy and time on target to overwhelm the retina’s capacity to wick away the heat, and the tissue is now burned. You have a scar. If the scar is off axis you will likely suffer no lasting perceptible effects because the brain is very good at averaging out small errors. If the burn is on the
fovea or optic nerve, the news is not so good. Most minor retinal burns heal over time and the individual can still function normally.

Figure 7.7.6-2 illustrates how a laser beam can impact a pilot very differently depending on the distance from the laser source to the pilot. Note the calculations for a 125 mW green laser. These types of lasers can be purchased on-line for less than $200 and cause distraction hazards at approximately 11 miles!

Figure 7.7.6-2. Laser Visual Effects Hazards and Distances (Laser Pointer Safety, 2015)

There are several factors that can contribute to a laser’s ability to damage human tissue. The damage mechanisms for various types of human tissue vary by the color or wavelength of laser in use. The region of the spectrum composed of wavelengths shorter than visible light is the ultraviolet (UV) region, and the region composed of wavelengths longer than visible light is the infrared (IR) region. In the UV region, the action is primarily photochemical, and the laser radiation is absorbed during the conversion from light into chemical energy, which, in turn, may damage tissue. Typically UV light will break down bonds between molecules in cells making long term exposure to UV light hazardous. Welders wear long sleeves to protect their skin from the UV light produced by the arc at the surface of the weld. This UV light causes sunburns and can eventually cause skin cancers. In the IR region, the action is primarily thermal. In the visible region, both effects are present. Pulsed lasers can present dangers at doses below the cell-death threshold including the production of...
proto-oncogenes (Obringer et al., 2000). The cornea, lens, and retina may be affected by several wavelengths of laser light. Figure 7.7.6-3 shows the damage mechanisms for various structures in the human eye (ACC Laser Awareness Training, 2008).

![Figure 7.7.6-3. Bio-Effects: Photochemical or Thermal](image)

The damage from laser radiation may range from relatively mild, from which there is complete recovery, to severe, where there is little, if any, recovery. The acute exposure symptoms include redness, tearing, conjunctival discharge, corneal surface exfoliation, and stromal haze and are extremely uncomfortable.

Invisible, near IR wavelengths are absorbed by the retina. The human visual cells are not stimulated by those wavelengths; hence, they pose a greater threat to the fovea and retina because the aversion response (blinking) will not limit exposure. The damage potential is increased because exposure will only be sensed when actual damage occurs in the fovea. Peripheral damage may not even be noticed until after extensive injury has occurred.

Due to lack of laser detection equipment onboard aircraft that could collect and characterize laser exposures, all exposure data currently comes from the eyes of the persons being exposed (ACC Laser Awareness Training, 2008). This is not good for several reasons:

- It is inherently dangerous.
- The eye cannot measure the amount of energy being received (radiant exposure).
- The brightness of the exposure makes judging the distance and color of the source difficult.
- The data are subjective.
Despite these facts, it is still very helpful if all exposures are reported promptly and as accurately as possible. It still benefits those who may be following the same flight path as the exposed aircraft by alerting others to the need to use eye protection.

Current DoD policy (12 Feb 2013) “...prohibits the use of lasers specifically designed to cause permanent blindness in humans” (DoD Laser Policy, 2013). Although it is a violation of international law to use weapons that are specifically designed to blind, violations could occur in the future.

We have talked a bit about how lasers have the potential to damage various structures in the human eye, but we haven’t discussed the actual damage mechanisms yet. It is important to understand that different wavelengths of light have different damage mechanisms when talking about human tissue. Also, CW lasers can have different damage mechanisms than pulsed lasers, especially ultrashort pulse systems.

Let’s start with the short wavelengths, UV-C, and work our way up the scale to the IR-C Band. UV-C is the region from 100 nm to 280 nm (0.1 μm – 0.28 μm). As can be seen by Figure 7.7.6-4, this is typically not a problem area for flight crews as this wavelength region is not transmitted through the atmosphere very well at all. This is mainly due to water and ozone absorption in the atmosphere. The transmission through the atmosphere is shown for the UV-C to the lower part of the IR-C band as a reference. The interested observer can note why specific laser wavelengths have been chosen for targeting and ranging lasers. Any remnants of UV-C that make it through the atmosphere at higher altitudes will be blocked by the materials in the aircraft windscreen (Nakagawara et al., 2007). This wavelength regime is only mentioned as it is a source of “welder’s flash,” or photokeratitis, and may possibly affect flight line personnel during repair operations if they are not wearing appropriate protective equipment.

Figure 7.7.6-4. Atmospheric Transmission Windows (Clark, 1999)

Like UV-C, UV-B is a not much more problematic. This wavelength region, from 280 nm to 315 nm is almost entirely blocked by the atmosphere and < 1% of the residual is transmitted by aircraft windscreen materials (Clark, 1999; Nakagawara et al., 2007). Both UV-C and UV-B are absorbed by the cornea, and the damage mechanism is photochemical in nature. In this case the short wavelength, high energy photons cause chemical reactions in the tissue of the eye. Specifically, chemical bonds are
broken in the corneal epithelium causing the characteristic symptoms of snow-blindness such as pain, redness, tearing and conjunctival discharge.

Approximately 10 – 40% of UV-A light, from 315 nm to 400 nm, is transmitted by the atmosphere. As can be seen in Figure 7.7.6-5, of that fraction transmitted by the atmosphere, approximately 40 – 50% actually makes it through aircraft windscreens to reach the eyes of unprotected flight personnel (Nakagawara et al., 2007). UV-A light is considered especially dangerous to the human eye as it is the wavelength region that causes cataracts in the lens. The damage mechanism here is photochemical as well, and the structures of the eye subject to damage from UV-A are the cornea and lens with 98% of the light being absorbed by these structures and the remaining 2% being absorbed in the vitreous (Clark, 1999). UV-A light is not considered to be a retinal hazard since such a minute amount actually reaches the retina. In addition, UV-A light is blocked almost entirely by most prescription lens wear. Polycarbonate lenses effectively block all transmission below 380 nm (Nakagawara et al., 2007).

Figure 7.7.6-5. Average UV and Visible Light Transmittance for Aircraft Windscreens (Nakagawara et al., 2007)

Visible light (VIS), 400 nm to 760 nm, is the area where most reports of laser exposures to flight crews occur for obvious reasons. The nearly 4,000 incidents of laser/aircraft interactions reported to the FAA in 2013 came from laser pointers or entertainment systems. Commercial laser pointers only transmit in the visible region of the spectrum and are trending heavily toward the center of the visible region around 500 nm to 550 nm where the apparent brightness is greatest (Nakagawara et al., no date). For both VIS and IR-A light, 760 nm to 1,400 nm, the primary structure of the eye subjected to damage is the retina. The retina is probably the most sensitive organ in the human body. Photoreceptors in the eye can respond to single photons although neural filters limit conscious responses to photon stimuli to larger numbers (typically 5 to 9 photons received in < 100 ms) to limit “background noise” (Gibbs, 1996). The extreme sensitivity of these photoreceptors, rods and cones, means that they are also susceptible to damage when too much light is received in a short period of time or over...
a sufficiently small area. The rods and cones only capture approximately 2% of the incoming light while about 50% is absorbed by the underlying retinal pigment epithelium (RPE) tissue (Sliney and Wolbarsht, 1980). Damage to the RPE is characterized as retinal burns and is considered to be largely caused by thermal effects for exposures ranging from 0.1 second to 10 seconds or more. In essence, the damage is caused because the normal energy removal system for the RPE is overwhelmed. The RPE is rich in blood vessels and these serve to carry away excess heat from the eye. However, since the human eye focuses wavelengths from both the VIS and IR-A regions onto the retina very well, optical concentrations of approximately 100,000 times are achieved for the entire VIS/IR-A region. An irradiance of 1 mW/cm² at the pupil will produce 100 W/cm² on the retina! The blood vessels in the RPE cannot remove that much heat fast enough and the temperature of the retinal image spot rapidly increases by 50°C or more causing the cells to denature or burn (Sliney and Wolbarsht, 1980).

IR-B and IR-C, 1,400 nm to 3,000 nm and 3,000 nm to 1 mm, commonly called mid-IR and far-IR light are absorbed by the cornea. Again, the damage mechanism here is thermal in nature. This is actually good news as the corneal epithelium has one of the highest metabolic rates in the human body and can typically regenerate in 24 to 48 hours. That means that superficial burns to the cornea will heal in a very short period of time; however, deeper burns can cause permanent opacity of the cornea and require corneal transplants to repair the damage (Sliney and Wolbarsht, 1980).

So far we have been talking about damage caused by CW or long pulse lasers (pulses > 1 ms in duration). COTS laser pointers are usually CW although some green or blue units have pulsed outputs. COTS systems typically have long pulses and can be treated as CW in terms of the damage mechanisms; however, military target designators are another story. Many military systems use short pulse Nd:YAG lasers to paint the target. The laser wavelength and a coded system of pulses help the incoming missile or weapons system lock onto the target and ignore extraneous light sources found on the battlefield. Q-switched Nd:YAG lasers have pulses in the nanosecond or picosecond regime due to the excited state lifetimes and physics involved in producing high peak power systems. Q-switched lasers essentially allow the gain medium to keep building up photons over a relatively long period of time, and then an optical shutter (the Q-switch) is opened and an enormous number of photons are released in a very short period of time. Systems like the Ground Laser Target Designator (GLTD) III utilize pulses of approximately 80 mJ to range or mark targets in excess of 10 km from the source. Assuming these are 10 ns pulses (a typical value for an Nd:YAG system), the peak power for this laser system is 8 MW! Yes, 8 million watts of peak power. The damage mechanism here is no longer thermal. Now we are ablating the material. Laser ablation simply means that the laser is used to remove material. When the laser flux is relatively low, the material is heated by absorption and evaporates or sublimates. At higher flux rates, the material is generally converted directly to plasma. In any case, the material is gone.

Pulsed lasers can also cause damage via a phenomenon called the photoacoustic or optoacoustic effect. As is suggested by the name, the light pulses actually produce sound waves. When ultrashort pulses of laser light contact the surface of a material, something like the skin or possibly the cornea, the material is heated and ablation occurs through evaporation, sublimation or plasma generation at the surface. This does damage to the surface whether intentionally or by accident. Intentional damage of this type can be seen in laser eye surgeries where the cornea is reshaped to provide more optimal focusing for the patient or in industrial machining practices.
However, when the material in question is inside another media, like the retina inside the human eye, much more catastrophic effects can occur due to optoacoustic damage. In optoacoustic damage, the material that has been heated due to photothermal absorption changes phase from a solid/liquid to gas. This expansion occurs rapidly when the short burst of laser energy hits the targeted material. Remember, short pulses from lasers can range from a few nanoseconds in length to a few femtoseconds. The peak powers of these pulses are typically a few MW to over a GW. The rapid pressure change when a substance like the human retina absorbs a MW or more in a few nanoseconds over an area of 100 microns or less has the following effect on the surrounding tissue: the initial impact site rapidly heats and material is ablated via evaporation, sublimation, or plasma generation. The ablated material can’t simply expand out into the atmosphere, instead it is trapped inside the vitreous humor, but now it takes up much more volume! This creates a pressure wave inside the eye. In normal eye surgeries where the target material is the cornea, laser pulses remove precise amounts of material and reshape the cornea for better focusing. It is almost as if the surface of the cornea has been sanded to produce a more optimum shape. However, when these laser pulses are focused on the retina instead of the cornea, the amount of material removed by ablation remains similar, but the optoacoustic pressure wave also rips the retina and produces much larger holes and tears. This is much more than a surface effect. The damage area can be as much as 10 times larger than for a simple burn or ablation site (Sliney and Wolbarsht, 1980).

7.7.7. Laser Exposure Mechanisms

As has already been discussed, lasers are being used extensively for target identification and designation, range finding, and weapons guidance. The battlespace of tomorrow will use lasers in more and different functions such as combined beams for directed energy applications, kinetic integrated air defense systems, and force protection systems.

Aircrews are often in the same space as battlefield targeting and ranging systems subjecting aircrew to unintended exposure. Many of these systems emit outside the visible region of the spectrum, and in addition, many of these systems are pulsed. Exposure to these types of lasers is especially dangerous because the victim may not be immediately aware of the incident.

The most common type of laser exposure experienced by aircrew is still caused by individuals on the ground lasing aircraft using hand held visible lasers. These lasers range from relatively low power systems (Class 3R devices) to high power hand held systems that are extremely dangerous Class 4 devices.

7.7.8. Laser Incidents in Aviation

In 2005, the Federal Aviation Administration (FAA) implemented a laser reporting system to track aviation lasing incidents. Since its implementation, the number of reported incidents in the United States has rapidly grown every year (Fiorino, 2012). In 2013, there were an average of almost 11 laser incidents reported every night (Murphy, 2013). By end of 2015, the average had increased to over 21 incidents per night, with 2016 on pace to top 32 per night (Murphy, 2016). On the night of 11 November 2015, more than 20 aircraft reported being struck by lasers in more than 10 states and Puerto Rico (Najarian, 2015). Figure 7.7.8-1 shows the rapid increase in laser incidents from
2007 through 2015. The predicted number of incidents for 2016 is based on the 1,413 reports filed with the FAA between 1 January and 9 April 2016 (Murphy, 2016).

Figure 7.7.8-1. Laser Incidents between 2007 and 2015 (Murphy, 2016)

Figure 7.7.8-2 shows the distribution of laser incidents within the United States during 2013.

Figure 7.7.8-2. Distribution of 2013 Laser Incidents (Murphy, 2013)
Despite the growing number of reports, not all incidents involve lasers actually entering the cockpit and interfering with aircrew operations. In 2011, Rockwell Laser Industries reported that out of the 6,903 reported to the FAA between 2004 to mid-March 2011, lasers were reported to have entered the cockpit only 27% of the time (Figure 7.7.8-3) (Murphy, 2011). Although 100 reported eye effects or injuries during the study period, no permanent or long-term damage was sustained by the affected aircrew. In fact, only about 1.5% of all FAA-reported laser incidents involve adverse eye effects with no documented reports of permanent eye injury in more than 21,000 reports to the FAA and Transport Canada Civil Aviation (Murphy, 2015a).

![Figure 7.7.8-3. Cockpit Illuminations, 2004 to mid-March 2011 (Murphy, 2011)](image_url)

There are a number of explanations for the growing number of reports. These include better education and awareness of aircrew, better report mechanisms, decreasing cost of laser devices, increasing availability via the internet, and increased power levels of available lasers. The combination of power, low cost, and the eye’s sensitivity to the wavelength used in green lasers has led them to be of the greatest concern for aircrew safety and they continue to be involved in the vast majority of incidents. Figure 7.7.8-4 shows the break-down of all reported laser incidents in the United States in 2013, with the trend continuing into 2015 (Murphy, 2013; Murphy 2015a).

![Figure 7.7.8-4. Laser Incidents by Beam Color (Murphy, 2013)](image_url)
While most laser incidents in the United States are not intentionally trying to interfere with the safe operation of the aircraft, the growing problem reflects not just hand-held lasers, but also unintentional or careless use of seasonal light displays. On 3 December 2015, a commercial aircraft flying at 13,000 feet was illuminated. The investigation determined that the light came from a holiday light display 22 miles east of the Dallas-Fort Worth airport (Murphy, 2015b). Three days later on 6 Dec 2015, three aircraft flying into Kansas City International Airport reported laser incidents that were traced to a home display 3 miles from the airport (Murphy, 2015b).

There are many challenges associated with tracking the source of a laser involved in an aircraft lasing incident. It can be difficult to pinpoint the exact location of the source while in an aircraft at altitude and even when able, the perpetrator has often disappeared before law enforcement agencies can respond. Between 2005 and 2013, there were 17,725 incidents reported to the FAA. Of those, only 134 arrests were made, with 80 resulting in convictions (Murphy, 2015a). Conviction of knowingly aiming a laser at an aircraft in the United States is a felony and punishable by fine and/or prison (Public Law 112-95, 2012).

7.7.9. Protection

A laser threat is defined as any laser light that could alter operations without causing catastrophic structural damage to platforms. With that definition in mind, even relatively low power hand held devices can pose a risk to aircraft during low level flight operations or during take-off or landing. Landing is the most vulnerable portion of any flight profile and gives the aircrew the fewest options. Laser eye protection (LEP) may be the most beneficial during this portion of the flight. Wavelength and optical density (OD) are the two most critical factors in determining what LEP should be used in a particular laser threat environment. Most LEP devices are developed and manufactured for a specified set of wavelengths so they are considered wavelength specific. Optical density is a measure of the amount of light transmitted by a filter, such as eye protection spectacles. This term is defined such that high OD equates to low transmission of light. The OD is critical because it specifies how much of a certain wavelength’s energy is blocked (reduced), thus the degree of protection. The OD, or protection factor of the LEP, is designed to bring the energy level to below the maximum permissible exposure. The range from the laser, the magnification of optics being used, and the energy density of the laser are used to calculate OD for unique situations. The scale is logarithmic such that an OD of 0 equates to 100% transmission and a reduction factor of 1, an OD of 1 equates to 10% transmission and a reduction factor of 10, an OD of 2 equates to 1% transmission and a reduction factor of 100, etc. Sunglasses have an OD of about 1. LEP choices will usually be a balance between protection from incident laser beams and visible light transmission allowing the aircrew to still see cockpit displays and the airspace around them. Many LEP devices resemble dark glasses with various degrees of wrap around for added protection.

The current spate of green laser pointer incidents is especially troubling because it is more difficult to protect aircrew from these lasers while still allowing aircrew members to have full access to cockpit displays. Many cockpit instruments use green in their displays, so blocking green light with LEPs can make it difficult to see all of the information displayed on cockpit instrumentation. Very narrow band LEPs are available; however, they are expensive and their protection can be of limited value for off-axis laser exposure.
LEP comes in many formats and styles, such as spectacles, goggles and wrap around. They also come in single band and multi-band wavelengths. There are also different types of filter technologies commonly used in LEP. Absorptive filters are one of the most popular types. Absorptive filters are dye-based and made of polycarbonate material. These LEP are designed to absorb particular bands of wavelengths by adjusting the concentration of dyes to protect from a band or bands of wavelengths. These LEP are popular because of their light weight, ballistics grade strength and flexible protective wavelength capabilities. There are two main drawbacks to absorptive filters: the broadband nature of the filter response, and the limited amount of visible light transmitted by the filters. The Kentek® model KXL-5301 is an example of this. This LEP blocks all VIS/UV from 190 nm to 532 nm and has only 42% visual light transmission (Kentek®). As an example, a typical polycarbonate LEP designed to fit over prescription eyeglasses with an OD of 7 at 532 nm (the most common wavelength for green laser pointers), blocks all visible light in the blue and green regions of the visible spectrum leaving only 40 – 50% of the visible light remaining. With no blue or green light transmitted by the LEP, the flight crew might be safe from attacks by over 90% of handheld lasers, but many cockpit instruments will be unavailable to the flight crew as their video or LED based displays rely heavily on green and blue light to convey information. Absorptive filters are also not “sharp-edge” filters. The dyes in the polycarbonate material absorb very well at their design wavelengths, but the transition from absorption to transmission is gradual and may extend for tens of nanometers before the transmission is close to 100% again.

Glass filters can have extremely narrowband blocking characteristics. Usually this is due to a system of reflective dielectric layer coatings applied to the glass substrate. In addition, glass laser eyewear with narrowband dielectric coatings can be made into custom prescriptions for each individual member of the flight crew as necessary. Glass lenses are more scratch resistant than polycarbonate lenses and they resist bleaching better than polycarbonate lenses due to the nature of the protective mechanism. The dyes in the polycarbonate lenses can be aged prematurely (the bleaching mentioned in the previous line) due to over exposure to sunlight or high power lasers—this usually happens if the LEP is left in the cockpit or on a dashboard instead of being properly stored. Glass lenses also have better optical quality than plastic, and the narrowband nature of the dielectric filter coatings means the visible light transmission will be much higher—often as high as 80%. An example of this is the Kentek® model KXL-C500C laser eye protection, which has an OD 7+ at 532 nm and 80% visual light transmission (Kentek®). Glass lenses have two major drawbacks: they are much more expensive than polycarbonate lenses, and they do not protect as well as polycarbonate against off-axis laser beams striking the surface of the LEP. Figure 7.7.9-1 shows how sensitive narrowband dielectric filters are to changes in the angle of incidence (AOI).
What this means in practical terms is that a mix of dielectric coatings to provide protection while still allowing a sufficient amount of visible light transmission is probably the best solution. Extremely narrowband filters like the one depicted in Figure 7.7.9-1 are not practical for flight crew. Broader filters that block a 30 or 40 nanometer region of the spectrum for common laser threats is more likely to provide adequate protection. However, care must be taken when choosing these filters as crucial flight information may be dimmed or completely blocked. Protecting flight crew from VIS lasers is not as simple as handing each individual a pair of LEP goggles. It will require close cooperation between cockpit instrument manufacturers, LEP manufacturers, and aerospace engineers.

One other option that has not been discussed yet is to replace or retrofit aircraft windscreens or canopies with new materials or films designed to block the more common VIS laser dangers. While this is an option, it would not only be very expensive in both the material and manpower required to make the modifications, it is not a very flexible option for the military community. If we design a windscreen to block 532 nm green lasers, the most common wavelength for laser/aircraft incidents today (Nakagawara et al., no date), adversaries can simply shift to a high intensity blue laser at 445 nm and still cause disruption to flight operations.

7.7.10. Summary

Lasers are a fact of the modern, technological world we live in. Civilian pilots, law enforcement personnel, and military airmen are being repeatedly subjected to lasers targeting their aircraft in flight. The most dangerous flight regimes, low level flight and take-off/landing, are where pilots are in the most danger from hand-held laser pointers, and even though most are relatively low power they can still pose dangerous distractions to flight deck personnel. In addition, laser pointers that far exceed the allowable limits for sale in the United States are increasingly available through on-line
sources (Wicked Lasers). Military aircrew are also exposed to a variety of laser designators and range finders. These are typically invisible, pulsed systems and thus may prove to be an even greater source of danger to aircrew due to the extremely damaging characteristics of short pulse, IR-A region lasers. While aircraft canopies protect against most UV threats, they do little to stop or deflect VIS and IR-A lasers. Individual LEP is available and is constantly being evaluated by USAF physiologists for use by flight crew as well as laboratory personnel. Lasers are not magic weapons, nor does every exposure mean permanent damage or blindness. Aircrew can be protected from accidental or deliberate exposure by ground-based handheld lasers with appropriate LEP/cockpit instrumentation choices as well as aircraft attitude and evasion maneuvers.

References


7-134
Recommended Readings

Belland KM. Aircrew Performance Cutting-Edge Tech: Emerging Human Performance Enhancement
AU/AWC/CSAT/2001-12. 1 Apr 2002.

Vocabulary

Continuous Wave (CW)
Dazzle
Divergence
Glare Blindness
Laser
Laser classes
Laser Eye Protection (LEP)
Mode
Optical Density
Peak Power
Pulsed
7.8. Physiological Responses to Thermal Environments

Mark White, M.S.

7.8.1. Thermal Stress

7.8.1.1. Introduction. Mission requirements impose a variety of threats to military personnel in the operational environment, thermal stress being one of significance. The operational environment can range greatly and subject personnel to a variety of environmental extremes, such as cold and hot exposure. For example, you might find yourself at the Yuma Proving Grounds, AZ, and exposed to heat stress on the ground while waiting to board an aircraft for High Altitude Low Opening (HALO) school, with the ambient temperature at 40°C (104°F). Then, within minutes you’re flying at 20,000 ft MSL in an unpressurized cabin and the temperature is now a cool 0°C (32°F). One minute you’re sweating it out on the flight line preparing for a jump and the next minute the sweat begins to turn ice-cold as the ramp opens and a 20-mph gust of wind drops the ambient temperature to a wind chill blown -8°C (17°F). Another scenario to consider: an F-15 pilot stationed at Elmendorf AFB, AK, is well versed on the aircraft’s life support and environmental control systems, which allow pilots the comfort of flying with minimal interference from the outside world. However, if the heater control core unit shorts out and the cockpit temperature equalizes with the ambient conditions while being more than 2 hours away from landing, normal cold weather flying attire may be inadequate. Regardless of the situational circumstances, the environment of a closed cabin can pose similar thermal stress to the crew who are ill-prepared for changes encountered with altitude. The following section focuses on the human body’s capacity to maintain physiological and psychological function under thermal stress of heat and cold exposure. A solid understanding of the physical and cognitive changes and protective defense strategies can provide potentially life-saving guidelines to air and ground crew faced with an environment that challenges human performance.

7.8.1.2. Biophysics of Environmental Stressors. For the human body to maintain homeostatic balance, it must manage internal systems based upon the input of external stressors. Examples of external stressors considered to be a threat to the human body include: thermal stressors (i.e., heat, cold), odor, food, water, hypoxia, noise, light, darkness, trauma or injury, electricity, physical threats, and bacteria or viruses. With exposure to thermal stressors, the human body must maintain the core body temperature ($T_C$) through the balance of heat gain and heat loss (Figure 7.8.1.2-1). If the rate of heat production (i.e., environmental, metabolic, hormonal, or food thermogenesis) is greater than the rate of heat debt, then the net is a heat gain and the positive heat storage results in hyperthermia. Conversely, if heat loss (i.e., through conduction, convection, radiation, and evaporation) is greater than heat gain, then the net is heat loss and the negative heat storage results in hypothermia.
The balance of heat storage, shown in Figure 7.8.1.2-1, can be mathematically expressed as follows:

\[ S = M - (\pm W) - E \pm (R + C) \pm K \]

where:
- \( S \) = storage of heat
- \( M \) = metabolic heat
- \( W \) = work
- \( E \) = evaporation
- \( R \) = radiation
- \( C \) = convection
- \( K \) = conduction

The transfer of heat between the body and the environment occurs through conduction, convection, radiation, or evaporation as shown in Figure 7.8.1.2-2. Environmental factors that affect the rate of heat exchange consist of air temperature, air movement (wind), air saturation (humidity), and water immersion (submersion); each of these will be addressed in further detail under hot and cold stressor sections as they relate to thermoregulation. Conduction is defined as the transfer of heat to or from one object to another, e.g., by direct contact with an external object or, within the body, through tissue-to-tissue, all of which occur due to a temperature gradient. The heat loss of a person in a resting state at normal room temperature is approximately 3%. The movement of heat to or from air or water across the body due to thermal currents, body-in-motion, and/or air (i.e., wind chill) or water (i.e., current) movement is convection. The primary source of heat loss, transferring heat out and away from the core body, is through convection and can account for approximately 12% of heat loss under normal room temperatures. Heat loss or gain occurs when the body is surrounded by air or water that is lower or higher in temperature than the body, thus the transfer of heat down the gradient. The loss of heat will be greatly increased if the air or water surrounding the body is of a turbulent nature; i.e., wind chill or turbulent current or flow of water. Conversely, radiation from heat exchange is independent of air movement and occurs with the radiant temperatures of solar, sky, large objects, and ground either being higher or lower than the body; thus, the body will gain heat or lose heat to the environment.
Environment. Evaporation, the most important method the human body uses for thermoregulation, consists of the transfer of heat from the body when sweat accumulates on the skin and evaporates into the air. Under normal room temperatures in a rested state, evaporation can be responsible for up to 25% of heat loss. As evaporation occurs, the heat transferred from the absorption of sweat is called the latent heat of evaporation and equals approximately 0.58 kcal/g of water absorbed. Under environmental conditions in which heat loss from the body is hindered, it is critical to understand that core body temperature may increase 0.2°C/minute during moderate activity levels, which may cause thermal injury within 15 – 20 minutes.

**Figure 7.8.1.2-2. Biophysics of Heat Exchange (TBMED507, 2003)**

7.8.1.3. **Thermoregulation of Normal Body Temperature.** From a biological perspective, the human body’s ability to maintain constancy of body temperature places us in a group of advanced animals called homeotherms, along with monkeys, dogs, bears, and birds. The homeothermic ability, that is the ability to function relatively independent of the environment because of the maintenance of the T_C, has allowed us to perform in extreme environments (Table 7.8.1.3-1). The constancy of T_C is of physiological significance, as other biological processes are temperature dependent. For example, impairment of oxygen transport, cellular metabolism, muscle contractions, and neural function might mean the difference between life and death when considering the overall performance of the human body.
The term “normal body temperature” is one that is not easily defined because there can be minor individual variations in one’s body temperature, especially when it’s measured in different areas of the body or under different circumstances. Normal body temperature ranges from 36.0°C to 37.5°C (96.8°F to 99.5°F), with the commonly known average temperature reading of 36.7°C to 37.0°C (98.0°F to 98.6°F), and can maintain function within the limits of 36.0°C (NOTE: experienced in the early morning sleep cycle) and 40°C (NOTE: may occur during maximal exercise). Although there can be considerable variability in temperatures throughout the body, it’s the core temperature that maintains constancy and is usually defined as the temperature of the hypothalamus, the primary temperature regulator (Figure 7.8.1.3-1).

The hypothalamus is known as the thermostat of the body, as one of its primary functions is to monitor the temperature of the body and then manage the physiological response. As the primary control center, the hypothalamus relies upon sensory input from thermoreceptors located throughout the body. Thermoreceptors can be categorized as central or peripheral, central being that of the anterior and posterior...
hypothalamus as well as the preoptic region, while the peripheral receptors consist of the nervous, vascular, and muscular systems and the abdominal cavity. Of the peripheral receptors, a small portion exists in the nerves, abdomen, and deep leg muscles and veins, while the greatest density is in the skin. However, the central thermoreceptors are the most important for core temperature regulation.

Figure 7.8.1.3-1. Thermoregulatory Center of the Brain; Anterior and Posterior Hypothalamus and Preoptic Area
(http://www.neurology.org/cgi/content/full/69/12/1293/F116)

The hypothalamus is responsible for dual regulation of hot and cold defense responses; thus, the response of the hypothalamus is dependent upon the thermoreceptor input (Figure 7.8.1.3-1). The anterior and preoptic areas of the hypothalamus are coupled with the posterior hypothalamus in a negative feedback loop inhibitor response. The heat defense response is initiated when the anterior hypothalamus and preoptic area are stimulated by local temperature changes in heat; especially in the blood where temperature changes of less than 0.01°C (0.018°F) can elicit a reaction. The heat defense response consists of the evaporative heat loss mechanism (i.e., stimulation of sweat glands), inhibition of the cardiovascular control center normal vasoconstrictor vascular tone to the skin (i.e., skin vasodilation), and inhibition of thermogenesis (i.e., shivering and metabolic rate). Conversely, when the peripheral thermoreceptors, primarily the skin, provide afferent input into the posterior hypothalamus, the cold defense response is stimulated, which inhibits the heat defense response. Thus, the heat loss mechanism is inhibited and vasoconstriction occurs in skin vasculature due to the stimulation of the cardiovascular control center (i.e., blood is shunted away from the periphery and redirected to the core thoracic/abdominal area). In addition, the stimulation of shivering and metabolic thermogenesis increase insulative factors and heat production. As a result of stimulation of the posterior hypothalamus, epinephrine and norepinephrine (other contributors to metabolic thermogenesis) are directly secreted and thyroxin production is indirectly increased due to an increase secretion of thyrotropin-releasing factor, which stimulates the secretion of thyrotropin from the pituitary gland. Albeit, the hypothalamus is responsible for the primary regulation of the physiological response, there is another response defense mechanism that aids in thermoregulation: human behavior. A factor easily overlooked, the human
behavior response to hot or cold exposures is of significance when considering the defense mechanisms of thermoregulation. In an extreme cold environment, one might consider adding additional layers of clothing, or moving under shelter; on the other hand, excessive heat might drive one to drink fluids, reduce clothing layers, reduce physical activity, and seek shade. Regardless of the environment, it’s both the physiological and behavioral response mechanisms that allow the human body’s homeothermic abilities to maintain core temperature constant under extreme conditions, especially those that are imposed upon our military personnel.

Mission requirements impose a variety of threats to military personnel in the operational environment, thermal stress being one of significance. But the environment of a closed cabin can pose similar thermal stress to the crew who are ill prepared for changes encountered with altitude.

7.8.2. Heat Stress

As part of a crew member in a deployed environment you are constantly counseled on the negative effects of dehydration and performance, and the idea of euhydration has been reiterated to the point that every drink you take is preventative and you’re not going to be the one that succumbs to the hot environment. You’ve been hydrating since the night before. In the hot preflight environment, you continue to hydrate but begin to develop a headache. During the first low level route the headache gets worse, but you decide not to mention it; the mission comes first, so let’s press. At the end of route you self-identify as having some sort of dehydration or heat-related problem. The crew performs a full stop of the aircraft and declares a ground emergency for medical attention. The ambulance is called and you are checked out on the aircraft. You feel good enough to get yourself to the doctor, so you decline transportation to the hospital and are on your way to flight medicine, exactly where you didn’t want to end up. The flight doctor performs a blood test and discovers that you’re well hydrated but your electrolytes have been almost completely depleted. The solution is simple: you receive an IV solution to replenish your electrolytes and are released back to duty. Your instructions are to eat a balanced diet which is meant to maintain electrolytes. Because of you there’s a new squadron policy; it has changed supplemental water procedures to include a performance drink during hot temperatures to aid in maintaining electrolytes....

7.8.2.1. Introduction. For the human body to maintain homeostatic balance, it must manage internal systems based upon the input of external stressors. External stressors considered to be a threat to the human body include thermal stressors (i.e., heat, cold), odor, food, water, hypoxia, noise, light, darkness, trauma or injury, electricity, physical threats, and bacteria or viruses. With exposure to thermal stress, the human body must maintain its core body temperature ($T_C$) through the management of heat loss and heat gain. The body will either dissipate or gain heat through convection, evaporation, conduction, or radiation and generate heat through metabolism, shivering, or physical activity as shown in Figures 7.8.1.2-1 and 7.8.1.2-2. When considering the thermal stress of heat gain, there are four significant environmental variables that must be considered: air temperature, air movement (wind...
Heat stress occurs when the balance of heat exchange between the environment and the human body favors heat gain, with the ultimate result being an increase in core body temperature ($T_C$). The physiological and behavioral effects of heat stress are referred to as heat strain. The environmental factors that greatly influence heat stress are high air temperature, high humidity, high radiation heat, and low air movement. The National Weather Service’s Heat Index Chart stratifies the risk of heat stress over time of exposure relative to air temperature plus humidity, as shown in Figure 7.8.2.1-1. The U.S. Army utilizes a heat stress index, called the wet bulb globe temperature (WBGT), that provides strategic guidelines to maximize human performance by quantifying physical activity exertion levels, work/rest ratios for times of exposure, and fluid replacement based upon air temperature, humidity, and air movement (Table 7.8.2.1-1). The U.S. Air Force employs the fighter index of thermal stress (FITS), as outlined in AFPAM48-151, 18 November 2002, that represents cockpit heat stress stratified across zones of risk; the FITS zones are called the caution and danger zones (Table 7.8.2.1-2). The FITS caution zone (32°C to 38°C) instructions for implementation consist of: 1) be alert for heat stress, 2) drink plenty of noncaffeinated fluids, 3) avoid exercise 4 hours prior to takeoff, and 4) limit ground operations to 90 minutes outside of an air-conditioned environment. The FITS danger zone (> 38°C) adds the following five instructions to the four instructions of the caution zone: 1) minimum recovery time, landing time to next take off between flights, is 2 hours, 2) limit ground operations to 45 minutes for fighter/trainer aircraft types and time outside air-conditioned environment, 3) if possible, wait in a cool, shaded area if the aircraft is not ready to fly, 4) complete a maximum of two aircraft inspections, two exterior inspections on initial sorties, and one exterior inspection on subsequent sorties for fighters and trainers, and 5) Undergraduate Flying Training solo students are to complete one exterior aircraft inspection per sortie.
Table 7.8.2.1-1. Wet Bulb Globe Temperature Heat Stress Index

Generally, evaporative heat loss is the most significant method for cooling of the body when the air temperature is greater than the skin temperature. If the conditions hold true and air temperature is higher than skin temperature and air humidity is less than the vaporization of water from skin to air, the evaporative cooling can account for all heat loss. Air movement, i.e., convection, is a major aid to the evaporative process as well.

Table 7.8.2.1-2. Fighter Index of Thermal Stress (FITS) Reference Values and Flag Colors

Generally, evaporative heat loss is the most significant method for cooling of the body when the air temperature is greater than the skin temperature. If the conditions hold true and air temperature is higher than skin temperature and air humidity is less than the vaporization of water from skin to air, the evaporative cooling can account for all heat loss. Air movement, i.e., convection, is a major aid to the evaporative process as well.
7.8.2.2. **Physiological Response to Heat Exposure.** To maintain the narrow range of core body temperature ($T_c$), in addition to the external environmental factors, the body will also depend upon physiological and behavioral responses for modification. Unfortunately, behaviorally, military personnel may be able to override the common-sense factor of the physiological responses simply due to motivation of mission completion and may succumb to overexposure to heat stress. The heat defense response (increased sweating and vasodilation of the skin) is the process that is responsible for body heat loss via evaporation, convection, and conduction by altering skin blood flow. Evaporation is dependent upon sweat rate production and is reduced due to the environmental factors of high air humidity and low air movement; hot, humidified air stays close to the body and minimizes the skin’s contact of cooler, less humid air.

As the $T_c$ of the body is maintained through the heat defense response, then heat balance is reestablished and further action is not required. However, if the heat gain exceeds the heat loss and the body cannot dissipate the heat, then the heat balance moves towards heat gain and the thermal heat strain will eventually ensue. With the increase in heat strain, the normal response is to vasodilate to increase skin blood flow. If successful, the $T_c$ will stabilize as the skin temperature begins to decrease proportional to the reduced heat strain. However, if the $T_c$ stays elevated and skin blood flow stays high, then there’s the potential the high skin blood flow might affect cardiovascular function. With increased peripheral blood flow, there’s less blood centrally and that negatively affects cardiac filling and stroke volume, so heart rate must increase to maintain cardiac function. The sympathetic nervous stimulation attempts to compensate the reduced cardiac function by increasing myocardial contractility to deliver oxygenated blood to the visceral tissue; i.e., muscle and skin. If excessive, the improper redistribution in blood flow might lead to heat stress injuries.

Dehydration might cause additional heat injury due to excessive water loss, especially in nonacclimatized bodies where the sweat rate is a maximum of 1.5 L/hr. Dehydration can reduce evaporative and convection heat loss, an increase in $T_c$ by 0.2°C (0.36°F) per every 1% body weight loss, increase in cardiac strain by approximately 5 BPM per 1% body weight, reduction in physical work capability, and reduction in $T_c$ tolerance. Physical work capacity may be reduced in temperate environments by approximately 50% when the human body experiences 4% body weight loss. Just as one might want to avoid dehydration, it is also essential to understand that one can overconsume water (hyperhydration) and induce a state of low blood sodium (hyponatremia).

7.8.2.3. **Psychological Response to Heat Exposure.** Unfortunately, the research on the effects of heat stress and mental performance is not as extensive as it is on the physiological response to heat stress. Mentally, heat stress reduces one’s ability to perform cognitive functions, primarily due to the thermal discomfort accompanied with high skin temperature, high skin moisture, and increased cardiovascular strain. Mental decrements in performance occur in boring, monotonous, repetitive tasks; tasks that require attention to detail, concentration, and short-term memory and tasks that are not self-paced may degrade from heat stress. Heat stress is responsible for slowed reaction and decision times, increase in error of omission, and slowed performance times in routine tasks, with slight degradation in vigilant task performance after 30 minutes progressing to a marked decrement within 2 to 3 hours. A minor 2% body weight loss due to dehydration affects serial addition, response time,
and word recognition during heat stress exposure and will probably be exacerbated as dehydration worsens.

7.8.2.4. Effect of Self-Imposed Factors on the Physiological Response to Heat Exposure. The consideration of individual- and mission-imposed factors and how they could negatively affect human performance under heat stress exposure are important. There is an increase in the likelihood of heat strain and/or heat casualties if one or more of the following risk factors are present close to or the day of the event: dehydration, electrolyte depletion, poor nutrition, lack of acclimatization, poor physical condition, excessive body weight, skin disorders, medications/over-the-counter medications, alcohol, illness/disease, and genetics. These heat exposure risk factors can be further divided into individual- and mission-imposed factors; individual imposed factors are defined as those risk factors that might negatively impact the mission and are under your complete control. For example, dehydration, electrolyte depletion, poor physical condition, excessive body weight, and alcohol are considered individual-imposed factors. Meanwhile, mission-imposed factors might include, but are not limited to, lack of acclimatization, poor physical condition, medications, illness/disease, or genetics.

The intent of understanding individual- and mission-imposed factors and their effect on human performance in heat stress exposure is simply a necessity of prevention. Prevention is paramount in the reduction of heat-related injuries and casualties; since the mid-1980s through the mid-1990s, there has been a decrease in heat casualties of military recruits from 60 per 100,000 soldiers to 30 per 100,000 soldiers. It was determined that strategic planning before combat operations was a basic requirement in order for prevention to be effective. With the proper implementation of strategic planning as a preventive measure, one can mitigate the problems of hostile conditions, mission requirements, supply problems, and poor physical conditioning. In addition to dehydration being one of the most significant factors to guard against heat stress exposure, military and civilian personnel have gathered significant data that support that low aerobic fitness level (> 12 minutes in 1.5-mile fitness run) and high body mass index (> 26 kg/m²) have a 9x greater risk of heat illness. Evidence also suggests that some are susceptible to malignant hyperthermia (e.g., an example of a mission-imposed threat in which a military member might have a genetic predisposition) and that approximately 17% of heat stroke victims were sick in the days prior to the heat stress exposure. Additionally, individual-imposed factors such as alcohol, poor physical conditioning, illness/disease, and medications/over-the-counter medications may impair the function of the heat defense response. Alcohol will cause dehydration and hypoglycemia, some drugs reduce sweating while others change blood flow distribution, and poor physical conditioning and illnesses reduce the human body’s ability for proper thermoregulation. The best method of prevention combines the knowledge of how to minimize individual- and mission-imposed factors with allowing time for acclimatization.

7.8.2.5. Acclimatization to Heat Exposure. Military personnel must be fit-to-fight, meaning that not only do they need to know the environmental-, individual-, and mission-imposed factors but they must also be able to understand the operational scenarios and effective management of the details to properly educate and train the troops about heat stress. Successful heat stress management begins with the implementation of procedures designed to mitigate the threats of heat stress.
exposure, and then intervention measures must be executed to reduce all the risk
factors. A military member who is prepared to handle any hot environment and able to
perform at a high level of efficiency will be fit, healthy, hydrated, well-nourished,
educated, and trained on all aspects of the current military operating environment and
has allowed ample time for heat acclimatization.

An effective heat acclimatization program for any military personnel getting ready
to either change operating locations or be deployed needs to consider frequency,
intensity, and duration of heat exposures as well as a thorough heat strain decision
process. Before the heat acclimatization program is implemented, the heat strain
decision process needs to be in place and ready for any emergency action necessary.
A simple heat strain decision process might have the following components: heat stress,
mission requirements, uniforms and equipment, identification of high- and low- risk
personnel, heat mitigation procedures, feedback, and review of the process for further
modifications and improvements (Figure 7.8.2.5-1). Once the heat strain decision
process is in place for safety, the acclimatization program details can be organized and
implemented.

<table>
<thead>
<tr>
<th>Heat Stress</th>
<th>WBGT</th>
<th>Microenvironment</th>
<th>Climate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Analyze Mission Requirements</td>
<td>Work rate</td>
<td>Duration</td>
<td>Airmen status</td>
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<tr>
<td>Determine Uniform &amp; Equipment</td>
<td>Body armor</td>
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<tr>
<td>Identify High Risk Airmen</td>
<td>Low Fitness/Overweight</td>
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<tr>
<td>Implement Heat Mitigation</td>
<td>Fluid Replacement</td>
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<tr>
<td>Observe / Supervise</td>
<td>Leadership</td>
<td>Wingman</td>
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**Figure 7.8.2.5-1. Heat Strain Decision Process**

Heat acclimatization is a 2 weeks, progressive process that limits the intensity,
frequency, and duration of heat exposure and physical activity with the focus being on
unacclimatized personnel. As soon as the second day you might expect to see small
changes of acclimatization, and the physiological strain of heat is reduced; however, by the end of the first and second week, the expectation is that personnel are approximately 50% and 80% physiologically adapted, respectively, for the “average soldier.” The greater the physical fitness status the airmen are starting out with the faster they will acclimate, approximately 70% heat acclimation by the end of the first week. At this point, if no further heat stress exposures occurred, then effects of the acclimatization would last about 1 week, at which point deacclimatization occurs at a rate of 75% over the next 3 week. All military personnel will reap benefits of the heat acclimatization program; it’s relative to the initial physical fitness and total heat stress exposure. The minimum heat exposure per day is 2 hours, which can be broken up into 1-hr intervals and must consist of aerobically based endurance activities (e.g., jogging or marching) that gradually progress up in workload and time. Personnel will only acclimate to the environment to which they have been exposed; if they rest in heat then that’s what their relative work capacity will be once placed in the operational environment. Once deployed, continue the heat stress exposures based upon the slowest participant and keep account of the performances of the least- and most-motivated personnel – these people are potential safety hazards – always monitoring the work/rest cycles and hydration consumption (Table 7.8.2.1-1).

7.8.2.6. Medical Considerations. The medical needs of heat stress exposure casualties should be viewed as a continuum of signs and symptoms that can overlap one another and quite often are not clear-cut in presentation, which can lead to diagnostic issues due to lack of recognition (Figure 7.8.2.6-1). The purpose of the following section is to present a simplified version of heat stress exposure injuries and illnesses to aid in the recognition. However, the aspect of treatment is minimized due to the fact that medically qualified personnel need to be the service providers when extreme conditions manifest. From a prevention perspective, it is important that all military personnel be knowledgeable about heat stress exposure, the recognition of heat stress signs and symptoms, and the basics in care and treatment of heat stress illnesses and injuries so as to provide supportive aid as necessary until medical interventions can be administered by qualified personnel.

As stated previously, the classification of heat stress exposure injuries and illnesses can be a difficult task due to the fact that an individual may not always present with a standard list of signs or symptoms in a particular order over a set period of time, every single time (Figure 7.8.2.6-2). Holding all things equal, generally speaking, heat stress exposure casualties will present with minor heat illnesses first that progress to exertional heat injuries and ultimately can terminate in a heat stroke, all of which can be caused by the imbalance between the heat gain (i.e., metabolic, physical activity, environmental) and heat loss (i.e., heat defense response), which culminates in the rise of core body temperature as a dysfunction of the body’s compromised thermoregulatory response.
Figure 7.8.2.6-1. Heat Stress Exposure Spectrum

Figure 7.8.2.6-2. Heat Stress Illness Signs and Symptoms
On the left end of the heat stress continuum (Figure 7.8.2.6-1), with moderate severity, the minor heat-related illnesses are heat edema, miliaria rubra, sunburn, heat tetany, heat syncope, and heat cramps. Heat edema is commonly known for swelling in the periphery of the hands and feet that usually manifests in tight-fit clothing on those areas. The cause of heat edema is unknown, so treatment is equally vague, with the focus on comforting the individual and evaluating the area as needed; time for acclimatization will usually resolve the problem. Miliaria rubra, also known as “prickly heat” or “heat rash,” is a condition of the skin occurring when eccrine secretory sweat glands become clogged, which can lead to redness and inflammation of the affected area. Treatment consists of conditions that keep the affected area cool and dry and the proper application of prescribed medication; good hygiene and loose clothing aid in the healing process. The primary concern should focus on the fact that the affected area cannot assist in thermoregulation; therefore, the person’s thermoregulatory system is compromised proportionally to the size and severity of the area. One must pay close attention to those with heat rash as to not exacerbate the condition by poor implementation of prevention measures. Heat tetany, i.e., long tonic spasm of muscle, is caused by simple hyperventilation in unacclimatized personnel and should be treated appropriately. The condition will include tightening of muscles with potential numbness and tingling; simply calm and control breathing to mimic a normal rate and depth of breath. Heat cramps should not be confused with heat tetany. Heat cramps are short, recurring muscle contractions lasting 2 to 3 minutes that may present in the form of a palpable ball after vigorous activity. The condition is thought to rise from sodium depletion with the resultant effect of a calcium increase in the intracellular muscle tissue, thus causing the cramps; muscle cramps usually subside upon electrolyte replenishment. The final minor heat-related illness is syncope. Heat syncope or “parade syncope” can range from lightheadedness to loss of consciousness due to peripheral blood pooling and a reduction in cardiovascular ejection fraction. The reason for the vasodilation and reduced venous return is focused on the heat stress exposure and the defense response; compounding variables that would exacerbate the condition include vigorous activity and weather conditions. Heat syncope presenting after more than 5 days of acclimatization might indicate other medical conditions such as heat exhaustion or exertional heat injury.

Heat exhaustion is the most common type of heat-related injury and falls under the leftmost aspect of the heat stress exposure continuum as moderate in severity. There is no organ damage from heat exhaustion, and personnel succumbing to the heat stress exposure with these signs and symptoms will normally recover quickly. The occurrence of heat exhaustion is preceded by a demand mismatch of blood flow between skin that is involved in the heat defense response and delivery to active muscle mass and organs. The cardiac output of the heart is unable to provide sufficient ejection fraction per beat to deliver oxygen-rich blood to all demanding tissues, thus exhaustion due to heat stress. The signs and symptoms personnel may experience might range from generalized weakness, fatigue, ataxia, dizziness, headaches, nausea, vomiting, malaise, hypotension, tachycardia, muscle cramps, hyperventilation, to transient changes in mental alertness. The onset and/or severity of these signs and symptoms can be exacerbated by dehydration, hypovolemia, peripheral blood pooling, and potentially failure of splanchnic vasoconstriction. With heat exhaustion, the individual is combating excessive cardiovascular demand and some level of electrolyte depletion but is still able to thermoregulate through sweating, and it may be more profuse than normal. Recovery from an episode consists of removal of the personnel
from the hot environment, replenishment of electrolytes, plus rest. If the casualty is young and fit, then field treatment is sufficient; however, if the condition continues to deteriorate, then medical intervention is required. The same holds true for recurrent episodes, which may indicate reduced heat tolerance.

As the intermediate level of severity on the heat injury continuum, exertional heat injury and rhabdomyolysis can be difficult to distinguish from heat exhaustion unless vital signs and blood chemistries are being monitored. Heat injury and rhabdomyolysis show evidence of organ (i.e., kidney or liver) and tissue (i.e., muscle) damage or dysfunction but do not include neurological aspects as seen with heat stroke. Personnel will still be sweating, possibly profusely, thus the heat defense response is still functioning and cooling can occur; however, precautionary measures dictate immediate active cooling to reduce core body temperature below 38.3°C (101°F) and rehydration plus electrolytes. Exertional rhabdomyolysis may not have an increase in $T_C$, but blood chemistries will reveal higher levels than normal in creatine kinase, phosphate, potassium, uric acid, and myoglobin, as they have been released from the muscle tissue due to cellular destruction. Medical intervention is necessary in these intermediate-level heat injuries with the application of rest in a cool environment and fluid-electrolyte replacement.

Heat stroke is the failure of the thermoregulatory system and must be handled as a catastrophic medical emergency. In heat stroke, the $T_C$ has elevated above 40°C (104°F), causing central nervous system dysfunction and organ system failure. The central nervous system disorders consist of an ambiguous set of signs or symptoms that may be transient or persistent, with the resultant dysfunction of delirium, convulsions, or coma. The heat stroke casualty might exhibit onset signs and symptoms of headaches, dizziness, drowsiness, restlessness, ataxia, confusion, and irrational or aggressive mental status. There are two categories of heat stroke, classical and exertional, presenting with dissimilar signs and symptoms because of the environmental settings and the affected personnel; Table 7.8.2.6-1 outlines the differences. The classical signs and symptoms of classical heat stroke (e.g., coma, convulsions, and cessation of sweating) may be late or delayed in exertional heat stroke, while rhabdomyolysis and renal failure will be present for exertional heat stroke. These diagnostic criteria can be misinterpreted because of a strict adherence, which often leads to an under- or misdiagnosis of exertional heat stroke.

The principal effect of heat stroke injury focuses on the damage to organ tissues, such as brain, liver, kidney, and muscle, which interrupts homeostasis. Compounding factors that contribute to the increased severity of the condition are mostly due to the magnitude of $T_C$, the duration of exposure, and any other additional underlying physiological conditions, for example, tissue ischemia, hypokalemia, exercise-induced lactic acidosis, and any systemic inflammatory responses. Cognitively, a heat stroke victim’s dysfunction may include delirium, euphoria, coma, hallucinations, rapid eye movement, and seizures; seizures might alternate with tonic contractions, tremors, and muscle cramps. Hepatic liver injury is generally seen in heat stroke conditions and can lead to hypoglycemia and jaundice 24 to 36 hours post-onset. Acute renal failure occurs in approximately 30% of exertional heat stroke cases and is preceded by rhabdomyolysis. The muscular system is often affected by the increase in plasma levels of myoglobinuria and muscle enzyme due to the presence of rhabdomyolysis accompanied by muscle rigidity. Coagulatory issues, e.g., microthrombi, arise in the blood due to rhabdomyolysis, hepatocyte, and vascular endothelium damage as well as thermal platelet activation. Additionally, cardiac (i.e., hyper- or hypodynamic circulatory
states) and gastrointestinal tract (i.e., diarrhea or vomiting) dysfunction or pulmonary edema may be present if the severity of the injury is extreme. The outcome of the heat stroke casualty is dependent upon the duration and magnitude of the elevated $T_C$ temperature, and the most important therapeutic measure is the rapid cooling of the body. Any means should be taken to rapidly cool the body, from immersion in cold water, to ice baths, to the use of ice packs; rapid cooling can reduce the mortality rate of heat stroke casualties from 50% to 5%. During mission planning and execution, the obstacle that often needs to be overcome regarding heat stress illness and injury is the possible perception that it is only a minor threat. The responsibility lies with all military personnel, directly and indirectly, to identify, assess, analyze, decide, implement, and review; prevention is the key factor.

### Table 7.8.2.6-1. Heat Stroke Categorical Differences

<table>
<thead>
<tr>
<th>Personnel Characteristics</th>
<th>Exertional</th>
<th>Classical</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>15 – 45 years of age</td>
<td>Child or elderly</td>
</tr>
<tr>
<td>Health</td>
<td>Usually healthy</td>
<td>Chronic illness common</td>
</tr>
<tr>
<td>History of illness</td>
<td>Common</td>
<td>Unusual</td>
</tr>
<tr>
<td>Environment</td>
<td>Variable</td>
<td>Prolonged heat</td>
</tr>
<tr>
<td>Activity</td>
<td>Strenuous exercise</td>
<td>Sedentary</td>
</tr>
<tr>
<td>Drug use</td>
<td>Usually none, some use of ergogenic aids or stimulants</td>
<td>Diuretics, antidepressants, anticholinergics, phenothiazines</td>
</tr>
<tr>
<td>Sweating</td>
<td>Present</td>
<td>Usually absent</td>
</tr>
<tr>
<td>Acid-base disturbances</td>
<td>Lactic acidosis</td>
<td>Respiratory alkalosis</td>
</tr>
<tr>
<td>Acute renal failure</td>
<td>Common (=30%)</td>
<td>Rare (&lt;5%)</td>
</tr>
<tr>
<td>Rhabdomyolysis</td>
<td>Common</td>
<td>Seldom severe</td>
</tr>
<tr>
<td>Hyperuricemia</td>
<td>Marked</td>
<td>Modest</td>
</tr>
<tr>
<td>Creatinine ratio (blood)</td>
<td>Elevated</td>
<td>1:10</td>
</tr>
<tr>
<td>Creatine-kinase, aldolase</td>
<td>Markedly elevated</td>
<td>Mild elevation</td>
</tr>
<tr>
<td>Hyperkalemia</td>
<td>Often present</td>
<td>Usually absent</td>
</tr>
<tr>
<td>Hypercalcemia</td>
<td>Common</td>
<td>Uncommon</td>
</tr>
<tr>
<td>Disseminating Intravascular Coagulation</td>
<td>Maybe marked</td>
<td>Mild</td>
</tr>
<tr>
<td>Hypoglycemia</td>
<td>Common</td>
<td>Uncommon</td>
</tr>
</tbody>
</table>

#### 7.8.2.7. Prevention. To successfully prevent a heat stress emergency, military personnel must be knowledgeable in the information, proficient in the application of procedures through education and training, and, ultimately, consistent in implementation of mission planning. Aspects of planning to consider are the environmental settings in which personnel will be exposed, the knowledge of how that will affect their performance, when to implement procedures to mitigate the threat of a heat stress casualty, and the efficient execution of emergency procedures when required. From a prevention perspective, military personnel must consider the things they have within their control to reduce their risk of succumbing to heat stress injury: fluid/electrolyte replacement, nutrition, and personal clothing ensembles.

First and foremost are the fluid and nutrition requirements for military personnel when they’re placed in an environment that exposes them to heat stress. Hydration and nutrition considerations for military personnel exposed to hot environments must be based on the simple premise that they will sweat and feel apathy towards food. With an increase in heat stress, there is a concomitant increase in sweat rate and, therefore, body water loss. Thirst is not a sufficient sign of hydration requirements because most often personnel have lost about 2% of body weight from water loss; they are already dehydrated once thirst is stimulated. Under normal conditions, water replacement is not
a primary concern, as rehydration occurs during mealtime. However, satiety for food is suppressed during heat stress, and thus the desire to drink might be suppressed simply because meals are eliminated, thus reducing water intake indirectly. Common methods to reensure water intake is sufficient consist of the frequency, volume, and clarity check method or the weight-loss method. Albeit a little unscientific, operationally the urine check method is easy to employ. Generally, a good sense of hydration is exemplified in frequent, large-volume, and clear urination; conversely, infrequent, small-volume, and dark-colored urine is an indication of dehydration. The weight method is based upon Archimedes’ principle and simply stated is as follows: 1 kg of body weight loss during a bout of physical activity under hot stress exposure is equal to 1 L of water loss; remember to subtract out the weight of the unsoaked and soaked clothing. Truly, the most effective method for consistent hydration status is to allocate work/rest cycles, water breaks, and mealtimes. The fluid supplied during the breaks should be cool (50°F to 60°F) for optimal palatability and contain water, electrolytes, and a carbohydrate replacement. Sweat rates can range considerably, as they relate to the physical activity level and the environment. Sweat rates for the most common military function can be 0.3 to 1.0 L/hr, with rates up to 2.2 L/hr while donning NBC protective clothing while working. Based upon Figure 7.8.2.7-1 and Table 7.8.2.7-1, these normative data provide caloric expenditures per job duty based upon the current environmental temperature and correlate that to a specific water requirement.

Figure 7.8.2.7-1. Nomogram for Daily Water and Nutritional Consumption
Table 7.8.2.7-1. Military Personnel Total Daily Caloric Expenditure per Job Duty

<table>
<thead>
<tr>
<th>Group</th>
<th>Activity</th>
<th>kcal/day</th>
</tr>
</thead>
<tbody>
<tr>
<td>Army Special Forces</td>
<td>Combat exercise, temperate</td>
<td>3,400</td>
</tr>
<tr>
<td>Army Engineers</td>
<td>Build road and airstrip @ altitude</td>
<td>3,549</td>
</tr>
<tr>
<td>Army Transportation Company</td>
<td>Garrison</td>
<td>3,568</td>
</tr>
<tr>
<td>Marine Combat Engineers</td>
<td>Construction</td>
<td>3,668</td>
</tr>
<tr>
<td>Israeli Infantry</td>
<td>Combat exercise, summer</td>
<td>3,937</td>
</tr>
<tr>
<td>Army</td>
<td>Support hospital</td>
<td>3,960</td>
</tr>
<tr>
<td>Army Ranger</td>
<td>Training course</td>
<td>4,010</td>
</tr>
<tr>
<td>Army Ranger</td>
<td>Training course</td>
<td>4,090</td>
</tr>
<tr>
<td>Marine</td>
<td>Artillery exercise, desert</td>
<td>4,115</td>
</tr>
<tr>
<td>Marine</td>
<td>Combat exercise, winter</td>
<td>4,198</td>
</tr>
<tr>
<td>Army</td>
<td>Artillery exercise, winter</td>
<td>4,253</td>
</tr>
<tr>
<td>Israeli Infantry</td>
<td>Combat exercise, winter</td>
<td>4,281</td>
</tr>
<tr>
<td>Army Special Forces</td>
<td>Combat exercise, winter</td>
<td>4,558</td>
</tr>
<tr>
<td>Marine</td>
<td>Crucible, women</td>
<td>4,679</td>
</tr>
<tr>
<td>Australian Infantry</td>
<td>Jungle training</td>
<td>4,750</td>
</tr>
<tr>
<td>Army Special Forces</td>
<td>Assessment school</td>
<td>5,183</td>
</tr>
<tr>
<td>Army Ranger</td>
<td>Combat exercise</td>
<td>5,185</td>
</tr>
<tr>
<td>Norwegian Ranger</td>
<td>Training course</td>
<td>6,250</td>
</tr>
<tr>
<td>Marine</td>
<td>Crucible, men</td>
<td>6,067</td>
</tr>
<tr>
<td><strong>Average</strong></td>
<td></td>
<td><strong>4,405</strong></td>
</tr>
</tbody>
</table>

**Electrolyte replacement** can take place in many different forms; most often under normal conditions, the normal meals throughout the day are adequate enough to replenish the daily turnover of electrolytes. However, the additional aspect of sweating from heat stress exposure increases sodium loss, ≈10 to 70 mmol/L, but that can be reduced by 50% through heat acclimatization. Most military personnel living and working in a hot environment, on average, lose approximately 4 to 9 g of sodium per day. If deployed, the consumption of three Meals, Ready to Eat (MREs) will adequately replace the sodium loss, but all the meal must be eaten including the salt packets as well. Similar to the water requirement nomogram, Figure 7.8.2.7-2 depicts the sodium requirement per day based upon the ambient temperature under specific caloric requirements.

From a thermal protection viewpoint, clothing can be either a major advantage or disadvantage, depending upon the individual- and mission-imposed stresses. The concept of multi-layers is essential in a hot environment. Long-sleeved shirts provide protection from radiant heat and skin protection from heat injuries such as sunburns and allow military personnel to take protective layers off as needed to provide a balance between radiant stress and thermal stress. The full spectrum begins with mission planning, when variables such as clothing layers required (e.g., cotton underwear, fire-retardant coveralls, G-suit, parachute harness, gloves, boots, etc.), environmental conditions (i.e., on earth, in the cockpit, in the back of the aircraft with the ramp deployed, at altitude, over the water, etc.), and nutritional and hydration status (e.g., pre-flight, during flight and post-flight) are considered. Mission planning is essential for the prevention of heat stress injuries/illnesses and must take into account individual- and mission-imposed stresses.
Ultimately, the responsibility of training and its proper implementation lie with the unit leadership. The United States Army Center of Health Promotion and Preventive Medicine has distributed a heat risk manual that outlines a five-step risk management tool to prevent heat stress causalities (Figure 7.8.2.7-3). The first step is to identify the hazards, such as weather conditions; inventory of required supplies; and self-imposed factors. The second step of hazard assessment focuses on the proper use of heat index charts relating to environmental conditions, work intensity, food and clothing, and injuries stratified into risk categories. The development of control points establishes limits for the third step. The implementation, supervision, and evaluation of the risk management plan are the final steps of the process, steps four and five. The heat stress exposure risk management tool is only effective if it remains as a dynamic loop that continues to take into account the ever-changing variables of the ambient environment, military personnel, and operational mission. Situational awareness is paramount when military personnel perform active and nonactive duties in hot environments, and if the proper preventive measures are implemented, heat stress exposure injuries such as hyperthermia, heat exhaustion, heat injuries, and heat stroke can be prevented. Prevention begins in pretraining preparation, use of heat stress index tables chart to manage personnel time of exposure to the hot environment, risk assessment by medical personnel on active duty personnel, an established buddy-system to advise one another in early detection of hazardous factors, and, importantly, leadership: leading by example.
Figure 7.8.2.7-3. Commanders, Senior NCPs and Instructors Guide to Risk Management of Heat Casualties
2. Assess Hazards

- When ambient temperature is over 75° F, constantly assess the **heat category** using Wet Bulb Globe Temperature (WBGT).
- Know your soldiers! Identify early who will be at increased risk based on **individual risk factors**.
- Check **hydration status** at the end of each training day. Give extra fluid at night and in the morning if hydration is inadequate.
  - Review Riley (water) card or Ogden cords
  - Ask about urine color. Urine is clear if well hydrated
- Daily assess the **overall risk** for developing a heat casualty (may use a risk matrix).

The following matrix has been used successfully through experience by Commanders.

### Example of a Heat Injury Risk Management Matrix

Scores assigned to different conditions based on risk for developing a heat injury.
This scoring system: 0 = Low risk, 1 = Medium risk, 2 = High risk, 3 = Extreme risk

<table>
<thead>
<tr>
<th>RISK FACTORS</th>
<th>Level of Risk</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Risk Management Worksheet</td>
<td>All control measures implemented</td>
<td>Not all control measures implemented</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heat (WBGT at site)</td>
<td>None</td>
<td>Category 1</td>
<td>Category 2 and 3</td>
<td>Category 4 and 5</td>
<td></td>
</tr>
<tr>
<td>No. Sequential Days Heat Cat 6</td>
<td>0</td>
<td>1</td>
<td>2-3</td>
<td>≥4</td>
<td></td>
</tr>
<tr>
<td>Heat Injuries in the unit in Past 2 Days</td>
<td>None</td>
<td>Heat Cramps</td>
<td>Heat Exhaustion</td>
<td>Heat Stroke*</td>
<td></td>
</tr>
<tr>
<td>Work in Past Two Days (see below)</td>
<td>Easy</td>
<td>Easy</td>
<td>Moderate</td>
<td>Hard</td>
<td></td>
</tr>
<tr>
<td>Projected Work for the Present Day</td>
<td>Easy</td>
<td>Easy</td>
<td>Moderate</td>
<td>Hard</td>
<td></td>
</tr>
<tr>
<td>Heat Acclimatization Days</td>
<td>&gt;13</td>
<td>7-13</td>
<td>3-6</td>
<td>&lt;3</td>
<td></td>
</tr>
<tr>
<td>Leader/Cadre Presence</td>
<td>Full time</td>
<td>Substantial</td>
<td>Minimal</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>Length of Duty Time of Cadre</td>
<td>18 Months</td>
<td>7-18 Months</td>
<td>1-6 Month</td>
<td>&lt; 1 Month</td>
<td></td>
</tr>
<tr>
<td>Communication System</td>
<td>Radio and Phone</td>
<td>Phone Only</td>
<td>Radio Only</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>Rest in Previous 24 Hours</td>
<td>&gt; 7 Hours</td>
<td>5-7 Hours</td>
<td>2-4 Hours</td>
<td>&lt; 2 Hours</td>
<td></td>
</tr>
</tbody>
</table>

Cumulative score: 25-33 = extreme risk, 16-24 = high risk, 7-15 = medium risk, 0-6 = low risk.
* if Heat Stroke has occurred in unit in past 2 days, risk level= extreme risk

<table>
<thead>
<tr>
<th>Easy Work</th>
<th>Moderate Work</th>
<th>Hard Work</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weapon Maintenance</td>
<td>Walking Loose Sand at 2.5 mph, no Load</td>
<td>Walking Hard Surface at 3.5 mph, ≥ 40 lb Load</td>
</tr>
<tr>
<td>Walking Hard Surface at 2.5 mph, &lt; 30 lb Load</td>
<td>Walking Hard Surface at 3.5 mph, &lt; 40 lb Load</td>
<td>Walking Loose Sand at 2.5 mph with Load</td>
</tr>
<tr>
<td>Marksmanship Training</td>
<td>Calisthenics</td>
<td>Field Assaults</td>
</tr>
<tr>
<td>Drill and Ceremony</td>
<td>Patrolling</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Individual Movement Techniques, i.e. low crawl, high crawl</td>
<td></td>
</tr>
</tbody>
</table>

![Figure 7.8.2.7-3. Commanders, Senior NCPs and Instructors Guide to Risk Management of Heat Casualties (continued)](image-url)
3 Develop Controls

Education

- Establish SOPs. Ensure all personnel are trained and follow SOPs for Heat Casualty Prevention.
- Ensure all bulletin boards have Heat Casualty Prevention posters and all leaders have Heat Casualty Prevention aids.

Planning

- Adjust the training schedule to minimize consecutive days of heavy physical training, especially if other heat stressors exist (e.g., heat exposure and lack of quality sleep).
- Plan communications, medical and evacuation support.
- Plan and provide adequate hydration for all personnel (including Cadre and Drill Instructors).
- When planning training events, keep in mind:
  1. Time of day the training is conducted — morning is cooler
  2. Location of training
     - Sun vs. shade. Rest in shade.
     - Open vs. protection from wind — wind has cooling effect
     - Open up the formation to decrease heat strain.
  3. Clothing
     - Heavy, restrictive vs. loose, lightweight
  4. Where in training cycle
     - Most Heat Casualties occur in the 2nd or 3rd week of Recruit training.
     - Acclimatization can take 7-14 days, depending on the physical condition of the trainee.
- After moderate to hard work in heat category ≥3; take cold, nude showers at the end of the day.

Identification

- Identify previous heat exhaustion or heat stroke soldiers and mark visibly on uniform (tape or cord).
- Identify overweight soldiers and soldiers who are unfit.
- Identify soldiers on medications and mark visibly on uniform (tape or cord).
- Seriously consider taking soldiers out of training who have had alcohol within the last 24h. Seriously consider having ill soldiers seen on sick call.
- Note and document heat category hourly. Position WBGT at site of training.

Develop a Hydration Monitoring System

- Examples of monitoring methods:
  - Riley (water) card. On the card, Battle buddy is to write the amount of water the soldier has drunk.
  - Ogden Cord is 550 cord, parachute cord or shoestring that is tied to a uniform buttonhole or ear protection case. Soldiers tie a knot in the cord each time they finish a canteen (1 quart) of water.

![Water Consumption Card](image)

Figure 7.8.2.7-3. Commanders, Senior NCPs and Instructors Guide to Risk Management of Heat Casualties (continued)
Develop Controls continued

Know Standardized Guidelines for Warm Weather Training Conditions

Fluid Replacement and Work/Rest Guide

*Acclimatized (after approx two weeks training) Wearing BDU, Hot Weather*

<table>
<thead>
<tr>
<th>Heat Category</th>
<th>WBGT Index, (°F)</th>
<th>Easy Work</th>
<th>Moderate Work</th>
<th>Hard Work</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Work/ Rest</td>
<td>Water Intake (Qt/h)</td>
<td>Water Intake (Qt/h)</td>
<td>Water Intake (Qt/h)</td>
</tr>
<tr>
<td>1</td>
<td>NL</td>
<td>½</td>
<td>NL</td>
<td>¼</td>
</tr>
<tr>
<td>2 (Green)</td>
<td>NL</td>
<td>½</td>
<td>50/10 min</td>
<td>¾</td>
</tr>
<tr>
<td>3 (Yellow)</td>
<td>NL</td>
<td>¾</td>
<td>40/20 min</td>
<td>¾</td>
</tr>
<tr>
<td>4 (Red)</td>
<td>NL</td>
<td>¾</td>
<td>30/30 min</td>
<td>¾</td>
</tr>
<tr>
<td>5 (Black)</td>
<td>&gt; 90</td>
<td>1</td>
<td>20/40 min</td>
<td>1</td>
</tr>
</tbody>
</table>

- The work-rest times and fluid replacement volumes will sustain performance and hydration for at least 4 h of work in the specified heat category. Fluid needs can vary based on individual differences (± ¼ qt/hr) and exposure to full sun or full shade (± ¼ qt/hr).
- NL= no limit to work time per hour.
- Rest means minimal physical activity (sitting or standing), accomplished in shade if possible.
- **CAUTION:** Hourly fluid intake should not exceed 1½ quarts.
- Daily fluid intake should not exceed 12 quarts.
- If wearing body armor add 5°F to WBGT in humid climates.
- If wearing NBC clothing (mission-oriented protective posture (MOPP 4)), add 10°F to WBGT index for easy work, and 20°F to WBGT index for moderate and hard work.

**Easy Work** = Walking hard surface 2.5 mph <30# load, Weapon maintenance, Marksmanship training  
**Moderate Work** = Patrolling, Walking sand 2.5 mph no load, Calisthenics  
**Hard Work** = Walking sand 2.5 mph w/load, Field assaults

**Continuous Work Duration and Fluid Replacement Guide**

*Acclimatized (after approx two weeks training) Wearing BDU, Hot Weather*

It is assumed the trainees performing these continuous effort tasks have not yet had heat stress or dehydration prior to this activity and will have several hours of rest afterwards.

<table>
<thead>
<tr>
<th>Heat Category</th>
<th>WBGT Index, (°F)</th>
<th>Easy Work</th>
<th>Moderate Work</th>
<th>Hard Work</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Work (min)</td>
<td>Water Intake (Qt/h)</td>
<td>Work (min)</td>
<td>Water Intake (Qt/h)</td>
</tr>
<tr>
<td>1</td>
<td>NL</td>
<td>½</td>
<td>NL</td>
<td>¾</td>
</tr>
<tr>
<td>2 (Green)</td>
<td>NL</td>
<td>½</td>
<td>150</td>
<td>1</td>
</tr>
<tr>
<td>3 (Yellow)</td>
<td>NL</td>
<td>¾</td>
<td>100</td>
<td>1</td>
</tr>
<tr>
<td>4 (Red)</td>
<td>NL</td>
<td>¾</td>
<td>80</td>
<td>1 ¼</td>
</tr>
<tr>
<td>5 (Black)</td>
<td>&gt; 90</td>
<td>1</td>
<td>70</td>
<td>1 ½</td>
</tr>
</tbody>
</table>

- NL can sustain work for at least 4 hours in the specified heat category.
- Fluid needs can vary based on individual differences (± ¼ qt/hr) and exposure to full sun or full shade (± ¼ qt/hr).

---

**Figure 7.8.2.7-3. Commanders, Senior NCPs and Instructors Guide to Risk Management of Heat Casualties (continued)**

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4 Implement Controls

Decision to accept risk is made at the appropriate level
- Made in accordance with appropriate MACOM regulation

Identified controls are in place
- Update WBGT hourly when ambient temperature is ≥ 75°F.
- Adhere to work/rest cycle in high heat categories. Rest in shade.
- For tasks requiring continuous effort, adhere to guideline and allow extended rest afterwards.
- Training event incorporates good prior planning.

Monitor and enforce hydration standard
- Encourage frequent drinking, but not to exceed 1 ½ quarts per hour or 12 quarts per day. Make water more palatable, if possible, by cooling.
- Do not allow soldiers or trainees to empty canteens to lighten load (consider imposing a penalty in timed events).
- Ensure soldiers are well hydrated before training. Ask about urine; urine is clear if well hydrated.
- Check Riley (water) card or Ogden Cord frequently.

Monitor and enforce eating meals
- Ensure all meals are eaten during the meal break
- Ensure adequate time to eat and drink meals
- Table salt may be added to food when the heat category is high. Salt tablets are not recommended

Execute random checks
- Spot checks by Cadre, Senior NCO’s, and Drill Instructors
- Enforce battle buddy checks – need to be aware of each other’s eating, drinking and frequency of urination
- Plan placement of leaders to observe and react to heat injuries in dispersed training

Follow clothing recommendations
- Heat category 1-2: no restrictions
- Heat category 3: Unblouse trouser legs, unbutton web belt
- Heat category 4-5
  - Unblouse trouser legs, unbutton web belt
  - Remove t-shirt from under BDU top or remove BDU top down to T-shirt (depends whether biting insects are present)
  - Remove helmets unless there are specific safety reasons to keep them on (e.g., range)
- MCPP 4: Add 10°F to WBGT index for easy work, and 20°F to WBGT index for moderate to hard work.

Figure 7.8.2.7-3. Commanders, Senior NCPs and Instructors Guide to Risk Management of Heat Casualties (continued)
Supervise & Evaluate

- Enforce SOPs
- Delegate authority to ensure control measures have been implemented
- Monitor adequacy/progress of implementation of control measures
- Conduct spot checks of cadre. Do cadre have current WBGT? Are cadre implementing work/rest/drink cycles? Make on-the-spot corrections. Lead by example.
- Conduct spot checks of recruits. Ask recruits questions while observing their mental status and physical capabilities. Look out for common signs and symptoms which can rapidly progress to serious signs and symptoms. Ask recruits when did they last urinate and was their urine clear?
- If 1-2 recruits become heat casualties, stop all training and evaluate each soldier for early signs and symptoms of becoming an impending heat casualty.
- When controls fail, heat injuries occur. The ability to recognize heat injury is paramount. Take immediate action if any heat injuries are observed or suspected. Stop-rest-cool then evaluate in accordance with warning signs and symptoms. If in doubt, evacuate.

Warning Signs and Symptoms of Heat Casualty and Water Intoxication

<table>
<thead>
<tr>
<th>More Common Signs / Symptoms</th>
<th>Immediate Actions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dizziness</td>
<td>Remove from training</td>
</tr>
<tr>
<td>Headache</td>
<td>Allow casualty to rest in shade</td>
</tr>
<tr>
<td>Nausea</td>
<td>Loosen clothing</td>
</tr>
<tr>
<td>Unsteady walk</td>
<td>Take sips of water</td>
</tr>
<tr>
<td>Weakness or fatigue</td>
<td>While doing the above, call for a Medic to evaluate the soldier (Medic will monitor temperature and check for mental confusion)</td>
</tr>
<tr>
<td>Muscle cramps</td>
<td>If no medic is available call for ambulance or Medevac</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Serious Signs / Symptoms</th>
<th>Immediately call Medevac or ambulance for emergency transport while doing the following:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hot body, high temperature</td>
<td>Lay person down in shade with feet elevated until Medevac or ambulance arrives</td>
</tr>
<tr>
<td>Confusion, agitation (Mental Status Assessment)</td>
<td>Undress as much as possible</td>
</tr>
<tr>
<td>Vomiting</td>
<td>Aggressively apply ice packs or ice sheets</td>
</tr>
<tr>
<td>Involuntary bowel movement</td>
<td>Pour cold water over casualty and fan.</td>
</tr>
<tr>
<td>Convulsions</td>
<td>Give sips of water while awaiting ambulance (if conscious)</td>
</tr>
<tr>
<td>Weak or rapid pulse</td>
<td>Monitor airway and breathing until ambulance or Medevac arrive</td>
</tr>
<tr>
<td>Unresponsiveness, coma</td>
<td></td>
</tr>
</tbody>
</table>

Figure 7.8.2.7-3. Commanders, Senior NCPs and Instructors Guide to Risk Management of Heat Casualties (continued)
### Indications of possible Water Intoxication (Over Hydration)

<table>
<thead>
<tr>
<th>Signs and Symptoms</th>
<th>What to do:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Confusion</td>
<td>Ask these questions to the soldier or battle buddy:</td>
</tr>
<tr>
<td>Weakness</td>
<td>1. Has soldier been eating? Check rucksack for # of MRE's left.</td>
</tr>
<tr>
<td>Nausea</td>
<td>2. Has soldier been drinking a lot? (suspect water intoxication if soldier has been drinking constantly).</td>
</tr>
<tr>
<td>Vomiting</td>
<td>3. How often has soldier urinated? (frequent urination seen with water intoxication; infrequent urination with heat illness).</td>
</tr>
<tr>
<td></td>
<td>4. What color is urine (clear urine may indicate over hydration).</td>
</tr>
</tbody>
</table>

*If soldier has been eating, drinking and urinating a lot, yet has these symptoms, immediately call Medevac or ambulance for emergency transport.*

### Mental Status Assessment

An important sign that the soldier is in a serious life-threatening condition is the presence of mental confusion (with or without increased temperature). Anyone can do a mental status assessment asking some simple questions.

**Call for emergency Medevac or ambulance if any of the following exist:**

- **What is your name?**  
  (Does not know their name.)
- **What month is it? What year is it?**  
  (Does not know the month or year.)
- **Where are we/you?**  
  (Is not aware of location or surroundings.)
- **What were you doing before you became ill?**  
  (Does not know the events that led to the present situation.)

---


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**Figure 7.8.2.7-3. Commanders, Senior NCPs and Instructors Guide to Risk Management of Heat Casualties (continued)**
### Hot Weather Injuries and Casualties Chart

<table>
<thead>
<tr>
<th>Cause</th>
<th>Symptoms</th>
<th>First-Aid</th>
<th>Prevention</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exposure to sun direct sun</td>
<td>- Red, hot skin</td>
<td>- Move to shelter, remove clothing if necessary</td>
<td>- Adequate sun protection</td>
</tr>
<tr>
<td>Can occur on overcast days</td>
<td>- May blister</td>
<td>- Apply cold compress or immerse in cool water</td>
<td>- Use sunblock liberally and apply often, especially when engaging in physical activity</td>
</tr>
<tr>
<td>- Moderate to severe pain</td>
<td>- Maintain cool temperature</td>
<td>- Cool affected areas</td>
<td>- Select SPF 30 or higher</td>
</tr>
<tr>
<td>- Can occur in fevers</td>
<td>- Administer analgesics for pain or fever</td>
<td>- Hypothermia and infection with prescribed medications</td>
<td>- Proper wear of clothing, cap</td>
</tr>
<tr>
<td>Sunburn</td>
<td>- Do not treat blisters</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Heat Rash (Prickly Heat)

- Excessive perspiration
- Causes intense itching
- Not a medical condition

<table>
<thead>
<tr>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Rash, itchy skin</td>
</tr>
<tr>
<td>- Blotchy skin due to blocked glands</td>
</tr>
<tr>
<td>- Moderate to severe itching</td>
</tr>
<tr>
<td>- Can occur in fevers</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>First-Aid</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Apply cold compress or immerse in cool water</td>
</tr>
<tr>
<td>- Wash area affected dry</td>
</tr>
<tr>
<td>- Control itching and infection with prescribed medications</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Prevention</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Finger wear of clothing</td>
</tr>
<tr>
<td>- Shroud neck after excessive sweating</td>
</tr>
</tbody>
</table>

### Heat Cramps

- Excessive loss of salt from body due to excessive sweating |
- Not associated to heat weather |

<table>
<thead>
<tr>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Muscle cramps</td>
</tr>
<tr>
<td>- Often muscle cramps</td>
</tr>
<tr>
<td>- Muscle cramps</td>
</tr>
<tr>
<td>- Sudden loss of blood</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>First-Aid</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Replace salt</td>
</tr>
<tr>
<td>- Intravenous saline solution</td>
</tr>
<tr>
<td>- Muscle cramps</td>
</tr>
<tr>
<td>- Intravenous saline solution</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Prevention</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Eat small meals replace salt</td>
</tr>
<tr>
<td>-Consumers must manage their salt intake</td>
</tr>
<tr>
<td>- Avoid prolonged periods of heavy sweating</td>
</tr>
<tr>
<td>- Ensure adequate hydration</td>
</tr>
</tbody>
</table>

### Heat Exhaustion

- Body fatigue and dizziness on heat due to overheating of sweat glands |
- Dehydration (see below) |
- Inadequate air intake |
- Inadequate air intake for air intake |
- Most common heat-related illness |

<table>
<thead>
<tr>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Cerebrovascular accident</td>
</tr>
<tr>
<td>- Headache, nausea</td>
</tr>
<tr>
<td>- Vomiting</td>
</tr>
<tr>
<td>- Dry, parched mouth</td>
</tr>
<tr>
<td>- Weakness</td>
</tr>
<tr>
<td>- Shortness of breath</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>First-Aid</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Initiate active cooling play best chance available</td>
</tr>
<tr>
<td>- Move to shade and loosen clothing</td>
</tr>
<tr>
<td>- Lay flat on a cool surface</td>
</tr>
<tr>
<td>- Stay cool by applying cool water to extremities</td>
</tr>
<tr>
<td>- Continue to drink cool water every 30 minutes</td>
</tr>
<tr>
<td>- If not improved in 30 minutes, evaluate for further medical care</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Prevention</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Allow for acclimatization</td>
</tr>
<tr>
<td>- Monitor vital signs and observe environmental conditions</td>
</tr>
<tr>
<td>- Keep cool in shade whenever possible</td>
</tr>
<tr>
<td>- Follow water replacement guidelines</td>
</tr>
<tr>
<td>- Identify high risk individuals</td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>

### Additional Medical Considerations in the Hot Weather Environment:

#### Dehydration

- Excessive loss of body fluids and possibility of salt loss |

<table>
<thead>
<tr>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Cerebral hemorrhage</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>First-Aid</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Replace lost water and salt</td>
</tr>
<tr>
<td>- Maintain intravenous fluids</td>
</tr>
<tr>
<td>- Cool boiled treatment</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Prevention</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
</tbody>
</table>

#### Over Hydration (Hyponatremia)

- Decreased need for dieting |
- Loss of body fluid |
- Kidney and/or treatment for dehydration |

<table>
<thead>
<tr>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Confusion</td>
</tr>
<tr>
<td>- Weakness</td>
</tr>
<tr>
<td>- Nausea, vomiting</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>First-Aid</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Replace salt loss</td>
</tr>
<tr>
<td>- Provide normal intake for dehydration</td>
</tr>
<tr>
<td>- Continue to monitor for dehydration</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Prevention</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
</tbody>
</table>

---

**Figure 7.8.2.7-3. Commanders, Senior NCPs and Instructors Guide to Risk Management of Heat Casualties (concluded)**

**Note:** See [http://phc.amedd.army.mil](http://phc.amedd.army.mil) for electronic versions of this document and other resources.

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Cleared, 88PA, Case # 2016-5592, 1 Nov 2016.
7.8.3. Cold Stress

7.8.3.1. Introduction.

Environmental challenges threaten performance during an HHQ tasking. Mission: Air defense sortie to provide cover and fire support for ground forces operating in mountainous terrain. Launch was from a forward base near the AOR and only a stone’s throw from the edge of the expanse of the sea. Crew was outfitted with MAC-10 flight suits, thermal gloves, and the requisite layers of flight gear including life preserver units laced to the collar of the survival vest. Loading the mission gear was uninterrupted for nearly 45 minutes; worked up a good sweat and the adrenaline of the mission sustained the heart rate through engine start, taxi, and take-off. Climbing out to the area, the sun was setting and the cabin temperature immediately began to plummet and the muscles and joints seemed to call out for more heat. First thing we noticed was how numb the hands became; there was no sanctuary from this bone chilling cold and handling any equipment that required dexterity was already a challenge. As the aircraft approached the area, the call was made to 'don the night vision goggles,' critical because the aircraft was going to 'go dark' in a few minutes. With only the NVG lighting to guide the icicles formerly called fingers, we each fumbled with the NVGs, the mounts, and toggles but finally secured the devices and switched on the frosty green glow of the tubes. Now as the aircraft motored deeper into the night, and temperature was traded for altitude, a deep chill penetrated the protective gear, every slight gap between layers became a wind tunnel to the cabin air...new problem; hypothermia began to settle in to one of the crewmen and the uncontrolled shaking could be seen across the darkened cabin...

7.8.3.2. Cold Stressors. For the human body to maintain homeostatic balance, it must manage internal systems based upon the input of external stressors. External stressors considered to be threats to the human body consist of thermal stressors (i.e., heat, cold), odor, food, water, hypoxia, noise, light, darkness, trauma or injury, electricity, physical threats, and bacteria or viruses. With exposure to thermal stress, the human body must maintain its core body temperature ($T_C$) through the management of heat loss and heat gain. The body will either dissipate or gain heat through convection, evaporation, conduction, or radiation and generate heat through metabolism, shivering, or physical activity as shown in Figures 7.8.1-2 and 7.8.1-2-2. When considering the thermal stress of cold exposure, there are four significant environmental variables that must be considered: low air temperature, high air movement, high air saturation, and cold water immersion (Figure 7.8.1-2-2).

Air temperature is the most obvious cold stressor; it begins to affect homeostasis of heat body balance at a temperature below $T_C$ and skin temperature ($T_{SK}$). If the balance of heat loss is greater than heat production, then cold stress injuries can result, e.g., frostbite occurring when the $T_{SK}$ reaches a range of -2°C to 0°C (28°F to 32°F) and hypothermic death with a rectal temperature of 22°C (72°F). These hypothermic conditions are a real and viable threat given the fact that some of the coldest places on earth are inhabited by humans; the coldest ambient air temperature of -87°C was recorded in 1958 at the manned Soviet Antarctic station Vostok. From an operational perspective, ascent into higher altitudes introduces reduction in air temperatures. The
lapse rate, i.e., air temperature decrease, is approximately \(-2^\circ\text{C} (3.5^\circ\text{F})\) per 1,000 ft increase in altitude. Air movement, i.e., wind chill, increases heat loss through convection and evaporation. The most profound effect of wind chill is through the release of the still-warmed air layer trapped underneath insulated protective cold-weather clothing. Cumulatively, wind chill and evaporation on wet clothing and wet-soaked skin surface will increase heat loss when compared to air temperature alone. The National Weather Institute’s Wind Chill Chart stratifies the risk of frostbite over time of exposure relative to air temperature plus wind chill as shown in Figure 7.8.3.2-1.

Wind Chill Chart

![Wind Chill Chart](image)

Figure 7.8.3.2-1. National Weather Service Wind Chill Chart (11 Nov 2001)

Generally, as the wind chill index becomes increasingly more severe, the risk of frostbite increases. Air saturation (i.e., humidity, which is commonly referred to as air dryness in cold weather) is the other major cold stressor the human body must endure. With regard to saturation, cold is most often considered “dry” air because of its low water content. Cold, dry air is primarily the culprit for exercise-induced bronchial spasms (EIB), as it negatively affects the smooth muscle in the bronchial airways and lungs.

Of the four main environmental cold stressors, cold water immersion has the greatest magnitude of heat loss. When submerged and at rest, the body will dissipate heat approximately two to four times faster than when in air of the same temperature. Once submerged, heat loss from the body is no longer possible through evaporation, except for those body parts not submerged (i.e., the head), which can be responsible for significant heat loss. However, primary heat exchange in water immersion happens through conduction and convection with the heat-conductivity ratio of water-to-air being 24:1 with calm, stable currents creating a boundary layer around the body. By simply changing the variable of water movement and keeping temperature constant, the body
heat loss, primarily through conductance, can be up to 70 times greater than equal air temperature. The factors that affect one’s survivability in addition to water temperature and movement consist of the duration of immersion, body mass, subcutaneous fat, activity level while submerged, position in the water (e.g., floating on top or submerged from the neckline down), and clothing insulation.

7.8.3.3. Physiological Response to Cold Exposure. The human body is designed to maintain a state of thermoneutrality wherein the body attempts to regulate $T_C$ in response to external stressors, for example, cold stress. During periods of extreme cold exposures, the body utilizes two strategies to regulate the normal homeostatic balance of $T_C$: behavioral and physiological temperature regulation. Psychologically, humans will change their behavior in an attempt to conserve heat by seeking shelter, wearing cold-exposure clothing, and increasing physical activity. Physiologically, heat conservation occurs via the primary cold-effector response of vasoconstriction and thermogenesis.

The full spectrum of behavioral and physiological declination as hypothermic severity increases is shown in Figure 7.8.3.3-1. The initial physiological response to cold is a decrease in $T_{SK}$, which leads to vasoconstriction of subcutaneous blood flow from the skin surface to deeper tissue to maintain $T_C$. The vasoconstrictor response begins when $T_{SK}$ is reduced below 35°C (95°F) and ends at approximately 31°C (88°F). The subcutaneous vasoconstriction reduces heat loss immediately by increasing skin insulation through the β-adrenergic action of blood vessel diameter reduction and the increased plasma norepinephrine reduction in peripheral blood flow. The shunting of blood to deeper tissue reduces heat transference from blood to skin by minimizing the blood’s exposure to the cold environment and thus loss of heat via conductive heat transfer. Blood flow distribution is controlled via local vasoconstrictor controls in two major regions of the body: apical and nonapical. The apical region (i.e., ears, nose, lips, hands, and feet) is primarily controlled by the adrenergic sympathetic nervous system, while the nonapical region (i.e., trunk and limbs) is regulated by the noradrenergic-active vasoconstrictor and the active vasodilator systems. The result of vasoconstrictor response to cold exposure is the stimulation of body-fluid regulation, water balance, and thoracic blood volume. With exposure to cold air, the acute effect is a 7% - 15% reduction in plasma volume and an increase in plasma osmolarity and sodium concentration. In comparison, cold water immersion reduces plasma volume by 15% - 20%. Cold-induced diuresis can increase hemoconcentration by a twofold increase in the loss of fluid; hypotheses are still unclear.
Figure 7.3.3-1. Declination of Physiological Status with Decreasing Core Body Temperature ($T_c$) (adapted from Table 11.1 in Pozos and Danzl, 2001)

- 37.6°C: Normal rectal temperature
- 37°C: Normal oral temperature
- 36°C: ↑ in metabolic rate, blood pressure, pre-shivering muscle tone
- 35°C: Maximum shivering thermogenesis
- 34°C: Amnesia, poor judgment develops, maladaptive behavior
- 33°C: Ataxia, apathy, ↓ cerebral metabolism, tachypnea, cold diuresis
- 32°C: Stupor, ↓ 25% VO$_2$
- 31°C: Shivering thermogenesis ceases
- 30°C: Atrial fibrillation, arrhythmias, CO 67% of normal, insulin ineffective
- 28°C: Hypoventilation, ↓ ventricular fibrillation threshold, ↓ 50% VO$_2$
- 27°C: Loss of reflexes and voluntary motion
- 26°C: No pain response, major acid-base disturbances
- 25°C: Cerebral blood flow 33% of normal, CO 45% of normal
- 24°C: Significant hypotension and bradycardia
- 23°C: Areflexia
- 22°C: Maximal risk for ventricular fibrillation, ↓ 75% VO$_2$
- 20°C: Pulse 20% of normal
- 19°C: Electroencephalographic silencing
- 18°C: Asystole
- 16°C: Lowest adult survival from accidental hypothermia
- 15.1°C: Lowest infant survival from accidental hypothermia
- 10°C: ↓ 92% VO$_2$
- 9°C: Lowest survival from therapeutic hypothermia

Hypothermic Severity

Mild
Moderate
Severe

T°C
As cold stress exposure continues, maintenance of thermoneutrality is not feasible and the rate of heat loss becomes greater than the heat generated through normal metabolism; the liberated metabolic heat can be as great as 80% of the total energy expended. Thus, thermogenesis is employed via two methods: the act of shivering and energy metabolism. Shivering thermogenesis is modulated via peripheral and central temperature sensors; if the $T_{SK}$ declines and is unable to maintain $T_C$, then central temperature sensors are stimulated to induce a magnitude of shivering to balance the heat loss. The intensity of shivering is dependent upon $T_C$ and $T_{SK}$.

For instance, if $T_C$ is elevated prior to the reduction in $T_{SK}$, then the shivering thermogenesis response will be less than expected. Conversely, a lower beginning $T_C$ will enhance the thermogenic response and thus a smaller change in $T_{SK}$ will elicit the same response. However, the significant influence $T_{SK}$ has in shivering thermogenesis is affected in the rate of $T_{SK}$ change. Shivering is considered to be an involuntary muscle contraction and can generate force equal to 15% - 20% of maximal voluntary muscle contraction. This type of intense shivering can increase metabolic rate five to six times greater than resting metabolic rate.

Macronutrients and metabolic constituents (i.e., lipids, glucose, glycogen, and ketones), modulated through their respective metabolic processes (i.e., lipolysis, glycolysis, gluconeogenesis, glycogenolysis, and hepatic ketone metabolism), are the primary fuel sources for the human body during cold stress exposure. Generally, while at rest in a cold stress exposure, the metabolic requirements for an individual are not increased. However, metabolic demands may increase if any combinations of the following variables are experienced, for example, extreme cold (i.e., air temperature, wind chill, water immersion), intense shivering, heavy clothing, and increased work or exercise. Although the idea of limited shivering thermogenesis due to depleted carbohydrate fuel sources seems a valid notion, research has shown dietary manipulation via carbohydrate loading does not improve thermal tolerance and thus does not limit shivering thermogenesis. This is because a shift occurs from the primary metabolic fuel source of carbohydrates to that of lipid oxidation. The shift between glycolytic metabolism and lipid oxidation can be seen within 4 hours of cold exposure without significant changes to metabolic heat production and shivering thermogenesis. Additionally, during normal cold exposure there is an increase in metabolic fuel utilization of plasma glucose, muscle glycogen, and lipid oxidation, one and a half fold, twofold and threefold, respectively, with no change in protein usage. The percent distribution of total heat production based upon glucose, muscle glycogen, fat, and protein utilization is 10%, 30%, 50%, and 10%, respectively. Therefore, the fundamental aspects of caloric consumption and content regarding nutritional requirements for cold exposure are generally within normal dietary recommendations. However, caloric requirements are mission dependent upon additional physical activities and/or long-term, prolonged exposures to extreme cold.

The physiological response to cold water immersion is similar to cold air exposure, but with additional responses. Cold water immersion is categorized into three stages: Stage 1, Initial Immersion (0 – 3 minutes); Stage 2, Short-Term Immersion (3 – 15 minutes); Stage 3, Long-Term Immersion (≥ 30 minutes). Often a Stage 4, Post-Immersion, is included to address recovery. Stages 1 and 2 have the aforementioned physiological response to air plus respiratory and cardiovascular issues. Respiration may become uncontrolled, with reflex gagging and hyperventilation due to the panic response, which might result in death through aspiration or drowning. The additional cardiovascular issue relates to the complete submersion (i.e., face and whole body) and
the parasympathetic-driven, bradycardic reflex of the “diving response,” also known as diving reflex; all other sympathetic responses are present. If Stage 4 is not reached, then Stages 2 and 3 of cold water immersion progress through the vasoconstriction response and shivering thermogenesis with the result of death.

7.8.3.4. **Psychological Response to Cold Exposure.** The physiological response to cold stress exposure is not the only variable military personnel need to be concerned about when performing operational duties: the psychological aspect of human performance is affected as well. Mission-critical duties might fail to be completed due to the decrease in morale and/or cognitive performance.

Water is known to have 1,000 times greater thermal conductivity and 25 times greater heat loss when compared to cold air at the same relative temperatures. Cold water immersion has the greatest psychological performance measure decrement when compared to cold air with duration and severity being compounding variables.

The psychological response to cold exposure will often manifest before physiological changes in $T_C$ occur, dependent upon the severity of the cold stressors. For prolonged exposure, i.e., $T_C$ cannot be maintained at 37°C (98.6°F), the effect on psychological behavior has been compared to a level of anesthetic impairment, wherein consciousness and alertness decline; reflexes and responses slow; and speech is slurred, accompanied by drowsiness and apathy. As $T_C$ drops below 35°C (95°F) towards 34°C (93°F), memory registration and concentration become impaired to the point of 20% loss of information from memory registration and complex tasks processing (e.g., mathematical calculations) decreases by approximately 17% - 20%; accuracy does not seem to be affected. Auditory and visual hallucinations are not uncommon, and at a $T_C$ of 30°C - 31°C (86°F - 88°F), the pupil's reflex to light may be so slow that it is misdiagnosed as absent. Deep tendon reflexes, muscle rigidity, and simple voluntary movements are difficult to control or even initiate. Most often, unconsciousness is reported between 30°C and 32°C (86°F and 90°F); however, a grunt-response to questions may occur, and failure to respond to pain-stimulus progresses to a coma state as $T_C$ falls below 26°C (79°F). The mechanisms hypothesized for these extreme-prolonged responses to cold stress exposure consist of a slowing of synaptic gap transmission, brain cooling, changes in cerebral blood flow and electrical brain activity, as well as muscular and neuronal tissue cooling.

The mild to moderate cold stress exposure mechanism has a cumulative effect on cognitive performance through subtle effects on the central and peripheral nervous systems, which result in a decrease in human performance. A review of performance measures consists of a decrease in manual skills (i.e., tactile sensitivity, dexterity, strength, and motor speed), equivocal results in vigilance, minor changes in simple reaction times, significant increase in complex-tasks choice reaction time, significant impairment of memory recall, and complex cognitive functioning decrement due to task complexity. Ultimately, an individual’s behavior and decision-making ability are based upon the subjective perception and evaluation of his/her surroundings more so than the objective evaluation of exposure time, physiological status, prior acclimatization, or core body temperature.

7.8.3.5. **Self-Imposed Factors Effect on the Physiological Response to Cold Exposure.** There are a few major individual- and mission-imposed factors that affect a person’s ability to respond to cold stress exposure. Individual-imposed factors pertain to a person’s physiological status and abilities that are not
under the immediate-direct control of the individual. Conversely, mission-imposed factors can be considered as variables outside the body that an individual does have immediate-direct control over and can change with minimal effort to aid in the response to cold exposure. The individual factors consist of anthropometric measures (i.e., body size, subcutaneous fat), gender, ethnicity, physical fitness, and age, while the mission factors pertain to physical fatigue, dehydration, nutrition, alcohol, and nicotine.

Of the individual-imposed factors, anthropometric measurement (i.e., body size, subcutaneous fat) is the factor with the greatest variability to an individual’s cold exposure response. Individuals with a long and lean body size (i.e., ectomorph) have a high body surface area to body mass ratio, thus losing more heat than their short and stocky counterparts (i.e., mesomorph). The following is true because the ectomorph has an increased convective heat loss due to greater transference via skin surface. The insulative property of subcutaneous fat adds an additional layer of protection that is inverse heat loss; thus, the greater the layer of subcutaneous fat the less heat loss from the body and a reduction in shivering thermogenesis. However, this should not promote total body weight gain from total fat tissue. The similar thermal resistance is exhibited in a person with increased lean muscle mass due to the additive vasoconstriction of blood flow to the skin and muscle, creating an improved insulative layer. Complementary to the improved insulative layer is the improved shivering thermogenesis due to a greater muscle mass when compared to the ectomorph. Gender, ethnicity, and physical fitness all relate to the anthropometric measurements and the resultant effect on an individual’s cold exposure response. Gender differences are attributed to the female’s greater body fat percentage and thicker subcutaneous fat layer when compared to a male the same age and weight. Ethnicity uses the same logic as gender differences. The relationship of physical fitness is based upon body composition differences and sustainable steady-state activity between fit and less fit individuals. Age, greater than 45 yr, affects cold exposure response due to the declination in physical fitness and reduced vasoconstrictor response.

Mission-imposed factors affect an individual’s response through his/her own specific methods and are commonly associated with military operations in extreme environments. Physical fatigue reduces shivering thermogenesis and vasoconstrictor response, resulting in a reduction in sustainable steady-state activity, thus reducing the ability to maintain $T_C$. Dehydration increases the susceptibility to cold stress injuries by reducing the sustainable steady-state activity as well. Poor nutrition can lead to a diminished glucose and glycogen availability, which can reduce the central nervous system modulated aspect of shivering thermogenesis and the peripheral heat production. The risk of hypothermia due to the reduction in shivering thermogenesis increases when a person is without food for more than 48 hours. The primary concern with consumption of alcohol focuses on peripheral vasodilation and the concomitant heat loss. In addition, alcohol can cause hypoglycemia, reducing the shivering thermogenesis response and the ability to sustain physical activity, as well as a false perception of warmth. Conversely, the vasoconstrictor response to nicotine consumption is of primary concern in cold stress exposure. Nicotine from “heavy” smoking (e.g., two-three packs per day) or chewing tobacco increases a person’s susceptibility to freeze-related injuries because of the vasoconstriction of peripheral blood flow.
7.8.3.6. **Acclimatization to Cold Exposure.** The resultant outcome of exposure to thermal stress is acclimatization; however, acclimatization to cold stress is less apparent when compared to heat stress acclimatization. The modest human pattern of acclimatization to cold stress (Figure 7.8.3.6-1) that an individual will undergo is dependent upon the nature and severity of the exposure (i.e., intensity, frequency, duration, and mode). There are three physiological adaptive mechanisms an individual might undergo in response to cold stressor exposure: habituation, insulative, and metabolic acclimatization. Habituation, the first and most common type of cold exposure acclimatization, is exemplified by the diminished physiological response to cold exposure of individuals with exposure versus those without exposure. Individuals who are habituated to cold exposure often show signs of reduced vasoconstrictor and shivering thermogenesis responses; they may also exhibit a higher peripheral $T_{SK}$, improved cold-induced vasodilation (CIVD) response, decreased sense of pain, and improved manual dexterity skills. Cold habituation can further be divided into two subcategories: short-term and long-term exposures. Short-term exposure is considered less than 1 hour per exposure a few times per week, producing habituation. Long-term exposure is the physiological response to 8 hours or more of moderate-cold exposure on successive days for a period of 2 weeks or more. The resultant effect of long-term exposure is known as hypothermic habituation, and those with long-term cold habituation may display a more pronounced reduction in $T_C$ when compared to noncold-habituated individuals.

![Figure 7.8.3.6-1. Human Pattern of Cold Acclimatization](image-url)
The second possible mechanism of physiological adaptation concerns heat conservation through insulative acclimatization. After several weeks of repeated cold exposures, an improved vasoconstrictor response due to a more rapid sympathetic nervous system response drops the $T_{SK}$ faster, thus decreasing skin thermal conductance. Convective heat loss through an improved countercurrent heat exchange is postulated to be a factor for insulative acclimatization. Individuals who have an acclimatized insulative physiological mechanism to cold stressors may also exhibit a lower resting $T_C$. The third physiological mechanism to cold stress exposure has not been unequivocally proven regarding metabolic acclimatization. Exaggerated shivering may be seen in individuals exposed to chronic cold, thus increasing heat production via shivering thermogenesis. However, the theory of increased heat production through metabolism, nonshivering thermogenesis, has not been unequivocally proven. The operational benefit of cold stress acclimatization, i.e., habituation, insulative, and metabolic mechanisms, consists of improving the ability to perform motor skills, increasing thermal comfort, increasing CIVD responses, and reducing susceptibility to cold injury in an overall effort to improve human performance. With regard to the acclimatization factors for psychological aspects, preliminary research reports that cold-climate training improves performance of manual skills, but future research should focus on those tasks vulnerable to cold stressor exposure distractions.

7.8.3.7. Medical Considerations. There are seven cold stress injuries that commonly occur: hypothermia, chilblain, pernio, trench foot, immersion foot, frostnip, and frostbite. The diagnosis and treatment of these injuries are important but are beyond the scope of this manual; professional licensure should perform such functions. However, the recognition and prevention of these conditions are of relevance and will be discussed.

Hypothermia, the most widely known cold exposure injury, is defined by a -2°C (3.5°F) drop in $T_C$ from the normal 37°C to 35°C (98.6°F to 95°F), the most commonly agreed upon definition. The onset of hypothermia will differ by case due to the variability in the conditions of the operational and ambient environments and the variability in the person's individual factors (e.g., age, fitness, and percent body fat). The best way to avoid getting hypothermia is to understand the common factors leading up to the onset as well as the signs and symptoms. There are two common factors that contribute to the onset of hypothermia: wet clothes and extended periods of exposure to the cold or any combination thereof. If one is unable to minimize the risk of hypothermia, then recognition of the signs and symptoms aid in the prevention and early detection. The signs and symptoms include shivering, grayish skin color, vague/slow/slurred speech, argumentativeness, irritability, immobile fingers, impaired motor skills, difficulty walking, drowsiness, and fatigue.

Of lesser severity are the cold exposure, nonfrozen injuries of chilblain and pernio. Both have similar causes of onset and pathology and present mostly in the hand, toes, legs, and ears of children, women, and women of middle age. Chilblain is seen between temperatures of 0.5°C - 16°C (33°F - 61°F) and is the result of cold-induced vasoconstriction that causes cell ischemia and limb edema. White blood cells are transported into deep tissue, causing swelling of the tissue and blood vessels, which leads to the manifestation of red skin or plaques that may blister or ulcer. Pernio is the next progression of chilblain; patches of dead skin are peeled off, while superficial burning and pain replace the milder symptoms of chilblain. An indirect protective mechanism against cold-induced vasoconstriction blood flow reduction is the CNS-
modulated, vasomotor cold-induced vasodilation. In response to the decrease in $T_{SK}$, CIVD will allow periodic increase of blood flow to the periphery, resulting in an oscillator $T_{SK}$ response over time. Preexisting medical conditions such as diabetes and Raynaud's syndrome or mission-imposed conditions with vibrations blunt the CIVD response, which might increase the risk of these cold injuries.

Trench foot and immersion foot are slightly more severe, nonfrozen injuries. Trench foot is considered to be the next progression of untreated, undiagnosed pernio. Trench and immersion foot distinction can be difficult because of the similarities in the onset and pathology. Both develop slowly over a period of hours to days due to an overexposure of wet feet to temperatures between 0.5°C and 10°C (33°F and 50°F) for more than 12 hours. The cause of the injuries is vasoneuropathy, which is damage to the nerve and blood vessels without the freezing of water in the tissue. Immersion foot starts off with the area being cold, swollen, or numb for a few days, followed by a 2- to 6-week period where blood vessels are destroyed, blistering or ulcerations appear, and/or significant temperature changes occur – all without a pulse. Finally, within a few months the affected area may present with no signs or symptoms; Raynaud’s disease may develop or there may be signs of itching, pain, swelling, or lack of sensation. Trench foot occurs on land and from exposure to wet footgear, malnutrition, and indifference. Ultimately, the loss of the limb or leg may be imminent.

Frostbite, considered to be a freezing injury, has three classifications: frostnip, superficial frostbite, and severe frostbite. Frostnip, lesser in severity, is considered to be a first-degree skin burn usually imposed by super-cooled liquid or metal and wind chill exposure. There may be some redness and peeling of skin, similar to a sunburn. Superficial frostbite is more severe and involves the burning of the full skin layer and tissue underneath. The area will appear white-gray with the superficial skin layer being tough and the tissue just beneath being soft-spongy with blisters appearing within 24 hours. Severe frostbite consists of tissue death due to freezing and crystallization of water within the tissue. Slow cooling of the tissue will produce the lesser of the damage because rapid cooling will form intracellular crystals, rupturing the cells, while slow cooling forms interstitial crystals without cellular rupture. Most of the damage is vascular in nature from the formation of blood clots, fibrin deposits in the arteriole walls, cell hypoxia, and vascular wall degradation. The affected area is often described as seemingly like a third-degree skin burn. The causes of severe frostbite are the same as for frostnip and superficial frostbite: temperature, wind chill, exposed skin, moisture in clothing against skin, poor insulation, direct skin contact with super-cool liquid or metal, cramped position, tight clothing or boots, dehydration, and localized pressure. The three compounding factors leading to these freezing injuries are cigarette smoking, mental and physical fatigue, and lengthy periods of time without body movement. The onset of hypothermia and freezing and nonfreezing cold injuries is of significant concern when considering cold stress exposure in the operational environment. The best method of avoiding these injuries is not the diagnosis, or prognosis or treatment, but rather the recognition and even more so prevention.

7.8.3.8. Prevention. Prevention of cold stress exposure injuries can be best confronted with preparation through training, nutrition and hydration, proper clothing, and proper management of time of exposure. Of course, knowledge of the signs and symptoms is necessary in the early recognition of potentially hazardous situations and environmental conditions that result in minor to severe injury or even death.
In cold environment training, physical training is one of the most important factors promoting prevention of injury or death. People who are physically fit have greater abilities to control their thermoregulatory system, greater metabolic heat production, and $T_C$ maintenance insulation via constriction. Physical training has also been shown to offset the deleterious effects of wind chill in two ways. The first is through an increase muscle mass activation, which can provide greater heat production during shivering thermogenesis when compared to untrained people. Secondly, metabolic heat production can reach higher levels for longer periods of time due to a physically fit person’s ability to perform greater workloads that are sustained for longer periods because of the person’s greater work capacity (i.e., VO$_2$ max). Individuals who are physically fit have greater capability of keeping their extremities from cooling too quickly.

Training in cold weather environments better acquaints personnel with the interaction between themselves; their physical knowledge, skills, and abilities (i.e., KSAs); the equipment; and situational awareness is important. Practice maneuvers relative to the operational environment will improve those training KSAs.

The potential nutritional intake in an operational environment that accounts for maximal physical activity in addition to exposure to cold stress may be as high as 4,500 kcal per day. Hot meal requirements have been suggested in 10-day rotations: the first 10 days, one hot meal; the second 10 days, one hot-wet meal per day; the third 10 days, two hot-wet meals per day. The other meals are provided through Meal, Ready-to-Eat (MRE) rations. Along with MRE meals, additional water is required; thus, at least 4 qt of water should be consumed each day per person. Recall, water is the most important survival requirement in an environment with exposure to cold stressors; therefore, extra care must be taken to make certain sufficient water intake is achieved when factoring level of physical activity and extra diuresis due to nutritional, supplemental, or medication ingestion.

The time must be taken prior to any cold stress exposure to be properly fitted with appropriate cold-weather clothing. Airmen should be outfitted with multilayer clothing, and each item must be fitted individually to the airman’s anthropometric measurements in an effort to optimize comfort and fit. With well-fitted, multilayer clothing, airmen are able to contain air close to the body, warm the air with their own natural body heat, and then keep the warm air close to the body while simultaneously keeping perspiration away from the skin. The overall idea is to be able to take off and put on multiple layers of clothes dependent upon the environment without letting the warm, trapped air out or the cold, moist air in. The layering of clothing is a simple concept; consider the three basic "Ws"; wicking, warming, and weathering. The wicking layer keeps moisture away from the skin and should be some type of polysynthetic material; cotton has a tendency to hold moisture and will keep it close to the skin, reducing thermal aspects of warming the layer of air as well as putting the airman at risk of freezing the cotton layer to the skin if exposed to the appropriate environment. The warming layer is just that, a looser layer of clothing, atop the whisking layer, that is meant to optimize the warming of air closest to the body. One of the best warming layers is a tightly knitted wool; just be careful not to get wool wet or it will trap cooler moisture closer to the body and reduce its thermal effect. Finally, the weathering layer’s purpose is to aid in protecting you from the harsh cold environment while keeping another layer of warm air on top of the warming layer of air. The weathering layer is mainly for protection of the warming layers. Boots must fit properly, not too snug, so try them on with all variations of socks provided. If the toes are too tight, then get a larger size boot. Remove and dry socks and boots two to three times
per day to prevent trench foot; do not sleep with footwear on. The variables one must consider when selecting clothing requirements should focus on higher insulation values for more extreme environments. Additionally, military personnel must take into account the type of material they might encounter (i.e., air, wood, stone, steel, aluminum) and its respective thermal conductance in relation to skin-to-material time of exposure. These factors, individual- and mission-imposed stresses, all play a significant role in the overall outcome/result of a given scenario, whether training or in the real world.

Ultimately, unit leadership is responsible for training and its proper implementation. The U.S. Army Center of Health Promotion and Preventive Medicine has distributed a cold risk manual that outlines a five-step risk management tool with the intent of preventing cold causalities (Figure 7.8.3.8-1). The first step is to identify the hazards, such as weather conditions, inventory of required supplies, and self-imposed factors. The second step of hazard assessment focuses on the proper use of wind chill index charts relating to environmental conditions, work intensity, food and clothing, and injuries stratified into risk categories. The development of control points establishes limits for the third step. The implementation, supervision, and evaluation of the risk management plan are the final steps of the process, steps four and five. The cold exposure risk management tool is only effective if it remains as a dynamic loop that continues to take into account the ever-changing variables of the ambient environment, military personnel, and operational mission. Situational awareness is paramount when military personnel perform active and nonactive duties in cold environments, and if the proper preventive measures are implemented, cold exposure injuries such as hypothermia and frozen and nonfrozen injuries are preventable. Prevention begins in pretraining preparation; use of wind chill charts to manage personnel time of exposure to the cold environment; risk assessment by medical personnel on active duty personnel; an established buddy system to advise one another in early detection of hazardous factors; and, most importantly, with leadership, leading by example.
Unit Leaders' and Instructors' Risk Management Steps for Preventing Cold Casualties
Risk Management is the Process of Identifying and Controlling Hazards to Protect the Force

Possible Outcomes of Inadequate Climatic Cold
- Chilblain (due to bare skin exposed to cold humid air)
- Immersion Foot (Trench Foot) (due to wet feet)
- Frostbite
- Hypothermia (whole body temperature dangerously low)
- Dehydration
- Snow Blindness
- Carbon Monoxide Poisoning

The Five Steps of Risk Management Are:

1. Identify Hazards
   - Cold (temperature 40°F and below)
   - Wet (rain, snow, ice, humidity) or wet clothes
   - Wind (wind speed 5 mph and higher)
   - Lack of adequate shelter/clothing
   - Lack of provisions/water
   - Other Risk Factors include:
     - Previous cold injuries or other significant injuries
     - Use of tobacco/nicotine or alcohol
     - Skipping meals/poor nutrition
     - Low activity
     - Fatigue/sleep deprivation
     - Little experience/training in cold weather
     - Cold casualties in the previous 2-3 days
     - Overly Motivated Soldiers

2. Assess Hazards
   Follow the Wind Chill Temperature Table to Determine the Danger Level
   Do individuals have adequate shelter/clothing?
   - Are clothes clean without stains, holes or blemishes (which could decrease heat-retaining function)?
   Have meals been consumed?
   - Are meals warm?
   Are there other circumstances?
   - Is there contact with bare metal or fuel/POL (petroleum, oils or lubricants)?
   - Is the environment wet? Is there contact with wet materials or wet ground?
   - Can soldier move around to keep warm?
   - Are feet dry and warm?
   - Is the soldier with a buddy who can assist/watch over to prevent cold injuries?

Figure 7.8.3.8-1. U.S. Army Center for Health Promotion and Preventive Medicine Five-Step Cold Risk Management Tool
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Assess Hazards continued

Using the Wind Chill Temperature Table

The wind chill index (see table below) gives the equivalent temperature of the cooling power of wind on exposed flesh.
- Any movement of air has the same effect as wind (running, riding in open vehicles, or helicopter downwash).
- Any dry clothing (mittens, scarves, masks) or material which reduces wind exposure will help protect the covered skin.

Trench foot injuries can occur at any point on the wind chill chart and -
- Are much more likely to occur than frostbite at “LITTLE DANGER” wind chill temperatures, especially on extended exercises/missions and/or in wet environments.
- Can lead to permanent disability, just like frostbite.

### Wind Chill Temperature Table

<table>
<thead>
<tr>
<th>Wind Speed (mph)</th>
<th>Air Temperature (°F)</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>-5</td>
</tr>
<tr>
<td>15</td>
<td>-10</td>
</tr>
<tr>
<td>20</td>
<td>-15</td>
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<td>30</td>
<td>-25</td>
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<tr>
<td>35</td>
<td>-30</td>
</tr>
<tr>
<td>40</td>
<td>-35</td>
</tr>
<tr>
<td>45</td>
<td>-40</td>
</tr>
</tbody>
</table>

**GREEN**
- LITTLE DANGER (frostbite occurs in >2 hours in dry, exposed skin)

**YELLOW**
- INCREASED DANGER (frostbite could occur in 45 minutes or less in dry exposed skin)
- Provide warming facilities

**RED**
- GREAT DANGER (frostbite could occur in 5 minutes or less in dry, exposed skin)
- Provide warming facilities

### Wind Chill Category (see Wind Chill Temperature Table above)

<table>
<thead>
<tr>
<th>Work Intensity</th>
<th>Little Danger</th>
<th>Increased Danger</th>
<th>Great Danger</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>High</strong> Digging foxhole, running, marching with rucksack, making or breaking bivouac</td>
<td>Increased surveillance by small unit leaders; Black gloves optional - mandatory below 0°F (-18°C);</td>
<td>ECWCS* or equivalent; Mittens with liners; No facial camouflage; Exposed skin covered and kept dry; Rest in warm, sheltered area; Vapor barrier boots below 0°F (-18°C); Provide warming facilities</td>
<td>Postpone non-essential training; Essential tasks only with &lt;15 minute exposure; Work groups of no less than 2; Cover all exposed skin, Provide warming facilities</td>
</tr>
<tr>
<td><strong>Low</strong> Walking, marching without rucksack, drill and ceremony</td>
<td>Increased surveillance; Cover exposed flesh when possible; Mittens with liner and no facial camouflage below 10°F (-12°C); Full head cover below 0°F (-18°C). Keep skin dry - especially around nose and mouth</td>
<td>Restrict non-essential training; 30-40 minute work cycles with frequent supervisory surveillance for essential tasks. See above.</td>
<td>Cancel Outdoor Training</td>
</tr>
<tr>
<td><strong>Sedentary</strong> Sentry duty, eating, resting, sleeping, clerical work</td>
<td>See above; Full head cover and no facial camouflage below 10°F (-12°C); Cold-weather boots (VB) below 0°F (-18°C); Shorten duty cycles; Provide warming facilities</td>
<td>Postpone non-essential training; 15-20 minute work cycles for essential tasks; Work groups of no less than 2 personnel; No exposed skin</td>
<td>Cancel Outdoor Training</td>
</tr>
</tbody>
</table>

*ECWCS – Extended Cold Weather Clothing System

Note: These guidelines are generalized for worldwide use. Commanders of units with extensive extreme cold-weather training and specialized equipment may opt to use less conservative guidelines.

Figure 7.8.3.8-1. U.S. Army Center for Health Promotion and Preventive Medicine Five-Step Cold Risk Management Tool (continued)
Develop Controls

Main Points to Stress to Soldiers
When using Cold-Weather Clothing, Remember...

C-O-L-D
- Keep it........... Clean
- Avoid........... Overheating
- Wear it........ Loose in layers
- Keep it......... Dry

Main Points to Stress to Leaders

Follow these Wind Chill Preventive Medicine Measures
Based on Wind Chill Temperature

30°F and below
Alert personnel to the potential for cold injuries

25°F and below
Leaders inspect personnel for wear of cold weather clothing.
Provide warm-up tents/areas/hot beverages.

0°F and below
Leaders inspect personnel for cold injuries. Increase the frequency of guard rotations to warming areas. Discourage smoking.

-10°F and below
Postpone non-essential outdoor training. For mission essential operations, initiate the buddy system - Have personnel check each other for cold injuries.

-20°F and below
Consider modifying or curtailing all but mission-essential field operations.

NOTE: Trench Foot can occur at any temperature - Always Keep Feet Warm

General Guidance for all Cold-Weather Training

Skin: Exposed skin is more likely to develop frostbite, therefore cover skin. Avoid wet skin (common around the nose and mouth). Inspect hands, feet, face and ears frequently for signs of frostbite.

Clothing: Soldiers must change into dry clothing at least daily and whenever clothing becomes wet. Soldiers must wash and dry feet and put on dry socks at least twice daily.

Nutrition: 4500 calories / day / soldier. Equivalent to 3 meal packets in meal-cold weather (MCW) or 3-4 MREs.

Hydration: 3-6 liters (canteens) / day / soldier. Warm, sweet drinks are useful for re-warming.

Camouflage: Obscures detection of cold injuries; consider not using below wind chill of 32°F; not recommended below wind chill of 10°F.

Responsibilities: Soldiers are responsible for preventing individual cold injuries. Unit NCOs are responsible for the health and safety of their troops. Cold injury prevention is a command responsibility.

Figure 7.8.3.8-1. U.S. Army Center for Health Promotion and Preventive Medicine Five-Step Cold Risk Management Tool (continued)

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Develop Controls continued

Personal Protection

Ensure Appropriate Clothes and Proper Wearing of Clothes –
- Wear clothing loose and in layers.
- Ensure all clothing is clean.
- Ensure proper boots are worn and are dry.
- Ensure clothes do not have holes, broken zippers, etc.
- Ensure hands, fingers, and head are covered and protected.
- Avoid spilling liquids on skin or clothes. Liquid stains will reduce clothing’s protective efforts.
- Change wet, damp clothes ASAP.

Keep Body Warm
- Keep moving.
- Exercise big muscles (arms, shoulders, trunk, and legs) to keep warm.
- Avoid alcohol use (alcohol impairs the body’s ability to shiver).
- Avoid standing on cold, wet ground.
- Avoid all tobacco products (they decrease blood flow to skin).
- Eat all meals to maintain energy.
- Drink water or warm non-alcoholic fluids to prevent dehydration.

Protect Feet
- Keep socks clean and dry.
- Wash feet daily, if possible.
- Carry extra pairs of socks.
- Change wet or damp socks ASAP; use foot powder on feet and boots.
- Avoid tight socks and boots; do not over-tighten boot or shoes.
- Wear overshoes to keep boots dry.

Protect Hands
- Wear gloves, mittens, or gloves/mittens with inserts.
- Warm hands under clothes if they become numb.
- Avoid skin contact with snow, fuel or bare metal. Wear proper gloves when handling fuel or bare metal.
- Waterproof gloves by treating with waterproofing compounds.

Physical Fitness Uniform
- Wind Chill >60 deg F: T-shirt and trunks
- Wind Chill 51-60 deg F: Add jacket
- Wind Chill <50 deg F: Add pants, cap, gloves

Figure 7.8.3.8-1. U.S. Army Center for Health Promotion and Preventive Medicine Five-Step Cold Risk Management Tool (continued)
### Develop Controls continued

#### Personal Protection continued

**Protect Face and Ears**
- Cover face and ears with scarf. Wear insulated cap with flaps over ears or balaclava.
- Warm face and ears by covering them with your hands. Do NOT rub face or ears.
- Consider not using face camouflage when wind chill is 32°F or below. Also not recommended below 10°F.
- Wear sunscreen.
- Exercise facial muscles.

**Protect Your Eyes**
- Wear sunglasses to prevent snow blindness.
- If sunglasses are not available, protective slit goggles can be made from cutting slits in cardboard (e.g., MRE cardboard box).

**Protect Each Other**
- Watch for signs of frostbite and other cold weather injuries in your buddy.
- Ask about and assist with re-warming of feet, hand, ears or face.

**Prevent Carbon Monoxide Poisoning**
- Use only Army-approved heaters in sleeping areas. (post Fire Guards)
- Do not sleep near exhaust of a vehicle while vehicle is running.
- Do not sleep in enclosed area where an open fire is burning.

### Leadership Controls

- Discontinue/limit activities/exercise during very cold weather (see chart page 2).
- Use covered vehicles for troop transport.
- Have warming tents available. (with Fire Guards)
- Have warm food and drink on hand.

### Facility Controls

- Use only Army-authorized heaters. (i.e., no kerosene or propane heaters).
- Ensure heaters are in working order and adequately ventilated.
- Ensure integrity of shelters for maximum protection from the cold.

---

**Figure 7.8.3.8-1. U.S. Army Center for Health Promotion and Preventive Medicine Five-Step Cold Risk Management Tool (continued)**
4 Implement Controls

- Identified controls are in place
- Controls are integrated into SOPs
  - Educate soldiers of hazards and controls (including newly arrived soldiers)
  - Implement buddy system to check clothes/personal protection
- Decision to accept risk is made at appropriate level
- Buddy system to check each other
- Self checks
- Lip Balm (for high altitude training)

5 Supervise and Evaluate

- Ensure all soldiers are educated about prevention, recognition and treatment of cold weather injuries.
- Delegate responsibilities to ensure control measures have been implemented.
- Monitor adequacy/progress of implementation of control measures.
- Do frequent spot checks of clothes, personal protection and hydration.
- Record and monitor indicators of increasing cold risks, for example:
  - Increasing number of cold weather injuries
  - Increased complaints/comments about cold
  - Observations of shivering, signs of cold weather injuries
- Evaluate current control measures and strategize new or more efficient ways to keep warm and avoid cold injuries

Figure 7.8.3.8-1. U.S. Army Center for Health Promotion and Preventive Medicine
Five-Step Cold Risk Management Tool (concluded)

Note: See http://phc.amedd.army.mil for electronic versions of this document and other resources.

References
7-180


Recommended Readings


Concepts

Acclimatization
Anesthetic impairment
Behavioral temperature regulation
Cold defense response
Cold-induced diuresis
Cold stressor injuries
Complex tasks processing
Complex tasks choice reaction time
Core body temperature (Tc)
Dual regulation
Electrolyte replacement
Exertional heat injuries
External stressors
Fighter index of thermal stress (FITS)
Habituation acclimatization
Heat acclimatization
Heat defense response
Heat gain / loss
Heat index chart
Heat strain decision process
Heat stress
Hot defense response
Hydration
Hyperhydration
Insulative acclimatization
Individual-imposed factors
Lapse rate
Metabolic fuel utilization
Metabolic acclimatization
Mission-imposed factors
Normal body temperature
Physiological temperature regulation
Prevention
Primary cold-effector response
Risk management
Shivering thermogenesis
Thermogenesis
Thermoneutrality
Thermoregulation
Vasoconstriction
Balance of heat storage
Wet bulb globe temperature (WBGT)

**Vocabulary**
Air movement
Air saturation
Air temperature
Chilblain
Conduction
Convection
Evaporation
Frostbite, severe / superficial
Frostnip
Heat cramps / tetany
Heat edema
Heat exhaustion
Heat illness
Heat stroke, exertional / classical
Heat syncope
Homeotherm
Hyponatremia
Hyperthermia / Hypothermia
Hypothalamus
Immersion foot
Manual skills
Memory recall
Memory registration
Miliaria rubra
Prevention
Radiation
Reaction times
Rhabdomyolysis
Sunburn
Sweat rate
Thermal stressors
Thermoreceptors
Trench foot
Vigilance
Water immersion
Wind chill chart
7.9. Physiological Effects of Noise

Richard L. McKinley, B.S., M.S.

7.9.1. Introduction

The human auditory system is the sensory system that makes humans feel most “present” in their environment and allows them to communicate easily with other persons (Gilkey and Weisenberger, 1995). It can accommodate a very wide range of sound levels, from 0 dB, the threshold of hearing, to 130 dB, the threshold of auditory pain. However, at the higher noise exposure levels (noise level combined with exposure time), the auditory system can suffer temporary and/or permanent damage. This may result from years of overexposure or occasionally from a single extreme exposure. This chapter will provide some basic information about noise; how it relates to your sense of hearing, including the performance of the human auditory system; and how to protect hearing in continuous and impulsive military noise environments.

Hearing loss is very prevalent among military personnel. High levels of noise, long duration exposures, and, all too frequently, the improper use of hearing protectors result in large amounts of hearing loss. High noise levels and hearing loss frequently degrade voice communication capability, which has a negative impact on mission effectiveness and safety. Additionally, annual disability payments by the Department of Veterans Affairs (VA) for hearing loss as primary disability have been growing over the past three decades. For example, Figure 7.9.1-1, provided by the Department of Defense (DoD) Hearing Center of Excellence, shows the rapid increase in hearing loss compensation payments for 1977 – 2006. If the costs associated with treatment or retraining are considered, then the total cost in 2006 alone were estimated to be over $3 billion (Yankaskas, 2006). Since 2006, the costs have continued to climb as seen in Table 7.9.1-1, also provided by the DoD Hearing Center of Excellence. The method in which these payments are made make it very difficult accurately compute the costs associated to hearing loss.

Cost of Hearing Loss for All US Veterans (1977-2006)

Total = $8,385,892,465 BILLION

Major VA Disability Only

- Total does not include annual cost for tinnitus disabilities
- Total does not include treatment costs, e.g., hearing aids, retraining
- Total does not include disability paid to the 3x more veterans with hearing loss as a non-primary disability

Costs are approaching $1 Billion annually

Figure 7.9.1-1. VA Hearing Loss Disability Payment Annual Costs
Table 7.9.1-1. VA Hearing Loss Disability Payment FY13 – FY14 Costs

7.9.2. Physics of Noise

Noise is undesired or unwanted sound. Sound or noise is usually described quantified in units of decibels (dB) although high level impulsive noise is sometimes described in Pascals (Pa). Decibels are not an absolute measure but a ratio of the measured sound pressure to a reference sound pressure. The most common reference is:

\[ 0 \text{ dB} = 20 \mu \text{Pa} = 2 \times 10^{-5} \text{ Pa} \]

Decibels in sound pressure level (dB SPL) relate an absolute measure of pressure (Pascals, Pa) to the reference pressure. Decibels for hearing function (dB HL – hearing level, or dB HTL – hearing threshold level) are expressed relative to the average thresholds for persons with normal hearing. At 1 kHz, 0 dB HL (about 0 dB SPL or 20 µPa or 0.0002 dynes/cm²) is the quietest sound that can be heard by an average person with normal hearing; it also corresponds with 0 dB SPL. However, as the frequency gets lower than 1 kHz or higher than 4 kHz, 0 dB HL will correspond to increasingly higher absolute pressures and higher sound pressure levels.

People are less sensitive to sound/noise at lower frequencies and more sensitive in the middle and higher frequencies. The A-weighting function shown in Figure 7.9.2-1 is used in the United States for assessments of hearing damage risk in the workplace and for environmental noise assessments. Currently, it is the best predictor of potential hearing loss in the workplace and of potential annoyance in the community. The C-weighting function is the second most often used weighting function. The other weighting functions such as B and D are almost never used. Sound level meters typically have both the A-weighting and C-weighting function. Measuring the overall C-weighted noise level and subtracting the overall A-weighted noise level gives an
indication of the amount of low frequency noise in the spectrum. A positive difference (C – A) indicates more low frequency spectral content. This information can be very useful when selecting hearing protection for a particular noisy environment.

Figure 7.9.2-1. Weighting Functions A, B, C, and D
(source: en.wikipedia.org)

Sound/noise is measured on a logarithmic scale. In engineering terms, a doubling of the sound energy results in a 3 dB increase in the sound level. For human listeners, a doubling of the perceived loudness corresponds to approximately a 10 dB increase in sound level. For an average untrained person, a just-noticeable change in sound level is a 3 dB change, and a trained listener can normally discriminate sound level changes as small as 1 dB (Nixon). However, level discrimination is very complex and difficult to measure accurately (Shepherd and Hautus 2007).

Humans with good hearing can “hear” an extremely large range of sound pressures. Remember 0 dB SPL is 20 µPa or 0.0002 dynes/cm². By calculating the logarithm of the pressure ratio (the ratio of the pressure relative to 0.0002 dynes/cm²) and multiplying the result by 20, the decibel level can be calculated.

\[
decibels = 10 \times \log_{10} \left( \frac{\text{pressure}}{0.0002 \text{ dynes/cm}^2} \right)^2 = 20 \times \log_{10} \left( \frac{\text{pressure}}{0.0002 \text{ dynes/cm}^2} \right)
\]

Table 7.9.2-1 shows the relationship between pressure and sound pressure levels (dB in air) relative to the standard reference level for 0 dB (0.0002 dynes/cm²). The pressures have a very large range most of which is efficiently transduced by the human auditory system.
Since decibels are based on the logarithm of the pressure ratios, we say that decibels are a logarithmic function. When adding sound pressure levels (SPL), logarithmic addition needs to be performed. For example, 93 dB + 93 dB = 96 dB (this also is an example of the 3-dB-per-doubling rule), and 93 dB + 83 dB = 93.4 dB. The equation below shows how to perform logarithmic addition of sound pressure levels.

\[
\text{sum SPL decibels} = 10 \times \log \left[ 10^{\left(\frac{\text{SPL dB}_1}{10}\right)} + 10^{\left(\frac{\text{SPL dB}_2}{10}\right)} \right]
\]

### 7.9.3. Noise Exposure Criteria

Noise exposure criteria for the U.S. military and Department of Defense (DoD) civilians are described in DoD instructions, military standards, and military instructions. The Army, Navy, and Air Force have separate instructions and hearing conversation programs in addition to the DoD regulations and hearing conservation programs. Additionally, the noise exposure criteria are described separately for continuous noise (noise with a duration longer than 1 second) and impulsive noise (noise with a rapidly (< 50 ms) rising peak and a duration less than 1 second). The noise exposure criteria have been developed generally based on long-term hearing loss studies and temporary threshold shift studies from a wide range of exposures.

Generally, noise exposure criteria are a matter of policy. Technical experts can predict the amount of permanent hearing loss or NIPTS (noise-induced permanent threshold shift) in a population from years of exposure to noise within given criteria. Noise exposure criteria are a balance between a policy of acceptable levels of hearing loss and the noise exposure levels in the criteria. One example from the 1998 National Institute for Occupational Safety and Health (NIOSH) Criteria Document is shown below in Table 7.9.3-1.
Table 7.9.3-1. Estimated Excess Risk of Incurring Material Hearing Impairment\textsuperscript{a} as a Function of Average Daily Noise Exposure over a 40-Year Working Lifetime\textsuperscript{b}

<table>
<thead>
<tr>
<th>Reporting Organization</th>
<th>Average Daily Noise Exposure [dB(A)]</th>
<th>Excess Risk (%)\textsuperscript{c}</th>
</tr>
</thead>
<tbody>
<tr>
<td>ISO\textsuperscript{d}</td>
<td>90</td>
<td>21</td>
</tr>
<tr>
<td></td>
<td>85</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>80</td>
<td>0</td>
</tr>
<tr>
<td>EPA\textsuperscript{e}</td>
<td>90</td>
<td>22</td>
</tr>
<tr>
<td></td>
<td>85</td>
<td>12</td>
</tr>
<tr>
<td></td>
<td>80</td>
<td>5</td>
</tr>
<tr>
<td>NIOSH</td>
<td>90</td>
<td>29</td>
</tr>
<tr>
<td></td>
<td>85</td>
<td>15</td>
</tr>
<tr>
<td></td>
<td>80</td>
<td>3</td>
</tr>
</tbody>
</table>

\textsuperscript{a}For purposes of comparison in this table, material hearing impairment is defined as an average of the HTLs for both ears at 500, 1000, and 2000 Hz that exceeds 25 dB.
\textsuperscript{b}Adapted from 39 Fed. Reg 43802 (1974b).
\textsuperscript{c}Percentage with material hearing impairment in an occupational-noise-exposed population after subtracting the percentage who would normally incur such impairment from other causes in an unexposed population.
\textsuperscript{d}International Standards Organization.
\textsuperscript{e}Environmental Protection Agency.

7.9.4. Continuous Noise Criteria

DoD Instruction 6055.12 specifies the noise exposure criterion for continuous noise at 85 dB(A) level equivalent ($L_{eq}$) or 8-hour TWA (time weighted average) with a 3-dB/doubling exchange rate. This criterion includes two important factors. First, the exposure is a combination of the noise level AND the exposure time. Second, for every 3-dB increase in exposure level, the allowable exposure time is reduced by a factor of two. Additionally, the DoD instruction exposure criterion is more protective, (i.e., allows less noise exposure than the Occupational Safety and Health Administration (OSHA) 90-dB(A) criterion 8-hour TWA and a 5-dB/doubling exchange rate which applies to DoD contract personnel and other U.S. industrial personnel). Table 7.9.4-1 shows the allowable exposure times in a 24-hour period for a wide range of A-weighted noise levels using the DoD criterion. For exposure times greater than 8 hours, the recovery time at less than 75 dB(A) should be equal to or greater than the exposure time.

Noise levels at the ear can be computed by subtracting the hearing protection level from the ambient noise level. Double hearing protection is usually recommended for levels above 115 dB(A). A non-auditory, whole body noise exposure limit has been established at 150 dB overall sound pressure level or 145 dB in any single 1/3 octave or octave band. This whole body limit was established empirically by a group of scientists who exposed themselves to high level whole body noise from 130 dB to approximately 170 dB and reported their findings (Cole, et. al. 1965). Since there were no unusual symptoms at or below 150 dB, the limit was established and has been reaffirmed on several occasions by teams measuring the near-field noise of high performance jet aircraft.
Table 7.9.4-1. Allowable Noise Levels at the Ear and Times in a 24-hour Period with Equal Rest at ≤ 75 dB(A)

<table>
<thead>
<tr>
<th>A-Weighted Level [dB(A)]</th>
<th>Allowable Exposure Time (hr)</th>
<th>Allowable Exposure Time (min)</th>
<th>Allowable Exposure Time (s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>80.2</td>
<td>24</td>
<td>1442</td>
<td>86502</td>
</tr>
<tr>
<td>82</td>
<td>16</td>
<td>960</td>
<td>57600</td>
</tr>
<tr>
<td>85</td>
<td>8</td>
<td>480</td>
<td>28800</td>
</tr>
<tr>
<td>88</td>
<td>4</td>
<td>240</td>
<td>14400</td>
</tr>
<tr>
<td>91</td>
<td>2</td>
<td>120</td>
<td>7200</td>
</tr>
<tr>
<td>94</td>
<td>1</td>
<td>60</td>
<td>3600</td>
</tr>
<tr>
<td>97</td>
<td>0.5</td>
<td>30</td>
<td>1800</td>
</tr>
<tr>
<td>100</td>
<td>0.25</td>
<td>15</td>
<td>900</td>
</tr>
<tr>
<td>103</td>
<td>0.125</td>
<td>7.5</td>
<td>450</td>
</tr>
<tr>
<td>106</td>
<td>0.0625</td>
<td>3.75</td>
<td>225</td>
</tr>
<tr>
<td>109</td>
<td>0.0313</td>
<td>1.875</td>
<td>112.5</td>
</tr>
<tr>
<td>112</td>
<td>0.0156</td>
<td>0.938</td>
<td>56.25</td>
</tr>
<tr>
<td>115</td>
<td>0.0078</td>
<td>0.469</td>
<td>28.13</td>
</tr>
<tr>
<td>118</td>
<td>0.0039</td>
<td>0.234</td>
<td>14.06</td>
</tr>
<tr>
<td>121</td>
<td>0.0020</td>
<td>0.117</td>
<td>7.03</td>
</tr>
<tr>
<td>124</td>
<td>0.0010</td>
<td>0.059</td>
<td>3.52</td>
</tr>
<tr>
<td>127</td>
<td>0.0005</td>
<td>0.029</td>
<td>1.76</td>
</tr>
<tr>
<td>130</td>
<td>0.0002</td>
<td>0.015</td>
<td>0.88</td>
</tr>
</tbody>
</table>

Figure 7.9.4-1 shows some of the worst-case near-field noise levels from high performance fighter aircraft. The levels range from 135 dB to 148 dB at military power and from 146 dB to 153 dB at afterburner power.

Ground Run-up/ Maintainer Noise

![Figure 7.9.4-1. Worst-Case Near-Field Noise Levels From High Performance Fighter Aircraft](image-url)
7.9.5. Impulsive Noise Criteria

Impulsive noise generally results from the firing of a weapon, small arms to artillery; mortars; or explosions (improvised explosive devices (IEDs), detonation cord – emergency egress or ejection – canopy cutting). The levels are usually much higher than continuous noise and also of a much shorter duration. Some typical levels are shown in Table 7.9.5-1.

### Table 7.9.5-1. Typical Impulsive Noise Levels from Weapons Fire

<table>
<thead>
<tr>
<th>Model</th>
<th>Name</th>
<th>Location</th>
<th>Sound Level (dB) (peak)</th>
</tr>
</thead>
<tbody>
<tr>
<td>M16A2</td>
<td>5.56 mm rifle</td>
<td>Shooter</td>
<td>157</td>
</tr>
<tr>
<td>M9</td>
<td>9 mm pistol</td>
<td>Shooter</td>
<td>157</td>
</tr>
<tr>
<td>M249</td>
<td>5.56 mm squad automatic weapon (SAW)</td>
<td>Gunner</td>
<td>159.5</td>
</tr>
<tr>
<td>M60</td>
<td>7.62 mm machine gun fired from a HMMWV</td>
<td>Gunner</td>
<td>155</td>
</tr>
<tr>
<td>M2</td>
<td>0.50 caliber machine gun fired from a HMMWV</td>
<td>Gunner</td>
<td>153</td>
</tr>
<tr>
<td>MK 19 Mod 3</td>
<td>Machine gun fired from a HMMWV</td>
<td>Gunner</td>
<td>145</td>
</tr>
<tr>
<td>M26</td>
<td>Grenade</td>
<td>At 50 ft</td>
<td>164.3</td>
</tr>
<tr>
<td>M3</td>
<td>MAAWS recoiless rifle</td>
<td>Gunner</td>
<td>190</td>
</tr>
<tr>
<td>M72A3</td>
<td>Light antitank weapon (LAW)</td>
<td>Gunner</td>
<td>182</td>
</tr>
<tr>
<td>M119</td>
<td>105 mm towed howitzer at charge 8</td>
<td>Gunner</td>
<td>183</td>
</tr>
<tr>
<td>M198</td>
<td>155 mm towed howitzer firing M203 propellant</td>
<td>Gunner</td>
<td>178</td>
</tr>
<tr>
<td>M109A5/6</td>
<td>Paladin, 155 mm self-propelled howitzer    In fighting compartment, hatches</td>
<td>166.1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>firing M4A2 zone 7 charge</td>
<td>open except driver's</td>
<td></td>
</tr>
<tr>
<td>M110A2</td>
<td>8-in self-propelled howitzer firing M106 projectile with a M188A1 zone 9 propelling charge</td>
<td>Gunner</td>
<td>176.9</td>
</tr>
</tbody>
</table>

The impulsive noise exposure criterion is currently described in Military Standard (MILSTD) 1474E as shown in the following equation. This is a new standard based on the equal energy principal used for continuous noise. The $L_{Aeq}$ (A-weighted level equivalent) based metric is currently used in the European Union (EU) for impulse noise damage risk assessment. The modified $L_{Aeq100ms}$ metric, below, in MILSTD 1474E (see the MILSTD for details) includes a mathematical correction factor to reduce the overestimation of hearing damage risk due to long duration impulses.

$$L_{Aeq100ms} = 10 \log_{10} \left\{ \left( \frac{1}{T} \right) \sum_{i=1}^{n} \left\{ 10 \log_{10} \left[ \frac{L_{Ai}}{10} \times \Delta t_i \right] \right\} \right\}$$

*where:*
- $T = 100$ ms is the integration time
- $L_{Ai} =$ The A-weighted Level of the $i^{th}$ sample of the impulsive waveform
- $\Delta t_i =$ The sample time of the $i^{th}$ sample of the impulsive waveform
The criteria described in detail in MILSTD 1474E also include computation methods when hearing protectors are used and allows for the combined risk from continuous and impulsive noise exposures to be computed. The 30+ year debate in the scientific community regarding exposure criteria for impulsive noise continues but this is the objectively most widely used metric. The problems with impulsive noise damage risk criteria are related to the multiple damage mechanisms and the wide range of levels and durations. Additionally, for impulses above 195 dB, blast lung or other soft tissue injury should be considered.

7.9.6. Noise Dose Calculation Method for Continuous Noise

Noise dose is a combination of noise level and duration evaluated relative to a noise exposure criterion and exchange rate. The goal for noise-exposed workers is to keep the noise dose to 100% or lower on a daily basis. Noise dose can also be computed/calculated for time durations longer or shorter than a standard 8-hour work day. An example would be the noise dose accumulated by a pilot during a given flight in the high noise of the cockpit. The flight might be only 1.5 hours in duration, but the associated noise dose could easily exceed 100%. Another example would be the noise dose accumulated in command and control centers where the noise is at a relatively lower intensity but the duration may be significantly greater than 8 hours. Once again the allowable noise dose could easily exceed 100%. Noise dose is an aggregate or summation of several individual noise doses calculated from noise spectra/levels and times at each noise level evaluated relative to the noise exposure criterion.

The equation below gives a method for calculating noise dose from \( n \) multiple events for any noise exposure criterion, and exchange rate.

\[
Dose = 100 \times \sum_{i=1}^{n} \frac{t_i \times 2^{(L_i - L)/E}}{T}
\]

where:

- \( Dose \) = percentage dose
- \( t_i \) = exposure time in minutes for the \( i \)th time period
- \( L_i \) = A-weighted level in dB(A) for the \( i \)th time period
- \( L \) = exposure criterion A-weighted level in dB(A) [e.g., 85 dB(A)]
- \( E \) = exposure criterion exchange rate in dB per doubling of exposure time (e.g., 3 dB/doubling)
- \( T \) = exposure criterion time in minutes (e.g., 8 hours would be 480 minutes)
- \( n \) = total number of exposure time periods (e.g., if you had 17 separate noise levels and time periods, then \( n = 17 \))

The exposure criterion can have a dramatic effect on the total dose. The following examples will show the equations, MS Excel™ spreadsheet cell code, and dose calculations using the DoD (85 dBA, 8 hours, 3 dB/doubling) and the OSHA (90 dBA, 8 hours, 5 dB/doubling) criteria both using the same noise levels and exposure times.
Below are two example equations. The first example is for a noise exposure criterion of 85 dB(A), for 8 hours (480 minutes), with a 3-dB-per-doubling exchange rate. The second example is for a noise exposure criterion of 90 dB(A), for 8 hours (480 minutes), with a 5-dB-per-doubling exchange rate.

\[
Dose = 100 \times \sum_{i=1}^{n} t_i \times 2^{\left(\frac{L_i - 85}{3}\right)} \quad 480
\]

\[
Dose = 100 \times \sum_{i=1}^{n} t_i \times 2^{\left(\frac{L_i - 90}{5}\right)} \quad 480
\]

Included below are the equations in MS Excel™ format, where A2 is the time in minutes, B2 is the exposure level in dB(A), and 8 hours is represented as 480 minutes.

\[
= 100*(((A2*2^((B2-85)/3)))/480)
= 100*(((A2*2^((B2-90)/5))/480)
\]

**DoD Criterion (85/8/3)**

**OSHA Criterion (90/8/5)**

Tables 7.9.6-1 and 7.9.6-2 show the spreadsheet cells used to calculate the noise dose in the preceding examples. The first table is for the DoD 85-dB(A), 8-hour, 3-dB/doubling criteria while the second table is for the OSHA 90-dB(A), 8-hour, 5-dB/doubling criteria.

| Table 7.9.6-1. DoD Criterion (85/8/3) |
|---|---|---|
| 1 | Time (min) | Level [dB(A)] | Dose (%) |
| 2 | A2 | B2 | =100*(((A2*2^((B2-85)/3)))/480) |
| 3 | A3 | B3 | =100*(((A3*2^((B3-85)/3)))/480) |
| 4 | A4 | B4 | =100*(((A4*2^((B4-85)/3)))/480) |
| 5 | Total Dose | =Sum(C2:C4) |

| Table 7.9.6-2. OSHA Criterion (90/8/5) |
|---|---|---|
| 1 | Time (min) | Level [dB(A)] | Dose (%) |
| 2 | A2 | B2 | =100*(((A2*2^((B2-90)/5))/480) |
| 3 | A3 | B3 | =100*(((A3*2^((B3-90)/5))/480) |
| 4 | A4 | B4 | =100*(((A4*2^((B4-90)/5))/480) |
| 5 | Total Dose | =Sum(C2:C4) |

Next are two examples of the dose calculation using each sample noise exposure criterion. First, we will calculate the dose for the situation where a person is exposed to 85 dB(A) at the ear for 4 hours, 88 dB(A) for 1 hour, and 91 dB(A) for 30 minutes.
The example dose calculations for the two exposure criteria are as follows: first, on the left of Table 7.9.6-3, the 85-dB(A)-with-3-dB/doubling criterion; second, on the right, the 90-dB(A)-with-5-dB/doubling criterion.

Table 7.9.6-3. First Example of Dose Calculations

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>Level [dB(A)]</th>
<th>Dose (%)</th>
<th>Time (min)</th>
<th>Level [dB(A)]</th>
<th>Dose (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>240</td>
<td>85</td>
<td>50</td>
<td>240</td>
<td>85</td>
<td>25</td>
</tr>
<tr>
<td>60</td>
<td>88</td>
<td>25</td>
<td>60</td>
<td>88</td>
<td>9</td>
</tr>
<tr>
<td>30</td>
<td>91</td>
<td>25</td>
<td>30</td>
<td>91</td>
<td>7</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>100</strong></td>
<td><strong>41</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

For the second example, we will calculate the dose for a different exposure. This time, a person is exposed to 95 dB(A) at the ear for 1 hour, 90 dB(A) for 2 hours, and 85 dB(A) for 4 hours (Table 7.9.6-4).

The dose calculations for the two exposure criteria are as follows: first, on the left, the 85-dB(A)-with-3-dB/doubling criterion; and second, on the right, the 90-dB(A)-with-5-dB/doubling criterion.

Table 7.9.6-4. Second Example of Dose Calculations

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>Level [dB(A)]</th>
<th>Dose (%)</th>
<th>Time (min)</th>
<th>Level [dB(A)]</th>
<th>Dose (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>60</td>
<td>95</td>
<td>126</td>
<td>60</td>
<td>95</td>
<td>25</td>
</tr>
<tr>
<td>120</td>
<td>90</td>
<td>79</td>
<td>120</td>
<td>90</td>
<td>25</td>
</tr>
<tr>
<td>240</td>
<td>85</td>
<td>50</td>
<td>240</td>
<td>85</td>
<td>25</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>255%</strong></td>
<td></td>
<td><strong>Total</strong></td>
<td><strong>75%</strong></td>
<td></td>
</tr>
</tbody>
</table>

As can be seen, the exposure criterion has a dramatic effect on the calculated total dose. The OSHA criteria allows significantly more exposure than does the DoD hearing conservation criteria. The examples and spreadsheets can be expanded/extended to add as many different noise exposure levels and time as desired for the calculation of the total noise dose. Once the noise dose is calculated for each level and time, all the noise doses are then summed to calculate the total noise dose.

The noise dose calculation method for impulsive noise is described in detail in MILSTD 1474E and follows the basic equation below.

$$D_I (%) = \frac{100}{2^{(85 - L_{IAeq8hr})/3}}$$

where:

- \(D_I\) = Impulsive noise dose, in percent, for a single impulse (multiply the number of impulses within an 8-hour period to compute the impulsive noise dose for the 8-hour period)
- \(L_{IAeq8hr}\) = Impulsive A-weighted level equivalent for 8 hours, where:

\[
L_{IAeq8hr} = (L_{IAeq100ms}) - 54.6 - 1.5 * 10 * \log_{10} \left( \frac{A - duration}{0.2 \text{ ms}} \right)
\]

\(L_{IAeq100ms}\) = see equation above in Section 7.9.5

\(A - duration\) = duration (in ms) of exposure to A-weighted impulse noise lasting less than 2.5 ms (see MILSTD 1474E)
7.9.7. Hearing Protection

Hearing protection technologies can be very simple (foam earplugs or simple earmuffs) to very complicated (active noise reduction earplugs with large volume earmuffs mounted in a special helmet to reduce bone conduction). The major factor in the effectiveness of a hearing protector is compliance. That is, is it worn and, if so, is it worn properly? Frequently, the highest noise exposure segment for an industrial worker is the 30 minutes at lunch when he or she is not wearing the hearing protector.

A recent survey of U.S. Navy flight deck crews found a startling fact. Most of the personnel were not getting anything close to the attenuation required because almost half of the users were not wearing the earplugs and those who were wearing earplugs were not wearing them properly. The data from the Navy study are shown in Figure 7.9.7-1. The USAF hearing conservation program uses hearing protection attenuation values which are the mean attenuation (dB) minus two standard deviations. This is to provide the stated attenuation (or better) value 98% of the personnel using the device.

![Figure 7.9.7-1. Percentage of Earplug Insertion Depth and Estimated Attenuation (Naval Air Systems Command Survey of 301 Flight Deck Personnel)](image)

In a controlled laboratory setting, earplugs typically provide approximately 1 to 30 dB of attenuation performance depending on the specific make and model of earplug. However, when used in a real-world setting, most well motivated using populations will only achieve attenuation values that are about 33% of the attenuation achieved in the laboratory. Typical laboratory performance of earmuffs or helmets range from 10 to 25 dB with about 90% laboratory value being realized in a well-motivated using population. Combination hearing protection of earmuffs and earplugs used together typically results in total attenuations of about 5 dB greater than the better performing hearing protector. Additional care and analysis needs to be given to flanking pathways such as bone conduction once the attenuation of the combination hearing protector is ≥ 40 – 45 dB.

Active noise reduction (ANR) earmuffs typically provide 10 to 15 dB additional protection in the frequency range from 125 Hz to 800 Hz and, therefore, have the most dramatic effect in low-frequency-dominated noises such as propeller aircraft, helicopters, tanks, etc., but also may be effective in many jet aircraft when the noise exposure (under the helmet or earmuff) is driven by exposures in low-frequencies, (i.e. below 1 kHz). Noise exposure calculations should be conducted with total mission scenarios—noise levels and times for each mission segment summed for the total mission.
Active noise reduction earplugs are just emerging but can provide 5 to 12 dB of additional protection in the frequency range from 125 Hz to 3 kHz. These systems have been originally developed for maintainers working in the very high noise levels found in the near-field of a high-performance jet. These systems initially have been very expensive but are expected to decrease in cost with higher production quantities.

Tactical hearing protection systems generally have been designed to attenuate both impulse noise and continuous noise while providing some ambient listening/localization capability and enhancing communication capability with interfaces to radio communication. These devices mostly are electronic earplugs or electronic earmuffs, although a few passive earplugs and passive earmuffs are available.

### 7.9.8. Sense of Hearing

The human sense of hearing has many functions, including the detection, identification, localization of sounds in the environment as well as speech communication. Auditory detection thresholds are determined by the ambient noise environment, which may or may not mask the event, and the hearing threshold of the listener. A 10-dB temporary threshold shift (TTS) can reduce the detection threshold such that distance at which an event or possible threat is greatly reduced potentially impacting the mission. For an example, an event that would have been detected at 100 feet prior to the TTS might not be detected until it is only at 31 feet after a 10-dB TTS. Clearly, a TTS can have a significant effect on auditory detection thresholds. Identification thresholds and speech communication effectiveness can be similarly affected.

The ability to determine the location of a sound source is defined as auditory localization. Most people can localize a sound source within a few degrees. The auditory system is integrated with the visual system and can very quickly and effectively direct the gaze of the eye to particular locations when a sound is presented from the location of the visual target. The effect of localization cues on visual search, as measured in experiments conducted by the Air Force Research Laboratory’s Battlespace Acoustics Branch, is shown in Figure 7.9.8-1 (Simpson et al., 2005). These localization cues are moderately disrupted by passive hearing protectors such as earmuffs and earplugs (2nd and 3rd data lines from the bottom), but in situations where there is time (1 – 2 seconds) and head motion is allowed, the disruptive effects are not large. However, in situations where double hearing protection is used, the localization cues seem to be completely disrupted, and the listeners cannot reasonably localize.
Look at the top two lines of Figure 7.9.8-1 and notice the close relationship between the "earmuffs+earplugs" protection response times line and the "no cue" response time line. Effectively, double hearing protection removes almost all of the information used to localize. The tactical hearing protection systems previously described have been designed in an attempt to restore or preserve many of the auditory cues used for localization.

Speech communication performance in noise is primarily dependent on the speech-to-noise ratio (SNR). The higher the SNR (up to 30 dB according to the Articulation Index calculation standard), the better the speech intelligibility. However, in military communication systems, speech intelligibility cannot be accurately predicted from SNR due to nonlinearity in the communication system. Behavioral speech intelligibility tests such as the Modified Rhyme Test (MRT) and the Diagnostic Rhyme Test (DRT) have been shown to be effective measures of speech intelligibility performance over communication systems and have been described in an American National Standards Institute (ANSI) standard, S3.2-2009.

### 7.9.9. Summary

The auditory system is a robust communication pathway critical to the well-being of the listener. The auditory system provides communication capability, detection, identification, and localization capability for threats and targets and works with the visual system to identify locations of interest. The auditory system is susceptible to damage at higher noise levels, and it must be protected by either restricting the exposure level and time or by providing hearing protection, or by doing both. Hearing loss due to military noise exposures not only affects operational readiness but the personal lives of those affected. Hearing loss should be considered in the same class of disabilities such as progressive blindness. Glasses or lenses filter light but do not restore vision. Likewise, hearing aids do not restore hearing when the sensory cells are permanently damaged.
References
MILSTD 1474E, Department of Defense Design Criteria Standard Noise Limits. 15 April 2015.

Recommended Readings

Concepts
Noise dose
Noise exposure criteria

Vocabulary
Decibels (dB)
Impulsive noise
Noise
7.10. Radiation

Lt Col Christy A. Kayser-Cook, USAF

Note: Unless otherwise noted, the following material comes from the USAF Radiation Safety Officers Course, Lessons 6 – 9.

Radiation is defined as the process in which energy is emitted by one body, transmitted through an intervening medium or space, in the form of waves or particles, and absorbed by another body.

All objects radiate energy to their surrounding environment. This energy, or radiation, is emitted as electromagnetic (EM) waves that travel at the speed of light. Many different types of radiation have been identified. Each of these types is defined by its wavelength. The wavelength of EM radiation can vary from being infinitely short to infinitely long (www.Physicalgeography.net).

7.10.1. Types of Radiation

There are various ways of categorizing radiation. One way is as either ionizing or non-ionizing radiation. Ionizing radiation has shorter wavelengths (higher frequency) and higher energy than non-ionizing radiation (Figure 7.10.1-1). Ionizing radiation is of greater concern than non-ionizing radiation because of the higher energy levels. Ionizing radiation, which includes x-rays, gamma rays, alpha and beta particles, and neutrons, carries sufficient energy to remove electrons from atoms in materials in which the radiation passes. Non-ionizing radiation, which includes microwaves, radio/TV waves, ultraviolet (UV), and visible light, deposits energy in the material through which it passes, but does not have enough energy to remove electrons or break molecular bonds. Other categorizations are based on if the radiation has mass (alphas, betas and neutrons) or are massless (gamma rays and x-rays). Another way radiation can be categorized is whether it is charged (alphas and betas) or not charged (gammas, x-rays, and neutrinos).

![Figure 7.10.1-1. Types of Radiation](http://pixshark.com/ionizing-radiation-sources.htm)
7.10.2. Particle Interactions

There are three types of high-energy, charged particle interactions: 1) ionization, 2) excitation, and 3) Bremsstrahlung. The type of radiation will determine its interaction with the material it encounters. Understanding these interactions will help to determine how the radiation can be detected with instrumentation, how the dose is delivered to the material, what makes an effect shield, and what types of radiation protection techniques will be effective. These interactions can be grouped into two main types: 1) charged particle interactions and 2) uncharged particle interactions.

7.10.2.1. Charged Particles. Due to their electric charge, charged particles, such as alpha particles or electrons, will continuously interact with the electrons present in any medium through which they pass. These interactions result in a full or partial transfer of the charged particle’s energy to either the electron or the nuclei of the medium’s atom and result in one of the three types of interactions mentioned above (ionization, excitation or Bremsstrahlung).

When the energy transferred from a charged particle to the electron belonging to an atom (or molecule) of matter is greater than the energy holding the electron (the binding energy), ionization occurs. The electron will leave the atom, producing an ion pair. The atom is left with a net positive charge and an electron shell vacancy. As electrons drop to lower energy levels to replace the removed electron, another electron is acquired to neutralize the atom and the positively charged ion will emit one or more very low energy photons in the ultraviolet range. Ionization is most likely to occur in atoms near the charged particle’s trajectory. Each ionization event reduces the charged particle’s velocity because kinetic energy is transferred each time.

Excitation is any process that adds enough energy to an electron that is bound to an atom (or molecule) so that it occupies a higher energy state within the atom (which means lower binding energy), but not enough energy to ionize the atom. Similar to ionized atoms, the excited atom will emit very low energy photons in the ultraviolet range when the electron returns to its ground state. In contrast to ionization, excitation produces no ions (the atom remains electrically neutral). Excitation is most likely to occur in atoms some distance from the charged particle’s trajectory. However, as with ionization, each excitation reduces the charged particle’s velocity because some of its kinetic energy is transferred each time.

Bremsstrahlung is a German term that means “braking rays.” It is an important phenomenon in the generation of x-rays. In the Bremsstrahlung process, a high speed electron traveling in a material is slowed, or completely stopped, by the forces of any atom it encounters. As a high speed electron approaches an atom, it will interact with the negative force from the electrons of the atom, and it may be slowed or completely stopped. If the electron is slowed down, it will exit the material with less energy. The law of conservation of energy tells us that this energy cannot be lost and must be absorbed by the atom or converted to another form of energy. The energy used to slow the electron is excessive to the atom and the energy will be radiated as x-ray radiation of equal energy. If the electron is completely stopped by the strong positive force of the nucleus, the radiated x-ray energy will have energy equal to the total kinetic energy of the electron. This type of action occurs with very large and heavy nuclei materials. The new x-rays and liberated electrons will interact with matter in a similar fashion to produce more radiation at lower energy levels until finally all that is left is a mass of long wavelength EM wave forms that fall outside the x-ray spectrum (https://www.nde-
Bremsstrahlung is almost exclusively associated with beta particles and high-energy electron radiations.

To summarize charged particle interactions:

- Charged particles interact because of their charge.
- It is certain that charged particles will interact (i.e., it is not a matter of probability).
- Alpha particles lose energy very quickly, so they do not penetrate very far. This makes them easy to shield. However, if alpha particles interact with live tissue, they will deliver a large absorbed dose.
- Beta particles do not lose their energy as quickly as alpha particles, so they can penetrate a little further into matter. Their range will depend on their energy, atomic number, and density of the material with which they interact.
- Negative beta particles and electrons can produce Bremsstrahlung photons in high Z (high atomic number of protons in the nucleus) materials. This can complicate counting situations and shielding or dosimetry. Bremsstrahlung production can be minimized by surrounding beta particles with low Z material such as Lucite®.
- Positive beta particles produce annihilation photons (511keV) when they interact with matter. Positron emitters will have 511 keV photons associated with them.

7.10.2.2. Uncharged Particles. Radiations that have no electrical charge (such as gamma rays, x-rays, and neutrons) cannot interact with electrical fields created by electrons and protons. They must actually collide with these particles to interact. Since matter is made up of mostly empty space, uncharged electromagnetic radiation and neutrons are able to move freely through matter and have a small probability of interacting with it. This is why uncharged radiation is also called penetrating radiation (Figure 7.10.3-1).

In contrast to charged particle interactions, uncharged radiations do not continuously lose energy by constantly interacting with the absorbing matter. Instead, they may penetrate material and move through many atoms or molecules before they physically collide with an electron or nucleus. For example, in a chest x-ray the image is the distribution of x-rays that made it to the film without interacting in the patient’s chest. When uncharged radiations do interact, they produce charged particles that cause secondary ionizations. The probability of the interaction is dependent upon the energy of the radiation and the density and atomic number of the absorbing matter.

A photon is a quantum (a discrete quantity of energy) of electromagnetic radiation. It has no mass, no charge, and travels in a straight line at the speed of light. Photons are frequently referred to as “bundles,” or quanta, of electromagnetic energy. Not all photons have enough energy to ionize matter. Radio waves and visible light are photons, but they have much less energy than the gamma rays, x-rays, and other photons. The gamma rays and x-rays emitted by radioactive materials are waves of...
pure energy. Like all electromagnetic waves, they travel at the speed of light and their energies are determined by their frequencies (alpha and beta particles are not part of the electromagnetic spectrum. They travel at much less than the speed of light and their energies are functions of their velocities and masses).

### 7.10.2.2.1. Photon Interactions

Even though photons have no mass, some of their behavior can be particle-like, and some wave-like. In general, after photon radiation interacts with matter, electrons are liberated from an atom. These freed electrons then interact by ionization and excitation. There are three principal types of photon interactions: 1) photoelectric effect, 2) Compton scattering, and 3) pair production. There are more interactions for either extremely low-energy or extremely high-energy photons. However, for the range of energies encountered the majority of the time, the three listed above are the ones discussed here. As we review these three principal interactions, you should bear in mind that all of the photons we have mentioned so far (gamma rays, x-rays, Bremsstrahlung photons, and annihilation photons) can interact via these mechanisms.

#### 7.10.2.2.1.1. Photoelectric Effect

In the photoelectric effect (PE interaction), a photon may undergo a “head on” collision with an orbital electron of an atom. It is possible for all of the photon’s energy to be transferred to the electron, which causes the electron to be ejected from the atom. The PE interaction is most probable when the energy of the incident (colliding) photon and the binding energy of the electron are about the same. This occurs most often with relatively lower energy photons and relatively higher atomic number atoms, which have higher electron binding energies. This means that the interaction tends to occur most often with the more tightly bound inner shell electrons than with the more loosely bound outer shell electrons.

The PE interaction is considered an absorption process since all of the photon’s energy is deposited in the atom. Because of this factor, photoelectric interactions result in effective shields against this type of radiation. Shielding design involves careful matching of photon energy with the atomic number of the shielding material to increase the probability of photoelectric interactions. On the other hand, PE interactions are not desirable from the standpoint of absorbed dose to human tissue. Reducing the probability of PE interactions in human tissue, if possible, will reduce the absorbed dose to that tissue.

#### 7.10.2.2.1.2. Compton Scattering

The second type of photon interaction for discussion is Compton scattering, in which a photon undergoes a “sideswipe” collision with a loosely bound (essentially “free”) electron of an atom. In contrast to the PE interaction, only some of the photon’s energy is transferred to the electron; the rest is carried away by the scattered photon. Both the photon and the electron are scattered off in different directions and go on to interact via their respective interaction mechanisms.

Compton scattering occurs at all photon energies and in all materials. It occurs when an incident particle collides with an electron whose binding energy is much lower than that of the incident photon. The potential for Compton scattering is always present. The scattered photons are produced in all directions by the scattering object. In some radiation safety applications the exposure to Compton scattered photons can be the significant source of exposure, for example, to medical personnel present during radiological procedures.
7.10.2.2.1.3. **Pair Production.** The last type of photon interaction for discussion is *pair production*, which occurs when a photon collides with (or undergoes an interaction with) the electric field of the nucleus of an atom. The photon completely disappears near the nucleus and its energy is expended in the creation of an electron and a positron. Since the electron and positron each have an energy equivalence of 511 keV, the incoming photon must have an energy greater than 1,022 keV in order for pair production to take place. Any photon energy in excess of 1,022 keV is given to the kinetic energy of the positron and the electron, but it is not always equally divided between them. Pair production is most likely to occur with relatively high photon energies and high atomic number materials. In shielding, spectroscopy, or dosimetry applications, the two 511 keV photons created after pair production must always be considered.

7.10.2.2.2. **Neutron Interactions.** The neutron has no charge and a mass number of 1. Because it has no charge, the neutron has a high penetrating ability in matter. Neutrons are produced in nuclear reactors, accelerators, and other miscellaneous sources called neutron generators. As with photon interactions, neutrons must collide with a massive object, such as the nucleus of an atom (since electrons are only about 1/1,800\(^\text{th}\) the size of a neutron, they cannot slow down or deflect a neutron). The size of the target nucleus and the velocity (i.e., the kinetic energy) of the neutron determine the kind of neutron interaction that occurs. Neutrons are classified by their kinetic energy. This classification is important for two reasons: 1) the interaction of neutrons with the nuclei of atoms differs with the neutron energy, and 2) the methods of producing, detecting, and shielding against the various classes of neutrons are different. The classification used for neutron interaction in tissue is shown in Table 7.10.2.2.2-1 below. Different sources may contain additional categories and slightly different energy levels for each category.

<table>
<thead>
<tr>
<th>Neutron Energies and Sources (<a href="http://ocw.mit.edu">http://ocw.mit.edu</a>)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Classification of Neutrons by Energy</strong></td>
</tr>
<tr>
<td>Thermal</td>
</tr>
<tr>
<td>Epithermal</td>
</tr>
<tr>
<td>Fast</td>
</tr>
<tr>
<td><strong>Neutron sources</strong></td>
</tr>
<tr>
<td>Reactors</td>
</tr>
<tr>
<td>Fusion Reactors</td>
</tr>
<tr>
<td>Large Accelerators</td>
</tr>
</tbody>
</table>

There are four different interactions for neutrons of different kinetic energies: 1) radiative capture with gamma emission, 2) charged particle emission, 3) fission, and 4) neutron scattering.

7.10.2.2.2.1. **Radiative Capture with Gamma Emission.** This is the most common type of interaction for slow neutrons. This interaction often results in product nuclei, which are radioactive.

7.10.2.2.2.2. **Charged Particle Emission.** The second type of general interaction for slow neutrons is the emission of a charged particle after a slow...
neutron is captured by the nucleus. Typical examples included interactions in which a proton, a deuteron, or an alpha particle is ejected from the target nucleus.

7.10.2.2.3. Fission. The third type of neutron-induced nuclear reaction is fission, which occurs after the absorption of a slow neutron by several of the very heavy elements. The nucleus splits into two smaller nuclei, which are the primary fission products or fission fragments. These fission products usually undergo radioactive decay to form secondary fission product nuclei. There are some 30 different ways that fission may take place with the production of about 60 primary fission fragments. These fragments, and the atoms which result from their decay, are called fission products. There are about 400 to 600 fission products, according to the type and number of nucleons their nuclei possess. In a fission reaction, an average of 2 to 3 neutrons is expelled along with associated gamma radiation. Many fission products have found application in medicine, industry, and research. A well-known example is iodine-131, which is used extensively in medicine as both a diagnostic and therapeutic agent.

7.10.2.2.4. Neutron Scattering. Finally, the fourth type of neutron interaction with a nucleus, neutron scattering, occurs when the original free neutron continues to be a free neutron following the interaction. Scattering is the dominant process for fast neutrons when the neutron is moving too fast to become a part of a nucleus. Multiple scattering by a neutron is the mechanism of slowing down or moderating fast neutrons to thermal (lower) energies. This process is sometimes called thermalizing fast neutrons. There are two kinds of scattering: elastic and inelastic. Elastic scattering occurs when a neutron strikes a nucleus, typically of approximately the same mass as that of the neutron. Depending on the size of the nucleus, the neutron can transfer much of its kinetic energy to the nucleus, which recoils off with the energy lost by the neutron. During elastic scattering, no gamma radiation is given off by the nucleus. It can be knocked away from its electrons and, since it is positively charged, cause ionization and excitation. Inelastic scattering occurs when a neutron strikes a large nucleus. The neutron penetrates the nucleus for a short time, transfers energy to a nucleon inside, and then exits with a small decrease in energy. The nucleus is left in an excited state emitting gamma radiation, which can cause ionization and/or excitation.

7.10.3. Radiation Penetration Depths

Radiation penetration depth is a topic that has important implications for the kind of shielding required to stop each type of radiation. Figure 7.10.3-1 shows radiation paths and penetrating ability for alpha particles, beta particles, gamma, x-ray, and neutron radiations. As you can see from the illustration, alpha particles are the most easily stopped and the least penetrating, although their size is the largest. As discussed earlier, their range is short; meaning the thickness of material needed to stop them is small. Beta particles are much smaller but penetrate much farther, and a different shield will be needed to stop them. Gamma rays, x-rays, and neutrons are the most penetrating of all and require a shield different from that of alpha or beta particles.
7.10.4. Measuring Radiation

Note: Unless otherwise noted, the material in Section 7.10.4 comes from http://www.nrc.gov/about-nrc/radiation/health-effects/measuring-radiation.html.

There are four different but interrelated units for measuring radioactivity, radiation exposure, absorbed radiation dose, and dose equivalent. These can be remembered by the mnemonic R-E-A-D, with both common (British, e.g., Ci) and international (metric, e.g., Bq) units in use:

- **Radioactivity** refers to the amount of ionizing radiation released by a material. Whether it emits alpha or beta particles, gamma rays, x-rays, or neutrons, a quantity of radioactive material is expressed in terms of its radioactivity (or simply its activity). This represents how many atoms in the material decay in a given time period. The units of measure for radioactivity are the curie (Ci) and becquerel (Bq) and are summarized in Table 7.10.4-1 below.

- **Exposure** describes the amount of radiation traveling through the air. Many radiation monitors measure exposure. The units for exposure are the roentgen (R) and coulomb/kilogram (C/kg).

- **Absorbed dose** describes the amount of radiation absorbed by an object or person (that is, the amount of energy that radioactive sources deposit in materials through which they pass). The units for absorbed dose are the radiation absorbed dose (rad) and gray (Gy). 1 rad = 0.01 Gy.

- **Dose equivalent** (or effective dose) combines the amount of radiation absorbed and the medical effects of that type of radiation. For beta and gamma radiation, the dose equivalent is the same as the absorbed dose. By contrast, the dose equivalent is larger than the absorbed dose for alpha and neutron radiation.

Figure 7.10.3-1. Radiation Penetration Depths (http://ansnuclearcafe.org/)
because these types of radiation are more damaging to the human body. Units for dose equivalent are the roentgen equivalent man (rem) and sievert (Sv), and biological dose equivalents are commonly measured in 1/1,000th of a rem (known as a millirem or mrem). \( 1 \text{ rem} = 0.01 \text{ Sv} \).

For practical purposes, \( 1 \text{ R (exposure)} = 1 \text{ rad (absorbed dose)} = 1 \text{ rem} \) or 1,000 mrem (dose equivalent).

<table>
<thead>
<tr>
<th>Table 7.10.4-1. Radiation Measurements (<a href="https://orise.orau.gov/reacts/guide/measure.htm">https://orise.orau.gov/reacts/guide/measure.htm</a>)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Common Units</td>
</tr>
<tr>
<td>SI Units</td>
</tr>
<tr>
<td>becquerel (Bq)</td>
</tr>
</tbody>
</table>

### 7.10.5. Sources of Radiation

Radiation is a natural part of our environment and we are exposed to it from all directions: space, the earth, and from inside our bodies from the food and water we consume (Figure 7.10.5-1). This radiation is measured in millirem (mrem). The average dose per person per year is about 620 mrems, but can vary based on where you live, how many hours you spend flying per year (add about 0.5 mrem for every hour of high altitude flying), and how many medical procedures you have each year. For example, if you live at sea level, the average dose is 26 mrem/year from cosmic radiation. If you live at 6,000 – 7,000 feet, then your average dose per year is 66 mrem/year. As for medical procedures, chest x-rays are about 10 mrem and CT scans of your heart are about 2,000 mrem, and each of these will increase your annual exposure to radiation.

**Figure 7.10.5-1. Sources of Radiation**
(http://www.ans.org/pi/resources/dosechart/)
7.10.6. Effects on Cells

The cells in our bodies are comprised of a complex system of molecules which are made up of atoms that are chemically bonded together in specific ratios and sequences. Electrons are the very nature of chemical bonds and are responsible for holding together atoms to form molecules. Ionizing radiation transfers kinetic energy directly to electrons. If the energy transferred to the electron exceeds the binding energy that holds the electron in place, the molecular bond can be broken, thus altering the function of the molecule. This molecular damage can then affect the functioning and/or survival of the cells. The effects of radiation on cells can take two forms: direct and indirect. Each form has a different probability of occurrence.

7.10.6.1. Direct Effects on Cells. To ensure its survival, each new cell must possess a complete and accurate copy of DNA. Damage from radiation can fragment the chromosome and destroy DNA.

It is generally accepted that the DNA strand is the most critical target in the cell, although there may be others. The complexity and importance of DNA to the survival of the cell make it very radiosensitive. Any change or alterations to the genetic blueprint can kill the cell or change the genetic code necessary for cellular activity and reproduction. Therefore, the DNA is considered to be a critical target in the cell. Ionization or excitation of the DNA strand itself is called a direct effect. This ionization can break the chemical bonds that hold the DNA strand molecules together. Once the strand breaks, there is a 95% probability that the DNA will repair itself and continue to function. That leaves a 5% probability that the DNA will not repair normally and will die at the next reproduction phase or survive with an incorrect genetic code which could, in theory, cause a cancer at a future date.

Whether the cell survives depends on how many “hits” (i.e., ionizing events) the DNA strand takes simultaneously. If the DNA strand takes two simultaneous hits (called a double strand break), the cell will die. Cellular repair can occur from a single strand break if the cell is given sufficient time to repair before the next interaction with radiation. It should be noted that single strand breaks to the DNA also occur after exposure to substances other than ionizing radiation, and that the body repairs about 100,000 single strand DNA breaks daily. Double strand breaks, however, are believed to be unique to radiation. These then lead to other chromosome aberrations, such as dicentrics (a chromosome having two centromeres), rings (a chromosome whose arms have fused together to form a ring), and fragments (chromosomes that lack centromeres) (Figure 7.10.6.1-1).
7.10.6.2. Indirect Effects on Cells. Indirect effects are produced by interaction of radiation creating byproducts that are toxic to the cell, causing cell damage or death. Indirect effects are more likely to be produced than direct effects since the DNA is such a small part of the cell. This means that the probability of radiation causing ionization or excitation on the DNA strand (a direct effect) is small also. Since the cell is comprised mostly of water (70 – 80%), the more likely scenario is that the radiation will ionize water molecules. If the water molecule breaks up (dissociates), some of the parts will be charged. These fragments are called free radicals and are very reactive chemically.

Free radicals are electrically neutral structures with one unpaired electron (e\(^-\)). For example, an excited \( \text{H}_2\text{O}^\ast \) molecule may dissociate. The hydrogen radical \( \text{H}^+ \) will have an unpaired electron, and the \( \text{OH}^- \) radical will have nine electrons, one of which will be unpaired. The free radicals are very reactive chemically, and when combining can produce hydrogen peroxide (\( \text{H}_2\text{O}_2 \)), which is chemical poison to the cell and is the most harmful free radical product. Hydrogen peroxide is a somewhat stable compound which can survive long enough to diffuse throughout the body, oxidizing molecules or cells which did not suffer from the original radiation damage. Additionally, \( \text{H}_2\text{O}_2 \) can readily become a peroxide radical with the ability to attach other bio-organic molecules to form stable organic peroxides. For example, if an important cellular enzyme were converted to peroxide, it would no longer be useable at its phase in the cell cycle, resulting in irreparable injury and ultimately cell death.

Not all cells have the same sensitivity to radiation. Generally, radiosensitivity is determined by two factors: rate of mitosis and degree of cell differentiation. A cell’s life cycle is divided into four phases, of which mitosis is the most radiosensitive. Cellular reproduction (mitosis) occurs in the mitosis phase. Mitosis is a process of reproduction where the DNA strands replicate into two exact copies. Most cells in the body undergo mitosis at some point, but the rate or life cycle period is different for different types of cells. The higher the rate of mitosis, the greater the radiosensitivity.

These differences in radiosensitivity are explained by the Law of Bergonie and Tribondeau that states “the radiosensitivity of a tissue is directly proportional to its...”
reproductive capacity and inversely proportional to its degree of differentiation.” In other words, radiation will be most harmful to cells most active in reproducing themselves and cells that are not fully mature. This law is considered to be the basis, with some cells and tissues showing exceptions. Since the time that the Law of Bergonie and Tribondeau was formulated, it is generally accepted that cells tend to be radiosensitive if they:

- Have a high division rate (i.e., short cell cycle time, or short time between divisions).
- Have a high metabolic rate (high oxygen consumption).
- Are of a non-specialized type (i.e., a cell which is capable of specialization into an adult cell type, such as a fertilized ovum).
- Are well nourished (good blood supply)

Since tissues are made up of cells, these differences in cellular radiosensitivity translate into differences in tissue radiosensitivity. Generally, tissues which are young and rapidly growing are the most radiosensitive. This is the reason that human embryos, fetuses, and children are believed to be more sensitive to radiation than adults.

7.10.7.  Acute Doses of Radiation

There are several ways radiation can affect the human body. These effects are usually categorized as either from acute doses of radiation or chronic exposure to radiation. An acute dose of radiation means a large dose in a short period of time (minutes or hours). There is not an accepted limit for a high dose. Most of the time, acute radiation dosage is reported in units of absorbed dose (measured in rad or gray [Gy]), rather than dose equivalent. For biological purposes, significant health effects begin to occur above 100 rad when it is received within seconds, minutes, or hours. For comparison, the yearly dose of cosmic radiation at sea level is only 26 mrem. The period of time between the exposure to radiation and the manifestations of any biological effects resulting from that exposure is called the latency period. The higher the dosage received the shorter the latency period. Acute effects occur within approximately 60 days of the exposure. Delayed effects occur more than 60 days (usually years) after the exposure and are possible following either acute or chronic exposure.

Radiation effects vary greatly between species, and children are much more sensitive than adults. Since there is so much biological variability, the dose that is lethal to 50% of the individuals exposed is used as a benchmark. This dose is called LD50/30, and is defined as an acute dose of radiation expected to cause death within 30 days in 50% of those exposed, without medical treatment. The amount of the body that is exposed will determine how much radiation is an acute dose.

7.10.7.1. Whole Body Exposure. There are three radiation syndromes: 1) Hematopoietic System Syndrome, 2) Gastrointestinal System Syndrome, and 3) the Central Nervous System Syndrome. A syndrome is a
combination of symptoms resulting from a single cause that occur together and that constitute a single clinical picture. The three syndromes listed can occur from acute doses of radiation, depending on the dose received. Of course, since there are differences in radiosensitivity between individuals, there will be variation in the dose ranges at which each of the three syndromes occur.

The hematopoietic and gastrointestinal syndromes can progress through four stages. The first is the prodromal (or initial) stage, which consists of the first set of symptoms that occurs following a sufficiently large acute dose. The symptoms may include nausea, vomiting and diarrhea, as well as loss of appetite and fatigue. The latent stage is an asymptomatic period between the initial stage and the onset of symptoms of later stages. The higher the dose, the shorter the latent stage. At sufficiently high doses the latent stage effectively disappears. The illness stage is the stage where many of the symptoms of the initial stage reoccur along with the additional symptoms of one or more of the three radiation syndromes. The final stage is either recovery or death. Death is likely with an acute dose above 1,000 rad, even with the best of medical care. It is generally believed that, without medical attention, death is certain above 600 rad.

7.10.7.1.1. **Hematopoietic System Syndrome.** The hematopoietic system syndrome involves a decrease in the number of red blood cells (RBC), leukocytes or white blood cells (WBC), and platelets. Doses from 300 to 500 rad can result in death without medical treatment. In this syndrome, the bone marrow (the critical blood-forming tissue) is the tissue that received the acute dose of radiation. Most of the bone marrow is located in the sternum, pelvis, and long bones of the upper leg and upper arm. As you would expect from the discussion on radiosensitivity, the mature RBC and WBCs are somewhat resistant to radiation, but the immature stem cells, precursors to WBCs and RBCs, are very radiosensitive. After an acute dose of radiation, the mature RBCs and WBCs will die from natural causes (age), but there will be no replacement cells, since the radiosensitive stem cells were killed. The body, depleted of its RBCs and WBCs, becomes susceptible to infection and hemorrhaging. Infection is an important cause of death, but may be controlled to a large extent by antibiotic therapy.

Following a dose of 200 – 1,000 rad, the prodromal stage of the hematopoietic system syndrome starts one to five days later with nausea, vomiting, and diarrhea. This is followed by an asymptomatic period of one to three weeks. After this latent stage follows a period of extreme illness. Characteristic symptoms include nausea, vomiting, and diarrhea, fatigue, anemia (brought about by the decrease in the RBC population), fever, epilation (loss of hair), anorexia, and petechial (pinpoint) hemorrhaging on the skin caused by damage to the lining of the capillaries. Finally, death or recovery will be within two to eight weeks of exposure. The most probable cause of death will be hemorrhaging and infection. The hemorrhaging is caused by damage to the radiosensitive cells lining the small blood vessels and is compounded by the reduced population of platelets that are important in blood clotting. The body’s ability to fight infection is reduced by the decrease in the number of WBC.

7.10.7.1.2. **Gastrointestinal System Syndrome.** The gastrointestinal (GI) tract is covered with small finger-like projections called villi, which add to the effective surface area of the lining and thereby increase the capacity of the body to absorb nutrients. The cells on the surface of the villi are constantly migrating towards
the tip of the projections where they are sloughed off. Mitotically-active cells (crypt cells) at the base of the villi replace those that are lost. The turnover rate of these epithelial cells is high—they have an average life span of a few days. Sufficiently large acute doses lead to the reproductive death of the rapidly dividing crypt cells. The cells covering the villi continue to be sloughed off, but are no longer replaced. This deterioration of the lining of the GI tract then leads to a loss of body fluid, inadequate absorption of nutrients, and infection from the intestinal bacteria.

At doses less than 1,000 rad, the crypt cells can recover in about a week. For acute whole-body doses greater than 1,000 rad, the crypt cells will not survive. Surgical replacement of the small intestine is not possible and therefore, survival is impossible. Death occurs within one to two weeks from both the damage to the lining of the GI tract (resulting in circulatory collapse) and damage to the hematopoietic system. A dose of 1,000 rad represents the maximum survivable whole-body dose, provided that extensive medical care is given.

Within a couple hours of exposure, the prodromal stage of the gastrointestinal system syndrome starts with a sharp loss of appetite, upset stomach, and apathy. Several hours later nausea, vomiting, and diarrhea will occur. By the third day after the exposure, the latent phase starts; previous symptoms disappear and the victim will appear healthy. This asymptomatic latent phase will last from one to seven days. This is followed by a period of severe illness which includes nausea, vomiting, diarrhea, fever, apathy, anorexia, and loss of weight. Finally, death or recovery follows. Death, if it occurs, will be within three to twelve days after the exposure. If the cell renewal mechanism of the GI tract has been completely destroyed and cannot be replaced, death is inevitable. The causes of death include fluid and electrolyte losses (circulatory collapse) brought about by the destruction of the lining of the GI tract. These fluid losses also account for the loss of weight, diarrhea, and the thickening of the blood associated with the GI syndrome. Another contributing cause of death is infection. The latter can occur within 24 hours of the exposure as the bacteria that invade the GI tract invade the body across the damaged lining. Damage to the hematopoietic system simultaneously reduced the body’s ability to cope with the infection.

7.10.7.1.3. Central Nervous System Syndrome. The central nervous system (CNS) syndrome is produced by acute whole-body exposures above 2,000 rad; exposure of the head alone may have similar effects. Death results from respiratory failure and/or brain edema (swelling) caused from direct or indirect effects on the central nervous system. Although the CNS syndrome is not well understood, it most likely involves a combination of cellular and vascular damage. In other words, there may be direct damage to the brain cells by the radiation and indirect damage mediated by effects on the blood vessels of the brain. The latter are known to be damaged by such doses of radiation. Fluid from the blood is lost through the damaged vessel walls into the cranial cavity causing an increase in intracranial pressure. Perhaps pressure on certain areas of the brain, such as the respiratory center, may be most important, or it may be the change in the blood supply to the brain.

At these high doses, the individual stages of the CNS syndrome become so short that they cannot be distinguished. Following such a dose, the individual may function coherently for a short while or immediately go into shock. Within hours the symptoms become very severe. Symptoms include vomiting, diarrhea, apathy, disorientation, and tremors. The victim is also likely to fall in to a coma. Death will be due to respiratory failure and/or brain edema and occurs within 30 hours. The hematopoietic and GI
syndromes do not have time to fully manifest themselves before death occurs and generally do not play a role in death.

7.10.7.2. Localized Exposure. The previously mentioned biological effects and radiation syndromes are applicable to acute doses of radiation to the whole body. Localized areas of irradiation will have different manifestations depending on the areas of the body exposed. In general, though, the individual can tolerate a higher dose if it is localized. Four types of effects from localized acute doses are: 1) skin damage, 2) epilation (hair loss), 3) sterility, and 4) cataracts. These biological effects are usually observed after radiation accidents. For example, open beam x-ray diffraction units, a type of analytical x-ray equipment, are quite hazardous if used improperly. “Open beam” means that the researchers using the device can inadvertently put their hands and fingers in the x-ray beam. The x-ray beam is very narrow but very intense (about 400,000 roentgen/min; 350,800 rad/min). Accidents like this usually result in the amputation of one or more fingers.

7.10.7.2.1. Skin. Immediate damage from an acute dose only occurs in the exposed region of the skin. The magnitude of the effect is primarily dependent on the dose. At 300 – 800 rad, erythema, or reddening of the skin will result. The skin will recover, but delayed biological effects, such as skin cancer may develop. At 800 to 1,000 rad, the skin will undergo dry desquamation (a dry scaling from the loss of epidermis). At about 2,000 rad, the skin will also undergo wet desquamation, in which the loss of epidermis is accompanied by ulceration and the seeping of fluid from the damaged area (Figure 7.10.7.2.1-1). Infection can easily occur under these circumstances. At 3,000 rad and greater, there will be necrosis (death) of the skin tissue. Treatment will require skin grafts and/or amputation, if the wound is on an extremity.

Figure 7.10.7.2.1-1. Wet Desquamation
(https://rpop.iaea.org/RPOP/RPoP/Content/InformationFor/HealthProfessionals/5_InterventionalCardiology/skin-injuries.htm)

7.10.7.2.2. Epilation. Acute doses from 300 – 600 rads may lead to temporary hair loss in the area of exposure approximately three weeks after the exposure. Acute doses of 700 rads and above will lead to a permanent hair loss within three weeks.

7.10.7.2.3. Sterility. In men, an acute dose of about 15 rads to the gonads will lead to a brief temporary sterility in many men. An acute dose of 250 rads can lead to sterility for one to two years. Permanent sterility will follow a dose of 350 – 7-211
600 rads. In woman, permanent sterility will follow a dose of 250 – 600 rads to the gonads.

7.10.7.2.4. Cataracts. The lens of the eye is highly susceptible to irreversible damage by radiation. When the cells of the lens become damaged, they lose their transparency and a cataract (opacity of the crystalline structure of the lens) is formed. An acute dose around 200 rad may produce a cataract, but the symptoms and signs may not be apparent for years after the exposure. Neutrons are particularly effective in producing cataracts due to the high water content of the eye, which is particularly effective in stopping neutrons.

7.10.8. Chronic Doses of Radiation

Chronic doses are low doses of radiation delivered over a long period of time, such as weeks or years. This low dose is what we are all exposed to from natural background radiation and what most radiation workers are exposed to on the job during the duration of their careers. While the biological effects of acute doses are fairly well known, the biological effects from low doses of radiation over long periods of time are not yet well understood. There are valid reasons for this. First, there is not a human population group that is not exposed to radiation who could serve as a control group. Secondly, the effects are not generally unique to radiation. Biological effects from chronic doses of radiation can be classified as: 1) somatic (effects to the exposed individual), 2) hereditary (effects to the progeny of the exposed individual), or 3) fetal (effects to the embryo/fetus exposed in utero).

The biological effects from chronic doses, if any, do not manifest themselves until years after the exposure. Other than radiation sickness associated with acute doses, there is no unique disease associated with chronic radiation doses, only a statistical increase in diseases that already occur in the population. The following sections discuss possible effects from chronic doses of ionizing radiation.

7.10.8.1. Genetic Mutations. A mutation is a change in the structure of the DNA, which may involve the base composition, the sequence, or both. An unrepaired alteration involving the substitution, gain, or loss of a single gene can be passed on as cells keep reproducing, perhaps eventually leading to cancer. Ionizing radiation is a known physical carcinogenic agent. There are also chemical carcinogenic agents (cigarettes), as well as biological carcinogenic agents (viruses). The human evidence of radiation induced cancer includes the population of radium dial painters, early radiologist and dentists, uranium miners, and Japanese atomic bomb survivors. The main sites of radiogenic solid tumors are the breast, thyroid, lung and some digestive organs. These tumors have long latent periods (approximately 10 to greater than 30 years) and occur in larger numbers than leukemia cases. The cancer incidence in the population as a whole is about 20%, so radiogenic cancers are difficult to detect against this background, unless there is a fairly large population who has received fairly large doses, the long latent period for the expression of a cancer further obscures the precise relationship between dose and cancer induction.

7.10.8.2. Leukemia. Leukemia is an abnormal increase in leukocytes, or white blood cells (WBCs), and was first noted in early radiologists who used radiation in their practices. The increase of leukemia was much greater for these early
radiologists than other physicians who did not use radiation. Also, atomic bomb survivors within 5,000 feet (1,500 meters) of the blast center showed significantly higher incidence of leukemia than those beyond this distance. Leukemia has a much shorter latent period than solid tumors. In an exposed population, the incidence of leukemia peaks at three to four years after radiation exposure and returns to normal levels after about 25 years. Leukemia induction is also a function of the type of radiation. In Nagasaki, leukemia induction was not seen in individuals with exposure less than 100 rad. In Hiroshima, which had a much greater neutron component in the dose than Nagasaki, leukemia was observed in individuals exposed to 20 to 40 rad.

**7.10.8.3. The Eye.** The lens of the eye is highly susceptible to irreversible damage by radiation. When the cells of the lens become damaged, they lose their transparency and a *cataract* is formed. As mentioned above, an acute dose around 200 rad may produce a cataract; however, the symptoms and signs may not be apparent for years after the exposure. The damaging effects of penetrating radiation to the lens of the eye may be cumulative and repeated small doses may also result in cataract formation. The reason for this is that the lens does not rid itself of damaged cells, as many other tissues do. Radiation induced cataracts are produced primarily by neutron and gamma radiation. Experiments with animals and human case histories indicate that neutron radiation constitutes the greatest danger, with gamma radiation of slightly less importance. It is interesting to note that radiation-induced cataracts differ from naturally occurring cataracts, in that they form on a different position in the lens of the eye.

**7.10.8.4. Inheritability.** Hereditary effects are those that manifest themselves in the offspring of exposed individuals. If a radiation induced mutation occurs in a germ cell (sperm or ovum) or in the tissues of the organ in which the germ cells are produced, no effects would be observable in the individual, but the effect may appear in future generations. Hereditary effects are presumed to occur in humans, but there are no definitive data to substantiate this. Data from the atomic bomb survivors leans in the direction of hereditary effects in humans, but the effect is not statistically significant. Information on hereditary effects is based almost entirely on animal experiments. Experiments with mice have yielded data for radiation induced hereditary effects that can be applied to humans with some measure of confidence.

About 10% of all live births involve some sort of spontaneous mutation ranging from mild to serious. It is impossible to determine whether the changes occurred naturally or if it was the result of exposure to radiation. Radiation does not produce new or unique hereditary effects, but increases the frequencies of mutations that already occur spontaneously in nature. The reasons for the vast majority of spontaneous mutations are not well understood, but it is estimated that not more than 1 to 6% of spontaneous mutations in humans are the result of background radiation. Approximately 99% of all mutations are considered to be undesirable. Hereditary damage in humans can result in a decreased life expectancy, inability to produce offspring, an increased susceptibility to disease, or any number of changes of lesser or greater importance.

**7.10.8.5. Age at Time of Exposure.** As previously discussed, the Law of Bergonie and Tribondeau predicts that the radiosensitivity of tissue is directly proportional to its reproductive capacity and inversely proportional to the degree of
differentiation. It follows that children could be expected to be more radiosensitive than adults, fetuses more radiosensitive than children, and embryos even more radiosensitive than fetuses. Most of the data involving fetal effects comes from the population of atomic bomb survivors, whose offspring (who were in utero at the time of the exposure) show evidence of both small head size and mental retardation. Most children in the study received doses ranging from 1 – 50 rad. The age at which the fetus was exposed was a critical factor in determining the type of effect observed. Any biological effect on the unborn child from radiation exposure depends on the stage of fetal development when exposure occurs. Again, radiation is an example of a physical teratogenic agent (substance that can cause malformation of the fetus), but there are chemical (thalidomide) and biological (German measles virus) teratogenic agents as well.

7.10.9. **High Altitude Effects**

Two populations in the Air Force are exposed to increased amounts of radiation: pilots and astronauts. Galactic cosmic radiation from outside the solar system can have a negative impact on human physiology. An occasional disturbance in the sun’s atmosphere may also lead to a surge in radiation particles. Protection from both sources is provided by the magnetic fields of the sun and the earth and from the earth’s atmosphere. Dose rates are dependent on the altitude, the geomagnetic latitude, and the solar cycle (Bagshaw, 2008).

7.10.9.1. **Risks During High Altitude Flight.** Exposure to galactic cosmic radiation roughly doubles with every 6,000 feet increase in altitude (Bailey, 2000). In recognition of this, in 1990 the International Commission on Radiation Protection (ICRP) identified flight crew, as a group, to be an occupation exposed to radiation and in 1994, the Federal Aviation Administration (FAA) formally recognized air carrier aircrews as being occupationally exposed to ionizing radiation and recommended they be informed about their exposure and potential health risks (Bailey, 2000; FAA, 1994). The FAA recommended limits are the same as those recommended by the International Commission on Radiological Protection (ICRP) (AC 120-61B, 2014). The ICRP’s recommendations for occupational exposure to ionizing radiation, which includes aircrew, are a maximum of 50 mSv in any one year, and a five-year average body effective dose limit of 20 mSv/yr for non-pregnant crew members (Bagshaw, 2008). If pregnant, fetal exposure is limited to 1 mSv during pregnancy and 0.5 mSv in any one month (Radiation; AC 120-61B, 2014). Typically,

Mean ambient equivalent dose rates are consistently reported in the region of 4 – 5 µSv/hour for long-haul pilots and 1 – 3 µSv/hour for short-haul, giving an annual mean effective exposure of the order 2 – 3 mSv for long-haul and 1 – 2 mSv for short-haul pilots (Bagshaw, 2008).

For aircrew that are concerned about exposure, or simply interested in what level of radiation they may be exposed to during flight, the FAA’s Civil Aerospace Medical Institute has developed the CARI-6 computer program that calculates the effective dose of galactic cosmic radiation received by an individual on an aircraft flying the shortest route between any two airports in the world (CARI-6). The program takes into account changes in altitude and geographic location during the course of a flight, as derived
from the flight profile entered by the user (CARI-6). A free copy can be downloaded from the website listed in the CARI-6 reference below.

In addition to the CARI-6 program, the FAA maintains a Solar Radiation Alert system that continuously evaluates measurements from geosynchronous operational environmental satellites of high-energy protons and issues an alert via the National Oceanic and Atmospheric Administration (NOAA) Weather Wire Service if there is a substantial elevation of effective dose rates at aircraft flight altitudes (Copeland et al., 2009). The surface of the earth is divided into 4 regions based on the degree of geomagnetic shielding (Figure 7.10.9.1-1). Due to the earth’s magnetic field providing very little protection from solar radiation, travelers in Region 1 (polar-latitude regions) received the highest dose rates, while Region 4 exposes travelers to the lowest dose rates. Alerts, watches, and warnings can also be found on the NOAA Space Weather Prediction Center’s website listed in the references (SWPC).

![Solar Radiation Alert Regions](http://www.faa.gov/data_research/research/med_humanfac/aeromedical/radiobiology/solarradiation/)

**Figure 7.10.9.1-1. Solar Radiation Alert Regions**

While early studies looking at the cancer risks and incidences among flight crews often were in conflict (Ballard et al., 2000; Diffey & Roscoe, 1990), more recent studies seem to suggest at least a strong correlation, and in some reports significance, between increased risk and exposure in those occupations (i.e. pilots and flight attendants) that routinely fly at high altitudes (> 30,000 ft). In 2008, a review of the current knowledge of cosmic radiation and its applicability to commercial aviation stated the following:

Epidemiological studies of flight crew have not shown conclusive evidence for any increase in cancer mortality or cancer incidence directly attributable to ionizing radiation exposure. Whilst it is accepted that there is no level of radiation exposure below which effects do not occur, all the current evidence indicates that the probability of airline crew or passengers suffering any abnormality or disease as a result of exposure to cosmic radiation is very
low...With respect to the suggestion that cabin crew may be at higher risk of contracting breast cancer...it is very difficult to effectively disentangle the relative contributions of occupational, reproductive, and other factors associated with breast cancer using the data currently available (Bagshaw, 2008).

Twenty years of radiation monitoring in the British Airways Concorde established that flight crew exposure levels were well under the occupational dose limit of 20 mSv/y recommended by the International Commission on Radiological Protection (Bagshaw et al., 1996). Despite this data, a 2015 meta-analysis looking at airline pilots and cabin crew found that the risk of melanoma incidence is approximately twice as high in this occupational group than in the general population (Sanlorenzo et al., 2015). This agrees with the conclusions of earlier studies looking at melanoma and breast cancer risk in female flight attendants (Rafnsson et al., 2001; Buja et al., 2006). While more work is necessary in order to clearly establish a causal relationship in these occupations, awareness and training on the risks and potential hazards, as well as medical monitoring, should be provided all crew members who operate in the high altitude environment.

7.10.9.2. Risks During Space Flight. Unlike air travel that operates within the earth’s atmosphere (even the reduced atmosphere of high altitude flight), astronauts no longer have the benefit of the atmosphere to shield them from radiation. As a result, NASA recognizes that

The most critical risk to humans in space is radiation exposure. Space radiation is qualitatively different from the radiation humans encounter on earth. It consists mainly of ionizing radiation in the form of charged atomic particles traveling at close to the speed of light. On earth, the planet’s magnetic field and thick atmosphere protect humans from this kind of ionizing radiation (NASA, Space Radiation, Part 1).

Highly charged, high-energy particles known as HZE particles pose the greatest risk to humans in space. Although not the most abundant form of ionizing radiation in space, HZE particles, because of their high energy and high charge, can do more damage to human tissue than other forms of space radiation. According to Francis A. Cucinotta, Ph.D., and manager of the Space Radiation Health Project at NASA’s Johnson Space Center in Houston, Texas,

Radiation from HZE particles results in an entirely different pattern of damage to human cells and DNA than do x-rays and gamma rays, the most commonly encountered forms of radiation of Earth. On earth, we know with a fair degree of certainty what the risk is from a given dose of radiation. For space radiation, however, we really don’t know that (NASA, Space Radiation, Part 1).

The potential “late effects” of radiation are a major concern. Cancer is currently considered the most important of the known late effects. Some experimental data also suggest the exposure to HZE particles may cause damage to the central nervous system, although this risk remains poorly understood. According to Cucinotta,
On a 10-day space mission, the risk is so small that if I multiply (the risk by 5) it’s still small. On the other hand, for astronauts who might go on several missions to the international Space Station, or who might be involved in a future mission to Mars, if our estimates of cancer risk are wrong by a factor of 5, the risk to their health would be well above the acceptable level (NASA, Space Radiation, Part 1).

To further complicate the problem, some people have a higher risk from space radiation than others. “Younger people are more susceptible because they have more years of life left for radiation damage to develop,” says Cucinotta. “Women are at greater risk because the breasts and ovaries are among the most radiation-sensitive human tissues and because their life expectancy is longer than men’s” (NASA, Space Radiation, Part 1).

Currently, astronauts’ exposure to radiation is reduced in two ways: 1) space activities can be carefully scheduled to coincide with periods of less intense space radiation, or 2) materials inside a spacecraft and in its structure can be used as shielding to reduce the intensity of radiation exposure.

Aluminum, which is both lightweight and dense, is the most commonly used shielding material. However, research conducted at NASA’s Langley Research Center and by NASA scientists at the Lawrence Berkeley National Laboratory in the late 1980s showed that materials that contain a lot of hydrogen provide the most effective shielding against radiation from HZE particles (NASA, Space Radiation, Part 2).

Experiments done on various Space Shuttle missions showed that Polyethylene was approximately 30 percent more effective than a comparable thickness of aluminum as an absorber of radiation from HZE particles...Among other findings, [additional] experiments showed that the internal organs absorbed about 80% of the radiation [HZE] dose absorbed by the skin. By contrast, when the human body is exposed to x-rays, the radiation dose is significantly weakened within a few millimeters of the skin and the dose absorbed by the organs is minimal (NASA, Space Radiation, Part 2).

The data collected from these experiments will help NASA to more accurately assess the space radiation risks faced by astronauts.

7.10.10. Summary

Radiation comes in various forms ranging from radio waves to UV light to x-rays and gamma rays; and we are exposed to it every day in one form or another. Some radiation is relatively harmless, while other, higher energy radiation can be deadly in high enough doses. The effects of radiation on humans depends on many things--energy of the radiation, is the radiation from charged particles or electrically neutral particles, body part(s) exposed, and the amount of exposure. Some effects can be felt immediately; while others many not manifest themselves for many years. Not all effects of radiation are known or completely understood. Therefore, understanding what radiation is and how it reacts with materials it comes in contact with, will help protect humans and minimize dangerous exposure to radiation.
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8. AEROSPACE PHYSIOLOGIST ACTIVITIES

8.1. Education Theory and Practice

Andrew D. Woodrow, Lt Col, USAF (Ret), BSC

8.1.1. Aerospace Physiology and Human Factors Education

The practice of aerospace physiology and human factors targets human performance and mitigation of error. The means most often employed is education of the operator; whether you are lecturing a topic in physiology for a group of undergraduate student pilots or developing a strategy for sleep-rest schedules for maintainers at a deployed location, the success of the program is linked to an understanding of learning theory and teaching practices. Learning professionals are in the "performance business, not the knowledge-gain business." This position is a role reversal for traditional learning and development organizations like universities and training centers. Instead of focusing on closing knowledge gaps, it pushes learning professionals to provide the tools to support learners when and where they need it.

The philosophy of training should be targeted at three primary points; make the training effective, make it efficient, and make it fun or interesting. There is evidence to suggest that retention of knowledge gained in a training environment is improved when the event is interactive. In a work setting, there is an inordinate amount of training that takes place. On balance when there is an option to deliver lessons via 'live instruction' versus by reading or audio-visual combinations, discussion and practice sessions tend to drive retention levels up above 50-75%. Much of the “training” we receive is delivered with the goal to stay current in familiar topics or learn a new skill. Cermak (2010) suggested that by tying curricula of training more closely to key performance metrics and then measuring its impact on them, organizations can generate greater value from training programs and find useful insights to improve programs consistently. For instance, developing a training program to ensure cockpit procedures are followed is fundamental to any flight operation. Instrument scanning and manual control skills are practiced early in the career to the point of implicitness, and are sustained in the foundation of flight skills. However, a study of cognitive skills that accompany manual flight revealed frequent and serious problems in a group of experienced pilots (Casner, 2014). In the study, several key hand-eye skills and cognitive-based skills were measured to determine proficiency of flight control. In the case of manual navigation, serious errors were observed when pilots failed to periodically reference the charted procedure, assess where they were, and determine what had to happen next in the flight profile. One suggestion from the data is that pilots who habitually turn to task-unrelated thoughts when automation is used may experience greater atrophy in their ability to sort out abnormal events. If the supposition that automation provides more time to concentrate on progress of the flight, then is simple disuse a reason for skills to atrophy? Navigation and troubleshooting instrument system failures is outside the lane of physiology, however the emphasis on practicing cognitive skills during flight or recurrent training is a topic that can be tied securely to the human factors lectures that are part of the aerospace physiology portfolio. Through it all, rules and procedures will never cover novel circumstances not covered during the instruction period. The concept of how we operate smartphones and tablets- not by plowing through thick manuals but by interaction and discovery- is the foundation of learning in
the 21st Century. The expectation is that the learner will take the basic information and apply general rules to a broader set of scenarios. Table 8.1.1-1 provides a comparison between training and instruction and exemplifies the major outcomes of both.

<table>
<thead>
<tr>
<th>Training allows you to…</th>
<th>Instruction allows you to…</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reproduce exactly what has been taught</td>
<td>Generalize beyond what has been taught</td>
</tr>
<tr>
<td>Act automatically</td>
<td>Act thoughtfully</td>
</tr>
<tr>
<td>Apply learning without variation, regardless of conditions</td>
<td>Adapt learning to each new set of conditions</td>
</tr>
</tbody>
</table>

Adapted from Stolovitch, HD in “Telling Ain’t Training”.

To balance the work of the physiologist in practice, there is an element of education that must be considered when approaching a lesson. The term “education” implies a longer term effort derived from life experiences and highly generalized learning principles. The result of the education process is usually measured in mental models and value systems. For instance, one can receive a computer-based training (CBT) segment on the importance of oxygen at altitude that includes oxygen tensions at various altitudes and aircraft-mounted equipment used to provide adequate oxygen. If the lesson ended with the CBT, the aviator may have enough information to defend against hypoxia at altitude. However, if the learning process continued with exposure to a simulated altitude to the extent that the student experiences personal symptoms of hypoxia and undergoes a performance-based measure of recovery with oxygen equipment, the lesson is reinforced. One further step in the education process occurs when the same aviator experiences the same hypoxia symptoms in flight and responds with the techniques taught and practiced during physiology training. There are technologies available that will track and record every key stroke, mouse movement, and interaction with simulation software, but effective training has moved away from ‘constant testing’ and migrated to allowing students the space and time to make errors and learn from their choices within a simulation. The benefits of learner-centered instruction seem to be borne out in the retention of key processes and the critical thinking developed during the training.

Learner-centered teaching is a central theme in aerospace physiology. We live in a time when technology is only as far as a keystroke on the computer. Content-based programs account for a majority of training programs that exist in work centers. The more appropriate style is to focus on the learners with specific needs, concerns, desires, fears, and characteristics. The standardized curriculum of aerospace physiology is designed to aid the instructor in tailoring an educational experience for the class. Information-based programs lead to telling and transmission—emphasis on the instructor. Student-centered programs lead to training and transformation—focus is on the learner.

Education researchers have, for decades, looked carefully at delivery techniques and outcomes of classroom experiences. One project adopted the hypothesis that stories ought to be a central feature of any strategy for helping concrete thinkers to grasp abstract and important ideas. These stories cannot serve merely to amuse or to help people concentrate; they must actually convey philosophic content. To explain what is meant by effective stories, and why they are difficult to find and construct, consider the following example of a story about a well-known philosophical figure that
helps to explain abstract ideas. The illustration acts as a principle for constructing stories well suited to the needs of concrete thinkers.

First, while many entertaining stories can be told of the life of Descartes (Figure 8.1.1-1), one is useful for explaining his fascination with clear and distinct ideas as a path to certain foundations for knowledge. It is the story of a mathematical dream he had as a young man, a dream of a perfect philosophy that had all the certainty of mathematics. To tell this story in the right way is effectively to convey the passion of his philosophy; it is biography and philosophy all at once. Most stories about a thinker’s life are of little philosophical use, however, and this leads to the first principle: biographical stories typically do not convey much philosophic content, as useful as they are for other purposes, and thoughtful research is necessary to construct appropriate stories. In a physiology example, to understand the first exposures to the “extreme” altitudes of balloons in the 18th century, one must first understand the initial purpose of the ascent. The French inventor Coutelle first demonstrated the balloon in 1794. He found that when he was at the end of the cables, he could clearly make out details as much as 18 mi (29 km) away through his telescope. The members of the commission were so impressed that they recommended formation of an air force, the world’s first, called the Compagnie d’Aeronautiers. It was established on March 29, 1794.

Across generations, inventors, scientists, and educators have tried to bridge personal theories of learning and discovery to practical application. On the surface, statements such as René Descartes’ (1596-1650) “I think; therefore I am” and Henry Ford’s (1863-1947) “The hardest thing in the world to do is to think, and that is why people do so little of it” appear trite and difficult to appreciate. A true teacher must do more than recite facts; he/she must build a bridge of facts and philosophies to practical application, effectively taking the step from thought to action. The best teacher is the one who can best kindle enthusiasm by a spark of electrical fire from his/her own soul. Personal experiences are, in many respects, the mortar that holds together the scientific foundation of an aerospace physiologist. Most arrive with a wealth of laboratory or academic experience. The key to success is to harness that knowledge to practical experiences in Air Force operations.

Learning theories aside, there comes a time in the education of aircrew that measurement must take place. One area that has gathered much attention is situational awareness; a critical factor in systems design and assessment of aircrew performance. The objective method of testing whether human factors instruction actually works is to assess reliability and validity. If one is measuring reliability and validity of teaching methods in a human factors lesson, the analogy of the sharp-shooter could be used. The reliability of the marksman refers to the grouping of the shots on the target whereas the validity refers to the closeness of the shots to the center of the target (Stanton and Young, 1999) Otherwise, we can use the terms repeatability and accuracy. The delivery of lessons should aim to improve the aircrews’ accuracy and repeatability in addressing human factors problems in flight. The method of delivering those lessons is the cornerstone of the human factor practitioners. In spite of the development of effective training aids and simulators, the device of instruction most frequently used in aerospace and operational physiology is the lecture. Many education gurus consider the lecture one of the weakest forms of educational methodology;
however, the much criticized lecture method has some significant strength when properly used. For example, to stimulate interest and convey information not otherwise available to the student, it is an economical and effective instrument of teaching. A carefully planned and well-delivered lecture is a platform a good teacher can use to allow students to learn and understand much more than they would through printed media or computer-based training. But it is easy for teachers to reduce their effectiveness by poor enunciation and distracting mannerisms. Many hours of student time have been wasted because the teacher went unprepared to lecture or because the material prepared was not planned to the best interest of the group attending. Professor I.A. Richards of Harvard used this analogy:

You have no stimulation for a person whatsoever if he thinks he understands what you are saying and why you are saying it…but how wide should the gap be between what the student is offered and what he gets? There is, as it were, a piece of elastic between you and your audience; you must not snap the elastic or you have lost them. On the other hand, you must have the biggest possible tension on the elastic and thus must be pulling at them most of the time. That is the whole point of teaching.

If we simply provide “consumer” level information—something easily consumed from color trifolds in the waiting room—then we fail to establish that tension.

The teacher must remember that the perceptual sensory mechanisms—eyes, ears, nervous system, pressure sensors, olfactory senses—are the means through which all our learning is accomplished. Thus, contrived experiences, such as the altitude chamber, Barany chair, parachute hanging harness, and ejection trainers, become very important adjuncts to the classroom methods. In aerospace and operational physiology, videos are also a useful tool but should not be used in place of another, more effective teaching method. A constant evaluation of materials presented (e.g., video, audio, photo) must be made; nothing is more uninspiring than presenting the very same lesson plan to a student five years after the last presentation. A combination of materials may do a better job than a single method, and, again, one may be more effective than another. The instructor must consider both the purpose of the material and the background and experiences of the aircrew in the class.

“What is perceived by the student is fixed in the mind more firmly than what is merely said over a hundred times. It is not the shadows of things but the things themselves that should be presented to the student” (Swett, 1880). From this observation, the instructor needs to, once again, assess the objectives of the lesson and the methods to most effectively convey the message. A well conducted class is choreography of auditory, visual and tactile input bound together with the enthusiasm for the importance of each lesson; not peppered with distractions of materials unrelated to the topic. The constant progress of excellence in teaching across the AOP spectrum is the goal. How will you meet that goal?

8.1.2. Education via Allegories

The balloon corps, or Aerostiers, transported L'Entrepremant to Mauberge, where Coutelle inflated it, and the air corps was ready to face the enemy, or at least to see the enemy. The air corps went into action against the Austrians in June, 1794. During the battle, Coutelle and Conté successfully spied on Dutch and Austrian troops
from high above Mauberge. They provided detailed reports of the location and composition of the Austrian and Dutch troops and directed ground fire against the forces. The Austrians protested that the use of a balloon was against the rules of war and attempted to shoot it down, but Coutelle had his ground crew let out more cable, and L'Entrepreneur easily rose out of range. As balloons soared higher and higher, there were reports from occupants of strange sensations and even visual illusions. It would be some years before the connection between altitude and lack of oxygen was made.

Using stories to support a complex topic may cause the learner to make a lasting connection in the process of memory. To keep an audience’s attention, avoid anything that breaks the student’s involvement in the story; in theatre terms this is the willing suspension of disbelief. Storytelling in films is a technical craft. In the classroom the same principles apply. If you were to use the story of the Aerostiers, you might first capture the attention of the students through visual or audio media. The effect of the altitude on the characters is what the audience follows in the lesson. We enter a character’s life and bridge from the pipe stem oxygen delivery system to modern oxygen systems by identifying something important about the outcome and bring the audience into the story.

Most storytelling has a visual component as well. Images of up-to-date aircraft systems or overviews of current mishaps that highlight the objective of the lesson begin to tap on the memory of the aircrew; how we tie into previous experiences (memory) to reinforce a current lesson is the result of tapping of levels of memory. The three categories of memory are sensory, short term, and long term. The sensory memories act as buffers for stimuli received through the senses. To best understand sensory memory, one should review the neurology of the human sensory systems. A sensory memory exists for each sensory channel: iconic memory for visual stimuli, echoic memory for aural stimuli, and haptic memory for touch. Information is passed from sensory memory into short-term memory by attention, thereby filtering the stimuli to only those that are of interest at a given time. When learning includes sensory information, such as parachute operations from a swing landing trainer, the student picks up on the verbal commands of the instructor along with the tactile input from the open shock during the initial drop from the tower.

8.1.3. Memory

Short-term memory is the wax tablet of memory for temporary recall of the information under process. For instance, a series of numbers related to radio frequencies or headings must be held in your mind from the beginning of the transmission to the end until it is time to act on the information. Short-term memory decays rapidly (200 ms) and also has a limited capacity (Partridge, 1993). A common term in human factors is “chunking,” wherein information is grouped for ease of storing more capacity in smaller bundles. The most common example of chunking is a hyphenated phone number, which is easier to remember than a single long number. Interference often causes disturbance in short-term memory retention. In aviation, if high-frequency radio communication is interrupted by interphone transmission, the receiver may lose some or all of the information presented. This accounts for the desire to complete the tasks held in short-term memory as soon as possible.

Long-term memory is intended for storage of information over a long time. Information from the working memory is transferred to it after a few seconds. Unlike
working memory, there is little decay. There are two types of long-term memory: episodic memory and semantic memory. Episodic memory represents our memory of events and experiences in a serial form. It is from this memory that we can reconstruct the actual events that took place at a given point in our lives. When teaching a topic that is unfamiliar, it may be appropriate to first identify the backgrounds and experiences of the audience to better tailor the presentation. Semantic memory, on the other end, is a structured record of facts, concepts, and skills that we have acquired. The information in semantic memory is derived from that in our own episodic memory, such that we can learn new facts or concepts from our experiences. Again, to understand the process of learning, the instructor must first understand the mechanics of processing new information and retrieving previously stored information.

There are three main activities related to long-term memory: storage, deletion, and retrieval. The most common analogy is the personal computer; however, instructional theory often neglects the impact of environment and motivation to learn—both are relevant and important features for accurate retrieval. Information from short-term memory is stored in long-term memory by rehearsal. The repeated exposure to a stimulus or the rehearsal of a piece of information transfers it into long-term memory.

Experiments also suggest that learning time is most effective if it is distributed over time. In a 2-day physiology course for aviators, time is not a function that can be easily manipulated. Reinforcement of basic concepts related to physiological changes in flight is the best means of distributing information over time: first in the classroom lecture, then in the oxygen equipment lab, and finally in the chamber while at altitude. Deletion of information or knowledge is mainly caused by decay and interference. Emotional factors also affect long-term memory. Most research indicates that we never really forget anything, but rather it becomes increasingly more difficult to access the memory. However, it is debatable whether we actually ever forget anything or whether it becomes increasingly difficult to access certain items from memory. The amount of information stored in any aircraft-mounted system typically exceeds the fluid memory capacity of the human brain, although the actual span of information held in the brain far exceeds any computer chip. It is simply the retrieval that may need prompting from time to time, a function of our attention and motivation to recall.

8.1.4. Retrieval

There are two types of information retrieval: recall and recognition. In recall, the information is reproduced from memory. Recall might be highlighted through the example of read-back of air traffic control (ATC) direction. All pilots are trained to recite a prescribed set of information in a prescribed order to the controller as the approach is made to the airfield; the prompt is not written but verbal from the ATC to the pilot. Association of symbols presented on a multifunction display to meaning for each symbol is an example of recognition. In recognition the presentation of the information provides the knowledge that the information has been seen before. Recognition is of lesser complexity, as the information is provided as a cue. However, the recall can be assisted by the provision of retrieval cues, which enable the subject to quickly access the information in memory.

Learning tends to increase or decrease the effectiveness of impulses that arrive at junctions between neurons. Prior to introducing new concepts, it is very helpful to the student if the instructor can make a connection between common items or preexisting knowledge. Raw-data flying skills could benefit from additional practice and the current
practice of manually operating flight controls in response to flight director (automated) systems probably falls short of keeping instrument scanning skills sharp (Farr, 1987). The initial foundation of mastery in the skills required to scan instruments and manually control the aircraft is critical in the retrieval of encoded information at a later date. Encoding is the first of three stages in the memory process, involving processes associated with receiving or registering stimuli through one or more of the senses and modifying that information. Before an instructor can use a concept like encoding, it is appropriate to review the wiring of the neural network.

The hippocampus receives input from all cortical association areas and serves as the final processing station of complex sensory information. Moreover, it is recognized as a central structure for the functional neuroanatomy of different memory processes. The interaction between the hippocampus and adjacent structures may help in understanding the following example: How might the association between the words “dishtowel” and “locomotive” be stored in neural network models of the hippocampus? First, recognition of the two words activates regions of the temporal lobe language cortex (Figure 8.1.4-1). Patterns of activity then spread into populations of neurons in the entorhinal cortex. Physiological and behavioral evidence suggests that the parahippocampal and entorhinal cortices provide the means for holding information about this event for a period of time (Stern, 1997). The neurons that receive the additional information do not represent the use of the words “dishtowel” and “locomotive” in all contexts but instead represent the specific use of the words in the specific behavioral context. The same neurons will play a role in portions of a wide variety of different memories. These neurons provide the basic code for the episodic memory.

![Figure 8.1.4-1 Brain Regions and Depiction of Neuron Pathway](image)

**Figure 8.1.4-1 Brain Regions and Depiction of Neuron Pathway**

### 8.1.5. Discussion

The effect of prior knowledge on learning cannot be underestimated. Adult learners come to physiology training with a unique set of prior learning sets, some with medical backgrounds, others with engineering, some with no more than high school science. If the instructor treats the learners as if they have little or no experience when they do, the instructor will lose them. It is critical to acknowledge the rich store of experience each has, and exploit it. In the aerospace physiology and human factors classroom, there should always be an objective to the lesson. The objective may be establishing a position on reducing error rates in aviation. The instructor holds the key to making the learning effective, but must understand that each student has a likely position on how to reduce error rates. How might position-driven discussions support
learning human factors? This technique causes the instructor to step away from the podium and actually maneuver the discussion throughout the students. First, position-driven discussions promote active participation before students are fully competent in a domain. In many cases, everyone is expected to commit to a position (and explain why), but it's perfectly fine to build on (or even copy) someone else's reasoning, provided the student states it in his or her own words. The instructor's role is to help students clarify and make explicit their position and the evidence for that position. Taken together, this kind of group discussion provides a great deal of support for students to listen to one another, build on one another's ideas, and take on new "ways with words" in the process. Because the goal is to have a well-developed position, which may or may not be right, students are willing to participate, even those who are most reluctant to participate in other kinds of group discussion. And each student is credited as a "player," a holder of a position or theory, whether or not his or her prediction or explanation wins out in the end. This provides a productive bridge from operational squadron level to the more academic forms of reasoning.

There are, however, some potential difficulties with this kind of group discussion talk format. First, it works most easily with science demos or cases where the question on the table has a definitive answer, one that can be demonstrated or revealed to the students after their discussion and arguments. Ideally, correctness rests in the world (as in the scientific domain), rather than with the instructor. The instructor does NOT evaluate student contributions as right or wrong, as might be more common in other kinds of instructor-guided discussion or recitation. Rather, in position-driven discussions, the instructor typically scaffolds students by "revoicing" their contributions and pushing for clarification, so that everyone has access to everyone else's reasoning (O'Connor, 1998). This a good method to use when discussing aircraft mishap investigations; everyone in the room has a vested interest in nurturing a safe approach to flight, and each crew member has a theory of how to make flight safe. Allowing open discussion and various viewpoints is more important to the learning process than nailing the "right theory" until the end of the discussion, where, for example, the video of an accident scenario is run and students see the actual outcome. At that point, the instructor's role changes, and a focus on correctness, getting the right theory, and actively explaining to the students how to think about the situation takes place.

Secondly, coming up with good framing questions is not easy. A planned discussion requires both a well-designed task and a carefully constructed framing question that will provoke a range of reasonable positions, no one of which is obviously correct. Additionally, the question must be carefully selected and sequenced among other tasks so as to advance the thinking of the group as a whole. It is unreasonable to expect an instructor to develop framing questions for programmed discussions without the support of a rigorous, coherent curriculum that emphasizes student reasoning and inquiry through group discussion. The goal of standardized curriculum is to provide the starting point for this discussion.

A third potential difficulty is that position-driven discussions crucially involve the instructor's active role in orchestrating, eliciting, and scaffolding students' predicting and theorizing. To pull off a "position-driven discussion" so that it engenders productive theorizing and learning, an instructor has to be comfortable with the domain and should know what kinds of assumptions learners are likely to make about the lesson. In guiding the discussion, the teacher must make productive use of students' nontechnical, everyday experiences as well as their observations and experiences of the current problem. Although well-designed and well-sequenced discussions on science topics

8-8
can carry a great deal of "intelligence" about the science under investigation, instructors must know the domain well to facilitate coherent and academically productive position-driven discussions.

8.1.6. Convey the Message

The methods used to convey a message and ultimately teach aircrew about aerospace physiology and human factors will vary based on the environment of your teaching platform. The methods used in aerospace physiology fit into the categories of informal learning and formal teaching; how you design for the delivery method is important. The term informal learning is often confused with a ‘time and place’, not a behavior that needs to be supported. For instance, e-learning in university or government platforms has been associated with the informal domain for decades because of the ‘anytime and anywhere’ flexibility, many learners would argue that it doesn’t meet their definition of informal learning. The method provides a good platform for technical information and easy to digest procedures that can be affirmed through simple on-line testing procedures.

The bridge between foundational understanding of a process or procedure, like teaching egress procedures in undergraduate pilot training, and the experiential execution of the process is the key to successful training. This bridge building begins with how educators are perceived and the deliverables our students feel we build. As a formal training system, we must design and engage flying organizations from an experience-first approach. The message from a platform instructor in Aerospace Physiology, then, must be grounded to information and tools that support experiential learning first, and supported by event-based instruction. Think about how that experiential learning feeds into classroom or e-learning. Put in the context of pilot training, if all the pilot candidates learned exclusively through experiential-based practice a degree of foundational learning is absent. The time in the classroom with instructor-based learning or submersed in lesson reviews provided through e-learning is the foundation that balances experience-only learning with theory and technical discussion. The classroom of the 21st century aviator is a world where experience can be a highly supported and structured experience, measured through technology and other modalities that surround the learner.

Performance support and other innovative approaches should be the dominant tools of our trade, supported by formal instruction that finds itself in a different but still powerful place in the learning landscape. This approach can be applied in high-risk environments and with abstract principles such as leadership and communication; likewise the approach is equally appropriate in the controlled environment of a classroom. The methods require instructors to develop an experience base that is supported by solid science and philosophy; although it is not easy, the process is doable and will absolutely cement the relationship between our audience and the scientific community. Whether you are in the hangar following a sortie, in front of the Wing staff during a safety day, or in a formal school house guiding through standardized curriculum, the impact you make through solid practice of educational theory is the foundation of the career field. Developing the skills to deliver a lesson that is relevant, substantive, and current is one of the most important tools to refine in your arsenal.

Use the research and practice of education professionals to bridge to the communities serviced by Aerospace Physiology.
References


Concepts

Computer-based training (CBT)
Information retrieval

Vocabulary

Episodic memory
Long-term memory
Short-term memory
8.2. High-Altitude Mission Support (HAMS)

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It’s 2045 hours, flying at FL250 over hostile territory, the dimly lit cargo compartment of the MC-130 is a scene of methodical activity. A team of seven Air Force combat controllers, two pararescuemen, two physiology technicians, and the loadmaster perform pre-jump duties. The droning of four 4,400 horsepower turboprop engines drowns out any other sounds in the cargo area, but the communication is practiced hand and body gestures. As the Hercules approaches the drop zone, the navigator and loadmaster make last inputs via intercom to line up the aircraft on the release point. The nine paratroopers disconnect from the oxygen consoles and activate their portable oxygen systems. Chemical lights flicker into view as the jumpers crack the glowsticks taped to their helmets. Each shuffles laboriously forward with nearly 100 pounds of gear hanging from their back, chest and legs toward the large black square of darkness that was once the ramp of the Hercules. The red light beaming on the darkened fuselage is suddenly changed to green, the jumpmaster raises his hand with one final gesture and the nine jumpers step off the ramp, one by one disappearing into the darkness. As the ramp begins to close, the aircraft makes a left bank and change of course back to the airfield. The warriors have been safely delivered to the drop zone, the aircrew have completed another high altitude mission, and the physiology techs are preparing the oxygen equipment for the next sortie. It is 0245 and the crew clocks out for a well deserved crew rest period.

8.2.1. Terminology

Several terms are used when referring to missions that require support from aerospace physiologists and physiology technicians (PTs):

High altitude low opening (HALO) is a term used to refer to all high-altitude airdrop missions that require a jumper to freefall to a lower parachute opening height. Traditionally, all high-altitude airdrop missions including leaflet drops and other cargo drops were referred to as HALO missions. More specifically, HALO procedures consist of parachutists jumping from an altitude above 5,000 ft then delaying parachute deployment until the last safe moment (approximately 2,000 ft). The purpose of HALO operations is to enable jumpers to depart inconspicuous aircraft and descend through the harsh, high-altitude environment and land with minimum exposure to enemy small arms fire. The high-altitude jump also protects the aircraft and crew from ground fire.

High altitude high opening (HAHO) is a term that describes parachute operations where the jumper delays deployment of the chute just 10 – 15 seconds following departure from the aircraft, then uses a high glide ratio parachute to steer to a landing zone. This procedure allows a drop zone and altitude that are well away from enemy activities. The square rigs allow parachutists to steer and glide considerable distances with minimum risk of exposure to themselves and the delivery aircraft to ground fire.
8.2.2. History of High-Altitude Mission Support

The 15th Physiological Training Flight at Kadena Air Base, Japan, began supporting HALO parachutists in 1965. The training missions were typically staged from 13,000 feet to 35,000 feet. In 1965, the U.S. Army’s 7th Psychological Operations Group tasked the 374th Tactical Airlift Wing (TAW) at Clark AB, Philippines, to provide aircraft and crews to support leaflet dropping missions in Viet Nam. The initial missions demonstrated that crewmembers involved with physically strenuous actions of leaflet dropping from unpressurized aircraft at FL250 often suffered physiological problems like hypoxia, hyperventilation, and decompression sickness. The C-130s used in the airdrop missions did not have enough oxygen stations plumbed for the crew and support teams, and the war efforts did not afford enough time to refit airframes with additional oxygen systems. In 1966, the 374th TAW requested assistance from the 15th Physiological Training Flight (PTF), and the oxygen solution was set in motion using newly designed, portable oxygen consoles. The 374th TAW/CC also requested that physiology technicians fly onboard to monitor the use of the new oxygen consoles along with the safety and well being of the crew.

In May 1971, the 15th PTF began regular support of COMMANDO VAULT missions, and in April of 1972, the PTF assumed physiological support of the container delivery system missions in Southeast Asia. Initial support was by temporary rotations from a detachment at Cam Rahn Bay and Tan Son Nhut, Republic of Viet Nam. In the period from 1966 to June of 1972, over 2087 missions were flown with PTs on board. The importance of PT support on these missions was summed up by Brig Gen Kelton Farris, the 374th TAW Commander, when he said,

> The utilization of personnel with years of experience in coping with similar problems experienced in physiological training proved to be a determining factor for mission success. The cases of decompression sickness, hyperventilation, and hypoxia encountered on initial missions were markedly reduced...their quick, expert, and initial treatment has safeguarded the well being of personnel and frequently corrected the difficulties encountered, prevented the abort of missions...we do not fly these missions without physiological training personnel.

Each of the PTs involved with airdrop missions was on flying status and received hostile fire pay, and many received Air Medals for support of the missions.

Aerospace Physiology Training Units (APTUs) in the continental U.S. (CONUS) were involved in HALO support missions starting in 1967. At the time, Tactical Air Command (TAC) was conducting special physiological training for aircrew and jumpers who performed missions above FL180. The training initially consisted of a modified passenger course to cover areas of concern when flying above 18,000 ft. In Europe, high-altitude airdrop support (HAAMS) physiological support requirements began in 1981. The APTU at Wiesbaden Air Base, West Germany, received five nonrated aircrew positions that were designated for HAAMS support. In 1977, the Military Airlift Command (MAC) Coordinator for Aerospace Physiology became the program manager for world-wide HAAMS support. In 1982, HALO became only part of the HAAMS requirement. HAAMS is the term that replaced HALO as a universal term for airdrop mission support. Most recently, in 2006, the term shifted again to high-altitude mission support (HAMS) to reflect a more universal use of PTs to support crew
as well as jumpers. Since much of the information on HAMS was written using the previous acronym, HAAMS, they are used interchangeably in this document. Several conferences were held through the 1980s to better refine the program, and in 1987, Air Force Regulation 50-27 formally designated the MAC Coordinator as the central point of contact for assigning missions to PTs. By 1988, there were eight functional APTUs designated across the MAJCOMs to provide HAAMS teams. The majority of qualified team members were 911X0/4MO with a few 916X/43A3 officers to assist.

In 1995, PTs flew 550 sorties aboard aircraft, including the C-17, C-130, C-141, C-5, and others. They launched from 114 locales, including Pakistan, Australia, Indonesia, Korea, Italy, and airfields throughout the United States. Because of the high demand for qualified PTs, the 1st Airlift Squadron is augmented by aerospace PT units at Shaw AFB, SC; Little Rock AFB, AR.; Andrews AFB, MD; Fairchild AFB, WA; Edwards AFB, CA; and Kadena Air Base, Japan.

In 2006, the School of Aerospace Medicine established a formal training course for newly assigned PTs to HAAMS Unit Type Codes (UTCs). Each team member must maintain currency in physiological training and medical clearance to fly, plus complete a series of training sorties and be proficient in emergency egress procedures for each platform flown.

8.2.3. The Mission

A large number of manned high-altitude jumps are HAHO and equipment drops, along with decades of experience in equipment trials at the parachute freefall school and test parachute center. HAMS for special assignment airlift missions, often top secret and clandestine affairs, carry elite troops from every branch of the service—Army Rangers and Special Forces, Marine recon forces, Navy SEAL teams, and Air Force special tactics units. Jumpers from all services may parachute at altitudes up to 35,000 ft with all of the accompanying hazards. High glide ratio parachutes utilize HALO and HAHO techniques during day and night operations and under all weather conditions.

The Joint Airborne/Air-Transportability Tactical Training planning meeting will project requirements for high-altitude mission support. Once identified, the requirement is coordinated through various channels including the physiological support representative at Air Mobility Command (AMC). The request for support is forwarded through the PT community, and the designated HAMS team then contacts the user group (USAF, Army Special Forces, Navy SEAL Team, etc.) to coordinate special requirements and mission details. One USAF PT is required for each 16 personnel on board the aircraft for all unpressurized high altitude missions conducted at or above 20,000 ft MSL. PT support for high altitude missions below 20,000 ft is by request to further mitigate risks and is highly recommended. Once in place, the HAMS team will set up oxygen equipment and support equipment and prepare for the lift. Close coordination with the aircraft loadmaster is essential to the PT for proper placement of the oxygen equipment and onboard support. When the crew is assembled for mission brief, the PT will conduct a short physiological hazard brief as outlined in AFI 11-409 and review emergency procedures in the event of a physiological reaction in flight. The PT also provides a physiological brief to the jump team prior to boarding the aircraft.

Throughout the flight, the PT monitors the jumpers for hyperventilation, oxygen discipline, and any other physiological reactions. The role on board an aircraft is very similar to that of an inside observer in the altitude chamber. If an airdrop is planned above FL200, the PT must coordinate with the crew to ensure the jumpers and crew
complete the requisite prebreathing below 16,000 ft MSL and prior to the 6-minute
warning preceding aircraft depressurization. Part of the PT’s job is to carefully record
the start time of prebreathing, the take-off time, and the time the cabin crosses through
critical altitudes like 10,000 ft, FL180, and the peak altitude. The PT notifies the crew of
any physiological reactions during flight. Once the jumpers have disconnected from the
console oxygen, the PT checks each jumper’s individual high pressure bottle, the
console, stows the extension hoses, and assists the loadmaster with equipment
storage.

8.2.4. Role of the Physiology Technician

The role of the physiology technician (PT) is to provide an experienced eye for
identification and response to physiological emergencies due to exposure to high
altitude. While on board, the PT is a critical part to ensuring a safe and successful
mission. The wartime task of high-altitude mission support is a niche that is specially
suited to the physiology community.

High-altitude, low-opening (HALO) techniques are used for missions to prevent
detection of the aircraft and the jumpers. Extreme accuracy is required since the
parachutes are deployed at a low altitude. HALO involves paratroopers jumping at
around 25,000 ft and freefalling down to 3,500 ft. Plummeting at a terminal velocity of
122 mph, parachutists can descend this distance within 2 minutes. A HALO jump gets
jumpers out of sight in a hurry, and they are less vulnerable to dangers. A drawback to
this technique is that the jumpers must exit the aircraft over, or close to, enemy territory,
thus making the aircraft a potential target for enemy surface-to-air or air-to-air defenses.

A high-altitude, high-opening (HAHO) jump is used for long-range insertion
operations. HAHO techniques are used for missions that require minimal detection of
the aircraft under conditions that restrict the aircraft from penetrating a certain area,
such as the border of a country. The jumpers will deploy the parachutes at very high
altitudes, which allows them to glide a considerable horizontal distance with a low
probability of detection. Jumpers are consequently exposed to hypoxia and cold
temperatures for extended periods. During high-altitude, high-opening missions, both
exit and deployment altitudes are high, and a special parachute lets them maneuver
more than 50 km as they quietly float into an area. HAHO allows the jump aircraft to
deliver its cargo from a significant standoff range, thereby reducing the odds of enemy
detection and increasing the survivability of the aircraft and the parachutists. The higher
the parachute-opening altitude and the flatter the glide slope of the parachute, the
greater the standoff distance attainable. Special Operations Forces (SOF) “hop and
pop” their chutes immediately, which is potentially a riskier maneuver because jumpers
are exposed to altitude and the enemy for a longer period. The opening shock is also
traumatic. It gives quite a jolt. Jumpers may be sore for a few days after a HAHO.

Given the same size parachutes, a heavier parachutist will descend more rapidly
than a lighter one. This variable rate of descent is not a problem in low-altitude airborne
work; military parachutists traditionally carry their individual combat gear with little
regard for weight considerations. However, that approach does not work in HAHO
operations. Because a HAHO team may travel more than 40 miles under their
canopies, a common rate of descent is a critical factor in keeping the team together. To
ensure the glide slopes are as uniform as possible, the team’s gear is carefully
apportioned so that all the team members weigh about the same—heavier troops jump
with lighter equipment containers and lighter troops jump with heavier containers. The team’s equipment can be redistributed into operational loads after landing.

The most hectic time is from the 2-minute warning until the jump. The team is switching over to their oxygen bottles, and you are double- and triple-checking equipment, connections, and bottle pressure and watching for symptoms and signs of hypoxia.

8.2.5. Hazard vs. Safety

The two greatest hazards they must contend with on HAAMS are hypoxia and decompression sickness. Hypoxia is a major concern during both techniques; there is one documented fatality associated with a high-altitude jump. To compensate for the body’s craving for oxygen, the heart and breathing rate increases. Hypoxia affects people uniquely, and its symptoms will change with age and lifestyle. That’s why all aircrew members are required to go through the altitude chamber periodically. At 10,000 ft, subtle changes take place in the body, and these multiply as you go higher. At 35,000 ft, you’ll have between 30 to 60 seconds of useful consciousness without supplemental oxygen. Ultimately, this leads to death.

Special Operations Forces regulations define the requirements for safe operation and mission completion. For day operations, supplemental oxygen must be used by all parachutists above 10,000 ft mean sea level (MSL) in the aircraft if exposure exceeds 30 minutes. Oxygen is supplied either by inline oxygen or from portable cylinders. If there are extremes in temperature or physical exertion, the jumpmaster can recommend supplemental oxygen at lower altitudes. Supplemental oxygen is used during the parachute descent for any jump between 13,000 ft MSL and 19,999 ft; however, 100% oxygen must be used about 19,999 ft. For night operations, supplemental oxygen is required in the aircraft for all parachutists above 10,000 ft MSL while flying to the drop zone and is encouraged for altitudes above 5,000 ft MSL at the discretion of the jumpmaster. HALO operations may be performed below 13,000 ft MSL once the parachutist has left the aircraft. HAHO operations above 10,000 ft MSL must be performed with supplemental oxygen both in the aircraft and under the parachute canopy. Aircraft oxygen delivery systems must be capable of delivering 100% oxygen and supplemental oxygen settings with a mask that conforms to physiologic PRICE check procedures. Parachute canopy oxygen delivery systems such as a simple oxygen cylinder and mask must maintain the jumper’s oxygen hemoglobin saturation greater than 92%.

The cold is another factor jumpers must contend with. For every 1,000 ft you climb, you lose 3.5°F (2°C) in temperature. In those conditions, knowing the wind-chill factor (a function of ambient temperature and wind speed) is important. A parachutist must have manual dexterity for a few minutes before exiting the aircraft to properly adjust the equipment and immediately after exiting to manipulate the parachute rip cord. The parachutist’s hands would become extremely cold unless over-gloves are pulled on.

As discussed above, any time a military flight drops personnel or cargo at altitudes above 20,000 ft, specially trained aerospace physiology technicians, nicknamed PTs, must fly on board. These technicians, who are experts in the field of human performance and the effects of flight on the body, monitor the aircrew and parachutists, looking for signs of impairment caused by altitude. A physiology tech’s
most critical duty is recognizing and treating those taken ill by the altitude. They tend to the injured until relieved by a flight surgeon.

Physiological technicians work hand-in-hand with the aircraft commander and jumpmaster. They brief aircrews and parachutists on the hazards of high-altitude operations and act as an in-flight oxygen equipment and physiological consultant. Physiology technicians also repair the oxygen equipment, which includes prebreathing consoles and oxygen bottles strapped to the paratroopers. All receive training from equipment manufacturers so they can troubleshoot and repair malfunctions on the spot.

They regulate the ascent to altitude, directing all on board to “prebreathe” 100% oxygen from a console for a half hour while holding the aircraft below 16,000 ft. This interval isn’t to “catch a breather”, but to purge nitrogen from the bloodstream, significantly decreasing the chances of decompression sickness.

Concepts
- Physiology technicians (PTs)
- Role of the PT

Vocabulary
- Aerospace Physiology Training Units (APTU)
- High Altitude, High Opening (HAHO)
- High Altitude, Low Opening (HALO)
8.3. High-Altitude Reconnaissance Mission Support (HARMS)

Andrew D. Woodrow, Lt Col, USAF (Ret), BSC and James D. Denniston, DAF

1933 First full-pressure suit—English firm for American balloonist Mr. Mark Ridge—Suit taken to 17 torr (84,000 ft) pressurized to 36,500 ft (Sears, 1995).

1934 Wiley Post Suit—B.F. Goodrich, full-pressure suit of double-ply rubberized parachute fabric, pigskin gloves, rubber boots, aluminum helmet pressurized to 7 psi, 10 flights before Post’s death in 1935 (Sears, 1995).

1943-46 Henry et al., at the University of Southern California, designed the capstan partial-pressure suit and exposed subjects to 80,000 ft--3 models (Sears, 1995).

1960-70s S901/970—A-12, YF-12A, and SR-71 full-pressure suit with integrated subsystems, parachute harness, automatic flotation system, urine collection device, redundant pressure control and breathing system, thermal protective garment, custom plus 12 sizes, various models; David Clark Company (Sears, 1995).

1960-70s S1010—Special projects full-pressure suit with integrated subsystems including parachute harness, automatic flotation, redundant pressure control and breathing system, thermal protective garment, custom plus 12 sizes, various models used in U-2R; David Clark Company (Sears, 1995).

1970-80s S1030—Upgraded SR-71 and S1031—Upgraded U-2, full-pressure suit, link net with integrated subsystems; David Clark Company (Sears, 1995).

1982 TLSS/ALSS—Tactical Life Support System. Developed by the USAF and Boeing/Gentex et al. to provide get-me-down protection from 60,000 ft. Incorporated many new features for a modular mask, vest anti-G suit ensemble integrated to provide PBG for high-G maneuvers and PBA for altitude with G trousers providing 4 times the breathing pressure from a molecular sieve oxygen concentration system. There are now many variants of similar protective design in the United Kingdom, Canada, Sweden, and an Advanced Oxygen System from France (Sears, 1995).

1987-89 AHAFS—Advanced High-Altitude Flight Suit. High pressure (5-6 psi) full-pressure suit developed for the USAF to increase mobility at higher operating pressures; ILC Dover (Sears, 1995).

1993-94 Partial-pressure suit developed for F-22 aircraft. Get-me-down partial-pressure ensemble combining mask/vest/uniform pressure anti-G garment for protection to 60,000 ft. USAF contractors include Boeing, ILC Dover, META, and Helmets Ltd. (Sears, 1995).


One of the two primary wartime skills supported by aerospace physiology is the High-Altitude Reconnaissance Mission Support (HARMS) program. This function is a highly specialized hybrid of life support and physiology and consists of survival continuation training, full-pressure suit maintenance, survival kit and parachute maintenance, high-altitude chamber flight training, and integration of the pilot to the cockpit. The program has been operating for over 52 years in support of various aircraft.
including the SR-71, WB-57, TR-1, and U-2. Along with daily worldwide flight operation support, the 9th Physiological Support Squadron (PSPTS) at Beale AFB, California, is the USAF Full-Pressure Suit Depot. As such, the depot is the single point of configuration control, maintenance, and procurement of all life support equipment assigned to the U-2. Additionally, all personnel assigned to the 9th PSPTS undergo a 10-month specialized training and certification program through each facet of the operation.

8.3.1. The Platform

Since the original design in 1955, the U-2 (Figure 8.3.1-1) has been a stalwart of the intelligence, surveillance, and reconnaissance (ISR) inventory. The aircraft carries highly sophisticated sensors capable of wide sweeps of terrain. Outside of military reconnaissance, this aircraft has proven useful in mapping forest fires, floods, and during rescue missions at sea. A single-seat, single-engine reconnaissance aircraft capable of altitudes in excess of 70,000 ft, the platform presents one of the most challenging human system integration (HSI) environments in the Air Force. The most recent challenge was the exposure of pilots to high altitudes for long durations. Even with aggressive denitrogenation efforts in place, altitude threats (evolved gas disorders) became an increasing problem. The fix was to reduce cabin altitude to a more benign altitude that would reduce the risk of decompression sickness (DCS). Prior to the Cockpit Altitude Reduction Effort (CARE), U-2 aircraft cabin altitude was normally no higher than 29,000 ft. After the CARE modification, cabin altitude is now reduced to no higher than 15,000 ft. Although the CARE modification has greatly reduced the risks associated with high altitude flight, pilot are still required to wear a full-pressure suit in the event of decompression.

With a 103-ft wing span and no aerial refueling capability, loitering time is one of the biggest advantages this aircraft has to sustain missions of over 10 hours. The aircraft is fitted with a Lockheed-Martin ejection system capable of safe ejection at maximum altitude.

Figure 8.3.1-1. U-2 High-Altitude Reconnaissance Aircraft
8.3.2. The Support Team

Composed primarily of aerospace and operational physiology (4M0) technicians and aircrew flight equipment (1P1) technicians and supported by aerospace and operational physiologists (43A3), the Physiological Support Squadron (PSPTS) technician (sometimes still referred to with the historical terminology of “PSD tech” from when the program was the Physiological Support Division) has considerably different responsibilities from normal duties in a physiology unit or aircrew flight equipment shop. A short summary of the primary responsibilities follows:

8.3.2.1. Depot Level Maintenance. Inspect, overhaul, repair and fit full-pressure suits (FPS) and associated hardware are accomplished at Beale. This includes oxygen regulators, anti-suffocation devices, pressure suit controllers, helmet components, gloves, FPS bladders, urine collection devices and valves, to name a few.

8.3.2.2. Periodic and Preflight Inspections. Parachutes, survival kits, and FPS assemblies are inspected, to include integration of the equipment to the aircraft. Also, a pilot preflight health survey is accomplished to assess the pilot’s basic physiologic condition, including the preflight meal and sleeping habits before each high flight.

8.3.2.3. Integration to the Cockpit. Installation and removal of the parachute and survival kit are completed, and the pilot is integrated with the aircraft. All necessary oxygen systems, ventilation systems, communication systems, and egress systems are correctly connected prior to launch.

8.3.2.4. Specialized Pilot Training. High-altitude chamber flights to FL700 are conducted as part of formal physiological training and as a fit and function check of the suit and related components.

8.3.3. The Mission Sequence

All Physiological Support Squadron (PSPTS) flight support operations include preflight, launch, recovery, and post flight elements. Pilots assigned to fly a mission are assisted by a mobile safety officer (“mobile”), who acts as the back-up pilot on mission day. The day prior to a mission, the FPS and associated equipment undergo preflight inspections. The PSPTS technicians prepare suits for both pilots if the mission is a high priority. The day prior to the mission, the FPS and associated components undergo preflight inspections. On the day of the sortie, the aircraft is loaded with inspected parachute and survival kits approximately 2 hours prior to launch. As part of the cockpit preparation, oxygen systems, ventilation systems, and intercom are checked. Prior to FPS donning, the pilot completes a health screening with one of the PSPTS technicians; this includes a blood pressure and temperature check, and a 12-hour eating, and 24-hour sleep history. Approximately 1.5 hours prior to launch, the pilot dons the FPS; requiring two technicians and a supervisor to complete all integration checks. One hour prior to launch, the pilot begins a denitrogenation period to mitigate evolved gas issues. An optional exercise enhancement prebreathing (EEPB) is available, however; must be accomplished prior to donning the FPS and requires stationary cardio exercise equipment and an additional 15-minutes before FPS donning.
At this point, the FPS is fully integrated with helmet in place and 100% oxygen delivered through the helmet regulator. Additional details about each component are described below.

While the mission pilot is prebreathing oxygen, the mobile is completing aircraft checks and configuring the cockpit for the mission pilot. This task sharing prevents excessive workload on the mission pilot while in the FPS. The mission pilot is transported to the aircraft in a specially designed step van by the three PSPTS technicians. Cooling air is provided to the FPS via a series of vent ducts integrated to the suit; the cooling air is delivered through a liquid oxygen converter. The pilot integration to the cockpit is completed by the technicians and checked by the supervisor prior to canopy closure. The PSPTS technician removes the safety pins from the parachute, prior to canopy closure, leaving the ejection seat (D-ring and secondary), and canopy jettison T-handle pins for the pilot to remove prior to take-off.

The process is reversed during the recovery phase. The PSPTS technicians debrief the mission pilot on any physiological and equipment issues encountered during flight and then complete post-flight inspections on all equipment.

8.3.4. The Equipment

A short description of the key components required to support the high-altitude reconnaissance mission is crucial to understanding the complexity of the operation. Each component undergoes thorough inspection prior to and following every mission. Since the approval of assigning women to combat aircraft in 1989, the U-2 equipment inventory has gone through a few modifications to better serve both genders; however, the principles of physiological support remain the same.

8.3.4.1. Pressure Suits. The protection given by oxygen regulators and oxygen masks is sufficient for the operational and emergency altitudes established for the various types of oxygen delivery systems. The limiting factor for sustained flight at, and above, FL500 is primarily an individual's respiratory and circulatory physiology rather than technology to develop oxygen regulators and masks. As the flight altitude is extended beyond FL430, the necessity for breathing 100% oxygen at increasingly higher pressures becomes critical. The flyer cannot tolerate the elevated breathing pressures for an extended period of time because the normal function of respiration and circulation becomes seriously impaired (Figure 8.3.4.1-1).
Figure 8.3.4.1-1. Time of Useful Consciousness (TUC) with Varying Types of Exposure at High Altitude
A. TUC after rapid decompression on oxygen
B. TUC after turning off oxygen at altitude
C. TUC after rapid decompression on air

The need for pressure suits arises primarily because of the hypoxia threat. A plane may ascend to an altitude above FL500, and the aircrew needs backup protection for the pressurization system. Sustained flight of the U-2 at very high altitudes results in an excessively high cabin altitude because of limitations in pressure ratio and compression temperature.

8.3.4.2. Design Criteria. Pressure suits are designed to embody three primary considerations: protection, mobility, and comfort. The primary protection provided by a pressure suit is the prevention of hypoxia. Pilots breathe 100% oxygen for one hour prior to take-off and throughout the mission eliminating the risk of hypoxia. Pressure breathing is not required while wearing a full pressure suit, because aircraft cabin altitude maintains the pilot at 15,000 ft. and in the event of a decompression, suit pressurization, holds the pilot at a physiological safe altitude of 35,000 ft. Counter-pressure may be achieved by using a pressure suit that applies a mechanical squeeze force on the flyer's body as with a partial pressure suit or by surrounding the body with a pressurized gas envelope as with a full pressure suit.

Some pressure suits are specially designed to guard against thermal extremes encountered in the flying or survival environments, while others may offer minimal protection. Pressure suits do not protect against decompression sickness or the effects of trapped gases in the body. Current pressure suit technology does not provide absolute or total protection without compromising the important design criteria of mobility and comfort.

The aircrew member whose mission demands sustained flight above FL500 is relatively secure in the aircraft pressurized cabin and receives breathing oxygen at a...
very slight positive pressure within a specially constructed helmet. During normal flight, the pressure suit is basically inactive, or depressurized, and the crewmember can move the body, arms, and legs with comparative ease. If the cabin decompresses, the flyer is immediately subjected to all of the environmental stresses of the ambient pressure altitude. However, when exposed to pressures at altitudes greater than FL350, the pressure suit assembly pressurizes almost instantaneously using the principles of Boyle’s Law, and the crewmember is again safe.

Freedom of movement is moderately or severely impaired by the pressurized suit assembly. The degree of mobility the flyer has depends upon the type of pressure suit assembly in use and the pressure altitude to which it is exposed. Proper suit size, adequate sizing adjustments, and equipment familiarity through training are important factors that contribute to optimum mobility required to perform flight duties. Finite movements of the arms and gloved hands are difficult, even for the experienced flyer. This is due to the rigidity or constraining force of the protection it must provide.

Comfort of the flyer is of primary importance when considering the design and use of any item of protective equipment. Pressure suit assemblies pose some special problems concerning thermal stress, body water balance, impaired circulation, fatigue, cramps, and general discomfort. To address the build-up of heat inside the current full pressure suit, cool air is passed throughout the suit via a ventilation “tree.” Vent air enters the pressure suit at the vent controller, commonly called the “T-Block,” (Figure 8.3.4.2-1) on the left abdomen area. From there it is ducted through the ventilation, or vent, tree to the extremities, back, and neck area where it is allowed to enter the suit (Figure 8.3.4.2-2). Since the only exit for the vent air is at the suit controller, on the right abdomen, the cool air flows through the suit picking up body heat and some moisture and carrying it out of the suit as it exits. The temperature and volume of the ventilation air can be controlled by cockpit vent and engine settings and the volume can be also be controlled by the T-Block.

Figure 8.3.4.2-1. T-Block (left) and Aircraft Vent Hose (right)
8.3.5. Types of Pressure Suits

The two basic types of pressure suits are the partial-pressure suit and full-pressure suit. The former applies counter-pressure directly to the body surface by means of mechanical squeeze and the latter by surrounding the body with an envelope of air or oxygen.

The earlier designs of pressure suit assemblies that applied mechanical counter-pressure directly to the surface of the body (figure 8.3.5.1-1) were subjectively less restricting to movement but caused superficial skin discomfort due to the pressure or pinching exerted by seams and adjustment lacements. When pressurized for long periods of time, these assemblies also reduced peripheral circulation of blood, caused tingling sensations on the body, and contributed to muscle cramps. Modifications have partially corrected these problems.

Pressure suits that cover the entire body in a virtually airtight enclosure (Figure 8.3.5.1-2) cause an accumulation of body heat and profuse sweating unless some method of cooling is provided as described above. Fluids for drinking are obtained by inserting tubes through a specially designed orifice in the helmet visor or shell, thereby maintaining body hydration.

8.3.5.1. Partial-Pressure Suits. The development of this series of suits began in 1947, and they are called partial-pressure suits because counter-pressure is applied only to the legs, torso, arms, and hands. The helmet, which seals around the neck of the user, provides breathing oxygen and pneumatic counter-pressure surrounding the entire head (Figure 8.3.5.1-1).
Figure 8.3.5.1-1. Partial Pressure Suit

The partial-pressure suit is virtually form-fitting and applies direct counter-pressure when the aircraft cabin altitude exceeds FL400. The variable squeeze effect is produced by means of inflatable capstans, which extend down the back and along the arms and legs. These tubes are attached to the suit by means of crossing tapes. The diameter of the capstan is approximately one-fifth the diameter of the area it protects. Therefore, the pressure introduced into the capstan must be five times the desired resultant pressure on the body. The objective is to supply the amount of counter-pressure that will just balance the breathing pressure necessary to prevent hypoxia at a given altitude.

For example, breathing pressure of 100 mmHg, or approximately 2 psi, requires balanced counter-pressure of 2 psi, obtained by applying a pressure of 10 psi to the capstan. Capstan pressures and breathing oxygen are delivered to the suit by means of a dual function oxygen regulator located in the seat kit of the aircraft. Partial-pressure suits are in limited use.

8.3.5.2. Full-Pressure Suits. The first operational full-pressure suit was produced for the U.S. Navy during the 1950s and has been succeeded by a number of other specially designed models. The full-pressure suit, helmet, and gloves surround the body with a pressurized gas envelope to provide counter-pressure usually when the aircraft cabin altitude exceeds FL350 (Figure 8.3.5.2-1).
Since the flyer is completely within the suit assembly, counter-pressure and oxygen breathing pressure are metered at a ratio of approximately 1:1. Full-pressure suits are unpressurized until the cabin altitude exceeds FL350. When the cabin altitude exceeds FL350, aneroids within a suit-mounted controller sense the decreasing pressure, expand, closing off vent ports which automatically causes the suit to inflate to a given pressure (Boyles Law) which, when added to the ambient pressure at that altitude, equals about 3.4 psi (FL350 equivalent). Additional air pressure into the suit is not required for it to provide full protection. Therefore, a flyer wearing a full-pressure suit is never exposed to a pressure altitude greater than FL350, regardless of aircraft altitude.

The following discussion provides a brief description of the components associated with the Full Pressure Suit.

**8.3.5.2.1. Special Modified Underwear.** Each U-2 aircrew member is supplied modified long cotton and Capilene underwear, modified jockey briefs, thin nylon or cotton glove inserts, and wool socks. The underwear modification consists of a hole cut out in the front of the briefs and drawers to accompany a urine collection device/system (UCD/UCS). The hole is surrounded by the soft part of Velcro® for males and an elastic material for females. The Velcro® holds the UCD in place, while the UCS pad uses a thin double-sided sticky tape to secure it to the female briefs. If a UCD/UCS is not worn, then a patch is attached over the hole of the men’s briefs/drawers.
8.3.5.2.2. Urine Collection Device (UCD) and Urine Collection System (UCS). The duration of U-2 missions requires the flyer to be able to void urine while in the FPS assembly. The urine collection device (UCD) is effectively an external catheter (Figure 8.3.5.2.2-1) while the urine collection system (UCS) has the form of a maxi-pad with a drainage tube that connects to the external valve assembly on the FPS leg (Figure 8.3.5.2.2-2). It contains thermo-sensitive gel that molds to the body to help improve the seal and reduce leaks. The UCD has a cone-shaped portion that is trimmed for size by the aircrew member to fit comfortably over the penis. It is then held in place by the modified underwear. There is a hole positioned on the top of the UCD to allow air from a slightly pressurized pressure suit to enter and then pass out the front end of the UCD through a tube. It then passes through the pressure suit at the UCD relief valve and into an external tube, which leads to a urine collection tank or a urine collection sponge device known as a pilot relief bag (a.k.a. piddle pack). The UCD is washed and maintained by the aircrew member at the end of each mission.

The female UCS is fabricated with Poise Pads (feminine product designed to protect against bladder leaks). It utilizes a unique “Blue-Loc Core” that helps lock away wetness for maximum protection. The absorbent material is a Superabsorbent Polymers (SAPs) also called slush powder that absorbs liquid to become a semi-solid gel. SAPs can absorb and retain extremely large amounts of a liquid relative to their own mass. Body temperature is not a factor. The UCS is designed so that most of the urine passes through the spacer layer (mesh stand-off layer), directly above the urine hose (target area). The material surrounding this target area is there to absorb and retain potential leakage. When the pad fills to capacity, the pilot opens a relief valve on the outer leg of the suit allowing fluids to exit through a tube into the aircraft urine collection tank or sponge filled pilot relief bag. The UCS is a single-use item.
8.3.5.2.3. **Full-Pressure Suit Assembly.** The full-pressure suit assembly comes in 13 sizes and can be fine-tuned through lace adjustments panels on the legs, arms, and torso. Each aircrew member is issued two pressure suit assemblies, called an S1034 coverall, but only one helmet. This assembly consists of a comfort liner layer that is held in place by Velcro®, a three-ply Gore® gas container to hold pressure, panels of mesh link net that surrounds the gas container to provide shape/conformity and as a platform for size adjustments and an outer cover made of a treated fire retardant material called Nomex®. The comfort liner and outer cover layers are periodically removed and washed. The pilot’s primary suit undergoes a 25-minute preflight check, and the back-up suit is given a cursory inspection prior to each high flight. A periodic inspection is performed every 120 days, or 125 flight hours whichever comes first.

9 PSPTS personnel perform three primary inspections of the FPS: a Periodic, Annual, and 40-month inspections. The Periodic inspection is extensive and takes about 4 hours to complete. The annual inspection covers a number of addition areas and may be included in conjunction with a Periodic or 40-month inspection. Each suit is overhauled during the 40-month inspection that entails an extensive breakdown and cleaning of the suit, a detailed visual inspection, replacement of worn and time sensitive parts; seals, diaphragms, and various pressure structural and leak tests. Maintaining the neck ring can be quite time consuming due to the approximately 78 ball bearings and 78 ceramic spacers that must be removed, cleaned, and reinserted one at a time. The 40-month overhaul requires about 8 man-hours to complete.

8.3.5.2.4. **S1034E Helmet Assy.** Each crew member is issued one helmet (Figure 8.3.5.2.4-1). Several additional helmets are maintained and periodically inspected to serve as back-ups in case the aircrew’s helmet has a mechanical problem during the suit up. All the helmets are the same size. Sizing is accomplished by different thickness helmet liners. The helmet is a fairly complex portion of the pressure suit system that requires considerable maintenance. Individual items that require a fair amount of maintenance and care include the exhalation valve; the anti-suffocation valve; and the dual oxygen regulator (lower right picture in Figure 8.3.5.2.4-1), which is located in the back of the helmet. Recalibrations of various functions of the dual oxygen regulator and suit pressure controller are also necessary and performed in our Oxygen Control Systems shop. The overhaul of these two pieces of hardware is very delicate work and requires highly trained and skilled PSD technician. Maintaining the correct adjustment of the bailor bar (visor lock down lever) is also critical. The visor must be
kept scrupulously clean. The microphone and ear phones in the helmet liner are notorious sources of problems and must be carefully maintained and inspected.

Figure 8.3.5.2.4-1. S1034E Helmet Assembly

**8.3.5.2.5. Gloves.** Each crew member is issued two pairs of gloves (Figure 8.3.5.2.5-1). Gloves are constructed by PSD personnel in the Suit Depot Element and take approximately 30 minutes to assemble each glove. Before being placed into service, each glove is tested for both structural integrity and to ensure minimal allowable leakage.

Figure 8.3.5.2.5-1. Full Pressure Suit Glove
8.3.5.2.6. **Torso Harness.** Each aircrew member is issued two retainer torso harnesses. This harness contains the support webbing and parachute Koch fittings to attach the crew member to the 35-ft canopy parachute. It also contains the automatic personal flotation device. As part of the FPS inspections, the harness also undergoes Periodic and 40-month inspections.

![Torso Harness](image1)

**Figure 8.3.5.2.6-1. Torso Harness**

8.3.5.2.7. **Boots and Spurs.** Each crew member is issued a pair of insulated boots two sizes larger than normal. This enables the pressure suit bootie to inflate in the boot. A set of spurs is attached to the heel of the boot during the dressing process and secured by nylon straps and Velcro (Figure 8.3.5.2.7-1). The spurs are then attached to a ball, spring and cable device attached to the ejection seat that retracts the heels in upon ejection to prevent leg flailing during bailout. These items take very little maintenance and are replaced when worn out.

![Boot with Spur Attached](image2)

**Figure 8.3.5.2.7-1. Boot with Spur Attached**
8.3.5.2.8. **Tube Food and Drinking Bottles.** In cooperation with the Army’s Natick Food Laboratory, pilots are provided 18 food varieties to consume during flight. The caloric intake is made through a feeding port in the helmet to prevent breaking the seal on the visor. This feeding port is automatically closed when not in use. When food or drink is required, the “tube food” with a feeding “pon tube” attached or a water bottle filled with various beverages is inserted into the feeding port (Figure 8.3.5.2.8-1). The specific food manufacture date must be monitored to ensure freshness, avoid potential spoilage and prevent aircrew from becoming ill. The aircrew's food and drink requirements have to be coordinated prior to every flight. After flight, the water bottles are washed and sterilized to be used again.

![Figure 8.3.5.2.8-1. Tube Food and Drinking Bottles](image)

8.3.5.2.9. **Survival Kit.** The survival kit used in the U-2 is unique. In addition to the standard components of a life raft and survival items, the kit houses two 2,200-psi, 45-in$^3$ oxygen cylinders (Figure 8.3.5.2.9-1). These provide oxygen during bailout or when required to supplement a malfunctioning aircraft oxygen system. The cylinders provide about 30 minutes of oxygen if there is no significant leak in the suit.

![Figure 8.3.5.2.9-1. Survival Kit and Oxygen System](image)
8.3.5.2.10. **Parachute.** The parachute used in the U-2 is the RQ-225 parachute (Figure 8.3.5.2.10-1). It is a 35 gore, 35 foot diameter backstyle chute rather than the 28 foot chute found on other aircraft to accommodate the additional weight of the FPS and survival kit. To increase the parachute opening speed and reliability, the chute has an integrated ballistic drogue gun designed to extract the main canopy from the pack during man-seat separation. The larger chute is designed to slow the descent rate and compensate for the additional weight of the FPS and seat kit. It will also sustain deployment at FL350, an altitude which would typically destroy typical aircraft emergency chutes. Also imbedded in the parachute pack is the minimum survival kit and the beacon. The parachute pack and harness are inspected daily prior to being used in flight. This daily inspection takes about 10 minutes. PSPTS personnel also perform a 30-day inspection involving a more in-depth examination. The parachute is installed and removed from the cockpit daily by PSPTS personnel. Parachute repack and repairs are accomplished by the local parachute fabrication shop.

![Figure 8.3.5.2.10-1. RQ 225 Parachute](image)

8.3.5.2.11. **Liquid Oxygen Ventilator.** The liquid oxygen (LOX) ventilator (a.k.a. cooler) is used as a portable source of 100% breathing oxygen and cool ventilation air (Figure 8.3.5.2.11-1). It weighs 35 pounds when full of the 2.5 L of LOX. PSPTS personnel perform all servicing, calibration and maintenance of these ventilators on a regular basis. The center white cylinder is an insulated storage container that holds the LOX. As the liquid warms it expands and changes into a gas. This gas continues traveling through the aluminum tubing until it meets a check valve at the top of the ventilator. An oxygen hose mates to the check valve and the helmet hoses to supply oxygen to the regulator in the back of the helmet and eventually as a breathable gas for the pilot.
8.3.6.  Training for the Dragon Lady (U-2) Pilot

The 9th PSPTS conducts physiological training for U-2 pilots in parallel with standard AFI 11-403 requirements. Due to the unique nature of the mission and hostile nature of the high-altitude environment, the curriculum is adapted to the needs of the pilot. From acceptance to the U-2 program, the pilot enters a series of specialized training programs. All SERE training (water and land survival) is re-accomplished locally and tailored to the wear of the full-pressure suit.

The altitude chamber training is outlined in ACCI 11-459 and includes a chamber flight to FL750 and rapid decompression from FL295 to FL630. The profile includes a hypoxia experience, fit and function checks of the FPS, and emergency procedures (BOLDFACE) check while in the chamber. The training is conducted one-on-one with the pilot integrated to an ejection seat mock-up and realistic oxygen and survival kit configuration.

Technicians assigned to the 9th PSPTS also undergo FPS training. If qualified, the technician will complete the same phase of FPS orientation that the pilot does in an effort to provide the qualified technician a solid foundation of the physiological stresses of being inside a FPS.

References

Recommended Reading
8.4. Safety and Accident Investigation

Donald J. White, Col, USAF (Ret), BSC; Dwayne Porter, Lt Col, USAF, BSC; and Andrew D. Woodrow, Lt Col, USAF (Ret), BSC

On February 12, 2009, about 2217 Eastern Standard Time, a Colgan Air, Inc., Bombardier DHC-8-400, N200WQ, operating as Continental Connection Flt 3407, was on an instrument approach to Buffalo-Niagara International Airport, Buffalo, New York, when it crashed into a residence in Clarence Center, NY, about 5 nautical miles northeast of the airport. The 2 pilots, 2 flight attendants, and 45 passengers aboard the airplane were killed, one person on the ground was killed, and the airplane was destroyed by impact forces and a post-crash fire. The flight was operating under the provisions of 14 Code of Federal Regulations Part 121. Night visual meteorological conditions prevailed at the time of the accident. (NTSB, NTSB/AAR-10-01, 2010).

The National Transportation Safety Board (NTSB) determined that the probable cause of this accident was the captain’s inappropriate response to the activation of the stick shaker, which led to an aerodynamic stall from which the airplane did not recover. From the human factors stand point, the investigation highlighted three critical elements: flight crew monitoring failures, pilot professionalism, and fatigue.

8.4.1. The Investigation Process

Investigations of aircraft accidents can be as simple as reviewing procedures that lead to a violation of air traffic rules and as complicated as the tragedy of Continental Connection Flight 3407. In all cases, the fundamental purpose of investigating accidents, from both the civil and military perspective, is to determine the circumstances and causes of the accident with the aim of preservation of life and the avoidance of accidents in the future; it is not to apportion blame or liability. In 1911, the first aviation legislation was enacted in the United Kingdom. This legislation took the form of the Aerial Navigation Act (AAIB, 2015). This legislation was primarily directed at ensuring the safety of people on the ground that might be struck by uncontrolled or otherwise descending aircraft. Clearly, the hazard to the earthbound public was officially regarded as of primary importance and was of public concern, while the welfare of the limited numbers of aviators and even fewer passengers was regarded as a distinctly secondary consideration. Since those early days of aviation and accident investigations a key to effective investigations has been rooted to the independent investigations of all aviation accidents and major accidents in the other modes of transportation.

Even before an investigation of an aircraft accident begins, the parties involved with the process must be aware of the hazards of aircraft wreckage sites. In the case of two USAF F-15C aircraft that crashed in the Cairngorm Mountains in Scotland on 26 Mar 2001, the first hazard to overcome for the investigation was the record-breaking snow and white-out conditions at the crash site; so bad were the conditions that the team was not able to reach the site for several days, and only after specially trained mountain rescue teams had provided the “all-clear” (BBC News, 2001). Investigators may be exposed to certain risks, including biohazards, airborne hazards, adverse
terrain and adverse climatic conditions. Typically, the safety team will coordinate with the local Incident Commander (local police, National Guard, or fire and rescue), if present, to determine hazards at the accident site and safety resources available to the investigative team. Personnel involved in the recovery examination, and documentation of wreckage are typically on-scene prior to the investigators and may be exposed to physical hazards from such things as hazardous cargo, flammable or toxic materials and vapors, sharp or heavy objects, pressurized equipment, and disease.

8.4.2. The National Transportation Safety Board and Other Investigation Agencies

The National Transportation Safety Board (NTSB) has a unique organizational relationship in mishap investigations in that it is not part of the Department of Transportation (DoT), nor organizationally affiliated with any of DoT’s modal agencies, including the Federal Aviation Administration. Additionally, the Safety Board has no regulatory or enforcement powers. To ensure that Safety Board investigations focus only on improving transportation safety, the Board’s analysis of factual information and its determination of probable cause cannot be entered as evidence in a court of law.

For civilian investigations, Title 49 CFR 845.50 states that all factual information, proposed findings that parties have submitted (that is, submissions), petitions for reconsideration, and the Investigation Board’s rulings will be placed in the public docket, which will be made available to the public (e-CFR, 2015). The investigating officer is responsible for assembling the public docket and retains the original copy of all docket material for one year from the date that the accident report is adopted.

The Air Accidents Investigation Branch (AAIB) (UK) is chartered to “To improve aviation safety by determining the causes of air accidents and serious incidents and making safety recommendations intended to prevent recurrence...It is not to apportion blame or liability” (AAIB, 2015). Very much like other federal aircraft accident investigation agencies, the AAIB is guided by conventions and guidelines that regulate the industry. One universal element to every safety investigation is the recommendation that result from the analysis; possibly the most important part of the safety investigation team’s mandate. The board must address safety deficiencies immediately, and therefore often issues recommendations before the completion of investigations. Recommendations are based on findings of the investigation, and may address deficiencies that do not pertain directly to what is ultimately determined to be the cause of the accident.

8.4.3. The “Go-Team”

At the core of any accident investigation is the ability to respond to an accident scene as soon after the event as possible. The NTSB investigations begin with the “Go Team.” The purpose of the safety board “Go Team” is simple and effective: begin the investigation of a major accident at the accident scene, as quickly as possible, assembling the broad spectrum of technical expertise that is needed to solve complex transportation safety problems (NTSB, 2015). The team can number from three or four to more than a dozen specialists from the Board’s headquarters staff in Washington, D.C., who are assigned on a rotational basis to respond as quickly as possible to the scene of the accident. In many instances, Go Teams travel by commercial airliner or government aircraft depending on circumstances and availability. Because of the depth
of experience, the NTSB has been winging teams to catastrophic airline crash sites for more than 35 years. They also routinely handle investigations of certain rail, highway, marine and pipeline accidents. The U.S. Air Force does not manage a Go Team in the same sense as other federal agencies. Instead, Major Command (MAJCOM) Safety Offices maintain a list of key consultants and qualified investigators that can be called upon at any time. Typically, each MAJCOM manages their own mishap investigation but does not recruit investigating team members from the organization involved in the accident. The remaining portion of this chapter will focus, primarily on the U.S. Air Force Accident Investigation process.

8.4.4. Applied Science in Investigations

Developing a theory of why an accident occurred must include more than a reconstruction of the events leading up to the accident and an analysis of crash dynamics. The human behavior alongside the actions is as critical as understanding the design and mission plan. Were the errors investigated the result of the individual or the design? The raft of human factors such as inaccurate perception, wrong expectancies, and hazardous activities stemming from deliberate behavior (to name a few) are specific to an investigation that must also consider inadequate design, deficient instructions/training, or improper integration with other systems. Studies have revealed that 70 to 80% of aviation accidents can be attributed at least in part to human error. A published study from the Directorate General Civil of Aviation (DGCA), India, reveals that 77.1% of the accidents studied had one or more human factors contributing to the accident causation (Gaur, 2005). The use of the categorization system like the Human Factors Analysis Classification System (HFACS) has a clear hierarchical structure, and has been shown to have reasonable levels of reliability for aviation mishap classification when the responses of pairs of well-trained experts have been compared. Reliably classifying the cause of a mishap is equally, if not more important, than reliably rejecting potential mishap causal factors. The DoD investigation process further refined the original HFACS model with the purpose of providing a common human error categorization system to accurately capture the human factors causes of all DoD mishaps (O’Connor, 2008). In many accidents, the role of the aviation human factors consultant and human factors forensics expert is paramount to explaining human behavior within a system…ultimately answering the question “Why were there errors made?” Evaluation of what went wrong must be explained by experts in the field of human factors and be grounded on current understanding of field studies and empirical data.

8.4.5. The Air Force Safety Organization

The Air Force Safety Center operates as a Field Operating Agency at Kirtland AFB, NM, under the Air Force Chief of Safety and Air Force Chief of Staff. It is organized into five main safety divisions including Aviation, Ground, Weapons, Space, and Training, whose primary mission is to prevent mishaps and preserve combat capability. Safety Center personnel engage a variety of tools to accomplish this mission such as proactive safety initiatives, risk management programs, strengthening safety culture and compliance, managing Air Force safety guidance and publications, educating DoD and international safety professionals, and investigating mishaps. A multi-disciplinary team of experts is required to address complex safety concerns and
the Safety Center relies on aerospace physiologists, flight surgeons, pilots, flight equipment specialists, engineers, bird and wildlife experts, psychologists, legal representatives, ground and weapons personnel, and many others to accomplish its mission.

8.4.6. **Mishap Classification**

Air Force mishaps are categorized according to total direct cost and/or severity of injury/illness. AFI 91-204, *Safety Investigations and Reports*, describes the following classifications:

- **Class A**: Mishap cost of $2 million or more, and/or a fatality or permanent disability, and/or aircraft destruction.
- **Class B**: Mishap cost of $500,000 or more but less than $2 million, and/or permanent partial disability.
- **Class C**: Mishap cost of $50,000 or more but less than $500,000, and/or injury/illness causing loss of work days or permanent change of job.
- **Class D**: Mishap cost of $20,000 or more but less than $50,000, and/or injury/illness not otherwise classified as Class A, B, or C.
- **Class E**: Events which do not meet reportable mishap classification criteria, but are deemed important to report for mishap prevention (such as physiological events).

Following a mishap, a Safety Investigation Board (SIB) may be convened IAW AFI 91-204. Many SIBs are preceded by an Interim Safety Board which responds immediately to initiate the investigative process and collect and preserve mishap site evidence. Safety investigations are conducted solely for the purpose of mishap prevention, and as such, all SIB findings and recommendations are privileged (not releasable to the public) and may not be used for any disciplinary actions. Following the SIB, an Accident Investigation Board (AIB) may be convened IAW AFI 51-503, *Aerospace Accident Investigations*, to produce a publicly releasable report of the facts surrounding a mishap. The Air Force Safety Center is primarily involved in the SIB process in its mishap prevention role.

8.4.7. **The Safety Investigation Board Structure**

A SIB is comprised of a Board President (BP) and various members depending on the class and type of mishap. Aviation SIB composition and personnel requirements, restrictions, and roles are specified in AFMAN 91-223, *Aviation Safety Investigations and Reports*. Aerospace and Operational Physiology (AOP) officers may be assigned as primary board members when physiological and/or human factors expertise is necessary. SIBs are generally convened for a period of 30 days; however, extenuating circumstances may require extensions.

The SIB gathers information, collects evidence, and analyzes all available data to determine how and why the mishap occurred. Ultimately it is the responsibility of the
SIB to produce a final report which explains relevant mishap factors, identifies findings (causal and contributory), and makes actionable prevention recommendations. An official mishap report includes several sections, or tabs, covering every aspect of the investigation. AOP officers assist the board as directed by the BP, but their primary responsibility is to identify, analyze, and explain all relevant human factors impacting the mishap sequence and effectively incorporate an accurate human factors analysis into the Tab T narrative. AOP officers are also frequently tasked with helping the flight surgeon in their reporting duties. The SIB report is submitted through the Air Force Safety Automated System (AFSAS), an online database containing mishap reports, investigation tools, statistical data, and other useful safety information.

8.4.8. DoD Human Factors Analysis and Classification System

Drawing upon Reason’s (1990) and Wiegmann and Shappell’s (2003) concept of active failures and latent failures/conditions, a new DoD taxonomy was developed to identify hazards and risks. DoDI 6055.7, Accident Investigation, Reporting, and Record Keeping, directs DoD components to “Establish procedures to provide for the cross-feed of human error data using a common human error categorization system that involves human factors taxonomy accepted among the DoD Components and U.S. Coast Guard.” The DoD Human Factors Analysis and Classification System (HFACS) was developed to meet the DoDI requirements and provide a standardized human factors investigation tool. It is intended for use by all persons who investigate, report, and analyze DoD mishaps and is particularly tailored to the needs of those assigned to ISBs and formal SIBs following all classes of mishaps. Human factors describe how our interaction with tools, tasks, working environments, and other people influence human performance. As in many other aviation venues, human factors are found as causal and/or contributory in 80 – 90% of all Air Force mishaps and are the leading cause of aircraft loss and personnel injury. The DoD HFACS model presents a systematic, multidimensional approach to error analysis and mishap prevention. Mishap investigators will use DoD HFACS including the applicable HFACS codes in the mishap analysis. Codes pertaining to Non-Factors Worthy of Discussion (NFWOD) and Other Findings of Significance (OFS) will also be included in the mishap analysis but will not be entered into AFSAS.

This tool provides a template that organizes the human factors identified in the investigation. It is designed for use by all members of an investigation board in order to accurately record all aspects of human performance associated with the individual and the mishap or event. DoD HFACS helps investigators to:

- Perform a more complete investigation
- Classify particular actions (or inactions) that sustained the mishap sequence
- Contribute to the AFSAS database as a repository for detecting mishap trends and preventing future mishaps

DoD HFACS is structured according to error type and the level at which the error occurred. As described by James Reason (1990), Active Failures are the actions or inactions of individuals that are believed to cause or contribute to the mishap. Traditionally referred to as “error,” they are the last “acts” committed by individuals,
often with immediate consequences. In contrast, *Latent Failures* are pre-existing conditions within an organization which indirectly affect the sequence of mishap events. These latent failures may lie undetected for some period of time prior to their manifestation as an influence on an individual’s actions during a mishap. Latent failures may include fatigue, organizational training deficiencies, leadership issues, poor risk management practices, equipment design flaws, and other conditions which have significant, but usually indirect, impact on the overall mishap circumstances.

Reason’s “Swiss Cheese” model describes the four levels within which active failures and latent failures may occur during complex operations (Figure 8.4.8-1). The holes in the layers represent failed or absent hazard mitigation controls which may contribute to the overall mishap circumstances. Working backward from the mishap, the first level of Reason’s model depicts those *Acts* that most immediately lead to a mishap. Most causal factors are uncovered at this level, however, Reason’s model forces investigators to address the latent failures, or “holes,” within the causal sequence of events which may be overlooked if the focus is limited to individual actions only. Latent failures and conditions are described within the context of Reason’s model as *Preconditions, Supervision, and Organizational Influences*.

![Figure 8.4.8-1. The “Swiss Cheese” Model (adapted from Reason, 1990)](image)

Mishaps are the result of individual and organizational factors that are further categorized as causal and/or contributory. Individuals whose actions impacted the outcome of the mishap should be identified as “mishap persons” and investigated to determine how various factors such as training, experience, physiological condition, and/or environmental issues affected their decision making process. Their acts and preconditions will be identified at the Person Level within AFSAS. The context in which these acts and preconditions occurred will be captured as supervisory and organizational factors and will be identified at the Mishap Level. These factors are attributed to the mishap itself and not to a specific person.

Investigators are guided on the utilization of HFACS v7.0 through a series of questions within AFSAS. These questions lead to specific HF classifications, called nanocodes, which identify and define the applicable factor/s as applied to each act or condition. For Class A, Class B, and Class E Physiological mishaps, investigators will
be required to answer all questions and provide input at the nanocode level. For Class C and D mishaps, investigators will answer all questions, however nanocode input will not be required. Each human factor code that the investigator identifies must be rated as causal or contributory for its influence on the mishap and assigned at the appropriate level. These coding rules have been embedded in AFSAS to guide the investigator and are navigated through a simple menu tree.

- Causal factors are deficiencies which, if corrected, would likely have prevented or mitigated damage and/or injury. Cause does not imply blame. Events/conditions that are highly probable results of other events/conditions are not causal and should be rated as contributory.

- Contributory factors are independent events/conditions that do not directly result in damage and/or injury, but are integral to the progression of the mishap sequence. Contributory factors allow progression of other events/conditions. If an event/condition is considered to be both contributory and causal, rate it only as causal.

8.4.9. Benefits of DoD HFACS

In an effort better understand causality with a view to improve performance and reduce accidents, a fair and open reporting system like DoD HFACS is fundamental to the efforts of the investigation process. The ease with which accident classification taxonomy under DoD HFACS can be turned into a practical tool within an operational setting should not be overlooked. In both the military and civil aviation investigation environment, there are limitations on the time available to train mishap classification system users, so using a prescribed formula like DoD HFACS eases the investigator’s task of sorting out the variety of behaviors and actions of those involved in the mishap. Simply put, DoD HFACS is:

1. Structured analysis of human error patterns
   - Detailed, complete and operationally focused

2. Gets to the “Why,” not just the “What”
   - More accurate root cause determination
   - Permits more effective risk management

3. Data-driven approach
   - Supports research across the DoD
   - Easily applied to both new mishaps and previous reports

4. Can be used for more than just operational situations
   - As a brainstorming tool for risk management
   - In developing interview questions
   - Applies to both on- and off-duty mishaps
References
Air Force Instruction 91-204, Safety Investigations and Reports.  10 Apr 14.
DoD Instruction 6055.07, Accident Investigation, Reporting, and Record Keeping.  6 Jun 2011.
National Transportation and Safety Board, NTSB/AAR-10/01, 2 February 2010.  490 L’Enfant Plaza, S.W. Washington, D.C.

Concepts
DoD Human Factors Analysis and Classification System (DoDHFACS)
Independent investigation team
Safety Investigation Board (SIB)
Applied Science in the Investigation Process
8.5. **Hypoxia Training**

8.5.1. **Hypobaric Training**

Since the earliest balloonists raised their gondolas to lofty heights above the Earth, the awareness of the effects of rarified air on human physiology has been documented. As aviators pointed the nose of their aircraft skyward and ascended to altitudes above 10,000 ft, then 18,000 ft, and again to a range above 20,000 ft without pressurization systems, oxygen systems and personal attention to the conditions linked to hypoxia have captured the attention of scientists and accident investigators alike. The environment universally accepted for hypoxia “training” is the hypobaric chamber. As an extension of the classroom, the altitude chamber is a training device that places the student at a designated altitude above field elevation. As the altitude climbs, the student completes a series of prescribed tasks including an hypoxic experience at 25,000 ft. The altitude chamber flight affords the student the opportunity to experience mild hypoxia, gas expansion, environmental temperature changes, practice with oxygen equipment, and demonstration of night vision.

Specific chamber flight profiles are described in AFI 11-403 (Aerospace and Operational Physiology Program), and objectives will be briefed in the classroom prior to each flight. An example chamber profile is shown in Figure 8.5.1-1.

![Initial Chamber Flight Profile](attachment:image.jpg)

**Figure 8.5.1-1. Initial Chamber Flight Profile**

8.5.2. **Reduced Oxygen Breathing Training**

The advent of the reduced oxygen breathing device (ROBD) has provided a new platform for hypoxia training into the 21st century. The introduction of a mixed gas during flight simulation places the crew member in a part-task training environment with the backdrop of a realistic flight scenario. The ROBD will be used only as a means of refresher physiological training where available and appropriate for the aircraft type (e.g., single and dual seat). While guiding a scripted profile on a flight simulator...
program, the ROBD operator will introduce a gas mixture representative of the ambient air conditions at the altitude of the simulation. The specific details of operation are part of a formal training program for aerospace and operational physiology staff. Once the crew member notices any change in performance or physiological conditions related to hypoxia, corrective actions are begun (“pilot demonstrates Emergency Procedures”), and the ROBD operator selects “100% Oxygen” on the device and monitors the student through recovery. The portable system does not replace the altitude chamber for the initial hypoxia experience but provides a flexible platform that can be co-located with squadrons without regard to chamber facilities.

8.5.3. Normobaric versus Hypobaric Hypoxia
Lt Col Ryan W. Maresh, USAF, BSC, Ph.D.

Within the scientific community, debate continues to exist as to whether there are physiological differences between exposures to normobaric or hypobaric hypoxia. However, the growing body of evidence supports the conclusion that hypobaric hypoxia is a more severe environmental stressor and results in different physiological responses than exposure to normobaric hypoxia (Millet et al., 2012).

When evaluating the differences between the two conditions and whether they are physiologically relevant, it is important to differentiate between the purpose of the exposure (research vs. training) and the duration of exposure (acute vs. chronic). In the research realm, physiological differences between normobaric and hypobaric hypoxia may be much more relevant and significant to understanding physiological processes and mechanisms. For example, overall ventilation during exposure to hypobaric hypoxia is lower, with a lower tidal volume and a higher breathing rate (Millet et al., 2012; Savourey et al., 2003). The result is faster, shallower breathing which increases dead space ventilation. There is a greater tendency for higher alveolar physiological dead space, ventilatory alkalosis, hypocapnia, and heart rate, while arterial O₂ saturation is lower (Millet et al., 2012; Savourey et al., 2003). Under hypobaric conditions, fluid movement at the alveolar-capillary border is altered and potentially leads to pulmonary vasoconstriction, thereby altering oxygen diffusion and transport (Millet et al., 2012). In addition, recent research indicates that postural instability may be more influenced by altered barometric pressure than by changes in the fraction of inspired oxygen, such as in normobaric hypoxia exposure (Degache et al., 2012).

Two research areas that commonly use normobaric hypoxia are studies on altitude sickness/acclimatization and training regimes for athletic performance. Some of the advantages of using normobaric hypoxia, rather than hypobaric hypoxia, include the cost (it is cheaper than purchasing a hypobaric chamber or traveling to altitude to conduct the protocol) and accessibility (it can be utilized anywhere, independent of location elevation). In addition, depending on the test altitude and protocol, there is a risk of decompression sickness and barotrauma during hypobaric exposure (when conducted in a hypobaric chamber) that is eliminated with the use of normobaric hypoxia.

Exposure to hypobaric hypoxia induces acute mountain sickness (AMS) to a much greater extent than normobaric hypoxia (Roach et al., 1996). Utilizing normobaric hypoxia to acclimate in preparation for ascent to higher altitude is appealing, but does not appear to be as effective as actual hypobaric exposure, with normobaric hypoxia providing little useful benefit during subsequent residence under hypobaric hypoxic conditions (Fulco et al., 2011). Recent evidence further supports different physiological
and performance responses during Live High-Train Low (LHTL) regimes depending on whether the “live high” conditions were hypobaric or normobaric (Saugy et al., 2014). Greater performance improvements were seen in those triathletes that lived in a hypobaric hypoxic environment compared to those that experienced normobaric hypoxia. While both groups increased hemoglobin mass and VO$_{2\text{max}}$, the hypobaric hypoxia group experienced greater increases in red blood cell number, hemoglobin concentration, hematocrit, higher breathing frequencies, and greater night desaturation levels (Saugy et al., 2014). Bonetti and Hopkins reported better performance improvements in elite athletes that experienced natural LHTL regimes compared to other methods of inducing hypoxia (Bonetti and Hopkins, 2009). The differences were most likely due to the difference in hypoxic dose, as well as the intermittent nature of the normobaric exposures. Given the current body of evidence, it appears that only hypobaric hypoxia (within limits) exposure reduces AMS and improves exercise performance. Proposed mechanisms for the physiological differences between normobaric and hypobaric hypoxia include: intravascular bubble formation resulting from a decrease in barometric pressure, increased alveolar dead space, a mismatch in ventilation and perfusion, and altered gas density or fluid permeability through the alveolar epithelium (Savourey et al., 2003).

Outside of the research and athletic performance environments, the use of normobaric hypoxia has been implemented to provide hypoxia familiarization training to pilots. In 1999, the Naval Aeromedical Research Laboratory developed a reduced oxygen breathing device (ROBD) to induce hypoxia by mixing aircrew breathing air with increasing amounts of nitrogen while at normobaric conditions (Artino et al., 2006). Since then, the device has been validated to replicate hypoxic conditions typically utilized in traditional hypoxia training in a hypobaric chamber. In 2001, the use of reduced oxygen breathing was demonstrated to induce hypoxia symptoms comparable to those seen in hypobaric training (Sausen et al., 2001). Westerman, using reduced oxygen breathing to simulate an altitude of 25,000 feet, reported symptoms of muscle incoordination, illegible writing, poor reproduction of geometric figures, impaired math ability, visual symptoms, dysaesthesia, and headache (Westerman, 2004). However, both studies consisted of only normobaric exposure and were therefore limited by the absence of a direct comparison with hypobaric hypoxia. Additional studies comparing the objective and subjective effects of normobaric versus hypobaric hypoxia in aircrew training reported no differences between the two training methods, suggesting that the effects of decreased tissue oxygenation are the same regardless of how it is induced (Vacchiano et al., 2004). Self et al. reported that, although differences exist in several physiological variables between normobaric and hypobaric hypoxia, the differences do not result in meaningful differences in the number of symptoms or pattern of occurrence (Self et al., 2011). They concluded that using reduced oxygen breathing under normobaric conditions is a reliable substitute for traditional hypobaric hypoxia training. This conclusion is further supported by Kumar et al. that found subjective symptoms experienced during normobaric hypoxia training are similar in type and severity, and that the physiological response and effect on psychomotor performance and similar to those during hypobaric hypoxia training (Kumar et al., 2013).

In 2004, the Naval Survival Training Institute began providing mobile hypoxia training using the ROBD in conjunction with aircraft simulators (Artino et al., 2006). Since then, the use of ROBD for aircrew training has expanded. In 2007, the Air Force began using it as an alternative option to the hypobaric chamber for refresher training (Arnsberg et al., 2013). Within the context of using normobaric hypoxia to train pilots,
such as occurs using a ROBD, any physiological differences at the cellular level appear to not be relevant as the temporary, outward hypoxia symptoms that a pilot recognizes closely resemble those experienced during hypobaric exposure. It appears that when exposed to acute but severe hypoxic conditions, normobaric and hypobaric hypoxia induce similar subjective symptoms and that the lack of major differences during these brief exposures reflects insufficient time for the body to reach a new state of equilibrium (Richard and Koehle, 2012).

Before leaving the discussion on normobaric versus hypobaric hypoxia, one other area where the use of reduced oxygen breathing is utilized is within the clinical setting with patients suffering pulmonary disease. While pulmonary disease is not a concern for military aviators, every year large numbers of patients with pulmonary diseases travel by commercial air and aeromedical transport of susceptible patients is a possibility within the military community. If a person suffering from pulmonary disease already has a reduced alveolar PO$_2$ at ground level, exposure to a higher altitude may result in a more significant decrease in P$_A$O$_2$, leading to significant oxygen desaturation (Dine and Kreider, 2008).

The reduced oxygen procedure, known as the hypoxia altitude simulation test or the hypoxia inhalation test, uses a gas mixture of 15.1% oxygen to simulate a cabin altitude of 8,000 feet (Dine and Kreider, 2008; Dillard et al., 1995). It allows the physician to screen a patient prior to traveling on an aircraft for adverse effects associated with mild hypoxia and either recommend against flying or the use of supplemental oxygen during flight. Guidelines vary as to which patients should be screened, but it is typically recommended that those with chronic obstructive lung diseases, restrictive lung disease, cystic fibrosis, a history of recent respiratory illness or infections, pulmonary tuberculosis, or past difficulties with air travel be evaluated (Dine and Kreider, 2008). Comparison of the hypoxia altitude simulation test using normobaric hypoxia and hypobaric hypoxia show the two are similar in their diagnostic abilities (Dillard et al., 1995).

References


8.5.4. Frequently Asked Questions Regarding Altitude Chamber Exposure.

The following list was compiled based on questions that have been asked by students during physiological chamber training. All answers were reviewed by Dr. Richard Sumrall, Lt Col, USAF, MC, CFS, USAF School of Aerospace Medicine, Centrifuge and Altitude Chamber Medical Director. The intent of these FAQs is to offer a brief explanation/answer for educational purposes, not to replace consultation with, or approval from, qualified medical personnel prior to exposure to hypobaria, either during chamber training or actual flight.

1. What medications should APOs be concerned about students taking? Why?

Mainly medications, both over-the-counter (OTC) and prescription, for colds/flu and/or allergies, such as decongestants, cough suppressants, nasal sprays, antihistamines, etc. Not only would some of these medications cause adverse effects, the underlying upper respiratory condition would be a problem in the chamber. A complete list is at: https://kx2.afms.mil/kx4/FlightMedicine/Documents/Med%20List%20Oct%202014.doc (published every 6 months, restricted access).

2. Do supplements count as “medications”? Are there any that APOs should be concerned with in regards to students?

Yes, even over-the-counter, “sports,” “healthy,” “natural.” Whatever they are called, they should be considered for safety. For the most part, supplements should not affect pressure change risk, as there are no supplements that affect the respiratory physiology, unless someone is taking something for allergies.

3. Is birth control safe for use in a hypobaran environment?

Yes. All methods, including oral, continuous oral, implant, intra-vaginal, and injected.
4. What if I’m taking an oral decongestant, like Sudafed, even if I have been taking it for a long time and have no symptoms?

Because you are taking it for congestion, you should not be exposed to pressure changes in the altitude chamber because of the risk of block. Long term use of topical nasal steroids (Flonase, Beconase) is acceptable if you have no congestion, as these are preventive.

5. Why can’t I take Afrin (or other vasoconstrictor) before going in the chamber?

Afrin will open up sinus ostia (passages) and Eustachian tubes (to the ears), making it less likely for a block. But it can wear off during the flight resulting in that passageway closing. That block on descent would be more severe, and repeated use of Afrin would not help. Afrin may be carried in the chamber for "get-me-down" use in order to open a passageway that blocks when descending. If you need Afrin before going up in the chamber, you shouldn’t be going up in the chamber.

6. Is it safe to go into the chamber with breast implants?

Yes, all types of breast implants (silicone and saline) are safe (Vann RD, et al. Mammary Implants, Diving and Altitude Exposure. Plastic and Reconstructive Surgery, 1988; 81(2):200-203). There have been anecdotal reports of transient changes or sensations during air travel, some of which last for short periods of time following re-pressurization. This is most likely due to gases coming out of solution from the tissues and filling a potential space between the implant and the surrounding tissues, not due to any changes in the implant. This gas is gradually reabsorbed following landing (Lewin JM. Peri-Implant Gas Following Air Travel to High Altitude. Plastic and Reconstructive Surgery, 2013; 131(3):465e-467e).

7. Why can’t I wear make-up in the chamber?

Many make-ups are oil-based, and even though we are at lower air pressure, there is concentrated oxygen used, and oil and oxygen (at higher than 21%) don’t mix (i.e., risk of fire increases). Plus, oxygen masks and other gear would smear it and be dirtied by make-up.

8. What if I had dental work within the last week, but longer than 72 hours prior to the chamber flight?

There are no hard-and-fast duties not to include flying (DNIF) times in AFI 47-101 (dental) or 48-123. If you had local injected anesthetic, we use 8 hours no-fly. If you are in Dental Readiness Class 3, you would be DNIF as aircrew, and would use same standard. Obviously, for other dental procedures, such as implant placement, complicated root canal, etc. would return to flying status (RTFS) when cleared by dentist, off pain medications, and pain free.
9. Can I wear contact lenses in the chamber?

Yes. Most are gas permeable lenses and should pose no problem, even during rapid decompression. Since the air at altitude is drier, the concern is for them to become irritated or fall out. If you really rely upon the lenses to see, it’s always good to have a spare pair of spectacles available.

10. Jewelry, watches, and pens: will they “explode” in the chamber?

Generally not an issue although any trapped gases in the items will expand and may cause damage or destruction during a decompression.

11. Can I take my cellphone in the chamber?

No. Although we may carry these items on commercial flight and, in fact, can use them on some flights today, all of those cabins are pressurized to 6 – 8,000 feet and will not likely cause any problems that you could have at 25,000 feet. Many electronic devices, such as the iPhone® and iPad®, have an operational exposure limit of 10,000 feet.

12. What if I got decompression sickness (DCS) the last time I went in the chamber?

Depends on the outcome. For aircrew, we follow the guidance outlined in the Medical Standards Directory. If a volunteer in a research subject, they would probably be deferred from future exposure based on Institutional Review Board (IRB) instruction, unless study was designed with DCS as possible end-point.

13. What is the worry/concern if I had a collapsed lung while in high school?

Depends on the reason: If traumatic, safe; if spontaneous, maybe. From USAF Waiver Guide:

A single episode of spontaneous pneumothorax does not require waiver if posterior-anterior (PA) inspiratory and expiratory chest radiograph and thin-cut CT-scan show full expansion of the lung and no demonstrable pathology which would predispose to recurrence. After a second pneumothorax, or if CT demonstrates residual blebs, waiver may be considered only after definitive surgery to prevent recurrence.

14. What is bleomycin chemotherapy and why is it a concern?

Bleomycin is a chemotherapeutic agent commonly used for testicular cancer, lymphoma, and other forms of cancer. There are case reports of serious pneumonitis with oxygen exposure (e.g., in surgery) after having had bleomycin, but most people do fine with O₂ exposure. See discussion in USAF Waiver Guide under lymphoma.
15. Will I really experience a pulmonary barotrauma (i.e. ruptured lung) if I hold my breath during the 4.5 psi rapid decompression?

Yes, it really is possible. Fortunately, the chances of the glottis being closed, and maintained in such a state, during a rapid decompression is extremely rare. See Sections 3.1.3 and 3.3.2.5 for a complete discussion/explanation of the risks associated with rapid expansion of air within the lungs.

16. What are the guidelines/restrictions for flying after diving?

For USAF aircrew, AFI 11-202v3, paragraph 2.7.1.5 is the governing guidance:

Aircrew members will not fly within 24 hours of compressed gas diving including SCUBA, surface supplied diving, hyperbaric (compression) chamber exposure or aircraft pressurization checks (to below sea level) that exceed 10 minutes duration (this restriction not applicable to UAS flight operations). Exceptions: Following Helicopter Emergency Egress Device System (HEEDS) training, aircrew may only fly within the 24-hour window if the aircraft’s maximum altitude remains below 10,000 ft. MSL. Air Force divers on aeronautical orders will follow guidelines IAW SS521-AG-PRO-010 U.S. Navy Diving Manual for flying and diving restrictions.

For non-USAF aircrew, the following guidance is provided for recreational divers from the Divers Alert Network (DAN) website (www.diversalertnetwork.org/medical/faq/Flying_After_Diving):

The following guidelines are from the consensus of attendees at the 2002 Flying After Recreational Diving Workshop. These guidelines apply to air dives followed by flights at cabin altitudes of 2,000 to 8,000 feet (610 to 2,438 meters) for divers who do not have symptoms of decompression sickness (DCS). The recommended preflight surface intervals do not guarantee avoidance of DCS. Longer surface intervals will reduce DCS risk further.

- For a **single no-decompression dive**, a minimum preflight surface interval of 12 hours is suggested.

- For **multiple dives per day or multiple days of diving**, a minimum preflight surface interval of 18 hours is suggested.

- For **dives requiring decompression stops**, there is little evidence on which to base a recommendation and a preflight surface interval substantially longer than 18 hours appears prudent.
17. Why can’t I exercise for 12 hours after my chamber exposure?

HAOP Section 3.4.4.3.3:

Exercise following a decompression exposure was shown to involve no more risk than resting after exposure (Webb JT, Pilmanis AA, and Fischer MD. Moderate Exercise After Altitude Exposure Fails to Induce Decompression Sickness. Aviat Space Environ Med 2002; 73:872-875). However, exercise-induced pain may be misdiagnosed as decompression sickness (DCS) pain, or vice versa in the case of latent/delayed DCS. It is therefore advised to avoid strenuous exercise for 12 hours after altitude chamber training.

18. Why should I avoid drinking alcohol for 12 hours after a chamber exposure?

Alcohol can mask decompression sickness (DCS) symptoms, and dehydration can increase DCS risk.

19. Is it safe to dive after flying?

From the Divers Alert Network (DAN) website:

Mild dehydration can occur on long flights, especially when travelers cross several time zones... Generally speaking, dehydration is thought to predispose a diver to decompression illness because the washout of inert gas (nitrogen, in diving) is less effective in a dehydrated individual.

Although no one can insist upon a 24-hour waiting period after flying, such a conservative approach to diving after flying is a reasonable idea [as] it gives divers an opportunity to rehydrate, adjust to a new climate and time zone, and rest up after a long flight.

20. From the para-rescue/battlefield setting: What are the guidelines/restrictions for aeromedical transport of a patient with TBI (real-world)?

Stick with ABCs: maintain airway (consider definitive or advanced airway before flight, even if patient doesn’t need it right then, as difficult to do procedure in flight), ensure breathing (O2, oximetry, capnography), watch circulation (how monitor HR, peripheral circulation in flight?). Protect hearing, eyes. Keep warm. If very long flight, make sure not on hard board that can give pressure sores. Also, consider bladder catheter (condom or indwelling). If risk of neck injury, cervical spine immobilization.
8.5.5. Sample Chamber Flight Lesson

BEGIN DENITROGENATION TIME (with concurrence from inside observers):

- Ensure all regulators are set to ON and 100% position. Caution students NOT TO DROP their masks or move the switches on the regulator.

INTERCOM CHECK:

- Perform intercom check with students/crew
  - Explain intercom system. There are two ways to communicate. One button is below the three switches on the regulator and the other button is located by the CRU 60P.
  - Have students give their NAME and SEAT POSITION (COMM).
  - Next have the IO's and outside crew do a COMM check on the COMM channel.
  - In case of intercom failure, you will need to use hand signals to the inside observers.

BEFORE THE FLIGHT BEGINS:

- Have everyone practice doing a Valsalva.

DENITROGENATION:

- Conduct ear and sinus (E and S) check.
  - Explain the reason why we conduct an E and S check from GL to 5,000 ft. GL to 5,000 is the area of the greatest pressure change in the Earth's atmosphere. If everyone is able to make it through the ear and sinus check without any problems, then they should be able to complete the entire chamber flight profile without any problems.
  - Have inside observers demonstrate “level-off” signal.
  - Remind students that they shouldn't need to accomplish the Valsalva maneuver on ascent. Middle ears normally clear automatically. To help assist with pressure equalization, you could yawn, swallow, or move your jaw from side to side.
  - Point out training aids.
    - Explain the Flask/Beaker arrangement if applicable or use a different training aid to show problems with trapped gases in the middle ears and sinuses. Tell students that they should not fly with a cold.
    - Gloves hanging in the chamber will increase in size as we go up in altitude in accordance with Boyle’s Law.
    - If there is an altimeter inside the chamber, describe how students can reference it to have a general idea of the altitude that they are passing through. Don’t forget to remind the students that the altimeter inside the chamber has been through a few rapid decompression profiles and it is not as accurate as the equipment that we have on the outside of the chamber.
o At 5,000 ft ask students if they have any problems with the ascent portion of the flight. Ask for a “thumbs up” from the students and observers before beginning descent.
o On descent remind the students that they need to do something to clear their ears because their ears will not clear automatically while the pressure is increasing.
  ▷ To do an effective Valsalva, keep your head straight or tilted back slightly, with your two forefingers or thumb and forefinger, pinch off the Valsalva ports on the mask and blow. The maneuver should be short and forceful. If you have a slow ear, put the slow ear up towards the ceiling and do a short and sharp Valsalva.
  ▷ The other ways to clear your ears include yawning, swallowing, wiggling your jaw back and forth, or delivering pressure from the regulator.
  ▷ Remember the level off sign, if you have a problem.

**PRICE CHECK** (relate to specific aircraft):

DO a PRICE CHECK (1) before the aircraft leaves the ground, (2) during flight, and (3) whenever hypoxia occurs or after a decompression occurs.

o P – Pressure
  o Low-Pressure Gaseous (Yellow)
    ▷ Full pressure: 425 ± 25 psi
    ▷ Operationally empty: 100 psi; Maintenance empty: 50 psi
  o High-Pressure Gaseous (Green)
    ▷ Full pressure:
      ◇ 1800 – 2,000 psi (A/C)
      ◇ 1,800 – 2,200 psi (Emergency)
  o Liquid Oxygen (No Color Code)
    ▷ Low pressure: 50 - 120 psi (Fighter)
    ▷ High pressure: 300 psi (Multiplace A/C)
    ▷ Quantity gauge:
      ◇ Full: 95% of converter capacity
      ◇ Empty: 10% of converter capacity
    ▷ Expansion ratio: 860:1 (gas:liquid)

o R – Regulator
  o Fly to fail equipment – Do thorough inspection
  o Visual inspection:
    ▷ Cracks in face/cover
    ▷ Oil or grease (petroleum products)
    ▷ Broken switches.
  o Functional inspection:
    ▷ Switches move easily and stay in desired settings
    ▷ Integrity check:
      ◇ All 3 switches up (On, MAX/100% Oxygen, Emergency)
      ◇ Three breaths - Hold - Monitor indicator
      ◇ Follow same procedures with automix lever in "normal" setting - Monitor indicator
  o I – Indicator
o Automix lever at MAX/100% setting
o Monitor for smooth operation
o Uses:
  ▪ Gas flow through system
  ▪ Leak check on system
  ▪ Monitor breathing rate
o C – Connections: Start as far back as possible and work toward the helmet (habit forming). Remind students to use positive checks.

NOTE: DO NOT ALLOW THE STUDENTS TO DISCONNECT DURING DENITROGENATION

o Check CRU-60/P for:
  ▪ 12 – 20 pounds disconnect tension - first connect the CRU 60/P with ship supply and then disconnect; from that point on all checks will be positive checks
  ▪ Silver “C” ring
  ▪ Neoprene washer: Color is green, rust (orange), or white
  ▪ O-ring
  ▪ Emergency oxygen hose connector
o Oxygen Equipment/Intercom System:
o Inspect mask hose for holes, kinks, and cuts (wear and tear).
o Inspect intercom cord that is wrapped around mask hose.
  ▪ Check Intercom cord for cuts, kinks, and frays.
  ▪ Check all intercom connections (positive).
o Bayonets set in receivers with two clicks. If you get your own oxygen equipment the oxygen mask straps will be tacked down by Life Support (your equipment).
o Chin strap
o E – Emergency. Do a PRICE CHECK on ALL emergency equipment.
o Low-pressure systems: MA-1 portable oxygen assemblies (what is commonly called the Walk a Round Bottle)
  ▪ Color code – Yellow. Check the condition of the bottle.
  ▪ Check the pressure: The pressure will be whatever the line pressure of the aircraft.
  ▪ If the pressure is 50 psi or lower, for longer than 2 hours, then you must purge the system. Fill and empty the bottle 3 times.
  ▪ Lift the dust cover up and look inside. Things could be in it.
  ▪ Provides a certain amount of oxygen to the user.
  ▪ Refiller ports on aircraft: Know where they are and how to use them.
  ▪ Refill whenever the walk around bottle is at 100 psi or lower.
o High-pressure systems: High-pressure emergency cylinder
  ▪ Color Code – Green. Check the condition of the bottle.
  ▪ Check the pressure: The pressure will be 1,800 – 2,200 psi.
  ▪ Also check the nozzle, hose, green apple, and red tag.
  ▪ Once activated, it gives a 10-minute continuous flow of oxygen.
  ▪ The pressure you fill in the chamber may not be the actual pressure you will fill when activating a real bottle.
Check ALL emergency oxygen sources: Could be an extra regulator, passenger oxygen kit (POK), personal breathing device (PBD), sodium chlorate candle.

Extra regulator – Do a PRICE CHECK.

Passenger Oxygen Kit (POK): A green bag with a high-pressure emergency cylinder and oxygen mask. You would check everything that you normally check with the high-pressure emergency cylinder and also do an inspection of the mask.

Personal Breathing Device (PBD): Four major components:
- Solid state oxygen supply source
- Chemical scrubber for carbon dioxide and vapor
- Loose-fitting hood with head harness and neck seal to provide the breathable environment
- Pumping arrangement powered by the chemical oxygen, which recirculates the breathing gas within the scrubber and hood
- One time use, totally self-contained. Duration is approximately 15 minutes
- To use: Remove unit from container, tear off pull strip, and remove from plastic wrapper. Pull actuation ring. Hold device by open end, with supply pack away from you. Bend over and grasp hood opening with thumbs; pull overhead. Rise to standing position and adjust hood and pack for comfort. Check neck seal for secure fit. Remove and stow on aircraft after use.

Sodium Chlorate Candle: Chemical Generated Oxygen
- Mainly a passenger source, stowed under seat on cargo compartment pallets.
- System contains pull out passenger mask with polyvinyl bag. Duration is 30 minutes. This system consists of canister containing a fuel-enriched cone and a sodium chlorate candle and a mask with bag.
- To use: Activate by pulling mask from canister, thereby breaking lanyards that trigger striking pins, which activate chemical generation.

Any personal gear available

NOTE: Ensure that a full 30 minutes of denitrogenation has been completed and perform student and crew intercom check prior to ascent to FL250.

ASCENT TO FL250:

- Have students stretch out prior to ascent.
- Discuss the value of frequent intercommunication checks in chamber and aboard military aircraft.
- Remember no Valsalvas on ascent. Ears will pop and click (clear) automatically.
- If there are any problems remember the level off signal.
- AFI 11-202v3, General Flight Rules, Chapter 6, Life Support Systems, Paragraph 6.4.1 states that “each crewmember SHALL use supplemental oxygen anytime the cabin altitude exceeds 10,000 feet.” Chapter 6 also discusses oxygen requirements when you are flying in helicopters, unpressurized aircraft, and pressurized aircraft.
INFORMATION FOR AEROMEDICAL EVACUATION CREWMEMBERS:

Message also came out that states all primary aeromedical evacuation crewmembers will have a portable walk around bottle at their flight position for preplanned flights above FL350.

<table>
<thead>
<tr>
<th>Altitude</th>
<th>Pilot</th>
<th>Flight Engineer</th>
<th>Other Flight Deck Crew</th>
<th>Cabin/Cargo Area Crew</th>
<th>Pax</th>
</tr>
</thead>
<tbody>
<tr>
<td>10,000 ft through FL250</td>
<td>R</td>
<td>R</td>
<td>R</td>
<td>A</td>
<td>N/A</td>
</tr>
<tr>
<td>Above FL250 through FL350</td>
<td>One I</td>
<td>One R</td>
<td>I</td>
<td>A</td>
<td>A</td>
</tr>
<tr>
<td>Above FL350 through FL410 (both pilots in seat)</td>
<td>I</td>
<td>I</td>
<td>R</td>
<td>A</td>
<td>A</td>
</tr>
<tr>
<td>Above FL350 through FL410 (only one pilot in seat)</td>
<td>One O</td>
<td>One A</td>
<td>I</td>
<td>R</td>
<td>A</td>
</tr>
<tr>
<td>Above FL410 through FL450</td>
<td>One O</td>
<td>One I</td>
<td>I</td>
<td>R</td>
<td>A</td>
</tr>
<tr>
<td>Above FL450 through FL500</td>
<td>One O</td>
<td>One I</td>
<td>I</td>
<td>A</td>
<td>A</td>
</tr>
<tr>
<td>Above FL500 through FL600 (pressure breathing for altitude system/get me down scenario)</td>
<td>G</td>
<td>G</td>
<td>G</td>
<td>G</td>
<td>G</td>
</tr>
<tr>
<td>Above FL500 (sustained)</td>
<td>S</td>
<td>S</td>
<td>S</td>
<td>S</td>
<td>S</td>
</tr>
</tbody>
</table>

*A – Have Oxygen Available: Individuals required to have oxygen available must carry portable oxygen (such as walk around bottles) on their person any time they are moving about the cabin/cargo area. The requirement to have oxygen available can also be satisfied by placing sufficient portable oxygen units or extra oxygen outlets with masks throughout the cabin/cargo area so that any crew member or passenger has quick access to oxygen regardless of where he/she is in the cabin/cargo area should a loss of pressurization occur.

R – Have Oxygen Readily Available: Individuals required to have oxygen readily available must have a functioning system and mask located within arms reach, and the regulator must be set to 100% and ON.

I – Have Oxygen Immediately Available: Crew members who are required to have oxygen immediately available must wear helmets with an oxygen mask attached to one side or have available an approved quick-donning/sweep-on mask properly adjusted and positioned. Regulator shall be set to 100% and ON.

O – Oxygen Mask ON: Regulator ON and Normal.

G – Wear a Partial Pressure Suit: Suit must provide 70 mmHg of assisted positive pressure breathing for altitude.

S – Wear a Pressure Suit: Suit must provide a total pressure (atmospheric plus suit differential) of at least 141 mmHg to the head and neck with adequate body coverage and pressurization to prevent edema and embolism.

Given the shortage of the MA-1 portable oxygen bottles, the emergency passenger oxygen system, protective breathing equipment, or emergency escape breathing device can be used for preplanned flights below FL350 as a primary oxygen source.

- At 16,500 ft, explain that wet gas expansion is double – pass the gas.
- At 18,000 ft, explain that dry gas expansion is double, also half the Earth's atmosphere, and refer to altitudes by flight levels (i.e., FL180).
- Trapped gases that you have are middle ear, sinuses, teeth, and gastrointestinal tract.
Approaching FL250

- Highest altitude that an unpressurized aircraft can go (T-37).
- Hypoxia becomes critical factor: at FL180, time of useful consciousness (TUC) is 20 – 30 minutes; at FL250, 3 to 5 minutes.
- Evolved gas decompression sickness is more likely to occur above FL200.
  - Limb pain symptoms
  - Skin symptoms
  - Respiratory symptoms
  - Neurologic symptoms

**NOTE:** IAW AFI 11-202v3, General Flight Rules (5 Apr 06), Chapter 6, paragraph 6.4.5.4. If an individual appears to be suffering decompression sickness, a crew member should administer 100 percent oxygen to that individual using a tight fitting aviator’s oxygen mask.

6.4.5.4.1. If an aviator’s mask is not available, an alternate source that can provide the greatest percentage of oxygen delivery should be used.

6.4.5.4.1.1. Individuals suspected of decompression sickness should remain on 100 percent oxygen until evaluated by a flight surgeon or competent medical authority.

6.4.5.4.2. The pilot must descend as soon as practical and land at the nearest suitable installation where medical assistance can be obtained. Decompression sickness may occur up to 12 hours after mission completion. The affected person shall not continue the flight unless authorized by a flight surgeon or civilian designated aviation medical examiner.

6.4.5.5. After a cabin decompression, the risk of decompression sickness increases with prolonged exposure to altitudes at or above FL 210 (unpressurized).

**LEVEL AT FL250:**

**NOTE:** Minimize lecture during demonstration to allow students to focus on symptoms. Do not give any time hacks as this may affect a student’s decision to recover instead of keeping track of hypoxia symptoms he/she may be experiencing.

- Explain how the hypoxia demonstration will work. Everyone will go off oxygen. Drop mask from right side and turn regulator off.
- To correct for hypoxia, put all three switches on regulator up. Remember to slow your breathing down. Put the red level in normal position once all symptoms are gone.
START CLOCK FOR TIME (Maximum Time OFF – 10 minutes):

- Remind students that there is no prize for who can remain off of oxygen for the longest amount of time. Tell them that the goal is to recognize hypoxia and correct for themselves.
- TUC at FL250 is 3 to 5 minutes.
- To remain off oxygen too long could result in their forgetting the symptoms just received or result in a moderate to severe headache.
- Students monitor and help one another while observing “outward” symptoms of hypoxia.
- Discuss common recognition symptoms of hypoxia.
  - Objective symptoms (OUTWARD SIGNS, what others can observe)
    - Increased rate and depth of breathing
    - Cyanosis (bluing) of lips and fingernail beds
    - Unconsciousness
  - Subjective symptoms (WHAT ONLY YOU FEEL)
    - Apprehensive feeling
    - Dizziness
    - Fatigue
    - Nausea – correct immediately if you feel this
    - Hot and cold flashes
    - Blurred vision
    - Tingling and/or numbness
    - Euphoria or belligerence

DESCENT FROM FL250 TO FL180:

- Ensure a normal descent rate Explain that they will be doing the visual acuity demonstration.

PASSING THROUGH FL220:

- All students remove oxygen mask from right hand side of face.
- Lecturer, observer, and recorder start stopwatches.
- Have inside observers (IOs) pass out visual acuity cards.
- Students should be advised to place cards upside down on their lap (white side up).

LEVEL AT FL180 – VISUAL ACUITY DEMONSTRATION:

- Turn off main chamber lights.
- Dim emergency lights via rheostat on chamber’s main console (ONLY WHEN LIGHTS ARE NOT PREADJUSTED).
- Tell students that if they feel their hypoxia symptoms with the same intensity that they felt at FL250 to abort the demonstration, get back on oxygen, and notify the IOs of their problem.
o Explain that the main purpose of this demonstration is to illustrate the dramatic loss of vision, especially in a low illumination environment. Simulates flying at about 9,000 ft for 4 hours. We take you to FL180 for 5 minutes. TUC at FL180 is 20 – 30 minutes.

o The students may feel that their vision is improving after a minute or two, but you as the lecturer must remind them that they are becoming slightly hypoxic (double edge sword effect).

o Explain that as light intensity decreases, colors gradually begin to disappear. Vision usually is affected by the lack of oxygen, and this demonstration will show this to you. Visual acuity decreases by 25% at 10,000 ft.

o Explain that peripheral vision and loss of color vision deteriorate in a darkened environment.

o Have students look around the chamber. Look at the number across from you on the wall.
  o Have students turn cards over and fixate their vision on the mini vision chart in the center.
  o Instruct students to take note of their peripheral vision.
    ◊ “Zs” around periphery of card begin to fade or completely disappear.
    ◊ Turn card over and notice colors on the map (chart).
  o Instruct students to note changes or merging of colors on card.

o 5-minute point:
  o Have students replace oxygen mask (with free hand) while still focusing on center of card.
  o Instruct students to note any changes in their vision.

o 20-second mark:
  o Have students place cards back in their laps and reconnect masks to helmet.
  o Gradually turn up chamber lighting; advise students to shield eyes.
  o End demonstration with questions concerning changes in vision.

o After terminating demonstration, ensure that all students have the regulator in the ON (green switch up) and NORMAL setting (white automix lever down).

o Discuss what happened during the visual acuity demo. Oxygen paradox is a phenomenon that typically occurs during the visual acuity demonstration. Explain why most of the students experienced oxygen paradox, if applicable.

AFTER THE VISUAL ACUITY DEMONSTRATION: Dial up pressure for High-Pressure Demonstration/HIGH-PRESSURE OXYGEN CYLINDER DEMONSTRATION:

Chamber will stay level until students are comfortable with demonstration.

o Have students attach emergency cylinder mockup to their CRU-60/P connector.

o Have students locate the Green apple.

o Have students PULL APPLE 1 to 2 inches – Activate the emergency oxygen system (cylinder).
  o Lean back if you’re getting pressure.
  o Use your positive breathing techniques.
    ◊ Disconnect the CRU-60/P from the ship supply hose.
BEGIN DESCENT TOWARDS GROUND LEVEL:

- Ensure a normal descent rate.
- Remind students to Valsalva.

PASSING THROUGH FL180:

- Dial pressure down.
- Explain about the system. The system lasts 10 minutes. It delivers 10 to 12 L of oxygen. During the first minute, after it’s been activated, it will deliver 12 to 14 inches of water pressure. It will become easier to breathe after a while.
- Tell students to look at bottom of CRU-60/P.
  - Prongs should move when they try to breathe.
  - Plug CRU-60/P back into ship supply hose.

MA-1 PORTABLE OXYGEN ASSEMBLIES DEMONSTRATION:

- Have students practice using MA-1 portable oxygen assemblies (yellow walk around bottles).
- Direct each student to compete a mini PRICE CHECK on the assemblies.
  - Look at bottle, check pressure, inspect CRU-60P receptacle on bottle for foreign objects before making connection.
  - Have students dial in various pressures from bottle.
  - Duration will vary based on altitude, your activity, pressure of bottle prior to use.
- At conclusion of demonstration, ensure that students have plugged CRU-60P connectors back into chamber oxygen supply hose and that all bottles are set to NORMAL configuration.

BELOW 10,000 FT HAVE STUDENTS DROP THEIR MASKS.

REGULATOR INTEGRITY DEMONSTRATION:

- Instruct students in the differences between the old regulator (CRU-68) and the CRU-73 style narrow panel regulator.
- Have students place their regulators in the OFF and NORMAL oxygen position.

NOTE: Students with new CRU-73 style regulator cannot perform this demonstration; they should drop their masks.

- As soon as they realize that they cannot breathe with regulator in this configuration, have them drop mask from right side of face.
- Have students with new style (CRU-73) regulators place their regulator in the OFF and NORMAL position.
- With this style regulator, it is impossible to breathe until you turn regulator to ON position. Have students experience this then remove mask from right side of face.
- At conclusion of demonstration, ensure that students understand the importance of post-flighting any narrow panel regulator in the 100% and OFF configuration.
CONCLUSION OF CHAMBER FLIGHT:

- Instruct them to remain ahead of their ears and sinuses as chamber continues towards ground level.
- Instruct students to remain on intercom until reaching ground level.
- Remind students of “postflight” briefing immediately following flight.

POSTFLIGHT CONSIDERATIONS:

The primary concerns for students and inside observers following exposure to reduced barometric pressure are decompression sickness and ear problems. To minimize potential problems, all crew and students should follow the guidance provided as part of the postflight briefing, including:

- No physical exercise, strenuous activity, or extended duty for 12 hours.
- Personnel may fly as crew or passengers after a chamber flight to or below FL250 as long as cabin altitude remains below 15,000 ft.
- No flying as crew for 12 hours if chamber flight exceeded FL250. Personnel may fly as passengers if cabin altitude remains below 10,000 ft.
- Perform periodic Valsalvas throughout the day/evening to prevent delayed ear blocks.
- Minimize consumption of alcohol for 12 hours after a hypobaric exposure. Alcohol may mask the symptoms of DCS. Increased dehydration can increase DCS risk.
- Monitor your fatigue level. If possible, do not drive great distances without proper rest, use the buddy system, and take frequent breaks.

References

Vocabulary

PRICE CHECK
- Passenger oxygen kit (POK)
- Personal breathing device (PBD)
- Reduced oxygen breathing device (ROBD)
8.6. Acceleration Training

Major Sean Sarsfield, USAF, BSC and Capt Elizabeth Combs, USAF, BSC

8.6.1. Centrifuge Operations

USAF acceleration training is conducted by Aerospace and Operational Physiology personnel assigned to USAFSAM Aerospace & Operational Physiology Division. As of early 2015, this mission is conducted at Brooks City-Base in San Antonio TX. Once the centrifuge at Wright-Patterson AFB is completed, the mission and personnel will transition to Ohio. The Wright-Patterson centrifuge will be a state-of-the-art device that can be utilized for aircrew training and advanced acceleration research. AFI 11-404 governs the USAF acceleration training program.

8.6.2. Training Requirements

Centrifuge training is designed primarily for aircrew (pilots and weapons system officers [WSO]) transitioning to high-G aircraft; however, other personnel that are assigned to active flying billets or that perform unrestricted flight in sustained high-G aircraft (SHGA) are also required to complete centrifuge training. Sustained high-G aircraft are generally defined as any aircraft capable of performing above 6.0 G for longer than 5 seconds and capable of rapid (> 3.0G/sec) G onset. Current U.S. aircraft in this category are the A-10, F-15C/E, F-16, F-22, F-35 and T-38.

The purpose of centrifuge training is to enhance combat capability and safety by optimizing aircrew defense against G-induced Loss of Consciousness (GLOC). This is accomplished by increasing aircrew awareness of GLOC, evaluating their execution of an Anti-G Straining Maneuver (AGSM) in a controlled environment, addressing strategies to improve performance under G-stress, developing an understanding of the importance of proper nutrition, rest, hydration and use of protective equipment. There are myriad publications that highlight the importance of G awareness; one such document is AFPAM 11-419, G Awareness for Aircrew, which is an additional reference for a thorough discussion of G performance.

8.6.3. Training Program Description

Undergraduate Pilot Training (UPT) students that track to the fighter/bomber track require centrifuge training prior to T-38 flight training. This selection occurs between Phase 1 and Phase 2 of UPT. As part of the US program, these students attend Initial Centrifuge Training and will complete the academics and centrifuge profiles as prescribed by AFI 11-404.

Students are required to complete academics, various centrifuge profiles and then debrief with an Aerospace Physiology Officer (APO). Academics cover the physiological effects of acceleration forces, characteristics of GLOC, effective AGSM techniques and the impact of lifestyle (i.e. nutrition, rest, physical conditioning) on G tolerance. During each of the centrifuge profiles, students are evaluated and graded on their performance. Their scores and comments are documented on a student grade sheet. Performance is graded from 0 (Failure) to 4 (Outstanding), and a minimum score of 2 (Average) is required to pass. During debrief, the APO will use the centrifuge video file and the student’s grade sheet to provide performance feedback and highlight areas
for improvement. Students must demonstrate a proficient AGSM at the G load of the aircraft they will fly in order to successfully complete training.

The five Initial Training centrifuge profiles consist of: 1) a gradual onset (0.1 G/sec) profile with no G-suit (to assess resting G tolerance), 2) 4.0 G for 15 seconds rapid (6.0 G/sec) onset with G-suit pressure, 3) 5.0 G for 30 seconds with G-suit pressure, 4) 7.5 G for 15 seconds with G-suit pressure, and 5) a 7.0 G Simulated Air Combat Maneuver (SACM) profile with G-suit pressure. The SACM profile consists of varying G levels from a minimum of 3 G to a maximum of 7 G and requires the student to perform the AGSM in a dynamic high-G flight environment while fatigued. These profiles are designed to be completed in succession and not only test the student’s ability to effectively perform an AGSM at varying G loads, but also their AGSM endurance. Students usually complete the five profiles in one session; however, if a student is unable to pass the T-38 centrifuge profiles on the first attempt, they have three consecutive days (not-including weekends), two sessions per day, to complete the training.

Once students complete the T-38 Initial Training, they are cleared to fly all aircraft that have a 7.5 G cap (i.e. A-10, F-15E and the F-35). For example, if a student receives notification of a follow-on assignment to the F-15E or A-10 no additional centrifuge training is required. However, if a student receives a follow-on assignment to a higher G airframe, advanced centrifuge training leading to Qualification Training upon graduating from UPT is required. Qualification Training is scheduled by the Air Force Personnel Center (AFPC) prior to the Introduction to Fight Fundamentals (IFF) course. Aircraft that require Qualification Training are the F-16, F-15C and the F-22. This training is more operationally focused to sustained high-G aircrew, but consists of the same basic outline of Initial Training. For Qualification Training profiles, training is conducted with an aircraft-specific seat (i.e. 13° or 30° reclined seat) and the maximum G load for the individual centrifuge profiles is adjusted to meet the specific requirements for the different aircraft (i.e. the SACM profile for the F-16 and F-22 consists of a minimum of 3 G and a maximum of 9 G).

Following successful completion of Qualification Training, centrifuge training does not expire. Aircrew do not complete additional centrifuge training unless they are directed to do so by their commander (typically used as a remedial training tool if a pilot ever exhibits problems mitigating the effects of Gs in an aircraft), they upgrade their aircraft (i.e. going from the A-10 to F-16), or they have a break in flying of 36 months or greater. Pilots exceeding the 36 month limit must accomplish Refresher Training prior to starting their flight requalification course.

8.6.4. Training Requirements for Other Personnel

In addition to Initial and Qualification Training for rated aircrew (i.e. pilots and WSOs), acceleration training is also provided for personnel that routinely fly in sustained high-G aircraft. These include any pilot, weapons system officer, flight surgeon, aerospace physiologist, student pilot or navigator in the fighter pipeline or other rated aircrew/operational support flyer assigned to an active flying billet or performing unrestricted flight in a SHGA.

Flight Surgeons (FS) assigned to high-G aircraft are required to successfully complete acceleration training in order to understand the environment and fulfill their monthly flying requirements. However, they do not go through the normal student pilot pipeline training to become qualified. When a FS receives an assignment to a high-G
aircraft squadron, they are required to schedule training directly with the centrifuge. Unlike pilots and WSOs, flight surgeons do not undergo initial training. Instead, they receive qualification training up to the G load that is required for their particular airframe. As with pilots and WSOs, flight surgeons do not need to return to the centrifuge unless they upgrade their aircraft or they go more than 36 months without logging flight time in a SHGA aircraft.

The centrifuge also provides two specialized classes: one for the Resident in Aerospace Medicine (RAM) program and one for the Aerospace and Operational Physiologist Officer (AOPO) course. These courses are conducted annually and are designed to provide a more in depth foundation of acceleration physiology and AGSM execution. Students in these classes get to experience different G suits and COMBAT EDGE configurations. The goal is for each student to experience different aircrew flight equipment (AFE) configurations of the aircraft they may be assigned to support.

8.6.5. Frequently Asked Questions Regarding Centrifuge Exposure.

The following list was compiled based on questions that have been asked by students during centrifuge training. All questions and answers were reviewed by Dr. Richard Sumrall, Lt Col, USAF, MC, CFS, USAF School of Aerospace Medicine, Centrifuge and Altitude Chamber Medical Director. The intent of these FAQs is to offer a brief explanation/answer for educational purposes, not to replace consultation with, or approval from, qualified medical personnel prior to exposure to increased acceleration forces, either during centrifuge training or actual flight.

1. What should I eat the morning of centrifuge training?
   
   Eat something, usually what you would eat before your PT test, but do have a little something in your stomach.

2. Do supplements count as “medications”? Are there any that APOs should be concerned with in regards to students?
   
   Yes, even over-the-counter, “sports,” “healthy,” and “natural” products. Whatever they are called, careful consideration for safety factors should be made prior to use. For centrifuge training, be aware of sports supplements: many have caffeine or other stimulant which could have adverse synergistic effects on cardiovascular system under G load.

3. Is it safe to go into the centrifuge with breast implants?
   
   Yes, but make sure you have good support, even for breast without implants. Jogging and jumping actually puts far more G forces on the body (albeit for a fraction of time) than the centrifuge.

4. Can I wear contact lenses in the centrifuge?
   
   Yes. Spectacles are fine, too.
5. Should I be worried about incontinence in the centrifuge after childbirth?

Most likely not. This would be very similar to other activities where you “bear down” and increase intra-abdominal pressure. If you leak a little with intense pressure, you may do so in the centrifuge with an anti-G straining maneuver, so plan for that possibility. Gz exposure will not cause pelvic floor damage leading to incontinence later on.

6. Are there any restrictions following centrifuge training?

Yes. Per AFI 11-404, there is a 12-hour, automatic duty-not-including-flying (DNIF) period following any centrifuge training for individuals flying as primary aircraft crewmembers due to fatigue and residual motion sickness.
8.7. Spatial Disorientation Training

William Ercoline, Lt Col, USAF (Ret) and Andrew D. Woodrow, Lt Col, USAF (Ret), BSC

8.7.1. Introduction

Although the term “Spatial Disorientation Training” itself has become the standard terminology for training against spatial disorientation (SD), a more appropriate term is arguably “SD Countermeasures Training.” The training we do is a countermeasure for this known pilot killer. Flight schools that provide SD training are actually teaching pilot-trainees how to prevent SD from causing an undesirable result or, better yet, how to overcome it should it occur—and it will occur. As mentioned throughout this text, SD is a natural physiological response to the accelerations and velocities associated with motion while in flight. All pilots experience it at one time or another—at different times and at different intensities—but they all experience it. That’s why we believe any flight training program should include a significant portion on the causes of and countermeasures for SD. This section will stay with convention and will use the term SD Trainer, which leads us to a discussion of the devices that were developed from the discovery of SD through some of the efforts to improve on the SD trainers up to the more current technologies now available for training to counter SD.

8.7.2. Background

The perception of one’s orientation, due to the motions experienced on the ground, appears to have been first described by the noted physicist Dr. Ernst Mach. His knowledge of the vestibular system played a major role in him describing a sensation experienced while riding on a train as it moved around a bend in the tracks. Researchers would eventually term this motion perception as a somatogravic illusion.

I myself became interested in consideration of movement sensation during a research project on fluids containing suspended particles, which one of my students carried out at my suggestion…My view at that time was that the whole body contributed to movement sensation. The supposition of a special organ for movement sensation was far away at that time…A chance happening led me back to the study of motion sensation. I noticed the tilting of houses and trees while I was traveling around a curve in a railroad. This is easily explained if one directly senses the resultant inertial acceleration. Although the physiological side of this subject was still very foreign to me even on reconsideration…this observation was nevertheless sufficient to stimulate my thoughts in their current direction (Mach, 1875).

As Dr. Mach expanded his studies of motion perception, he would eventually study what we now call the somatogyral, somatogravic, oculogravic, oculogyral, and Coriolis illusions. In addition to the vestibular generated perceptions, he modified a Bárány Chair (named after the noted Austrian otologist Robert Bárány), which was used to study the response of the human vestibular system to rotation, to include a rotating striped cylinder. This addition allowed Dr. Mach to examine the effects of the visual system on the vestibular one, likely the first to study interactions of the two important orientation sensory systems. The Bárány chair is still used today to demonstrate the
effects of rotation and head movement on pilot-trainees. Figure 8.7.2-1 is one such device that was used at Brooks AFB by the physiologists who taught there.

![Figure 8.7.2-1. The Bárány Chair](image)

Our understanding of the vestibular system was fairly well studied by the time the airplane came about and The Great War was over, but no one tied these perceptions of motion to what would happen while flying in an airplane. There were numerous observations of peculiar events when coming out of a cloud, but most just assumed it was something they would deal with as they learned to fly. The flight environment remained largely unknown—especially at night or in obscure visibility, i.e. flying without a good visual horizon. At this point in time, there was no need for an SD Trainer because there was no need to better understand SD. It just didn’t happen to “good” pilots.

### 8.7.3. Discovery

The remarkable discovery of SD, which led to the construction of the first SD Trainer, happened in 1926 by a young U.S. Army pilot by the name of Bill Ocker. Bill happened to be taking his semi-annual physical at Crissy Field, CA, while under the medical examination of Dr. David Myers. Dr. Myers gave Bill the usual rotating test in the Bárány chair, and after completing it, Dr. Myers asked him if he’d be willing to participate in a study he was doing. Bill obliged and Dr. Myers rotated Bill in the Bárány chair, but this time he asked Bill to keep his eyes closed. During the rotations, Dr. Myers would query him as to which way he was rotating. In most cases none of the pilots could ever get it right. This repeatedly incorrect perception (now a misperception) bothered Bill very much. Soon after departing Dr. Myers’ office, Bill devised a contraption that he thought would allow him to test his sensation against reality. He rigged up a wooden box about the size of a large shoe box. The box was opened on one end so he could put his head in the box. He mounted a Turn Indicator (some airplanes at the time had such an instrument but no one used it) on the opposite side of the box from where his head would be inserted, and he taped a flashlight in the box so he could see the direction of the turn indicator. He then connected a tube to the back of the Turn Indicator so he could blow on the gyro to make it spin. An example of the modified Turn Indicator used by Ocker is shown in Figure 8.7.3-1.
Once he had put it all together he returned to Dr. Myers to take the test again. This time, instead of using a blindfold, he asked if it would be alright to put his head in the black box. Dr. Myers didn’t mind as long as he couldn’t see what was going on with the motion of the chair. Figure 8.7.3-2 shows the set-up Ocker used for this modified test.

Once all was acceptable by both Ocker and Myers, the test began. Dr. Myers slowly spun Bill in the chair and at the appropriate time (upon slowing down the rotation) he asked Bill which way he was turning. Much to Dr. Myer’s surprise, Bill indicated the correct direction. He did the test again and asked Bill the direction he was turning.
Again Bill responded with the correct answer. No one had been able to do this before! At this point Dr. Myers wanted to know what Bill had in the box. It wasn’t anything special, other than Bill was reporting what he saw on the Turn Indicator and not what he was sensing his body was doing. This was the first time, internationally, never challenged discovery of what would eventually be called spatial disorientation. Figure 8.7.3-3 shows the illustration from the patent Bill received about 2 years after filing for the patent. There were several made which varied in size and portability.

![Illustration from the Vertigo Stopper Box Patent](image)

**Figure 8.7.3-3. Illustration from the Vertigo Stopper Box Patent**

Unfortunately, Bill met a lot of resistance when he tried to get pilots to use the device. He wanted to show them that you could not tell correctly your orientation if you relied solely on your body’s sensation. At the time, pilots flew by the seat of their pants and did not need instruments. Most thought he was crazy and didn’t want anything to do with his contraption. Bill was soon assigned from Crissy Field, CA, to Brooks Field, TX, where he wouldn’t have such a negative impact on experienced pilots. Brooks Field was the pilot training base for the U.S. Army Air Corps at the time. However, Bill did find a few allies. The School of Aviation Medicine (SAM) also happened to be located at Brooks Field at this time, and the instructors from the school knew Bill was on to something important. They wanted to learn more about this misperception thing Bill had discovered. His device, referred to many as the Ocker Box, became a routine part of the school curriculum (much to the dismay of the pilots). Figure 8.7.3-4 shows one of the early SAM academic classes showing the instructor demonstrating the erroneous sensation, which would eventually be termed spatial disorientation (sometime around WWII).
8.7.4. Development of SD Training Devices

Over the years, more SD trainers would evolve with a variety of setups, but they all had one thing in common. They all had continual rotation which mimicked the basic motion of the Bárány chair. At about this same time flight simulation was beginning to evolve into a remarkable ground-based training tool for pilots. SD Trainers never seemed to receive the same level of importance as the flight simulators. SD Trainers were for the medical community, not the operational side. It is ironic more of the early Link Trainers were not used for demonstrating SD because they had the motion platform to do so. Most likely it was due to the negative attitude the pilots had for ground-based training as a whole. SD Trainers were just something the medical corps had to show pilots they knew something about an airplane.

The medical corps continued to develop these devices independent of the flight simulation industry. Some of the more successful SD trainers are shown in the figures below. There were many problems with these devices but the idea of an SD trainer had emerged and quickly became a part of Specialized Undergraduate Pilot Training (SUPT) because of all the accidents and many associated with misperceptions. The pros and cons of these devices would take an entire chapter to review. Let’s just look at this technology and leave it at that. Figures 8.7.4-1 through 8.7.4-6 were all SD Trainers used by the U.S. Air Force School of Aerospace Medicine (USAFSAM) at one time or another, and a couple were used for training pilots during pilot training.
Figure 8.7.4-1. The USAFSAM Vista-Vertigon®

Figure 8.7.4-2. The USAFSAM Spatial Disorientation Demonstrator, circa 1964
Figure 8.7.4-3. The USAFSAM Spatial Orientation Trainer, circa 1968

Figure 8.7.4-4. The Multi-Station Disorientation Demonstrator (U.S. Navy), circa 1965
As you may be able to tell from the evolution of these SD trainers, there was always an effort to make the devices more and more like the airplane. The technology pushed the limits of the motion systems and the visual displays. Many were criticized for failing to be anything like the airplane, so the use and acceptability of the operational community was always tentative at best. Even after the long and thorough development of the Advanced Spatial Disorientation Trainer and/or Demonstrator (ASDT/ASDD) (Figure 8.7.4-6), the operational community still doubted the validity of such a device. Although it created motion cues needed for many of the vestibular illusions and most of the visual illusions, the need for such a device dwindled as the number of SD-related mishaps decreased (the rate never decreased, only the numbers). Except for one unit at Randolph AFB, TX, this type of SD trainer was never placed into operational use. The primary reasons were cost and need. The ASDT was removed from Randolph AFB in 2004 and replaced with the General Aviation Trainer (GAT) II (Figure 8.7.5-1).

Figure 8.7.4-5. The USAFSAM Vertifuge, circa 1980
8.7.5. Current Devices

There were several other SD trainers under development when the USAF had an interest in finding an alternative to the Advanced Spatial Disorientation Trainer. The interest within the physiology career field varied somewhat but there was always enough interest to ask the question as to the status of the SD Trainers. This interest usually peeked after an SD accident. Because the interest within the DoD decreased, the commercial companies who developed this technology were left to find a market outside of the military flying community. A few of the SD Trainers that did make it to the market are shown in Figures 8.7.5-1 through 8.7.5-4. They are all made by either Environmental Tectonics Corporation (ETC) of Southampton, PA, or AMST of Ranshofen, Austria.
Figure 8.7.5-2. GYRO IPT II™ Spatial Disorientation Trainer (by ETC)—Currently Being Used in USAF SUPT

Figure 8.7.5-3. The DISO Trainer (by AMST)
8.7.6. Future of SD Trainers

The two companies mentioned above have also developed what is arguably the most complex motion platform anywhere in the world. They were specifically designed to study the causes of and countermeasures for SD. It is difficult to imagine a more complete SD trainer. Two of these devices exist—one at WPAFB, OH and the other in Soesteberg, Netherlands (NL). The device at Wright-Patterson AFB was made by ETC and the device at Soesteberg was made by AMST. Although the cost is expensive compared to the earlier SD trainers, it has the best capabilities to date for exposing pilots to SD. Their operational relevance will be determined in the next several years.
8.7.7. Current U.S. Air Force Spatial Disorientation Training Program

8.7.7.1. Philosophy and Rationale for Training. Beginning in May 2015, the United States Air Force began bringing advanced spatial disorientation training to pilot candidates across Specialized Undergraduate Flying Training (SUPT) in Air Education and Training Command. The devices were installed at Sheppard AFB TX, followed by Vance AFB OK, Columbus AFB MS, Laughlin AFB TX, and Joint Base San Antonio-Randolph. Inclusion of the devices will provide all pilot candidates and instructor pilots formal SD events as part of their syllabus.

8.7.7.2. Program Description. In an effort between Air Education and Training Command and the Air Force Life Cycle Management Center, a contract was signed with Environmental Tectonics Corporation (ETC) to provide five GYRO IPT II™ (Integrated Physiological Trainer) spatial disorientation flight simulators to pilot training bases (Figure 8.7.5-2). In conjunction with classroom academics, Bárány chair demonstrations, and in-flight SD demonstrations, the GYRO IPT II™ will support the SD training requirements in the pilot training syllabus by exposing each student to typical vestibular and visual illusions found in aviation, enabling them to recognize, confirm, prevent, and recover from SD before they strap into an aircraft. The GYRO IPT II™ will establish a solid foundation of recognition and recovery from illusions at the earliest instructional point of pilot preparation, ultimately leading to increased safety as the pilot advances to other aircraft platforms.

ETC’s GYRO IPT II™ cockpit assembly resembles a T-6A Texan II cockpit and provides the student with a hands-on, realistic, full motion, SD flight training experience (Figure 8.7.7.2-1).

While in control of a simulated flight, the pilot can be exposed to a variety of selected disorienting illusions. Unlike simple disorientation demonstrators, a pilot in the GYRO IPT II™ has full closed loop control of the simulation before, during, and after the illusion. This capability creates a fully interactive flight training environment where the pilot must maintain control of the simulator and fly through the illusion to a successful resolution during training.
Prior to 2015, spatial disorientation training was limited to 2.5 hours of academic instruction and Bárány chair demonstration as part of the undergraduate flying training curriculum. The curriculum directs the instructor to “…demonstrate types of spatial disorientation…” and for the student to “…practice/perform methods to maintain aircraft control while disorientated; this will include the leans and nystagmus.” Limitations of transferring classroom-based experience to the flight environment reduced the practical application of combating SD for new pilots. And further, the foundation of understanding SD (“recognize and recover”) relied upon the skill of the instructor pilot (IP) to discuss and demonstrate the concept in-flight. The advent of advanced spatial disorientation in undergraduate pilot training puts in place a realistic training device and, equally as important, a requirement to complete the training at key stages of flight training.

The spatial disorientation program includes, in addition to the classroom academic and demonstrations, flight training with in-flight SD demonstrations during instrument flight sorties. Before the first night mission, instructor pilots brief the students on problems associated with night flying with particular emphasis on spatial disorientation. The inclusion of the GYRO IPT II™ in the T-6 syllabus aims to increase exposure to SD illusions early in the pilot training timeline and includes advanced training prior to the first night sorties.

References
8.8 Aircraft Identification

Capt James A. Dreibelbis, USAF, BSC

8.8.1. Aircraft Mission Design Series

The current designation system for identifying U.S. military aircraft was introduced by the Department of Defense on 1961 (AFI 16-401). This joint system of mission-based designations has been used, with most of these restarting from 1. Various previously-designated models from the pre-1962 Army-Air Force system were not re-designated. The Secretary of the Air Force serves as the DoD Executive Agent for the MDS program, and has delegated this authority to HAF/A8PE (AFI 16-401, 2014). This systematic approach provides guidance and procedures for naming defense military aerospace vehicles from all military branches. According to Air Force Instruction 16-401 (also known as Army Regulation 70-50 and NAVAIRINST 13100.16), Designating and Naming Defense Military Aerospace Vehicles, “All aircraft operated by U.S. military services are to receive an official designation” (Parsch, 2014). Each branch of the military follows this guidance, but each does operate a few specific aircraft under the manufacturers’ original designations.

8.8.2. The Designation System

A “Mission Design Series” designation, or MDS, is the official designation given to all U.S. military aerospace vehicles. It represents a specific category of vehicles for operations, support, and documentation purposes (AFI 16-401, 2014). The system uses letters and numbers to symbolize identifying characteristics of specific DoD aerospace vehicles. The alpha-numeric designation describes the vehicle in two components: the first, comprised only of alpha characters, describes the mission, and the second, comprised of both alpha-numeric characters, the design number and series. An example of an MDS will appear as, F-16, which will be further discussed below.

8.8.3. Basic Mission Designation

The basic mission of an aircraft identifies its primary function and capability and is the initial symbol assigned to that series. For standard vehicles, it appears to the immediate left of the design number separated by a dash. Vehicles can have the designation of either basic or modified mission. The basic mission symbols used for DoD aircraft are listed in Table 8.8.3-1.
### Table 8.8.3-1. Basic Mission Designation Symbols for DoD Aircraft

<table>
<thead>
<tr>
<th>Basic Mission Designation Letter</th>
<th>Basic Mission Designation Role</th>
<th>Description of Basic Mission Symbols</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Attack</td>
<td>Find, Attack, Destroy enemy targets using conventional or special weapons; Also describes aircraft use for interdiction and close air support missions.</td>
</tr>
<tr>
<td>B</td>
<td>Bomber</td>
<td>Bombing of enemy targets</td>
</tr>
<tr>
<td>C</td>
<td>Transport</td>
<td>Carry personnel, cargo, or both</td>
</tr>
<tr>
<td>E</td>
<td>Special Electronic Mission</td>
<td>1: Electronic countermeasures 2: Airborne early warning radar 3: Airborne command and control including communications relay 4: Tactical data communications link</td>
</tr>
<tr>
<td>F</td>
<td>Fighter</td>
<td>Intercept and destroy other aircraft/missiles</td>
</tr>
<tr>
<td>L</td>
<td>Laser-Equipped</td>
<td>Employing high-energy laser weapon</td>
</tr>
<tr>
<td>O</td>
<td>Observation</td>
<td>Observe (through visual or other means) and report tactical information concerning composition/disposition of forces</td>
</tr>
<tr>
<td>P</td>
<td>Patrol/Maritime Patrol</td>
<td>Long range, all weather, multiengine aircraft operating from land or water bases designed for independent submarine warfare, maritime reconnaissance, and mining</td>
</tr>
<tr>
<td>R</td>
<td>Reconnaissance</td>
<td>Photographic or electronic reconnaissance missions</td>
</tr>
<tr>
<td>S</td>
<td>Antisubmarine Warfare</td>
<td>Find, detect, identify, attack, and destroy enemy submarines</td>
</tr>
<tr>
<td>T</td>
<td>Trainer</td>
<td>Training aircraft</td>
</tr>
<tr>
<td>U</td>
<td>Utility</td>
<td>Perform multiple missions such as battlefield support, localized transport, and special light missions; included are aircraft designated for small payloads.</td>
</tr>
<tr>
<td>X</td>
<td>Special Research</td>
<td>Testing highly experimental configurations; Not generally intended as operational aircraft</td>
</tr>
</tbody>
</table>


The modified mission will be another letter to the left of a basic mission symbol (i.e. AC-130). This is utilized when the aircraft is used for another purpose than originally designed. Typically, only one modified mission symbol will be attached to a basic mission symbol, but this rule has been adjusted for certain aircraft over the years. The modified mission symbols used for DoD aircraft are listed in Table 8.8.3-2.
### Table 8.8.3-2. Modified Mission Designation Symbols for DoD Aircraft

<table>
<thead>
<tr>
<th>Basic Mission Designation Letter</th>
<th>Basic Mission Designation Role</th>
<th>Description of Basic Mission Symbols</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Attack</td>
<td>Modified to find, Attack, Destroy enemy targets using conventional or special weapons; Also describes aircraft use for interdiction and close air support missions.</td>
</tr>
<tr>
<td>C</td>
<td>Transport</td>
<td>Modified to carry personnel, cargo, or both</td>
</tr>
<tr>
<td>D</td>
<td>Drone Director</td>
<td>Modified for controlling drone aircraft or missiles</td>
</tr>
</tbody>
</table>
| E                               | Special Electronic Installation | Modified for:  
1: Electronic countermeasures  
2: Airborne early warning radar  
3: Airborne command and control including communications relay  
4: Tactical data communications link |
| F                               | Fighter                         | Modified to intercept and destroy other aircraft/missiles |
| H                               | Search/Rescue/MEDEVAC           | Modified for search and rescue and/or MEDEVAC missions |
| K                               | Tanker                          | Modified to refuel other aircraft in flight |
| L                               | Cold Weather                    | Modified for operation in Arctic and Antarctic regions; Includes skis, special insulation, and other cold weather operations equipment |
| M                               | Multi-Mission                   | Modified to perform several different missions |
| O                               | Observation                     | Modified to observe (through visual or other means) and report tactical information concerning composition/disposition of forces |
| P                               | Patrol/Maritime Patrol          | Modified for long range, all weather, multiengine aircraft operating from land or water bases designed for independent submarine warfare, maritime reconnaissance, and mining |
| Q                               | (Unmanned) Drone                | Modified for remote or automatic control; powered aerial vehicle that does not carry a human operator, uses aerodynamic forces to provide vehicle life, can fly autonomously or is piloted remotely, can be expendable or recoverable, and can carry lethal or nonlethal payloads. Ballistic or semi-ballistic vehicles, cruise missiles, and artillery projectiles are not considered drones. |
| R                               | Reconnaissance                  | Modified for photographic or electronic reconnaissance missions |
| S                               | Antisubmarine Warfare           | Modified to find, detect, identify, attack, and destroy enemy submarines |
| T                               | Trainer                         | Modified training aircraft |
| U                               | Utility                         | Modified to perform multiple missions such as battlefield support, localized transport, and special light missions |
| V                               | Staff Transport (POTUS/VPOTUS/FLOTUS) | Modified to provide support for the President/Vice President/First Lady of the United States |
| W                               | Weather Reconnaissance          | Modified and equipped for meteorological conditions |

8.8.4. Design Number Designation

Each aircraft mission symbol is accompanied with a series of design numbers. The design numbers will appear to the immediate right of the basic/modified mission or vehicle type symbols, separated by a dash. For the previous example of F-16C, the design number “16” is the sixteenth MDS requested for fighter aircraft under the current system (USAFSAM, 2015). These numbers identify the major design changes to the aircraft but still uphold the same basic mission. The design numbers run consecutively from 1-999. These numbers are usually assigned in a strict numerical sequence without any reference to the original manufacturers’ model numbers, but again some aircraft will violate this guidance. The next aircraft design number will not always be the next chronological number (i.e. F-22 followed by F-35). Sometimes numbers are completely skipped in a series to avoid confusion because they can be in use by another series at the same time (i.e. C-34 was skipped due to conflict with T-34) (Parsch, 2006).

8.8.5. Series Designation

Variants of the basic or modified aircraft type are designated by a suffix letter. This letter identifies the production model of a specific design number that represents major modifications that will significantly alter or improve the operational use of the aircraft. The series modification can include altered systems, cockpit ergonomics and design, logistics support, and ordnance systems/capacity (USAFSAM, 2015). The series designation will consist of consecutive symbols starting with letter “A” and will appear to the immediate right of the design number (not separated by a dash). For example, the F-16A variant (first production model of the F-16) will precede the more recently altered F-16C (third production model of the F-16) variant. Letters are intended to run in sequence but there are many aircraft designations with out-of-sequence suffixes. It is also important to note that an earlier production of a certain series aircraft can be much different from a more current production of that same aircraft (i.e. early production of F-16C vs. newer production of F-16C).

8.8.6. Vehicle Type

Vehicle type is the process of naming non-standard aircraft only. A vehicle type symbol must accompany the basic mission type symbol. An example is the CV-22A Osprey, where the basic mission type is Cargo (C) and the vehicle type is VTOL/STOL (V). Table 8.8.6-1 lists the vehicle type symbols.
Table 8.8.6-1. Vehicle Type Designation Symbols for DoD Aircraft

<table>
<thead>
<tr>
<th>Vehicle Type Designation Letter</th>
<th>Vehicle Type Designation</th>
<th>Specifications</th>
</tr>
</thead>
<tbody>
<tr>
<td>D</td>
<td>UAV Control Segment</td>
<td>Applies to ground control equipment for unmanned aerial vehicles</td>
</tr>
<tr>
<td>G</td>
<td>Glider</td>
<td>Includes motor gliders, routinely used in unpowered flight</td>
</tr>
<tr>
<td>H</td>
<td>Helicopter</td>
<td>Helicopter</td>
</tr>
<tr>
<td>Q</td>
<td>UAV (Unmanned Aerial Vehicle)</td>
<td>Reusable unmanned aerial vehicle operations</td>
</tr>
<tr>
<td>S</td>
<td>Spaceplane</td>
<td>Aircraft that can operate both within and outside of the atmosphere</td>
</tr>
<tr>
<td>V</td>
<td>VTOL/STOL</td>
<td>Vertical-Takeoff and Landing (hover, forward/backward/lateral flight)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Short-Takeoff and Landing (short runway requirement for takeoff and landing)</td>
</tr>
<tr>
<td>Z</td>
<td>Lighter-Than-Air Vehicle</td>
<td>Non-rigid or semi-rigid aircraft that gains lift from hot gases or lighter than air gases (Blimps and Balloons)</td>
</tr>
</tbody>
</table>


8.8.7. Block Number

Block numbers are used to further identify specific vehicles. Block numbers are used for some production aircraft but not all. They denote a production group of identical aircraft within a given design series. From the prior example, the F-16C Blocks 25 – 32 was put into operation in 1981, the F-16C Blocks 40 – 42 in 1989, and the F-16C Blocks 50 – 52 in 1994 (Air Force Fact Sheets, 2015). AFI 16-401 currently indicates block numbers as option and do not state any specific rules for their actual application. Depending on the type of aircraft, but the USAF does not refer to the typical dash nomenclature when adding blocks to the MDS. The “F-16C Block 50” will appear as such and not as “F-16C-50” (Parsch, 2014). Please refer to aircraft specific Dash-1 for more information.

8.8.8. Popular Name

Many U.S. Military aircraft have a “popular name” or nickname, associated with the official name of the aircraft. This name is not the official manufacturer or DoD-given name of the vehicle but has been verified through an approval process to check for conflicts with existing names and political correctness. The official name of the aircraft tends to be disregarded by the aviator and the nickname becomes more prevalent in the community. An example is the F-16, which has the official name as the Fighting Falcon but is more commonly known as a “Viper.” The F-15 Eagle is also known as the F-15E Strike Eagle or “Stikes.” In the heavy aircraft community, the C-130H is known as the “Hercules” or “Herc” and the B-52 is commonly referred to as the “BUFF.”
8.8.9. Vertical Stabilizer Information

The vertical stabilizer, or tail flash or tail codes, of military aircraft often contain markings that help to identify the unit operating the aircraft, the base assigned, and information relating to the aircraft manufacture date. Since 1993, all USAF components, including Air Force Reserve and Air National Guard (ANG), utilize the tail flash system. Two large letters identify the home base, or in some organizations, an historic legacy, such as "FF" ("First Fighter") for the 1st Fighter Wing or "WP" ("Wolf Pack") for the 8th Fighter Wing. Air National Guard units usually use the two-letter state abbreviation as a tail code, although there are exceptions. The tail marking also contains two digits indicating the fiscal year the aircraft was ordered, followed by the last three digits of the airframe's serial number (Figure 8.8.9-1).

![Figure 8.8.9-2. Detail of Tail Flash Information](image)

Air Mobility Command (AMC) aircraft do not use two-letter identification codes, but instead have the name of the base written inside the tail flash. AMC aircraft also use a different standard to identify the aircraft serial number. They use a 5-digit number in which the first digit represents the last digit of the fiscal year (FY) and the remaining digits identify the 4-digit sequence number (Parsch, 2014). Figure 8.8.9-2 shows the base abbreviation code, as well as aircraft type and unit location for aircraft being operated by the U.S. Air Force in 2005 (Airman Magazine, 2005).
<table>
<thead>
<tr>
<th>Code</th>
<th>Aircraft</th>
<th>Location and Command</th>
</tr>
</thead>
<tbody>
<tr>
<td>AC</td>
<td>F-16BC/D, T-38A, T-38G</td>
<td>177th Fighter Wing, Atlantic City AFB, N.J. (ATC)</td>
</tr>
<tr>
<td>AK</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>1441st AW, Bolling AB, D.C. (ATC)</td>
</tr>
<tr>
<td>AL</td>
<td>E-13EH, E-12C, E-12D, E-12G</td>
<td>177th Wing, Kingsville AFB, Texas (ATC)</td>
</tr>
<tr>
<td>AM</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>177th Wing, Detroit ANGB, Michigan (ATC)</td>
</tr>
<tr>
<td>AN</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>173rd Wing, Orlando ANGB, Florida (ATC)</td>
</tr>
<tr>
<td>AO</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>171st Wing, Sheppard AFB, Texas (ATC)</td>
</tr>
<tr>
<td>AP</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>169th Wing, Nellis AFB, Nevada (ATC)</td>
</tr>
<tr>
<td>AR</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>146th Wing, Chicago AFB, Illinois (ATC)</td>
</tr>
<tr>
<td>AS</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>156th Wing, Sheppard AFB, Texas (ATC)</td>
</tr>
<tr>
<td>AT</td>
<td>E-13EH, E-13C/DC</td>
<td>152nd Wing, Goodfellow AFB, Texas (ATC)</td>
</tr>
<tr>
<td>AU</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>153rd Wing, Keesler AFB, Mississippi (ATC)</td>
</tr>
<tr>
<td>AW</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>159th Wing, Keesler AFB, Mississippi (ATC)</td>
</tr>
<tr>
<td>AX</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>148th Wing, Ellington AFB, Georgia (ATC)</td>
</tr>
<tr>
<td>BX</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>147th Wing, Seymour Johnson AFB, North Carolina (ATC)</td>
</tr>
<tr>
<td>C1</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>146th Wing, Chicago AFB, Illinois (ATC)</td>
</tr>
<tr>
<td>C2</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>142nd Wing, Westover AFB, Massachusetts (ATC)</td>
</tr>
<tr>
<td>C3</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>143rd Wing, Campbellsburg ANG, Indiana (ATC)</td>
</tr>
<tr>
<td>C4</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>141st Wing, McChord ANG, Washington (ATC)</td>
</tr>
<tr>
<td>C5</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>140th Wing, Mountain Home AFB, Idaho (ATC)</td>
</tr>
<tr>
<td>C6</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>139th Wing, Rexford ANG, New York (ATC)</td>
</tr>
<tr>
<td>C7</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>138th Wing, McAlester ANG, Oklahoma (ATC)</td>
</tr>
<tr>
<td>C8</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>137th Wing, Mountain Home AFB, Idaho (ATC)</td>
</tr>
<tr>
<td>C9</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>136th Wing, Niagara Falls ANG, New York (ATC)</td>
</tr>
<tr>
<td>C10</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>135th Wing, Fairchild ANG, Washington (ATC)</td>
</tr>
<tr>
<td>C11</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>134th Wing, Memphis ANG, Tennessee (ATC)</td>
</tr>
<tr>
<td>C12</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>133rd Wing, Scott AFB, Illinois (ATC)</td>
</tr>
<tr>
<td>C13</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>132nd Wing, Dyess AFB, Texas (ATC)</td>
</tr>
<tr>
<td>C14</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>131st Wing, Keesler AFB, Mississippi (ATC)</td>
</tr>
<tr>
<td>C15</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>130th Wing, Keesler AFB, Mississippi (ATC)</td>
</tr>
<tr>
<td>C16</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>129th Wing, Arkansas Air National Guard (ATC)</td>
</tr>
<tr>
<td>C17</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>128th Wing, Keesler AFB, Mississippi (ATC)</td>
</tr>
<tr>
<td>C18</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>127th Wing, Keesler AFB, Mississippi (ATC)</td>
</tr>
<tr>
<td>C19</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>126th Wing, Keesler AFB, Mississippi (ATC)</td>
</tr>
<tr>
<td>C20</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>125th Wing, Keesler AFB, Mississippi (ATC)</td>
</tr>
<tr>
<td>C21</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>124th Wing, Keesler AFB, Mississippi (ATC)</td>
</tr>
<tr>
<td>C22</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>123rd Wing, Keesler AFB, Mississippi (ATC)</td>
</tr>
<tr>
<td>C23</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>122nd Wing, Keesler AFB, Mississippi (ATC)</td>
</tr>
<tr>
<td>C24</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>121st Wing, Keesler AFB, Mississippi (ATC)</td>
</tr>
<tr>
<td>C25</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>120th Wing, Keesler AFB, Mississippi (ATC)</td>
</tr>
<tr>
<td>C26</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>119th Wing, Keesler AFB, Mississippi (ATC)</td>
</tr>
<tr>
<td>C27</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>118th Wing, Keesler AFB, Mississippi (ATC)</td>
</tr>
<tr>
<td>C28</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>117th Wing, Keesler AFB, Mississippi (ATC)</td>
</tr>
<tr>
<td>C29</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>116th Wing, Keesler AFB, Mississippi (ATC)</td>
</tr>
<tr>
<td>C30</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>115th Wing, Keesler AFB, Mississippi (ATC)</td>
</tr>
<tr>
<td>C31</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>114th Wing, Keesler AFB, Mississippi (ATC)</td>
</tr>
<tr>
<td>C32</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>113th Wing, Keesler AFB, Mississippi (ATC)</td>
</tr>
<tr>
<td>C33</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>112th Wing, Keesler AFB, Mississippi (ATC)</td>
</tr>
<tr>
<td>C34</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>111th Wing, Keesler AFB, Mississippi (ATC)</td>
</tr>
<tr>
<td>C35</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>110th Wing, Keesler AFB, Mississippi (ATC)</td>
</tr>
<tr>
<td>C36</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>109th Wing, Keesler AFB, Mississippi (ATC)</td>
</tr>
<tr>
<td>C37</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>108th Wing, Keesler AFB, Mississippi (ATC)</td>
</tr>
<tr>
<td>C38</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>107th Wing, Keesler AFB, Mississippi (ATC)</td>
</tr>
<tr>
<td>C39</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>106th Wing, Keesler AFB, Mississippi (ATC)</td>
</tr>
<tr>
<td>C40</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>105th Wing, Keesler AFB, Mississippi (ATC)</td>
</tr>
<tr>
<td>C41</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>104th Wing, Keesler AFB, Mississippi (ATC)</td>
</tr>
<tr>
<td>C42</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>103rd Wing, Keesler AFB, Mississippi (ATC)</td>
</tr>
<tr>
<td>C43</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>102nd Wing, Keesler AFB, Mississippi (ATC)</td>
</tr>
<tr>
<td>C44</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>101st Wing, Keesler AFB, Mississippi (ATC)</td>
</tr>
<tr>
<td>C45</td>
<td>C-130J, E-13EH, E-13C/DC</td>
<td>100th Wing, Keesler AFB, Mississippi (ATC)</td>
</tr>
</tbody>
</table>

**Figure 8.8.9-2. U.S. Air Force Aircraft Tail Markings, 2005 (Airman Magazine, 2005)***

*Note AMC aircraft do not have tail code markings.*

**This issue:** 149th FW, San Antonio, TX. (AN)
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United States Air Force School of Aerospace Medicine (USAFSAM). Aerospace and Operational Physiologist Officer Course 101 Study Guide. 2015.
8.9. Flight Principles and Terminology

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8.9.1. Basic Terminology

The purpose of this chapter is to introduce basic flight terminology and aerodynamic principles. With the ever-changing improvements and modernization of aircraft, it is important that we understand the blueprints of the flying community and the procedures they are expected to follow. By recognizing basic terminology and the aerodynamic forces, we will gain a better understanding of the stresses placed on both the aviator and the aircraft.

8.9.1.1. Dash “-1”. An official United States Air Force Technical Order (T.O.) is the basic flight manual. The proper usage of “-1” will be preceded by aircraft designation (i.e. T.O. 1-T6A-1) and is considered to be the pilot’s guide to the aircraft. The -1 contains descriptive information about the aircraft systems, normal procedures, emergency procedures, auxiliary equipment, operating limitations, flight characteristics, systems operation, and performance information. Refer to specific aircraft MDS for Dash -1 guidance.

8.9.1.2. Emergency Procedures (EPs). Procedures requiring immediate action to avoid damage to the aircraft and/or the crewmember(s) are required to be committed to memory (exact wording) and tested on every check ride that crewmembers receive each year. The procedures in many aircraft, can each involve many steps and number over 10. EPs can often be found on the crewmember(s) boldface checklist carried with them during flight as well as the full emergency procedure checklist binder per that specific aircraft. See Figure 8.9.1.2-1 for an example of in EP checklist (Dauntless Aviation, 2016).
8.9.1.3. Flight Checklists. Checklists have been the foundation of pilot standardization and cockpit safety for years. The checklist is an aid to the memory and helps to ensure that critical items necessary for the safe operation of aircraft are not overlooked or forgotten. They are a step-by-step, guide and their items are usually numbered and state a control and an action required. The action required is verbal acknowledgement of procedures for pre-flight/in-flight/post-flight guidance from both the Instructor Pilot and Student Pilot and/or all crewmembers. Typically, in multi-place aircraft, the pilot will perform the items read by the co-pilot or other crewmember(s) and respond with the result of that item’s accomplishment (i.e. Co-pilot: “Throttles”, Pilot: “Idle”). Sometimes the checklist is accomplished and a “clean-up” reading of the checklist is accomplished by the co-pilot. All crewmembers will have specific checklists for guidance per their role on the aircraft. Failure to accomplish checklists can result in inappropriate settings critical to flight or other ground operations. An example is shown in Figure 8.9.1.2-2 (Dauntless Aviation, 2016).
Cessna 182T Checklist

PREFLIGHT INSPECTION

CABIN
1. Pilot Tube Cover - REMOVE, check opening for blockage
2. Documents (AROW) - AVAILABLE IN THE AIRPLANE
3. OKWG Form 761A - CHECK
4. OKWG Form 761 - ENTER Hobbs and Tach Times
5. Parking Brake - SET
6. Control Wheel Lock - REMOVE
7. Ignition Switch - OFF, Key Removed
8. Avionics Master Switch - OFF

WARNING
When turning on the master switch, using an external power source, or pulling the propeller by hand, treat the propeller as if the ignition switch were on. Do not stand, nor allow anyone else to stand, within the arc of the propeller, since a loose or broken wire or a component malfunction could cause the propeller to rotate. Hand propped starts are prohibited by CAPR 60-1.

9. Master Switch - ON
10. Fuel Quantity Indicators - CHECK QUANTITY and ENSURE LOW FUEL ANNUNCIATORS (L LOW FUEL R) ARE EXTINGUISHED
11. Avionics Master Switch - ON
12. Avionics Cooling Fan - CHECK AUDIBLY FOR OPERATION
13. Avionics Master Switch - OFF
14. Static Pressure Alternate Source Valve - OFF
15. Annunciator Panel Switch - PLACE and HOLD IN TST POSITION and ensure all annunciators illuminate
16. Annunciator Panel Test Switch - RELEASE. Check that appropriate annunciators remain on

N-1

TAXI
1. Brakes - CHECK
2. Nose Wheel Steering - CHECK
3. Cross Wind Controls - APPLY

BEFORE TAKEOFF

1. Parking Brake - SET
2. Passenger Seat Backs - MOST UPRIGHT POSITION
3. Seats and Seat Belts - CHECK SECURE
4. Cabin Doors - CLOSED and LOCKED
5. Flight Controls - FREE and CORRECT
6. Flight instruments - CHECK and SET
7. Fuel Quantity - CHECK
8. Mixture - RICH
9. Fuel Selector Valve - RECHECK BOTH
10. Elevator and Rudder Trim - SET for Takeoff
11. Throttle - 1800 RPM
   a. Magnetos - CHECK (rpm drop should not exceed 150 rotor on either magneto or 50 RPM differential between magnetos)
   b. Propeller - Cycle from High to Low RPM, Return to High
   c. Vacuum Gage - CHECK
   d. Engine Instruments and Ammeter - CHECK
12. Annunciator Panel - ENSURE no annunciators are illuminated
13. Throttle - CHECK IDLE
14. Throttle Friction Lock - ADJUST
15. Strobe Lights - AS DESIRED
16. Pulse Light - ON
17. Radios and Avionics - SET
18. Transponder - ALT
19. NAV/GPS Switch (if installed) - SET
20. Autopilot (if installed) - OFF
21. Wing Flaps - SET for Takeoff (0° to 20°)

N-7

17. Fuel Selector Valve - BOTH
18. Flaps - EXTEND
19. Pitot Heat - ON (Carefully check that pitot tube is warm to touch within 30 seconds)
20. Stall Warning - CHECK (Push Vane Upward)
22. Pitot Heat - OFF
23. Master Switch - OFF
24. Trim Controls - NEUTRAL
25. Baggage Compartments - INVENTORY, SECURE CONTENTS - Tow Bar, Chocks, Pitot Tube Cover, Ladder, First Aid Kit, Tie Down Straps and Ropes, Survival Kit, Fuel Sampler, Cleaning Materials, 1 QT Oil, Landing/Taxi Light Bulbs, Avionics Control Lock
26. Baggage Door - CHECK, Lock with Key

EMPENNAGE
1. Ruddor Gust Lock (if installed) - REMOVE
2. Tail Tie-Down - DISCONNECT
3. Control Surfaces - CHECK freedom of movement and security
4. Trim Tab - CHECK security
5. Antennas - CHECK for security of attachment and general condition

RIGHT WING Trailling Edge
1. Flap - CHECK for security and condition
2. Aileron - CHECK freedom of movement and security

RIGHT WING
1. Nav and Strobe Light - CHECK VISUALLY
2. Wing Tie-Down - DISCONNECT
3. Main Wheel Tire - CHECK for proper inflation and general condition (42 PSI)

N-2

23. Takeoff Checklist - REVIEWED
   VR 55 KIAS Vx 65 KIAS Vy 82 KIAS Best Glide 75 KIAS
   Heading/Altitude After Takeoff - REVIEW
24. Takeoff Emergencies Briefing - COMPLETE ENGINE FAILURE OR SYSTEM MALFUNCTION
   Before VR: Stop Aircraft on Runway
   After VR: Land on remaining runway or straight ahead with only small turns. Flaps as necessary to slow touchdown speed.
25. Cowl Flaps - OPEN
26. Brakes - RELEASE

TAKEOFF

NORMAL TAKEOFF
1. Wing Flaps - 0°-20°
2. Power - FULL THROTTLE and 2400 RPM
3. Mixture - RICH (mixture may be leaned to Maximum Power Fuel Flow placard value)
4. Elevator Control - LIFT NOSE WHEEL (at 50-60 KIAS)
5. Climb Speed - 70 KIAS (Flaps 20°)
6. 80 KIAS (Flaps 0°)
6. Wing Flaps - RETRACT

Short Field Takeoff
1. Wing Flaps - 20°
2. Brakes - APPLY
3. Power - FULL THROTTLE and 2400 RPM
4. Mixture - Lean to obtain Maximum Power Fuel Flow
5. Brakes - RELEASE
6. Elevator Control - MAINTAIN SLIGHTLY TAIL LOW
7. Climb Speed - 60 KIAS (Until all obstacles are cleared)
8. Wing Flaps - RETRACT after reaching 70 KIAS

Figure 8.9.1.2-2. Cessna 182T In-Flight Checklist Card
8.9.1.4. **Notice to Airmen (NOTAM).** NOTAMs are a notice containing information concerning the establishment, condition, or change in any aeronautical facility, service, procedures, or hazards. The timely knowledge of which is essential to personnel concerned with flight operations. NOTAMs are available at: [https://www.notams.jcs.mil](https://www.notams.jcs.mil).

8.9.1.5. **Air Traffic Control (ATC).** Air traffic control represent the Federal Aviation Administration (FAA), military, or other controlling facility which provide information to pilots and direct their flight path, altitude, speed, and approach clearances.

8.9.1.6. **Flight Level (FL).** Flight levels are an abbreviation of altitude in hundreds of feet. It is used to indicate altitude by pilots and flight controllers, starting at 18,000 ft (FL180) and above. Proper altimeter readings must be set to correspond with proper communication transmissions at given altitudes.

8.9.1.7. **Greenwich Mean Time (GMT).** Greenwich Mean Time is the clock time at Greenwich, England on the Prime Meridian; also known as Zulu time, at the zero longitude line of the earth. By using GMT time, all aircraft activities can be scheduled to avoid conflicts when flying across time zones.

8.9.2. **Aircraft Equipment**

8.9.2.1. **Ultra-High Frequency (UHF).** Frequency band of communications equipment used mostly for military aircraft. 225 – 399.9 MHz.

8.9.2.2. **Very-High Frequency (VHF).** Frequency band of communications equipment used in civil and military aircraft. 118 – 136.975 MHz.

8.9.2.3. **Identification, Friend or Foe (IFF).** Part of the aircraft transponder system used to identify the aircraft to air traffic control agencies. The discrete identification friend or foe code assigned to a particular aircraft by electronic means.

8.9.2.4. **Selective Identification Feature (SIF).** A capability that, when added to the basic identification friend or foe system, provides the means to transmit, receive, and display selected coded replies.
8.9.3. **Basic Aerodynamic Forces on Aircraft**

Aerodynamics concerns the motion of air and other gaseous fluids and other forces acting on objects in motion through the air. In effect, aerodynamics is concerned with the object (aircraft), the movement (relative wind), and the air (atmosphere) (DynamicFlight, 2002).

![Aerodynamic Forces Acting on and Aircraft](image)

**Figure 8.9.3-1. Aerodynamic Forces Acting on and Aircraft (FAA, 2016)**

8.9.3.1. **Thrust.** Thrust is the resultant force in the direction of motion due to the components of the pressure forces in excess of ambient atmospheric pressure acting on all inner surfaces of the vehicle propulsion system parallel to the direction of motion. Thrust is the force which propels the aircraft forward into the wind stream to create lift, parallel to the axis of the engine (Figure 8.9.3-1). It can come from rotation of propeller/s which force air toward the rear of the aircraft via lift of the blades, a jet engine via increased velocity of the exhaust gas versus the intake velocity, or artificial thrust as in a glider being dragged or an aircraft being catapulted from an aircraft carrier. In the latter two cases, once the artificial thrust ends, the aircraft will lose all thrust and descend or rely on aircraft sources of thrust as in a carrier-launched aircraft. Power and/or torque is another term associated with thrust or an aircraft.

8.9.3.2. **Weight.** Weight acts vertically, downward, and concentrated at the center of gravity (Figure 8.9.3-1). The weight of any given aircraft is affected by its:

- Basic, empty weight
- Fuel weight
- Cargo weight
- Occupant total weight
- The effect of acceleration in the vertical axis, pulling G’s, part of any level or climbing turn.
8.9.3.3. Drag. Drag is the aerodynamic force in a direction opposite to that of flight and due to the resistance to movement brought to bear on an aerospace vehicle by the atmosphere through which it passes (Figure 8.9.3-1). Drag will have rearward action parallel to the wind. Induced drag, in subsonic flow over a finite airfoil or other body that part of the drag caused by lift; i.e., induced by the downwash.

8.9.3.3.1. Parasite drag. Friction of air flowing over the aircraft and not associated with the production of lift is parasite drag. It is composed of form drag caused by the separation of the boundary layer from a surface and the wake created by that. Profile drag is caused by friction and the viscosity of the air. Interference drag caused by air flowing around the fuselage mixing with air flowing around an external store or flow around the fuselage mixing with the flow around the wing where it attaches to the fuselage.

8.9.3.3.2. Induced Drag. Drag caused by the production of lift and varies inversely as the square of the airspeed. If the airspeed is reduced to one-half its original value, the induced drag increases 4 times.

8.9.3.4. Lift. Lift is the component of the total aerodynamic force acting on a body perpendicular to the direction of the undisturbed airflow relative to the body (Figure 8.9.3-1). This lift, sometimes called aerodynamic lift, acts on such as an airfoil, a fuselage, an airplane, a rotor, etc., at a suitable angle of attack (AOA) in the airflow. Lift is concentrated on the center of pressure. It is affected by the relative speed of air moving over and under an airfoil such as the wing, which is, in turn, affected by the shape or camber of that airfoil and its angle of attack.

8.9.3.5. Angle of Attack (AOA). The angle at which body, such an airfoil, wing, or fuselage, or a system of bodies, such as a helicopter rotor, meets a flow. This is typically measured between a reference line in the body, e.g. the chord of a wing, and a line the direction of the flow or in the direction of movement of the body. The angle of attack controls the pressures above and below the wing. As the angle of attack is increased, the pressure differential becomes greater until the stalling angle of attack is reached (Figure 8.9.3.5-1). The AOA meter on the T-6A reaches 18 units. Once the AOA of the aircraft hits 14-15 units, the “stick shaker” will be activated notifying the pilot to correct. (FAA, 2016; and HPE Flt Princ).
8.9.3.6. **Stall.** A stall occurs when the smooth airflow over the airplane’s wing is disrupted, and lift degenerates rapidly (Figure 8.9.3.6-1). This is caused when the wing exceeds its critical angle of attack. This can occur at any airspeed, in any attitude, with any power setting.

![Figure 8.9.3.5-1. AOA and Center of Pressure. (FAA, 2016)](image-url)
8.9.3.7. **Stall Recovery.** At the first indication of a stall, the pitch attitude and angle of attack must be decreased positively and immediately. Since the basic cause of a stall is always an excessive angle of attack, the cause must first be eliminated by releasing the back-elevator pressure that was necessary to attain that angle of attack or by moving the elevator control forward.

8.9.3.8. **Spiral.** A maneuver or performance, in which the aircraft ascends or descends in a corkscrew path, distinguished from a spin in that the angle of attack is within the normal range of flight angles; the flight path of an aircraft so ascending or descending.

8.9.4. **Aircraft Flight Components**

8.9.4.1. **Wing.** An airfoil that provides, or that is designed to provide, sustentation for an airplane, either: extending on either side of the airplane, separated from its mate by the fuselage or hull (thus a monoplane so constructed is said to have “wings”), but sometimes with both wings considered as a single unit, or extending uninterrupted on both sides of the airplane; e.g., as on a parasol monoplane. Some common wings types include anhedral, dihedral, straight, swept, rectangular, elliptical, tapered, and delta. Wing types will correspond to aircraft designation for performance output.

8.9.4.2. **Stabilizer.** A fixed or adjustable airfoil or vane that provides stability for an aircraft; i.e., a fin, specifically, the horizontal stabilizer on an airplane.

8.9.4.3. **Pitch.** Rotation about the horizontal axis of the aircraft. The primary control surface is the elevator.

8.9.4.4. **Elevator.** A control surface, usually hinged to a horizontal stabilizer on the tail section, deflected to produce a pitching moment; i.e., to make the aircraft or other flying body of which it is a part to rotate about its lateral axis. An elevator may be one of a pair, each one of the pair being situated to either side of the centerline (hence the frequent use of the plural, elevators), or it may be a continuous surface running from end to end of the stabilizer (Figure 8.9.4.4-1).
8.9.4.5. **Roll.** Rotation about the longitudinal axis of the aircraft. The primary control surfaces are the ailerons.

8.9.4.6. **Aileron.** A movable control surface or device, one of a pair or set located in or attached to the wings on sides of an airplane, the primary usefulness of which is controlling the airplane laterally or in roll by creating unequal or opposing lifting forces on opposite sides of the airplane. An aileron commonly consists of a flap-like surface at the rear of a wing, although other devices are sometimes used (Figure 8.9.4.6-1)

8.9.4.7. **Bank.** The position or attitude of an airplane, glider, helicopter, or aircraft when its lateral axis is inclined to the horizontal, as, to put an airplane into a bank. This is the position normally assumed by an aircraft when turning. A right bank is a bank in which the lateral axis inclines downward to starboard. The inclination of the lateral axis of an airplane, etc., to the horizontal, as, the degree of bank.

8.9.4.8. **Vertical Stabilizer.** A fin mounted approximately parallel to the plane of symmetry of an airplane, airship, or other aircraft, to which the rudder, when present, is attached. Also called a vertical fin.
8.9.4.9. **Yaw.** Rotation about the vertical axis of the aircraft. The primary control surface is the rudder.

8.9.4.10. **Rudder.** An upright control surface that is deflected to create a yaw (rotation) about the vertical axis. Application of rudder in the direction of the desired turn during the beginning of any aileron application to initiate a turn may avoid an uncoordinated turn which could impart a side force known as a “slip” or “skid” turn (Figure 8.9.4.10-1).

![Rudder](image)

**Figure 8.9.4.10-1. Rudder to Control Aircraft Yaw (HPE, 2011)**

8.9.4.11. **Flap.** Specifically, a hinged, pivoted, or sliding airfoil or plate, or a combination of such objects regarded as a single surface, normally located at the rear of a wing. Flaps will be extended or deflected for increasing camber, especially at takeoff or during landing. Greater camber = more lift.

8.9.4.12. **Spoiler.** A plate, series of plates, comb, tube, bar, or other device that projects into the airstream from an aircraft to break up or spoil the smoothness of the flow. Especially such a device that projects from the upper surface of an airfoil, giving an increased drag and a decreased lift.

8.9.4.13. **Trim.** The condition of an aircraft in which it maintains a fixed attitude with respect to the wind axes, the moments about the aircraft’s axes being in equilibrium. The word trim is often used with special reference to the balance of control forces.

8.9.4.14. **Trim Control.** An airplane is designed so that the primary flight controls (rudder, aileron, and elevator) are streamlined with the non-movable airplane surfaces when the airplane is cruising straight-and-level at normal weight and loading. If the airplane is flying out of that basic balanced condition, one or more of the control surfaces is going to have to be held out of its streamlined position by continuous control input. The use of trim tabs relieves the pilot of this requirement.

8.9.4.15. **Trim Tab.** A tab that is deflected to a position where it remains to keep the aircraft in the desired trim.
8.9.4.16. Power Control Lever (PCL)/Throttles. The power control lever (PCL) or throttles (based of aircraft MDS) is responsible for setting the desired power level. The throttle controls the mass flow-rate of air (fuel-injected engines) or air/fuel mixture (carbureted engines) delivered to the cylinders. The PCL/throttles will often be measure in terms of torque percentages.

8.9.5. Basic Flight Maneuvers

All flying tasks are based upon: straight-and level flight, turns, climbs, and descents. All controlled flight consists of either one, or a combination or more than one, of these basic maneuvers (USAFSAM 2015; HPE, 2011)

8.9.5.1. The Four Fundamentals of Flight. There are four fundamental basic flight maneuvers upon which all flying tasks are based: straight-and-level flight, turns, climbs, and descents. All controlled flight consists of either one, or a combination, of these basic maneuvers. In attitude flying, aircraft control is comprised of pitch control, bank control, power control, and trim. Examples of cockpit instruments for each of these is shown in Figure 8.9.5.1-1. The acronyms REA (rudder (yaw), elevator (pitch) and ailerons (roll)) and PAT (pitch, attitude, trim) are be vital in maintaining proper attitude and making smooth corrections in flight. The primary rule of attitude flying: Attitude + Power = Performance (USAFSAM, 2015).

Figure 8.9.5.1-1. Pitch (1), Bank (1), Power (2), and Trim (3) Instrumentation.

8.9.5.2. Attitude. The position of a body as determined by the inclination of the axes to some frame of reference. If not otherwise specified, this frame of reference is fixed to the Earth.
8.9.5.3. **Turns.** A turn in an aircraft requires use of ailerons, rudder, and elevator to maintain a consistent altitude. The aileron initiates the bank with coordinating rudder and the elevator acts to maintain altitude and control the rate of the turn once the aileron has established the proper degrees of bank. Without application of elevator, back pressure on the yoke or stick, as done to initiate a climb, the bank of an aircraft would only reduce lift and the aircraft would lose altitude.

8.9.5.4. **Steep Turns.** A steep turn is typically any turn with over 30 degrees of bank. During a 60-degree bank level turn, the elevator use to maintain level flight results in a G-force of 2 \(+G_z\) since the lift vector is half of the lift vector in level flight. The G-force increases with bank angle in level flight, reaching infinity at a 90-degree bank. It is impossible to maintain level flight during a 90-degree bank coordinated turn, but use of the rudder as an elevator and the fuselage as an airfoil while in a 90-degree bank, it may be possible to maintain altitude as done by some airshow stunt pilots. This is partially true because the thrust vector is typically angled up, providing an additional upward vector of force.

8.9.5.5. **Standard Rate Turns.** A standard rate turn is one which will produce a change in heading of 3 degrees per second (180 degrees in one minute). See turn indicator in Figure 8.9.5.1-1.

8.9.6. **Basic Flight: Control Instruments**

8.9.6.1. **Control Instruments.** Instruments which display information relating to control of the aircraft. These are the attitude indicator(s) and engine instruments which show the level of thrust or power being applied, e.g. engine tachometers, exhaust pressure ratio, fuel flow, and other indications of engine performance depending on the type of aircraft.

8.9.6.2. **Attitude Director Indicator (ADI).** The attitude director indicator, or more commonly known as the attitude indicator, is displayed on a cockpit instrument panel in two basic forms. It may be a gyro-stabilized sphere with indications of pitch and roll or a flat-panel display which depicts the same information.

8.9.6.3. **Engine Tachometer.** Engine tachometer are usually indicated in % of maximal RPM, the engine tachometers indicate the relative level of thrust depending on throttle setting.

8.9.6.4. **Exhaust Pressure Ratio (EPR) Gauges.** The EPR gauges indicate the difference in pressure from two stages of the jet engines which provide an accurate measure of the level of thrust being developed.

8.9.6.5. **Fuel Flow Gauges.** The fuel flow gauges indicate how much energy is being supplied to the engines, thus how much thrust is expected from the engines.
8.9.7. Basic Flight: Performance Instruments

8.9.7.1. Performance Instruments. The altimeter, airspeed, Mach indicator, vertical velocity indicator, heading indicator, angle of attack indicator and turn and slip indicator indicate the actual performance of the aircraft. See Figure 8.9.7.1-1 for an example of a control instrument panel (USAFSAM, 2015; HPE, 2011)

![Control Instrument Panel](image)

Figure 8.9.7.1-1. Control Instrument Panel (HPE, 2011)

8.9.7.2. Altimeter. Any of various types of instruments for measuring altitude; specifically, an instrument similar to an aneroid barometer that utilizes the change of atmospheric pressure with altitude to indicate the approximate elevation above a given point or plane used as a reference.

8.9.7.3. Pitot-Static Tube. A parallel or coaxial combination of a pitot and static tube used for sensing the difference between impact total pressure and static pressure. The difference between the impact pressure and the static pressure is a function of the velocity of flow past the tube and may be used to indicate airspeed of an aircraft in flight.

8.9.7.4. Airspeed Indicator. Any instrument or meter designed to indicate airspeed; commonly, an instrument designed either to show measurements of airspeed related to assumed air conditions, or to show airspeed measurements automatically corrected for certain existing air conditions. Sometimes called an airspeed meter.

8.9.7.4.1. True Airspeed (TAS). True airspeed is equivalent airspeed corrected for error due to air density (altitude and temperature). With no wind component, the true airspeed is the ground speed.

8.9.7.4.2. Mach Indicator. Pronounced “mock” after Ernst Mach (1838-1916), Austrian scientist. Indicates the ratio of the velocity of a body to that of sound in the surrounding medium. Thus, a Mach number of 1.0 indicated a speed equal to the speed of sound. 0.5 is a speed one-half the speed of sound. 5.0 is a speed five times the speed of sound.
8.9.7.5. **Vertical Velocity Indicator (VVI).** The instrument which indicates rate of climb and descent in 100 foot increments.

8.9.7.6. **G-Meter/Accelerometer.** The instrument which indicates that amount of “G's” pulled and sustained by the aircraft. Each aircraft will have specific g-load capabilities and do not exceed requirements.

8.9.7.7. **Heading Indicator.** The magnetic compass indicates the heading of the aircraft with reference to magnetic north. Although very useful when a stabilized and corrected heading indicator is not available or inoperative, the magnetic compass is unstable in turns and subject to several errors even during straight-and-level flight.

8.9.7.8. **Horizontal Situation Indicator (HSI).** As a combination of a heading indicator, radio magnetic indicator, course indicator, and range indicator, the HSI provides the pilot with indications of heading and position relative to selected navigational aids.

8.9.7.9. **Angle of Attack Indicator (AOA).** AOA information is displayed with an indication of the stall AOA and may include reference to the AOA which provides maximum lift/drag, L/D max. L/D max produces the minimum total drag and is the most efficient AOA, thus providing the best fuel efficiency. When nearing AOA limitations, the aircraft will respond with a physical warning for the aviator to make necessary corrections to maintain proper attitude.

8.9.7.10. **Turn and Slip Indicator.** An instrument which combines the functions of a turn indicator and a slip indicator (Figure 8.9.7.10-1). The principal functions of the turn and slip indicator are to provide an alternate source of bank control and to indicate a need for yaw trim (see Rudder).

![Figure 8.9.7.10-1. Turn Indicator (HPE, 2011)](image-url)
8.9.8. **Navigation Equipment**

Equipment used to determine position; radar, TACAN, VOR, DME, ILS, Marker beacon, and GPS. Also listed is terrain/collision avoidance systems GPWS, GCAS, ACAS, TCAS, and TAWS.

8.9.8.1. **Radar.** Air Traffic Control (ATC) radar allows ATC controllers to inform pilots of their aircraft position and verify or inform them of other air traffic in the area.

8.9.8.2. **Tactical Air Navigation (TACAN).** Tactical Air Navigation requires a station and aircraft equipment to provide the pilot of radial, course, and DME information relative to a specific TACAN station.

8.9.8.3. **Very High Frequency Omnidirectional Range (VOR).** Very high frequency Omnidirectional Range navigation requires a station and aircraft equipment so the pilot can be informed of radial and course information relative to a specific VOR station.

8.9.8.4. **Very High Frequency Omnidirectional Range and Tactical Air Navigation (VORTAC).** Very high frequency omnidirectional range station and tactical air navigation aid collocated to aid navigation.

8.9.8.5. **Ground Proximity Warning System (GPWS).** A ground proximity warning system (GPWS) is a system designed to alert pilots if their aircraft is in immediate danger of flying into the ground or an obstacle. GPWS uses the ground elevation direction below the aircraft. GPWS is utilized in many “heavy” aircraft.

8.9.8.6. **Ground Collision Avoidance System (GCAS).** A system and method for avoiding collision between objects and wingtips of an aircraft when the aircraft is on the ground. More modern versions of GCAS have become Automatic or “Auto-GCAS” which is popular in F-16, F-22, and F-35 models (Merlin P, 2015).

8.9.8.7. **Airborne Collision Avoidance System (ACAS).** ACAS is a type of Ground Collision Avoidance Technology (GCAT) that operates independently of ground-based equipment and air traffic control in warning pilots of the presence of other aircraft that may present a threat of collision.

8.9.8.8. **Traffic Collision Avoidance System (TCAS).** Actively interrogates the transponders of other aircraft and negotiates collision-avoidance tactics with them in case of a threat. They are effective in avoiding collisions only with other aircraft that are equipped with functioning transponders with altitude reporting.

8.9.8.9. **Terrain Avoidance Warning System (TAWS).** TAWS uses a digital terrain map, together with position information from a navigation system such as GPS, to predict whether the aircraft’s current flight path could put it in conflict with obstacles such as mountains or high towers, that would not be detected by GPWS. TAWS is utilized in many “heavy” aircraft.
8.9.9. **Aerobatics**

Aerobatic maneuvers develop techniques for obtaining maximum flight performance from the aircraft. Aerobatics explore the entire performance envelope of the aircraft and should be smoothly executed. Aerobatic practice improves feel for the aircraft and the ability to coordinate the flight controls, while remaining oriented, regardless of attitude. Aerobatics increase confidence, familiarize the pilot with all attitudes of flight, and increase the ability to fly an aircraft throughout a wide performance range. The concepts learned from aerobatic practice are applicable in formation maneuvering and other advanced missions. Table 8.9.9-1 summarizes the entry airspeeds and power settings for aerobatic maneuvers (AFPAM 11-248, 2016).

**Table 8.9.9-1. Summary of Entry Airspeeds and Power Settings for Aerobatics.**

(AFPAM 11-248)

<table>
<thead>
<tr>
<th>ITEM</th>
<th>Maneuver</th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
<th>E</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Aileron Roll</td>
<td>180</td>
<td>220</td>
<td>80% to MAX</td>
<td>+1,000 feet</td>
<td>Neutral</td>
</tr>
<tr>
<td>2</td>
<td>Barrel Roll</td>
<td>200</td>
<td>220</td>
<td>80% to MAX</td>
<td>+1,500 feet -2,000 feet</td>
<td>Neutral</td>
</tr>
<tr>
<td>3</td>
<td>Chandelle</td>
<td>200</td>
<td>250</td>
<td>MAX</td>
<td>+3,000 feet</td>
<td>Gaining</td>
</tr>
<tr>
<td>4</td>
<td>Cloverleaf</td>
<td>200</td>
<td>220</td>
<td>MAX</td>
<td>+3,000 feet -1,000 feet</td>
<td>Slightly losing</td>
</tr>
<tr>
<td>5</td>
<td>Cuban Eight</td>
<td>230</td>
<td>250</td>
<td>MAX</td>
<td>+3,000 feet</td>
<td>Neutral</td>
</tr>
<tr>
<td>6</td>
<td>Immelmann</td>
<td>230</td>
<td>250</td>
<td>MAX</td>
<td>+3,000 feet</td>
<td>Gaining</td>
</tr>
<tr>
<td>7</td>
<td>Lazy Eight</td>
<td>200</td>
<td>220</td>
<td>80% to MAX</td>
<td>+2,000 feet -1,000 feet</td>
<td>Neutral</td>
</tr>
<tr>
<td>8</td>
<td>Loop</td>
<td>230</td>
<td>250</td>
<td>MAX</td>
<td>+3,000 feet</td>
<td>Neutral</td>
</tr>
<tr>
<td>9</td>
<td>Split-S</td>
<td>120</td>
<td>140</td>
<td>Idle to 80%</td>
<td>+500 feet - 2,000 feet (75%) -2,500 feet (IDLE)</td>
<td>Losing</td>
</tr>
</tbody>
</table>
8.9.9.1. **Aileron Roll.** An aileron roll is a 360° roll with at a constant roll rate about the longitudinal axis of the aircraft. The maneuver is complete when the wings are again parallel to the horizon and the aircraft has completed (Figure 8.9.9.1-1).

![Figure 8.9.9.1-1. Aileron Roll](image)

8.9.9.2. **Barrel Roll.** A barrel roll is a coordinated roll through a 360° in which the nose of the aircraft describes a circle around a point on or near the horizon. Definite seat pressure should be felt throughout the roll. Practice in both directions. The maneuver is complete when the aircraft is wings level, abeam the reference point on the original side, at approximately entry airspeed (Figure 8.9.9.2-1).

![Figure 8.9.9.2-1. Barrel Roll](image)
8.9.9.3. **Chandelle.** The Chandelle is a precision, constant bank 180° steep climbing turn that achieves a maximum gain of altitude for a given power setting. The maneuver is complete after 180° of turn (Figure 8.9.9.3-1).

![Chandelle Diagram](image1)

**Figure 8.9.9.3-1. Chandelle**

8.9.9.4. **Cloverleaf.** The cloverleaf is composed of four identical maneuvers, each of which changes heading by 90°. The pull up is similar to the loop, although with less G-load. The top part is a rolling pull to the horizon 90° displaced from the original heading. The pulling roll resembles a nose-high recovery. The lower part or pull through is flown like a Split-S. The maneuver is complete in level flight after four leaves in the same direction. Fewer than four leaves may be performed when practicing the maneuver (Figure 8.9.9.4-1).

![Cloverleaf Diagram](image2)

**Figure 8.9.9.4-1. Cloverleaf**
8.9.9.5. **Cuban Eight.** Each half of this maneuver is a combination of a slightly modified loop and Immelman. The first portion of each leaf is approximately the first five-eighths of a loop followed by a half roll. The pull and roll is then repeated in the opposite direction. The maneuver looks like an 8 on its side. The maneuver is complete at level flight, with entry airspeed and on original heading (Figure 8.9.9.5-1).

![Cuban Eight Diagram](image)

Figure 8.9.9.5-1. Cuban Eight

8.9.9.6. **Immelman.** The Immelman is a half loop followed by a half roll, all flown in the same vertical plane. The maneuver is complete after a momentary pause in level flight with wings level on an opposite heading from entry (Figure 8.9.9.6-1).

![Immelman Diagram](image)

Figure 8.9.9.6-1. Immelman
8.9.9.7. **Lazy Eight.** This is a slow, lazy maneuver that describes a horizontal figure eight at the horizon. The horizon line bisects this figure eight lengthwise. Pitch, bank, and airspeed constantly change. The maneuver is complete after two 180° turns with the aircraft in level flight (Figure 8.9.9.7-1).

![Figure 8.9.9.7-1. Lazy Eight](image)

8.9.9.8. **Loop.** The loop is a 360° turn in the vertical plane with constant heading and nose track. Because it is executed in a single plane, the elevator is the principle control surface. Ailerons and rudder are used to maintain directional control and coordinated flight. The maneuver is complete when wings are level at the horizon on the same heading as at entry (Figure 8.9.9.8-1).

![Figure 8.9.9.8-1. Loop](image)
8.9.9.9. **Split S.** The Split-S combines the first half of an aileron roll with the last half of a loop. It demonstrates how much altitude is lost if recovery from inverted flight is attempted by pulling through the horizon. The aircraft climbs during entry and descends during recovery. The maneuver is complete when the aircraft returns to level flight.

![Figure 8.9.9.9-1. Split S](image)

**References**


9. GLOSSARY

Absolute altitude see AGL
Absolute pressure Gauge pressure + local atmospheric pressure
AC Aircraft Commander
ACC Air Combat Command
ACCES Attenuating Custom Communications Earpiece System
Acclimatization The adjustments of a human body or other organism to a new environment; the bodily changes that tend to increase efficiency and reduce energy loss.
ACPM American College of Preventive Medicine
AEF Aerospace Expeditionary Force
Aero-otitis media An inflammatory reaction of the middle ear resulting from a difference in pressure between the gas in the middle ear and the surrounding atmosphere. Also called otitic barotraumas.
Aerospace medicine That branch of medicine dealing with the effects of flight through the atmosphere or in space upon the human body and with the prevention or cure of physiological or psychological malfunctions arising from these effects.
Aerospace (From aeronautics and space). 1. Of or pertaining to both the Earth's atmosphere and space, as in aerospace industries. 2. Earth's envelope of air and space above it; the two considered as a single realm for activity in the flight of air vehicles and in the launching, guidance, and control of ballistic missiles, earth satellites, dirigible space vehicles, and the like. Used primarily by the U.S. Air Force. The term aerospace first appeared in print in the Interim Glossary; Aero-Space Terms (edited by Woodford Agee Heflin), published in February 1958 at the Air University, Maxwell Air Force Base, Alabama.
AETC Air Education and Training Command
AF Air Force
AFB Air Force Base
AFFSA Air Force Flight Standards Agency
AFMOA Air Force Medical Operations Agency
AFRL Air Force Research Laboratory
AFTO Air Force Technical Order
AGL Above Ground Level, height above the ground; also referred to as absolute altitude
AGSM Anti-G straining maneuver
AHFA Aerospace Human Factors Association
<table>
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<tr>
<th>Acronym</th>
<th>Description</th>
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</thead>
<tbody>
<tr>
<td>AIM</td>
<td>Aeronautical Information Manual</td>
</tr>
<tr>
<td>Altitude</td>
<td>The vertical distance of a level, a point, or an object considered as a point, measured from a reference point, usually taken to be mean sea level (MSL)</td>
</tr>
<tr>
<td>AMDS</td>
<td>Aerospace Medicine Squadron</td>
</tr>
<tr>
<td>AOA</td>
<td>Angle of attack</td>
</tr>
<tr>
<td>AOPT</td>
<td>Aerospace and Operational Physiology Team</td>
</tr>
<tr>
<td>APA</td>
<td>Aerospace Physiology Apprentice</td>
</tr>
<tr>
<td>APA</td>
<td>American Psychiatric Association</td>
</tr>
<tr>
<td>APC</td>
<td>Aerospace Physiology Craftsman</td>
</tr>
<tr>
<td>APIMS</td>
<td>Aerospace Physiology Information Management System</td>
</tr>
<tr>
<td>APO</td>
<td>Aerospace Physiology Officer</td>
</tr>
<tr>
<td>APPB</td>
<td>Assisted Positive Pressure Breathing for Altitude</td>
</tr>
<tr>
<td>APTF</td>
<td>Aerospace Physiology Training Flight</td>
</tr>
<tr>
<td>AsMA</td>
<td>Aerospace Medical Association</td>
</tr>
<tr>
<td>ATC</td>
<td>Air Traffic Control</td>
</tr>
<tr>
<td>ATD</td>
<td>Aircrew Training Device</td>
</tr>
<tr>
<td>ATIS</td>
<td>Air Terminal Information System</td>
</tr>
<tr>
<td>atm</td>
<td>Atmosphere, pressure at Earth’s surface is nominally one atmosphere (1 atm); but varies slightly depending on the weather conditions.</td>
</tr>
<tr>
<td>BIC</td>
<td>Basic Instructor Course</td>
</tr>
<tr>
<td>C2</td>
<td>Command and Control</td>
</tr>
<tr>
<td>C3</td>
<td>Command, Control, and Communications</td>
</tr>
<tr>
<td>C3I</td>
<td>Command, Control, Communications, and Information</td>
</tr>
<tr>
<td>CAS</td>
<td>Close Air Support</td>
</tr>
<tr>
<td>CATM</td>
<td>Curriculum, Administration, Training, Management</td>
</tr>
<tr>
<td>CCAF</td>
<td>Community College of the Air Force</td>
</tr>
<tr>
<td>CDC</td>
<td>Career Development Course</td>
</tr>
<tr>
<td>CDI</td>
<td>Course Deviation Indicator</td>
</tr>
<tr>
<td>CFETP</td>
<td>Career Field Enlisted Training Program</td>
</tr>
<tr>
<td>CINC</td>
<td>Commander-in-Chief</td>
</tr>
<tr>
<td>Communication</td>
<td>The act of sharing information with others to cause some kind of action: to direct, to inform, to question, or to persuade.</td>
</tr>
<tr>
<td>CONOPs</td>
<td>Concept of Operations</td>
</tr>
</tbody>
</table>
CONUS
Continental United States

Crew Coordination
As used in this instruction, the act of working with all the members of the crew to accomplish the tasks of the mission.

Crew
As used in this instruction, any collection of Air Force personnel who routinely work together to accomplish an Air Force mission. For example, an air task order-designated team of fighter pilots and airborne battle managers prosecuting an interdiction mission uses "crew" skills to maximize its effectiveness.

CRM
Cockpit/Crew Resource Management. The effective use of all available resources--people, weapon systems, facilities, and equipment, and environment--by individuals or crews to safely and efficiently accomplish an assigned mission or task. The term “CRM” will be used to refer to the training program, objectives, and key skills directed to this end. MAJCOMs may implement their programs as either “cockpit” or “crew” resource management based on their respective missions.

CSW
Course Selector Window

CTS
Course Training Standards

CWS
Cockpit Warning System

dB
Decibel, unit of sound intensity

DCI
Decompression Illness; general term encompassing all pathological changes secondary to reduction of environmental pressure; includes DCS, embolism, ebullism, trapped gas, hypoxia, anoxia.

DCS
Decompression Sickness; symptoms caused by nitrogen gas dissolved in body fluids and tissues forming a gas phase during decompression and interacting with nervous tissue and blocking blood flow.

Decision Making
The ability to choose a course of action using logical and sound judgment based on available information.

Dew Point
The point at which the air at a certain temperature contains all the moisture possible without precipitation occurring. When the dew point is 65 °F, one begins to feel the humidity. The higher the temperature associated with the dew point, the more uncomfortable one feels.

DME
Distance Measuring Equipment

DNIF
Duty Not Including Flying

DoD
Department of Defense

DR
Dead Reckoning

DRU
Direct Reporting Unit
DISTRIBUTION STATEMENT A. Approved for public release. Distribution is unlimited.
FL

Flight Level; hundreds of feet; used to indicate altitude by pilots and flight controllers, typically at 18,000 ft (FL180) and above.

Flight Discipline

The judgment and actions exercised by AF personnel to adhere to the spirit, intent, and written word of governing guidelines in the presence of temptation to do otherwise while executing the Air Force flying mission. It also includes the prioritization of tasks based on crew responsibilities to ensure safe mission accomplishment while demonstrating the highest degree of integrity in the performance of flight duties.

Flight Integrity

Utilizing all the members of a flying package to accomplish the mission at hand.

FLIP

Flight Information Publications

FLIR

Forward Looking Infrared systems

FOA

Field Operating Agency

FOD

Foreign Object Damage

fpm

feet per minute

FS

Flight Surgeon

FSA

Future Strike Aircraft

G or g

Acceleration of gravity. The standard value of gravity, or normal gravity, g, is defined as $g_0 = 980.665$ centimeters per second squared, or $32.1741$ feet per second squared.

Geometric altitude

The scale we are most familiar with; it is the altitude we would measure with a tape measure.

GLOC

G-induced Loss of Consciousness

GMT

Greenwich Mean Time; same as Zulu time

GPS

Global Positioning System

GSTF

Global Strike Task Force

GWOT

Global War on Terrorism

HAAMS

High Altitude Airdrop Mission Support

HAHO

High Altitude High Opening

HALO

High Altitude Low Opening

HAMS

High Altitude Mission Support

HAP

High Altitude Parachutists

HARMS

High Altitude Reconnaissance Mission Support

HATR

Hazardous Air Traffic Report

Health

A human condition with physical, social, and psychological dimensions, each characterized on a continuum with positive
and negative poles. Positive health is associated with a capacity to enjoy life and to withstand challenges; it is not merely the absence of disease. Negative health is associated with morbidity and, in the extreme, premature mortality.

HFACS  DoD Human Factors Analysis and Classification System
HMD    Helmet-Mounted Display
hPa    hectoPascals, or hundreds of Pascals (newtons per square meter, the metric unit of pressure); the preferred unit for atmospheric science. Standard atmospheric pressure is 1013.25 hPa (29.92 inches of Hg; 1013.25 mb).
HPE    Human Performance Enhancement
HPT    Human Performance Team
HPTT   Human Performance Training Team
HPW    Human Performance Wing (711th)
HSI    Human Systems Integration
HUD    Heads-Up Display
IAASM  International Academy of Aviation and Space Medicine
IAF    Initial Approach Fix
IAP    Instrument Approach Plates
ICAO   International Civil Aeronautical Organization; the international body governing the operation of commercial aircraft.
IFE    In-Flight Emergency
IFF    Identification, Friend or Foe (part of the aircraft transponder system)
IFR    Instrument Flight Rules
ILS    Instrument Landing System
IMC    Instrument Meteorological Conditions
IMSAFE Checklist on Self-Medication
inches Hg Inches of Mercury, and the number given in most weather reports in the United States. Standard pressure is 29.92 inches Hg.
inches  Inches of water pressure. Used to describe mask pressure in some documents.
INS    Inertial Navigation System
IP     Instructor Pilot
IRB    Institutional Review Board
Instrument Takeoff
Joint Airborne/Air Transportability Tasking
Jet Stream
Strong winds concentrated within a narrow zone in the atmosphere in the upper troposphere, about 30,000 ft aloft, that generally move in an easterly direction and drive weather systems around the globe. In North America, jet streams are more pronounced in winter.

Joint Helmet-Mounted Cueing System
Joint Specialized Undergraduate Pilot Training
Knots Indicated Airspeed
 Knock It Off (stop current operations immediately)
The window in an altimeter where the altimeter setting in inches of Hg is displayed. Standard setting while flying above 18,000 ft is 29.92.
The decrease of temperature with height, considered positive when temperature decreases with height.
Laser Guided Bomb
Loss of Consciousness
Line-of-Sight
Long-Range Strike Aircraft
Mach number. The Mach number is a ratio between the aircraft's speed, $v$, and the speed of sound, $a$; $M = v/a$.
Major Command
Maximum thrust position
Millibar or thousandths of a bar, where a bar is approximately the same as an atmosphere; 1 mb = 1 hPa.
Mission Capable
Minimum Descent Altitude
Minimum En Route Altitude. The altitude in effect between radio fixes that ensures acceptable navigational signal coverage and meets obstruction clearance requirements between those fixes.
Reviewing and discussing mission accomplishment looking at what was achieved, what barriers were encountered, and how the mission could be accomplished better next time.
Taking all of the information for a mission and developing short-term, long-term, and contingency plans to coordinate, allocate, and monitor crew/flight and aircraft resources. Effective planning leads to flight conduct that removes
uncertainty, increases mission effectiveness, and enhances safety.

mmHg  Millimeters of Mercury. Standard pressure is 760.0 mmHg.
MOA  Military Operations Area
MOCA  Minimum Obstruction Clearance Altitude. That specified altitude in effect between radio fixes on VOR airways, off-airway routes, or route segments that meets obstruction clearance requirements for the entire route segment and that ensures acceptable navigational signal coverage only within 22 nautical miles of a VOR.
MOST  Mission-Oriented Simulator Training. Training presented as a part of a CRM program in a realistic, operationally based simulator environment in real time.
MSL  Mean Sea Level, the height above
MTR  Military Training Route
Muscle Contraction  Muscle contraction has both mechanical and metabolic categories. Mechanical classification stresses whether the muscle contraction produces movement of the limb:
Classification of Muscle Contraction:
Isometric (same length) or static exercise where there is no movement of the limb
Isotonic (same tension) or dynamic exercise if there is movement of the limb
Isokinetic (same velocity) dynamic exercise contraction of constant torque or tension at a set speed at all points in the range of motion
Metabolic classification involves the availability of oxygen for the contraction process and includes aerobic (oxygen available) or anaerobic (oxygen unavailable) processes. Whether an activity is aerobic or anaerobic depends primarily on its intensity. Most activities involve both static and dynamic contractions and aerobic and anaerobic metabolism. Thus, activities tend to be classified according to their dominant features.
Contextual Classification: physical activity categorized by the context in which it occurs. Common categories include occupational, household, leisure time, and transportation. Leisure-time activity can be further subdivided into categories such as competitive sports, recreational activities, and exercise training.
NACWS  Naval Aircraft Collision Warning System
NASA  National Aeronautics and Space Administration
<table>
<thead>
<tr>
<th>Acronym</th>
<th>Definition</th>
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<tr>
<td>NAVAID</td>
<td>Navigational aid</td>
</tr>
<tr>
<td>NCA</td>
<td>National Command Authority</td>
</tr>
<tr>
<td>NCOIC</td>
<td>Noncommissioned Officer in Charge</td>
</tr>
<tr>
<td>nm</td>
<td>Nautical mile</td>
</tr>
<tr>
<td>NOAA</td>
<td>National Oceanic and Atmospheric Administration</td>
</tr>
<tr>
<td>NORDO</td>
<td>No radio</td>
</tr>
<tr>
<td>NOTAM</td>
<td>Notice to Airman</td>
</tr>
<tr>
<td>NTSB</td>
<td>National Transportation Safety Board</td>
</tr>
<tr>
<td>NVD</td>
<td>Night-Vision Device/s</td>
</tr>
<tr>
<td>NVG</td>
<td>Night-Vision Goggles</td>
</tr>
<tr>
<td>OBOGS</td>
<td>Onboard Oxygen-Generating System</td>
</tr>
<tr>
<td>OIC</td>
<td>Officer-in-Charge</td>
</tr>
<tr>
<td>ORM</td>
<td>Operational Risk Management</td>
</tr>
<tr>
<td>Ozone</td>
<td>A molecule consisting of three oxygen atoms that is formed by a reaction of oxygen and ultraviolet radiation. In the stratosphere, ozone has beneficial properties: it forms an ozone shield that prevents dangerous radiation from reaching the Earth's surface. Closer to the planet's surface, ozone is considered an air pollutant that adversely affects humans, plants, and animals as well as acts like a greenhouse gas.</td>
</tr>
<tr>
<td>PA or Pressure Altitude</td>
<td>A type of geopotential height used so that aircraft, which use static pressure to determine altitude, can agree upon what &quot;altitude&quot; they are flying at without having to continually update their altimeters with local pressure corrections. Technically, this is only true above 18,000 ft (FL180). Altitude in the Earth's atmosphere above the standard datum plane, standard sea level pressure, measured by a pressure altimeter.</td>
</tr>
<tr>
<td>PACAF</td>
<td>Pacific Air Force</td>
</tr>
<tr>
<td>PAPI</td>
<td>Principles of Aerospace Physiology Instruction</td>
</tr>
<tr>
<td>Physical Activity</td>
<td>Bodily movement produced by the contraction of skeletal muscle that increases energy expenditure above the basal level. Health benefits increase with the intensity of the activity and the time you engage in it each week. Exercise is a type of regular physical activity that we perform to maintain or improve physical fitness. Physical activity can be classified in various ways.</td>
</tr>
<tr>
<td>Leisure-Time Physical Activity</td>
<td>one category of physical activity; physical activity that a person or group chooses to undertake during discretionary time.</td>
</tr>
</tbody>
</table>
Physical Fitness

Several general definitions:

A set of attributes that people have or achieve that relates to the ability to perform physical activity.

The ability to carry out daily tasks with vigor and alertness, without undue fatigue, and with ample energy to enjoy leisure-time pursuits and to meet unforeseen emergencies (President’s Council on Physical Fitness and Sport).

The ability to perform moderate to vigorous levels of physical activity without undue fatigue and the capability of maintaining such ability throughout life (ACSM).

The ability to last, to bear up, to withstand stress, and to persevere under difficult circumstances when an unfit person would quit. The opposite to becoming fatigued from ordinary efforts; to lacking energy to enter zestfully into life’s activities; and to becoming exhausted from unexpected, demanding physical exertion.

These are some of the myriad definitions of physical fitness, but they are somewhat lacking in the means for objective, simple measurement. One must consider components of fitness for such.

Physical Inactivity

Lack of regular exercise. Physical inactivity denotes a level of activity less than that needed to maintain good health.

POI

Plan/s of Instruction

psi

Pounds per square inch, the standard unit used in the aeronautical industry in the United States. Standard atmospheric pressure is 14.7 psi.

psia

Pounds per square inch absolute; see Absolute pressure

PT

Physiology Technician

RandM

Reliability and Maintainability

RCR

Runway Condition Reading

RDTandE

Research, Development, Test, and Evaluation

RESCAP

Rescue combat air patrol

RH

Relative Humidity. The ratio of the ambient vapor pressure of water to the saturated vapor pressure at the particular temperature. It is usually calculated with respect to liquid water even when the temperature is below the melting point.

Risk Management

Logic-based, common sense approach to making calculated decisions on human, material, and environmental factors before, during, and after Air Force mission activities and operations, i.e., on- and off-the-job.

ROBD

Reduced Oxygen Breathing Device
ROBE  Reduced Oxygen Breathing Environment
rpm  Revolutions per minute
RSU  Runway Supervisory Unit
SA  Situational Awareness. In flying, this refers to an aircrew member’s continuous perception of self and aircraft in relation to the dynamic environment of flight, threats, and mission and the ability to forecast, then execute, tasks based upon that perception.
SAAM  Special Assignment Air Mission
SCUBA  Self-Contained Underwater Breathing Apparatus
Sedentary Lifestyle  Synonymous with physical inactivity
SFL  Simulated Forced Landing
Signs  Evidence of disease; an indication of the presence of a disease or disorder, especially one observed by a doctor but not apparent to the patient.
SII  Special interest item
Skills Criteria  Defined skills used as the basis for operational training and evaluation. The characteristics of the skill are that they are easily identifiable and offer consistency in grading evaluation.
SLT  Swing Landing Training
SOF  Supervisor of Flying
SRB  Safety Review Board
Stratospheric ozone  In the stratosphere, ozone has beneficial properties: it forms an ozone shield that prevents dangerous radiation from reaching the Earth’s surface. Recently, it was discovered that in certain parts of the world, especially over the poles, stratospheric ozone was disappearing, creating an ozone hole.
STS  Specialty Training Standard
Symptoms  Indication of illness felt by patient; an indication of a disease or other disorder, especially one experienced by the patient, e.g., pain, dizziness, or itching, as opposed to one observed by the doctor.
TACAN  TACTical Air Navigation
TCAS  Traffic Collision Avoidance System
TAS  True Airspeed
Task Management  The ability to alter a course of action based on new information, maintain constructive behavior under pressure, and adapt to internal and external environment changes.
TDY  Temporary Duty
TO   Technical Order
torr This unit is named after the scientist Torricelli and is another name for mmHg; standard pressure in this unit is also 760.0 torr.
TUC  Time of Useful Consciousness; time of consciousness with the ability to take corrective action; now called Effective Performance Time (EPT).
UHF  Ultra High Frequency
UMD  Unit Manning Document
UMPR Unit Personnel Management Roster
USAF United States Air Force
USAFE United States Air Forces in Europe
USAFSAM USAF School of Aerospace Medicine
USUHS Uniformed Services University of the Health Sciences
USSTRATCOM United States Strategic Command
UTC  Universal Time Coordinated; substituted for GMT
UV   Ultraviolet radiation from the sun plays a role in the formation of the ozone layer by acting as a catalyst for a chemical reaction that breaks apart oxygen molecules which then recombine to form ozone. The absorption of UV by stratospheric ozone and atmospheric oxygen prevents very little ultraviolet radiation from reaching Earth's surfaces where it can have detrimental effects on human health and property.
VFR  Visual Flight Rules
VHF  Very High Frequency
VMC  Visual Meteorological Conditions
VOR  Very high frequency omnidirectional range station
VORTAC Very high frequency omnidirectional range station and TACtical air navigation
VSI  Vertical Speed Indicator
VVI  Vertical Velocity Indicator
Z or Zulu Zulu (military and aviation) time; GMT

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Physical fitness, Physical fatigue, Physiological divisions of the atmosphere, Radiation, Quick donning oxygen masks, Pressure Breathing for Altitude (PBA), Pressure Breathing for G (PBG), Pressure demand oxygen masks, Pressure suits, Pressurization schedule, Pressurization systems, Proprioception, Proprioceptive System, Psychophysics, Pulmonary barotrauma, Pulmonary circuit, Pulmonary ventilation, Purkinje shift, Radiation, Red blood cells (RBCs), Reduced Oxygen Breathing Device (ROBD), Referred tooth pain, Regulated Emergency Oxygen System (REOS), Relative Motion or Linear Vection, Remotely Piloted Aircraft (RPA), Accidents, Crew Resource Management, Ground control station, Ground crews, Home-based crews, Undergraduate RPA Training (URT), Video game players, Respiratory alkalosis, Respiratory bronchioles, Respiratory center, Respiratory system, Respiratory zone, Resting Metabolic Rate (RMR), Restraint system, Rod cells, Role of the PT, Ruffini endings, Runway Illusions, Saccule, SAFE Association, Safety and Accident Investigation, Safety Investigation Board (SIB), Scopolamine, Scotopic vision, Seat-of-the-pants, Self-medication, Semicircular canal adaptation, Semicircular canals, Semicircular Canals, Shallow water blackout, Shift lag, Shivering thermogenesis, Short-term memory, Sickle cell, Simulator sickness, Sinus block, Sinus cavities, Situational Awareness (SA), Sleep, Activity, Fatigue and Task Effectiveness (SAFTE), Smoke and fumes, Smokeless tobacco, Smoking tobacco, Society for Human Performance in Extreme Environments (HPEE), Somatographic Illusions, Somatogyril Illusions, Somatosensory, Space Motion Sickness (SMS), Space Myopia or Empty Visual Field, Spaceflight, Bone Physiology, Cardiovascular Physiology, Exercise, Fluid shifts, Muscle Physiology, Neurovestibular Physiology, Nutrition, Orthostatic intolerance, Radiation, Vision, Spatial disorientation, Spatial disorientation training.
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**Notes:**

- DISTRIBUTION STATEMENT A. Approved for public release. Distribution is unlimited.
- Cleared, 88PA, Case # 2016-5592, 1 Nov 2016.
11. APPENDICES

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Appendix 1b: Altitude of Exposure vs. Alveolar Partial Pressure of Oxygen – 25,000 to 50,000 ft Breathing 100% O₂ + PBA

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Oxygen – Sea Level to 45,000 ft Breathing Air
Alt
(ft)

Alt
(m)

Pressure
(psi)

Pressure
(mb)

PB
PACO2
(mmHg) (mmHg)

0

0

14.70

1013.3

760.0

40.0

0.85

149.7

104.2

15.0

1,000

305

14.17

977.2

732.9

39.5

0.85

144.0

99.2

13.0

2,000

610

13.67

942.1

706.7

39.0

0.86

138.5

94.4

11.0

3,000

914

13.17

908.1

681.2

38.5

0.86

133.2

89.8

9.1

4,000

1,219

12.69

875.1

656.4

38.0

0.87

128.0

85.3

7.1

5,000

1,524

12.23

843.1

632.4

37.5

0.87

122.9

81.0

5.1

6,000

1,829

11.78

812.0

609.1

37.0

0.88

118.0

76.9

3.1

7,000

2,134

11.34

781.9

586.5

36.5

0.88

113.3

72.9

1.1

912

8,000

2,438

10.92

752.7

564.6

36.0

0.89

108.7

69.1

-0.8

931

R

PIO2
PAO2
Temp
(mmHg) (mmHg) (°C)

%Hb
Sat

EPT
(min)

971,972

942

9,000

2,743

10.51

724.4

543.3

35.5

0.89

104.2

65.4

-2.8

10,000

3,048

10.11

696.9

522.7

35.0

0.90

99.9

61.8

-4.8

11,000

3,353

9.72

670.3

502.8

34.4

0.91

95.7

58.6

-6.8

862

12,000

3,658

9.35

644.6

483.5

33.8

0.92

91.7

55.5

-8.8

841

13,000

3,962

8.99

619.6

464.8

33.2

0.93

87.7

52.6

-10.7

14,000

4,267

8.64

595.5

446.6

32.6

0.94

83.9

49.7

-12.7

15,000

4,572

8.30

572.1

429.1

32.0

0.95

80.2

46.9

-14.7

16,000

4,877

7.97

549.4

412.1

31.5

0.96

76.7

44.1

-16.7

17,000

5,182

7.65

527.5

395.7

31.0

0.97

73.2

41.5

-18.7

18,000

5,486

7.35

506.3

379.8

30.4

0.98

69.9

39.0

-20.6

721

19,000

5,791

7.05

485.8

364.4

29.9

0.99

66.7

36.5

-22.6

682

20,000

6,096

6.76

466.0

349.5

29.4

1.00

63.5

34.1

-24.6

661

20 - 303

21,000

6,401

6.48

446.8

335.2

28.9

1.02

60.5

32.0

-26.6

22,000

6,706

6.21

428.3

321.3

28.4

1.03

57.6

29.9

-28.5

681

103

23,000

7,010

5.95

410.5

307.9

27.9

1.05

54.8

27.8

-30.5

24,000

7,315

5.70

393.2

294.9

27.4

1.06

52.1

25.9

-32.5

25,000

7,620

5.46

376.5

282.4

27.0

1.08

49.4

24.0

-34.5

26,000

7,925

5.23

360.4

270.3

26.6

1.08

46.9

21.8

-36.4

27,000

8,230

5.00

344.9

258.7

26.2

1.09

44.5

19.9

-38.4

28,000

8,534

4.78

329.9

247.4

25.8

1.09

42.1

18.0

-40.4

29,000

8,839

4.58

315.4

236.6

25.4

1.10

39.8

16.2

-42.4

30,000

9,144

4.37

301.5

226.1

25.0

1.10

37.6

14.4

-44.4

31,000

9,449

4.18

288.1

216.1

24.6

1.11

35.5

12.8

-46.3

32,000

9,754

3.99

275.1

206.3

24.2

1.11

33.5

11.1

-48.3

33,000

10,058

3.81

262.6

197.0

23.8

1.12

31.5

9.6

-50.3

34,000

10,363

3.64

250.6

188.0

23.4

1.12

29.6

8.2

-52.3

35,000

10,668

3.47

239.1

179.3

23.0

1.13

27.8

6.8

-54.2

36,000

10,973

3.31

228.0

171.0

22.6

1.13

26.0

5.5

-56.5

37,000

11,278

3.15

217.3

163.0

22.2

1.14

24.4

4.2

-56.5

38,000

11,582

3.01

207.1

155.4

21.8

1.14

22.8

3.1

-56.5

39,000

11,887

2.86

197.5

148.1

21.4

1.15

21.2

2.0

-56.5

40,000

12,192

2.73

188.2

141.2

21.0

1.15

19.8

0.9

-56.5

41,000

12,497

2.60

179.4

134.6

20.6

1.16

18.4

0.0

-56.5

42,000

12,802

2.48

171.0

128.3

20.2

1.16

17.1

-0.9

-56.5

43,000

13,106

2.37

163.0

122.3

19.8

1.17

15.8

-1.8

-56.5

44,000

13,411

2.26

155.4

116.6

19.4

1.17

14.6

-2.6

-56.5

45,000

13,716

2.15

148.2

111.1

19.0

1.18

13.5

-3.3

-56.5

781,752

562

3 - 53

2.5 - 33
1 - 23

0.5 - 13

0.25 - 0.333

0.15 - 0.203

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Appendix 1b: Altitude of Exposure vs. Alveolar Partial Pressure of Oxygen – 25,000 to 50,000 ft Breathing 100% O₂ + PBA

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<th>Alt (ft)</th>
<th>Alt (m)</th>
<th>Pressure (psi)</th>
<th>Pressure (mb)</th>
<th>Pₙ (mmHg)</th>
<th>PₐCO₂ (mmHg)</th>
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Altitudes shown in Appendix 1 are geometric altitude; U.S. Standard Atmosphere

Notes for Appendix 1a:
- **EPT (effective performance time)** = maximum time the crewmember has to make rational, life-saving decisions and carry them out at a given altitude without supplemental oxygen.

Notes for Appendices 1b and 1c:
- The levels of additional pressure from PBA are included in the PO₂ column; 1 mmHg = 1 torr; PO₂ values are for acute exposure during rest.
- The values for minimum positive pressure for altitude (PBA) were obtained for two oxygen regulators, which deliver within 2.5 mmHg of the same pressure at any comparable altitude in the specifications.
- **CRU 93**: Lockheed Martin Amendment No. 2 (28 Oct 1996) to Specification 16ZK048D dated 1 Oct 1990 for F-15 and F-16 PBG at 10 L/min ambient flow [Table II; Sheet 15 Rev D, SP1638050].
- **CRU 98**: MSOC-specific F-15-E PBG at 10 L/min ambient flow [Table IV; Sheet 10 Rev. H, SD1638058].
- **BRAG**: Data not available as of 12 Dec 06 for FA-22.
Appendix 1c: Altitude of Exposure vs. Alveolar Partial Pressure of Oxygen – 50,000 to 70,000 ft Breathing 100% O₂ + PBA

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<th>PₐCO₂ (mmHg)</th>
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Based on CRU-93 and CRU-98 oxygen regulators.

Appendix 1c: Altitude of Exposure vs. Alveolar Partial Pressure of Oxygen – 50,000 to 70,000 ft Breathing 100% O₂ + PBA

Fₒ₂, fraction of the total pressure which is oxygen; breathing air = 0.21; breathing 100% oxygen = 1.0

Pᵦ, barometric pressure in mmHg

PᵦCO₂, partial pressure of carbon dioxide in the alveoli, 40 mmHg at sea level.

Pₒ₂, calculated partial pressure of oxygen in the inspired air in the trachea

Pₒ₂, calculated partial pressure of oxygen in the alveoli

References


http://www.faa.gov/pilots/training/airman_education/media/AC%2061-107A.pdf
[87% @ 12K]
http://history.nasa.gov/SP-4701/session%201.pdf [65% @ 20K]
http://www.usaisr.amedd.army.mil/ewsh/Chp4AeroMed.pdf [98 - 100% at SL; 90% @ 8K]
http://www.alma.nrao.edu/memos/html-memos/alma162/memo162.html [87 - 95% @ 8K; 81-88% @ 12K; 71 - 85% @ 15K; 65 - 83% @ 17K; 56 - 73% @ 19.5K]

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Cleared, 88PA, Case # 2016-5592, 1 Nov 2016.
### Appendix 2a: Unit Conversion Table; Linear Units

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<td>1.152</td>
</tr>
</tbody>
</table>

### Appendix 2b: Unit Conversion Table; Pressure Units

<table>
<thead>
<tr>
<th>Unit</th>
<th>atm</th>
<th>N/m²</th>
<th>kg/cm²</th>
<th>g/cm²</th>
<th>psi</th>
<th>bars</th>
<th>mb or hPa</th>
<th>mmHg</th>
<th>inHg</th>
<th>inH₂O</th>
</tr>
</thead>
<tbody>
<tr>
<td>atm</td>
<td>=</td>
<td>1.013 X 10⁵</td>
<td>1.033</td>
<td>1.033 X 10³</td>
<td>14.70</td>
<td>1.013</td>
<td>1.013 X 10³</td>
<td>7.600 X 10²</td>
<td>29.92</td>
<td>4.067 X 10²</td>
</tr>
<tr>
<td>Newton/m²</td>
<td>=</td>
<td>9.872 X 10⁻⁶</td>
<td>1</td>
<td>1.020 X 10⁻⁵</td>
<td>1.020 X 10⁻²</td>
<td>1.452 X 10⁻⁴</td>
<td>1.000 X 10⁻⁵</td>
<td>1.000 X 10⁻²</td>
<td>7.501 X 10⁻³</td>
<td>2.953 X 10⁻⁴</td>
</tr>
<tr>
<td>kg/cm²</td>
<td>=</td>
<td>0.9681</td>
<td>9.807 X 10⁴</td>
<td>1</td>
<td>1.000 X 10³</td>
<td>14.22</td>
<td>0.9807</td>
<td>9.807 X 10²</td>
<td>7.356 X 10²</td>
<td>28.94</td>
</tr>
<tr>
<td>g/cm²</td>
<td>=</td>
<td>9.681 X 10⁻⁴</td>
<td>98.07</td>
<td>1.000 X 10⁻³</td>
<td>1</td>
<td>0.01422</td>
<td>9.807 X 10⁻⁴</td>
<td>0.9807</td>
<td>0.7356</td>
<td>2.894 X 10⁻²</td>
</tr>
<tr>
<td>psi</td>
<td>=</td>
<td>6.803 X 10⁻²</td>
<td>6.895 X 10⁻³</td>
<td>7.030 X 10⁻²</td>
<td>70.3</td>
<td>1</td>
<td>6.895 X 10⁻²</td>
<td>68.95</td>
<td>51.72</td>
<td>2.036</td>
</tr>
<tr>
<td>bar</td>
<td>=</td>
<td>0.9869</td>
<td>1.000 X 10⁵</td>
<td>1.019</td>
<td>1.019 X 10³</td>
<td>14.50</td>
<td>1</td>
<td>1.000 X 10³</td>
<td>7.501 X 10²</td>
<td>29.53</td>
</tr>
<tr>
<td>mb or hPa</td>
<td>=</td>
<td>9.869 X 10⁻⁴</td>
<td>1.000 X 10²</td>
<td>1.019 X 10³</td>
<td>1.020</td>
<td>1.450 X 10⁻²</td>
<td>1.000 X 10⁻³</td>
<td>1</td>
<td>0.7501</td>
<td>2.953 X 10⁻²</td>
</tr>
<tr>
<td>mmHg</td>
<td>=</td>
<td>1.316 X 10⁻³</td>
<td>1.333 X 10²</td>
<td>1.360 X 10⁻³</td>
<td>1.360</td>
<td>1.934 X 10⁻²</td>
<td>1.333 X 10⁻³</td>
<td>1.333</td>
<td>1</td>
<td>3.937 X 10⁻²</td>
</tr>
<tr>
<td>inHg</td>
<td>=</td>
<td>3.342 X 10⁻²</td>
<td>3.386 X 10²</td>
<td>3.453 X 10⁻²</td>
<td>34.55</td>
<td>0.4912</td>
<td>3.386 X 10⁻²</td>
<td>33.86</td>
<td>25.4</td>
<td>1</td>
</tr>
<tr>
<td>inH₂O</td>
<td>=</td>
<td>2.459 X 10⁻³</td>
<td>2.491 X 10²</td>
<td>2.540 X 10⁻³</td>
<td>2.54</td>
<td>3.614 X 10⁻²</td>
<td>2.491 X 10⁻³</td>
<td>2.49</td>
<td>1.868</td>
<td>7.355 X 10⁻²</td>
</tr>
</tbody>
</table>

atm = atmosphere; N/m² = Newton/m² = Pascal = Pa = 10⁵ dynes/cm²; psi = lb/in.²
mb = millibar = hPa = hectoPascals = 0.1 kPa = 1000 dynes/cm²
g/cm² = cm H₂O since 1 cm³ of water weighs very close to 1 g
Density of air at sea level is 1.229 kg/m³
Fahrenheit to Celsius: (5/9 * °F) – 32 = °C
Celsius to Fahrenheit: (9/5 * °C) + 32 = °F

11-5

DISTRIBUTION STATEMENT A. Approved for public release. Distribution is unlimited.
Cleared, 88PA, Case # 2016-5592, 1 Nov 2016.
Appendix 3: Prebreathe Requirements

AFI 11-409 (1 Dec 99, Certified Current 1 Feb 2011) Table 2.1. Prebreathing (100% O\textsubscript{2}) Requirements and Exposure Limits for High Altitude Operations.

<table>
<thead>
<tr>
<th>Altitude</th>
<th>Prebreathing Times (min)</th>
<th>Maximum Exposure Time Per Sortie (min)</th>
<th>Maximum Sorties Per 24-hr Period</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Aircrew</td>
<td>Jumpers</td>
<td></td>
</tr>
<tr>
<td>From FL200 to FL249</td>
<td>30</td>
<td>30</td>
<td>110</td>
</tr>
<tr>
<td>From FL250 to FL299</td>
<td>30</td>
<td>30</td>
<td>60</td>
</tr>
<tr>
<td>From FL300 to FL349</td>
<td>45</td>
<td>45</td>
<td>30</td>
</tr>
<tr>
<td>FL350 or above</td>
<td>75</td>
<td>75</td>
<td>30</td>
</tr>
</tbody>
</table>
Appendix 4: Checklists

A. Checklist for Treatment of Hypoxia and Hyperventilation
   1. All 3 switches up - On - 100% Oxygen - Emergency
   2. Mask - ON
   3. Check regulator and connections
   4. Control rate and depth of breathing
   5. Notify aircraft commander, lead, or other flight members

B. Checklist for Treatment of Ear and Sinus Pain on Descent
   1. Level off and try a Valsalva
   2. Climb to relieve pressure
   3. Try a Valsalva and decrease descent rate
   4. Consider using Afrin or other vasoconstrictor
   5. Declare an IFE?
   6. Land as soon as practical

C. Checklist for Treatment of Ear and Sinus Pain on Ascent
   1. DESCEND!!
   2. Land as soon as practical

D. Checklist for Treatment of Tooth Pain
   1. Descend and see Flight Surgeon/Dentist

E. Checklist for Treatment of GI Tract Pain
   1. Release the gas and/or descend

F. Checklist for Treatment of DCS
   1. 100% oxygen³
   2. Descend as soon as practical
   3. Declare IFE
   4. Land at the nearest airfield with qualified medical assistance available

³ Remain on 100% oxygen after landing, especially if symptoms are still present.
Appendix 5: USAF and Other FAA Directives Relevant to Aerospace Physiology

AETCI 11-406  Flying Operations: Fighter Aircrew Conditioning Program (FACP)
AETCI 36-2223  Flying Training Student Information Management
AFH 11-203v1and2  Weather for Aircrews
AFI 10-248  Fitness Program
AFI 11-202v3  General Flight Rules
AFI 11-217v1and2  Instrument Flight Procedures
AFI 11-290  Cockpit/Crew Resource Management Training Program
AFI 11-401  Aviation Management
AFI 11-403  Aerospace Physiological Training Program
AFI 11-403Sup1  Aerospace Physiological Training Program, AETC
AFI 11-404  Centrifuge Training for High-G Aircrew
AFI 11-409  High Altitude Airdrop Mission Support Program
AFI 11-409  High Altitude Airdrop Mission Support Program
AFI 11-419  G-Awareness for Aircrew
AFI 41-105  Medical Training Programs
AFI 41-120  Medical Resource Operations
AFI 41-209  Medical Logistics Support
AFI 48-123v1-4  Medical Examinations and Standards
AFJI 41-204  Joint Field Operating Agencies of the Surgeon General of the Army
AFMAN 10-100  Airman’s Manual
AFMAN11-248  T-6 Primary Flying
AFMCI 11-206  Mobility Force Management
AL-TR-1993-0022  Spatial Orientation in Flight

Federal Aviation Regulation PART 91--General Operating and Flight Rules
Federal Aviation Regulation PART 121--Operating Requirements: Domestic, Flag, and Supplemental Operations

ICAO Training of Operational Personnel in Human Factors.
Human Factors Digest No 3. ICAO Circular 227-AN/136. 1991
Flight Crew Training: Cockpit Resource Management (CRM) and Line-Oriented Flight Training (LOFT).
Appendix 6: Physiology Demonstrations

The Bárány chair, named for the Austro-Hungarian otologist and physiologist Róbert Bárány, is a device used in Aerospace Medicine to demonstrate the effects of spatial disorientation and to treat motion sickness (Figure A6-1). Bárány used this device in his work on the physiology and pathology of the vestibular apparatus of the ear, for which he received the 1914 Nobel Prize in Physiology or Medicine.

In 1926, Capt William Ocker used a device of his own invention, consisting of a wooden box containing primary flight instruments, to correctly identify his direction of motion while seated on a Bárány chair. He effectively demonstrated that by relying on instruments, and not your subjective perceptions or instincts, a pilot could accurately determine his direction of flight. Capt Ocker’s work laid the early foundations for spatial disorientation training and instrument flying.

Today, the Bárány chair is still used to demonstrate spatial disorientation and vestibular illusions to student pilots. It is also used to help overcome motion sickness by helping an individual acclimate by practicing relaxation techniques, visual dominance, and diaphragmatic breathing while being exposed to provocative motion.

![Figure A6-1. The Bárány Chair](image)

Practical demonstrations of physiologic principles often provide a mechanism of learning that is very effective and can lead to better retention than a recitation of the facts involved. The following examples can be tailored to an individual’s style of presentation.
**Bárány Chair Demonstrations**
SSgt Blake Lapp and SSgt Shawn Rose

**NOTE:** Ensure that seat belt is fastened during all demonstrations. Make sure someone is available to assist the student if needed. Allow the student time in the seat before sitting up to prevent falling out. Normal rotation speed for the chair is 25 – 30 rotations per minute.

1. **Nystagmus.** This is an involuntary movement of the eyes caused by stimulation of any one of the semicircular canals. When the canals respond to rotation in one direction, the eyes tend to sweep slowly in the opposite direction and then jerk to the center position. The canal that is affected will determine which way the eyes move (up, right, etc.)

   **Check Instruments**
   - Have student sit upright in chair
   - Eyes closed
   - Rotate chair for approximately 20 – 30 seconds
   - Have three other students stand in front of where the chair will stop to act as “instruments” with different numbers held up with their fingers
   - After 20 – 30 seconds, stop chair and have student open eyes
   - Student should try to cross check “instruments”

2. **Graveyard Spin.** When a turn is initiated, the fluid in the semicircular canals tends to lag behind and causes the hair cells to bend. The bending of the hair cells is what causes the sensation of turning. If you stay in the turn long enough, then the fluid will “catch up” with the canal and the hair cells will no longer bend. If the turn is stopped, the fluid continues to move and bends the hairs once again. This is what gives the student the false sensation of turning.

   **Indicate Turn Direction**
   - Have student sit upright in chair
   - Place goggles and earmuffs on student
   - The room must remain as quiet as possible during this demonstration
   - The student will indicate the direction of the turn with his/her thumbs (Figure A6-2)
   - Rotate the chair as smoothly as possible as the student indicates the direction
• Continue to rotate until fluid reaches equilibrium (approximately 20 – 30 seconds). The student should point thumbs in up position indicating no turning sensation.
• Slow the turn enough to let the student feel the change or abruptly stop the chair and student should indicate thumbs in the opposite direction of the actual initial turn

![Image of a person in a chair demonstrating the Graveyard Spin Illusion]

Figure A6-2. Graveyard Spin Illusion

3. **Coriolis.** This illusion occurs when there is a stimulation of two or more semicircular canals that gives the false sense of rotation in the third canal. The result is a tumbling sensation.

**Leans**
- Have student sit with head forward on the arms, resting on the bar
- Keep eyes closed throughout the entire demonstration
- Rotate until fluid reaches equilibrium (student senses no turn)
- Stop the chair, have student sit upright and throw arms up overhead in a “touchdown” fashion, eyes still closed
- Student will lean in direction of turn (Figure A6-3)

**Double Axis**
- Have student sit with head forward on the arms, with face turned to one side
- Keep eyes closed throughout entire demonstration
- Rotate chair until equilibrium is reached
- Stop the chair and have student sit upright and point at a fixed object
- Student should react as follows:
  - Right ear down, rotate toward face: **Movement: forward/right**
  - Right ear down, rotate opposite face: **Movement: backward/right**
  - Left ear down, rotate toward face: **Movement: forward/left**
  - Left ear down, rotate opposite face: **Movement: backward/left**
4. Other Demonstrations

**Disorientation 1**
- Have student sit upright with eyes closed and arms crossed on chest
- Rotate the chair until equilibrium is reached
- Have student touch chin to chest, right ear to right shoulder, left ear to left shoulder, look right, look left, tilt head back, rest forehead on arms on the bar
- Keep eyes closed throughout entire demonstration
- Have student describe sensation of direction during each movement

**Disorientation 2**
- Have student sit with head down and turned to one side on the arms, resting on the bar
- Rotate the chair until equilibrium is reached
- Have student lift head and put other ear down on bar
- Repeat if comfortable
- Stop chair and have the student raise arms overhead in a “touchdown” fashion with eyes opened or closed
Appendix 7: Phonetic Alphabet

A  Alfa
B  Bravo
C  Charlie
D  Delta
E  Echo
F  Foxtrot
G  Golf
H  Hotel
I  India
J  Juliet
K  Kilo
L  Lima
M  Mike
N  November
O  Oscar
P  Papa
Q  Quebec
R  Romeo
S  Sierra
T  Tango
U  Uniform
V  Victor
W  Whiskey
X  X-ray
Y  Yankee
Z  Zulu

For the Air Force vocabulary, visit:
http://www.nationalmuseum.af.mil/factsheets/factsheet.asp?id=7654 and
Appendix 8: Internet Resources

1.2. Respiration
http://www.faa.gov/pilots/training/airman_education/topics_of_interest/hypoxia/rem_hypoxia/index.cfm (hypoxia; 7 Oct 07)

1.9. Nutrition
http://www.faa.gov/library/reports/medical/hop/media/topics_issues.pdf (health of pilots; 8 Oct 07)
http://www.fitness.gov/exerciseweight.htm (diet, exercise expenditures; 8 Oct 07)
http://dietary-supplements.info.nih.gov/Health_Information/Dietary_Reference_Intakes.aspx (Water and electrolyte guidance; 26 Jun 08)

2.1. Constituents and Properties of the Atmosphere
http://history.nasa.gov/SP-367/chapt2.htm (aerodynamics of flight; 7 Oct 07)
http://www.lerc.nasa.gov/WWW/K-12/airplane/sound.html (speed of sound; 7 Oct 07)
http://www.adl.gatech.edu/classes/dci/aerodesn/dci03aero.html (aerodynamics of flight; 7 Oct 07)
http://www.cpc.ncep.noaa.gov/products/stratosphere (stratospheric filtering of UV radiation; 7 Oct 07)
http://www.chm.davidson.edu/ChemistryApplets/GasLaws/index.html (Gas Laws; 7 Oct 07)
http://www.nndb.com/people/278/000049131/ (Dalton’s Law; 7 Oct 07)
http://hyperphysics.phy-astr.gsu.edu/hbase/kinetic/relhum.html (relative humidity; 7 Oct 07)
http://mtl.jpl.nasa.gov/notes/altitude/altitude.html (altitude definitions; 8 Oct 07)
http://www.faa.gov/pilots/training/airman_education/topics_of_interest/hypoxia/rem_hypoxia/index.cfm (FAA hypoxia info; 8 Oct 07)

3.1. Hypoxia
http://mtl.jpl.nasa.gov/notes/altitude/altitude.html (altitude definitions; 8 Oct 07)
http://www.faa.gov/pilots/training/airman_education/topics_of_interest/hypoxia/rem_hypoxia/index.cfm (FAA hypoxia info; 8 Oct 07)
http://www.bordeninstitute.army.mil/published_volumes/harshEnv2/harshEnv2.html (Medical Aspects of Harsh Environments, Volume 2)

3.3. Trapped Gas
http://www.faa.gov/other_visit/aviation_industry/designees_delegations/designee_types/ame/media/Section%20II.1.4%20Trapped%20Gases.doc (Frenzel and Valsalva maneuvers; 8 Oct 07)

3.4. Altitude Decompression Sickness (DCS)

---

4 urls are shown with topic number/title and last date checked in parentheses.
3.4. Altitude DCS Risk Assessment Computer (ADRAC) Model

https://biodyn.istdayton.com/CBDN/Login/Login.aspx  (ADRAC registration/login; 29 Aug 2016)

The ADRAC model is available via the Air Force Research Laboratory. User registration must be completed and a username/password established via the web site before the model can be accessed.

Once logged into the AFRL Aircrew Performance and Protection Data Bank site, clicking on the ADRAC button and Go To Lab Program button will display the screen shown in Figure A8-1 (sans table on the right). Entering the altitude, prebreathe time, and exercise level followed by clicking Calculate Risk will result in update of the screen with a table like the one shown in Figure A8-1 with time at altitude and predicted DCS risk throughout the exposure time available. A chart that shows the level of risk throughout the exposure time available may be obtained by clicking Create Graph as shown below (Figure A8-2).

![Altitude Decompression Sickness Risk Assessment Computer (ADRAC)](https://biodyn.istdayton.com/CBDN/Login/Login.aspx)

**Figure A8-1. ADRAC Prediction of DCS Risk at 25,000 ft with 30 Minutes of Ground-Level Prebreathe and Exercise Level at Altitude = Rest**
Figure A8-2. ADRAC Graph of DCS Risk at 25,000 ft with with 30 Minutes of Ground-Level Prebreathe and Exercise Level at Altitude = Rest

4.1. Human Performance
http://www.coloradofirecamp.com/swiss-cheese/introduction.htm (Human Error)

4.1.4. Crew Resource Management

4.2. Self-Medication
National Center for Complimentary and Alternative Medicine:
http://nccam.nih.gov/health/herbsataglance.htm
USAF Policy Letter on Nutritional Supplements:
https://kx.afms.mil/kxweb/dotmil/file/web/ctb_010712.pdf;jsessionid=BF4DE1D36113C80FC52C787C4670FB4D
USAF Policy Letter on the Use of Ephedra Containing Nutritional Supplements:
https://kx.afms.mil/kxweb/dotmil/file/web/ctb_012653.pdf;jsessionid=8C00D0A4D2FB4D1D2A450150C09DB879
SF 600 Overprint for review of nutritional supplements:
Aircrew Medication List and Over the Counter Medication List:
https://kx.afms.mil/aersopacemedicine

4.2. Smoking and Alcohol
http://www.airpower.maxwell.af.mil/airchronicles/aureview/1979/may-jun/bronson.html (Airpower article, well documented; 25 Feb 09)
http://legacy.library.ucsf.edu/tid/qzx62f00 (report on Smoking: Its adverse effects on airline pilot performance)
4.3. Fatigue and Fatigue Countermeasures References
http://faid.interdynamics.com/ (Fatigue Audit InterDyne (FAID); 9 Oct 07)
http://www.sleepfoundation.org (National Sleep Foundation website; 9 Oct 07)

5.2. Oxygen Systems

Use of oxygen for breathing and prebreathing in chambers carries a risk of producing an atmosphere of increased burning rate as defined by the National Fire Protection Association (NFPA) Publication 99B (2005). The NFPA 99B, Standard for Hypobaric Facilities (2005; 3.3.3.3), defines an atmosphere of increased burning rate based on a 12-mm/s burning rate (at 23.5% oxygen at 1 ATA). The equation defining such an atmosphere (NFPA 99B Chapter 3 Definitions; 3.3.3.3) is:

\[
NFPA \text{ max } O_2 \% = \frac{23.45}{\left(\frac{P_B}{760}\right)^{0.5}}
\]

where:

\[
NFPA \text{ max } O_2 \% = \text{ max } O_2 \% \text{ without being an atmosphere of increased burning rate (see Table A8-1 for examples)}
\]

\[
P_B = \text{ barometric pressure (mmHg)}
\]

<table>
<thead>
<tr>
<th>Altitude (ft)</th>
<th>(P_B) (mmHg)</th>
<th>(P_B) (psia)</th>
<th>TP_\text{atmospheres}</th>
<th>NFPA Max O_2 (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>760.1</td>
<td>14.7</td>
<td>1.000</td>
<td>23.4</td>
</tr>
<tr>
<td>5,000</td>
<td>632.4</td>
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<td>0.832</td>
<td>25.7</td>
</tr>
<tr>
<td>8,000</td>
<td>564.6</td>
<td>10.9</td>
<td>0.743</td>
<td>27.2</td>
</tr>
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<td>10.1</td>
<td>0.688</td>
<td>28.3</td>
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<td>428.6</td>
<td>8.3</td>
<td>0.564</td>
<td>31.2</td>
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<td>0.510</td>
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<td>4.4</td>
<td>0.297</td>
<td>43.1</td>
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<tr>
<td>35,000</td>
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<td>3.5</td>
<td>0.235</td>
<td>48.3</td>
</tr>
<tr>
<td>40,000</td>
<td>141.2</td>
<td>2.7</td>
<td>0.186</td>
<td>54.4</td>
</tr>
</tbody>
</table>

7.3. Spatial Disorientation
http://www.faa.gov/pilots/training/airman_education/topics_of_interest/spatial_disorientation/index.cfm (FAA AC No: 120-51E on CRM; 7 Apr 08)

7.4. Motion Sickness
http://www.stens-biofeedback.com/about_biofeedback.html (Stens Corp. web site; 9 Oct 07)
http://www.bordeninstitute.army.mil/published_volumes/harshEnv2/harshEnv2.html (Medical Aspects of Harsh Environments, Volume 2)
http://www.bordeninstitute.army.mil/published_volumes/harshEnv2/HE2ch35.pdf (Chapter 35. Motion Sickness of the above text on military medicine)
http://www.flixxy.com/aerobic-flight.htm (aerobatics video showing head movements of performers)

7.5. Impact and Ejection
http://www.aeromuseum.org/aircraft_b58.html

7.7. Laser Awareness Training
http://www.acc.af.mil/xf/ARMED/ASD/A8D/a8dr.htm (Laser Information Training and Education (LITE) training program, Aircrew Laser Eye Protection Specifications, etc.
http://www.a3a5.hq.af.mil/a5r/a5re/docs/directenergy.htm (Directed Energy Task Force (DETF); historical information about lasers and the DE Roadmap offers guidance and boundaries for more detailed roadmaps; Eye Protection Task Force
http://www.decaag.af.mil.html (DECAAG website; a collection of documents on DE written by aircrew at the USAF Warfare Center)
http://stinet.dtic.mil/cgi-bin/GetTRDoc?AD=ADA464670 and Location=U2 and doc=GetTRDoc.pdf (based on FM 8-50)

7.8. Thermal Stress

Glossary, Abbreviations, Acronyms, and Definitions
http://www.cpc.noaa.gov/products/outreach/glossary.shtml#Z (NWS Glossary; 11Dec07)
http://www.azdot.gov/aviation/library/MP_PDF/1G4_MP_APP_B.pdf (Arizona Department of Transportation; 11 Dec 07)
http://www.e-publishing.af.mil/shared/media/epubs/AT-M-06A.pdf (Glossary on CRM; 7 Apr 08)
Appendix 9: AFI 11-403 Original Course Objectives

Physiological Effects of Altitude

1) Know the characteristics of the Earth’s atmosphere.
   a. Recognize the functions of the atmosphere.
   b. Identify which gases are present in the atmosphere and their associated percentage of the total composition.
   c. Recall the common units of measurement for atmospheric pressure.
   d. Recognize the description and common unit of measure of the U.S. Standard Atmosphere.
   e. Recognize the definition of the standard temperature lapse rate.
   f. Compute the temperature for a given altitude using standard temperature lapse rate.
   g. List the physiological divisions of the atmosphere.

2) Know the impact of the gas laws.
   a. Define partial pressure and identify its notation.
   b. List the gas laws. Identify the following gas laws and their definition:
      Dalton’s law, Boyle’s law, Henry’s law, Gay-Lussac’s law, Graham’s law
   c. Describe the physiological effects of each gas law.

3) Know the structures and functions of the respiratory system.
   a. List the phases of respiration.
   b. Describe the functions of each structure in the respiratory system.
   c. List structures that are important to respiration.

4) Know the structures and functions of the circulatory system.
   a. Recall functions of the structures of the circulatory system.
   b. List the structures of the circulatory system.
   c. Identify factors affecting oxygen delivery to the tissues.

5) Know the characteristics of hypoxia.
   a. Identify the definition of hypoxia.
   b. Recognize the characteristics of hypoxia.
   c. Recall the types of hypoxia and associated causes.
   d. Identify the factors that induce hypoxia and change onset rate of symptoms.

6) Know the signals for recognizing hypoxia and methods for preventing it.
   a. Recognize the importance of immediately correcting for hypoxia after a rapid decompression.
   b. Identify the signs and symptoms of hypoxia onset.
   c. Identify the procedures to treat hypoxia.
   d. Observe hypoxia symptoms exhibited during altitude exposure.
7) Know the characteristics of hyperventilation.
   a. Recall the definition and causes of hyperventilation.
   b. Recall the signs of and symptoms of hyperventilation.
   c. Identify the procedures to treat hyperventilation.
   d. Describe in-flight emergency procedures for treating hyperventilation.
   e. Recall the similarities of treatment for hypoxia and hyperventilation.

8) Know the characteristics of trapped gas disorders.
   a. Identify the various trapped gas disorders and areas of the body most likely to be affected.
   b. Recall symptoms of trapped gas disorders and the phase of flight in which symptoms are likely to occur.
   c. Explain how to treat and prevent trapped gas disorders.

9) Know the characteristics of decompression sickness.
   a. Identify the common types and causes of decompression sickness.
   b. Identify the impact various factors have on DCS incidence and severity.
   c. Identify the symptoms associated with each type of decompression sickness.
   d. Recall the corrective actions for suspected decompression sickness.
   e. List methods used to treat decompression sickness.

**Performance Threats**

1) Know the effects over-the-counter (OTC) medications have on the crewmember.
   a. Identify the effects of OTC medications.
   b. List the types of OTC medications.
   c. Describe the potential for nutritional supplements to affect in-flight performance.
   d. Recall Air Force policy on OTC medications and nutritional supplements.

2) Know the residual effects of alcohol on a crewmember in-flight.
   a. Identify the effects of alcohol on the body, specifically impacts on in-flight performance.
   b. Identify Air Force policy concerning alcohol consumption by crewmembers.

3) Know the hazards associated with smoking and chewing tobacco products.
   a. Describe the effects of smoking and smokeless tobacco, specifically impacts on in-flight performance.
   b. Recall the physiological effects of carbon monoxide.

4) Know the physiological need for proper diet and nutrition.
   a. Describe the effects of hypoglycemia on in-flight performance.

5) Know the adverse impact of dehydration on crewmember performance.
   a. List the signs and symptoms associated with dehydration.
   b. Recall methods to combat dehydration.
6) Know the causes of acute and chronic fatigue.
   a. List the causes and cures for acute and chronic fatigue.
   b. Recognize ways to reduce the effects of fatigue.
   c. Describe how to minimize the effects of circadian rhythm disruptions.

7) Know the role of GO / NO-GO pills as fatigue management strategy.
   a. Describe the aircrew constraints and mission implications of GO / NO-GO pill usage.

8) Know the effects of caffeine on the body.
   a. List the effects of caffeine on the body.
   b. Understand the impacts of caffeine on in-flight performance.

9) Know the importance of physical fitness on aircrew situations.
   a. Recall proper aerobic and anaerobic fitness principles.

10) Know the effects of thermal stress on human performance.
    a. Identify impacts to in-flight performance resulting from hot / cold stress.
    b. Understand the recommendations for protection from cold stress.
    c. Understand the recommendations for protection from heat stress.
    d. Identify thermal stress impacts on physiological problems in flight.

   **Aircrew Flight Equipment**

1) Identify and operate aircraft oxygen systems.
   a. Describe safety concerns and characteristics of aviator’s breathing oxygen.
   b. Describe the characteristics of aircraft oxygen storage systems.
   c. Understand the function of pressure demand regulators.
   d. Identify the proper use of helmet, mask, and connector assemblies.
   e. Identify and operate emergency oxygen sources.
   f. Demonstrate and explain the P.R.I.C.E. check.

   **Cabin Pressurization and Decompression**

1) Know how aircraft pressurization affects aircrew members.
   a. Recall the process for maintaining cabin pressurization.
   b. Describe the different pressurization systems.
   c. Identify the advantages and disadvantages of pressurization systems.
   d. Recall the types of decompression and characteristics of each.
   e. List the physical indications of rapid decompression.
   f. Describe procedures for preventing physiological problems after rapid decompression.

   **Pressure Breathing**

1) Know the pressure breathing requirements and techniques.
   a. Describe why and when pressure breathing is necessary for the aircrew member.
   b. Describe the normal respiration cycle.
c. Describe changes required to normal respiration resulting from pressure breathing.

**Vision**

1) Know the anatomy and function of the eye.
   a. Recognize parts of the eye that are critical to vision.
   b. Identify the function of each part of the eye.
   c. Recognize the physiological blind spots associated with parts of the eye.

2) Know the characteristics of the visual field.
   a. Identify the characteristics of both focal and peripheral vision.
   b. Describe the limitations of focal and peripheral vision.

3) Know the limitations and visual illusions associated with daytime flight.
   a. Recognize how visual contrast, target shape, target movement, environmental conditions, and empty-field myopia limit the ability to perceive objects in the visual field.
   b. Identify the effect that perception, reaction time, visual acquisition, and scanning have on midair collision avoidance.
   c. Describe the correct scanning technique used to identify objects.
   d. List factors that can cause daytime visual illusions.

4) Know the limitations and visual illusions associated with low-light level and night flying environments.
   a. List the physiological characteristics of night vision.
   b. Describe dark adaptation’s influence on night vision.
   c. Define the night blind spot.
   d. Identify the correct technique to keep an object in sight at night or under low-light conditions.

5) Comprehend night vision conditions that contribute to illusions.
   a. Demonstrate the method used to prevent the autokinesis illusion.
   b. Demonstrate how flash blindness produces debilitating effects on dark adaptation.

6) Know the measures that help ensure maximum visual acuity in night flying conditions.
   a. Describe techniques to maximize visual acuity in night environments.
   b. Identify methods to prevent night visual illusions.
   c. Understand basic night vision goggle (NVG) components and how they function.
   d. Understand basic limitations of NVGs such as the effects of illumination, contrast, and shadows on image quality.
   e. List methods to improve NVG use.

7) Know the characteristics of lasers and associated actions upon exposure.
a. List the physiological hazards associated with laser exposures.
b. Identify procedures to prevent laser exposure injuries.
c. Identify how and when to report exposure to lasers.

Spatial Disorientation
1) Know the characteristics of spatial disorientation.
   a. Know the threats and impacts of the different types of spatial disorientation.
   b. List the four sensory systems used in orientation.
   c. Define the relationship of the sensory systems to spatial disorientation.

2) Know the characteristics of the orientation sensory systems.
   a. Select the sensory system that provides the strongest, and usually most reliable orientation information.
   b. Describe the vestibular system.
   c. Define the relationship of the vestibular system and the two subsystems: semicircular canals and the otolith organs.
   d. Explain the reason for the somatosensory system’s unreliability in-flight.
   e. Describe the somatosensory system’s function in-flight.

3) Know the characteristics of the types of vestibular induced spatial disorientation.
   a. Identify the cause of somatogyral illusions.
   b. Identify the cause of somatogravic illusions.

4) Know the factors affecting spatial disorientation. (Note 2)
   a. Recall affecting environmental factors of spatial disorientation.
   b. Recall affecting physiological factors of spatial disorientation.

5) Know how to prevent and / or overcome spatial disorientation.
   a. Recall methods used to prevent spatial disorientation.
   b. Recall procedures used to overcome spatial disorientation.

6) Know the causes of and techniques to prevent / overcome motion sickness in-flight.
   a. Identify the most widely accepted theory of the cause for motion sickness.
   b. Identify techniques to help prevent and / or treat motion sickness.

Noise and Vibration
1) Know the characteristics of noise.
   a. Define noise.
   b. List characteristics of noise that affect hearing.
   c. Recall the definitions and units of measure of frequency, intensity, and duration.

2) Know the effects of hazardous noise on hearing capability.
   a. List types of hearing loss associated with high intensity noise.
   b. Identify the potential non-auditory effects of noise on crewmembers in-flight performance.
3) Know protective measures used to minimize hazardous noise exposure.
   a. List devices that help minimize hazardous noise.
   b. Describe techniques for minimizing hazardous noise exposure.

4) Know the potential effects of prolonged exposure to aircraft vibration.
   a. Recall the definition of vibration.
   b. Identify how vibration energy is passed through the body.
   c. Describe symptoms of vibration exposure.

   **Attention Management Threats to Situational Awareness**

1) Know the levels of awareness.
   a. Identify the two primary levels of awareness.
   b. Outline the subconscious level of awareness.

2) Comprehend some of the cognitive causes of a loss of situational awareness.
   a. Identify attention threats.
   b. Identify temporal distortion.
   c. Summarize task saturation.
   d. Identify inappropriate motivations.

3) Know how to recognize, prevent, and treat lost situational awareness resulting from attention management threats.
   a. Identify the tools for preventing lost situational awareness.
   b. Identify cues for recognizing a loss of situational awareness.
   c. Identify the techniques for recovering from lost situational awareness.

4) Comprehend the impacts of physiological issues on situational awareness.
   a. Identify physiological issues that can potentially degrade an individual’s situational awareness.
   b. Identify physiological issues that can degrade an individual’s decision-making.

   **Acceleration (Required only for personnel flying in high-G aircraft)**

1) Know the definition and characteristics of G forces.
   a. Identify the types of acceleration.
   b. Identify the types of G force.
   c. Recall the definition of each type of G force.
   d. Identify the physical symptoms associated with each type of G force.

2) Know the characteristics of the factors that determine the effects of G forces on a crewmember’s body.
   a. List the factors determining the effects of G force on a crewmember’s body.
   b. Identify principle physiological effects and associated symptoms of exposure to G forces.
c. Recognize what causes blackout and how it is different than G-Induced Loss of Consciousness (G-LOC).

3) Comprehend the characteristics of G-LOC.
   a. Describe the symptoms of each of the phases of incapacitation.
   b. Explain the impact of relative incapacitation on the total time required to regain control of the aircraft after G-LOC.

4) Know the methods used to help prevent G-LOC.
   a. List methods to increase G tolerance.
   b. Detail g-suit function and level of protection provided.
   c. Describe the elements involved in correctly performing the Antit-G Straining Maneuver (AGSM).

5) Know the common errors in performing the AGSM.
   a. List errors involved in performing the AGSM.
   b. Detail common mission characteristics that are likely to cause AGSM errors.

6) Know the characteristics of the methods used to increase a crewmember's tolerance to positive G-forces.
   a. Describe physiological factors related to increased performance in a positive G force environment.
   b. Recognize the role self-imposed stressors play in decreasing G force tolerance.

Aircraft Egress

1) Know the principle courses of action to minimize injury during aircraft egress.
   a. Understand basic principles of aided escape in relation to design.
   b. Describe common aided escape injuries.
   c. Understand basic principles of unaided escape.
   d. Describe common unaided escape injuries.
   e. Describe the physiological threats of high altitude egress.
   f. Describe ways to improve survivability before, during, and after a crash.
Appendix 10: Objectives for Board Certification in Aerospace Physiology

The Executive Council of the Aerospace Medical Association (AsMA), acting upon recommendations of the Aerospace Physiology Certification Board, grants certification in Aerospace Physiology (CAaP). Board certification in Aerospace Physiology was established by the Aerospace Medical Association to encourage the study, improve the practice, and elevate the standards of excellence in Aerospace Physiology. For details regarding eligibility to sit for the exam, as well as exam references and guidance, please visit the Aerospace Physiology Society’s website (http://aspsociety.org/). The following objectives are established and maintained by the Aerospace Physiology Certification Board:

UNIT 1 - ACCELERATION

Background Physics
  1.1 Describe the XYZ coordinate system used in describing aircraft acceleration.
  1.2 Define linear, angular, and centripetal acceleration.
  1.3 Define “G force” as it applies to the aviation environment.

Physiological Aspects
  1.4 State the effects of $+G_z$ on the body.
  1.5 State the effects of $-G_z$ on the body.
  1.6 Describe the “push/pull” effect and the resultant reduction in G tolerance.
  1.7 Describe the principal central nervous system factors that limit short-duration, high $+G_z$ exposure tolerance.
  1.8 Given a G-time Tolerance curve, describe the regions of the curve and how they relate to human performance while experiencing Gs.
  1.9 List the symptoms of grayout, blackout, and G-induced loss of consciousness (GLOC), as they apply to the $+G_z$ environment.
  1.10 Describe the differences between absolute and relative incapacitation as they relate to GLOC.
  1.11 Differentiate between Almost Loss of Consciousness (ALOC) and GLOC.
  1.12 List physical and physiological factors that may reduce an individual’s tolerance and/or endurance to G forces.
  1.13 Describe potential harmful effects of sustained G

Prevention
  1.14 List the methods available to increase a crewmember’s tolerance to $+G_z$ forces.
  1.15 State the benefits of wearing an anti-G suit.
  1.16 Describe the elements of the anti-G straining maneuver (AGSM) and how they interrelate.
1.17 Describe common errors in performing the AGSM.
1.18 Describe exercises (physical training) that can be performed in order to improve an individual’s G-tolerance.

UNIT 2 - THE ATMOSPHERE

Atmospheric Zones/Pressure/Temperature
2.1 Describe the composition of the earth’s atmosphere.
2.2 Describe atmospheric pressure and how it is caused.
2.3 Define the U.S. Standard Day (pressure and temperature).
2.4 Describe standard units used to measure atmospheric pressure and convert a given pressure from one unit to another.
2.5 Compute a temperature for a given altitude using the standard temperature lapse rate.
2.6 Define the physiological divisions/zones of the atmosphere.
2.7 Define the temperature divisions/zones of the atmosphere.

Gas Laws
2.8 Define Boyle’s Law and apply it to specific calculation examples.
2.9 Define Charles’ Law and apply it to specific calculation examples.
2.10 Define Henry’s Law and apply it to specific calculation examples.
2.11 Define Dalton’s Law of Partial Pressure and apply it to specific calculation examples.

UNIT 3 - CARDIOVASCULAR PHYSIOLOGY

Muscle Physiology
3.1 Describe the basic physiological process involved with muscle contraction.
3.2 Describe the differences between isometric and isotonic muscle contraction.
3.3 Describe the structures associated with the neuromuscular junction.
3.4 Describe the effects of acetylcholine in the neuromuscular junction.
3.5 Describe the charge and time of the muscle action potential.
3.6 Describe the function of T-tubules in the spreading of a muscle action potential.
3.7 Describe the function of calcium ions with respect to muscle action potential propagation.
3.8 Describe the different types of smooth muscle.
3.9 Describe the differences between striated and smooth muscle anatomy.
3.10 Describe the differences between striated and smooth muscle physiology.
3.11 Describe the physiological function of a smooth muscle neuromuscular junction.
3.12 Describe the physiological function of smooth muscle action potentials.
3.13 Describe the effect of calcium ions on smooth muscle action potential generation/propagation.

Cardiac Function and Physiology
3.14 Describe physiological concerns specific to cardiac muscle.
3.15 Describe the role of the atria, ventricles, and valves in each phase of the cardiac cycle.
3.16 Describe the five cardiac fraction/volume measurements.
3.17 Describe the Frank-Starling mechanism of cardiac pumping regulation.
3.18 Describe the roles of the A-V node, sinus node, A-V bundle, and Perkinje fibers in the control of the cardiac cycle.
3.19 Describe the effects of parasympathetic and sympathetic stimulation on cardiac function.
3.20 Describe the basic components of an EKG reading.

Circulatory Physiology and Blood Pressure Regulation
3.21 Describe the primary functions of the circulatory system.
3.22 Describe the general functional anatomy of the circulatory system.
3.23 Describe the basic process and physical principles of blood flow.
3.24 Describe factors that determine/impact resistance to blood flow.
3.25 Describe Poiseuille’s Law.
3.26 Describe the effects of vascular distensibility on blood pressure/volume control.
3.27 Describe the effects of vascular compliance on blood pressure/volume control.
3.28 Describe the physical/physiological factors that affect arterial pressure.
3.29 Describe the physical/physiological factors that affect venous pressure.
3.30 Describe components of the microcirculatory anatomy.
3.31 Describe the mechanism for blood flow through the capillaries.
3.32 Describe the physiology of nutrient exchange in the capillaries.
3.33 Describe the composition of the interstitium and interstitial fluid.
3.34 Describe the forces/pressures that determine the distribution of fluid across the capillary membrane.
3.35 Describe the basic anatomy of the lymphatic system.
3.36 Describe the purpose/function of the lymphatic system.
3.37 Describe factors that acutely control local blood flow.
3.38 Describe factors that are long term controls of local blood flow.
3.39 Describe the effects of various hormones on the regulation of circulation.
3.40 Describe the effects of ionic concentration on the regulation of blood flow.
3.41 Describe autonomic nervous system regulation of the circulatory system.
3.42 Describe the physiology of the arterial baroreceptor reflex control system.
3.43 Describe the function of the carotid and aortic chemoreceptors.
3.44 Describe the function of the cardiopulmonary reflex in arterial pressure regulation.
3.45 Describe the mechanism of the renal-body fluid system of blood volume/pressure regulation.
3.46 Describe the mechanism of the renin-angiotensin system of blood volume/pressure regulation.
3.47 Describe factors which effect coronary vessel output and venous return.
3.48 Describe the muscular blood flow regulating systems during exercise.
3.49 Describe the cardiovascular changes that occur during exercise.
3.50 Describe the effects of exercise on coronary circulation.
3.51 Describe the factors that lead to ischemic heart disease.
3.52 Describe the factors that lead to heart failure.

UNIT 4 - DECOMPRESSION PHYSIOLOGY

Physics of Decompression and Pressurization Systems
4.1 Describe the primary purpose for aircraft cabin pressurization.
4.2 Describe the physical indications of a rapid decompression.
4.3 Describe factors that can affect the rate of a cabin depressurization.
4.4 Relate the rate of cabin depressurization to respiratory, trapped gas, and evolved gas related disorders.

Physiology of Trapped Gas Disorders (Barotraumas)
4.5 Compare the relative effects of depressurization on wet and dry gasses.
4.6 State the anatomical structures most likely to be affected by a decrease and/or increase in ambient pressure.
4.7 Describe the physical, anatomical, and physiological causes of an ear block.
4.8 List the symptoms of an ear block.
4.9 Describe the technique for the prevention of ear blocks on ascent.
4.10 Describe the physical, anatomical, and physiological causes of a sinus block.
4.11 List the steps for treating a sinus block.
4.12 Describe the physical, anatomical, and physiological causes of baro-induced tooth pain.
4.13 List the steps for the treatment of tooth pain.
4.14 List the steps for the treatment of trapped gas expansion in the intestines.
Physiology of Evolved Gas Disorders (Decompression Sickness/Illness)
4.15 State the effects of atmospheric pressure on the incidence of DCS.
4.16 Describe the relationship between micro-bubble nuclei and DCS
4.17 Relate supersaturation and critical supersaturation to the onset of DCS.
4.18 Describe Haldane’s theory for DCS.
4.19 List the symptoms of the four manifestations of DCS.
4.20 Describe factors which may predispose a crewmember to DCS.
4.21 List the steps for treating DCS.
4.22 Describe the physical and physiological benefits of hyperbaric therapy in the treatment of DCS.

Physiology of Pulmonary Over-inflation Disorders
4.23 Describe the causes and symptoms of pneumothorax.
4.24 Describe the causes and symptoms of substernal emphysema.
4.25 Describe the causes and symptoms of aero-embolism.
4.26 Describe the standard treatment protocol(s) for pulmonary over-inflation injuries.

UNIT 5 - HYPERBARIC PHYSIOLOGY

Hyperbaric Physics
5.1 Describe the relationship between depth (in sea water) and ambient pressure.
5.2 Calculate volumetric changes in gas as a function of change in sea water depth.
5.3 Describe the relationship between hyperbaric exposure and hypobaric exposure.

Nitrogen Narcosis
5.4 Describe the physiological mechanism that causes nitrogen narcosis.
5.5 State the depth in sea water at which nitrogen narcosis can become a physiologic concern.
5.6 Describe the symptoms of nitrogen narcosis.

Oxygen Toxicity
5.7 Describe the physiological mechanism that causes pulmonary oxygen toxicity.
5.8 Describe the symptoms of pulmonary oxygen toxicity.
5.9 Describe the physiological mechanism that causes neural oxygen toxicity.
5.10 Describe the symptoms of neural oxygen toxicity.

Recompression Therapy for Dysbarism Injuries/Syndromes
5.11 Describe the physical and physiological explanation for the use of recompression therapy in the management of DCS.
5.12 Describe the physical and physiological explanation for the use of recompression therapy in the management of aero-embolism.

**Hyperbaric Oxygen Therapy**

5.13 List (non-pressure related) medical conditions that may be treated with hyperbaric oxygen therapy.

5.14 Describe the physiological basis for using HBO therapy for the treatment of carbon monoxide poisoning.

5.15 Describe the physiological basis for using HBO therapy for wound healing.

5.16 Describe the physiological basis for using HBO therapy for treating radial osteonecrosis.

5.17 Describe the physiological basis for using HBO therapy for treating gas gangrene.

**UNIT 6 - METABOLISM, THERMOREGULATION, AND HOMEOSTASIS**

**Homeostasis and Body Fluids**

6.1 Define homeostasis.

6.2 List examples of extracellular fluid.

6.3 Describe the mechanisms by which nutrients are supplied to and waste products are removed from extracellular fluid.

6.4 Match physiologic control systems (e.g. nervous system, endocrine system, etc.) to the action which they control.

6.5 Describe the features of a negative feedback control system.

6.6 Describe the features of a feed-forward control system.

6.7 Describe the differences in composition of intra and extracellular fluid.

6.8 Describe the differences between channel proteins and carrier proteins.

6.9 Describe the differences between simple and facilitated diffusion.

6.10 Describe the features of protein channels.

6.11 Describe the features of protein gates, differentiating between the various mechanisms (e.g. chemical and voltage).

6.12 Describe the steps involved in facilitated diffusion.

6.13 Describe how each of the following affects the rate of diffusion across a membrane: permeability, concentration, electric potential, and pressure.

6.14 Describe osmosis, osmotic pressure, and osmolality.

6.15 Describe the processes of active transport.

6.16 Describe the mechanism of the sodium/potassium pump.

6.17 Describe the differences between co-transport and counter-transport.

6.18 State the fluid volumes of intracellular and extracellular fluid compartments.

6.19 Discuss the physiological causes of edema
Renal Function and Fluid Level Balance
6.20 Describe the physiologic anatomy of the kidney.
6.21 Describe the principal mechanism by which the nephron filters the blood plasma.
6.22 Describe the roles of the glomerulus and the tubules in the reabsorbing of fluid and electrolytes.
6.23 Describe the overall mechanism for blood volume control by the renal system.
6.24 Describe the renal system’s role in the control of extracellular fluid volume.

Thermoregulation
6.25 State the normal skin and core body temperatures.
6.26 State the factors that determine the rate of metabolic heat production.
6.27 Describe the insulatory effects of the skin.
6.28 Describe the effect of the circulatory system on body cooling.
6.29 Describe how conduction, convection, and evaporation effect thermoregulation with regards to the skin.
6.30 Describe the physiological mechanism involved in the production of sweat.
6.31 Describe the role of the hypothalamus in the regulation of body temperature.
6.32 Describe the physiological mechanism that produces fever.
6.33 State the symptoms of heat stroke.
6.34 Describe the harmful physiological effects of untreated heat stroke.
6.35 Describe the physiological effects of exposure to extreme cold.

Acid Base Balance
6.36 Define the terms “acid” and “base” as they apply to human physiology.
6.37 State the “normal” pH of various fluids and tissues of the human body.
6.38 Describe the bicarbonate buffer system.
6.39 Describe the phosphate buffer system.
6.40 Describe the protein buffer system.
6.41 Describe the function of the respiratory system in the regulation of acid-base balance.
6.42 Describe the function of the renal system in the regulation of acid-base balance.
6.43 Describe the causes of metabolic acidosis and alkalosis.
6.44 Describe the effects of acidosis and alkalosis on the body.

UNIT 7 - NERVOUS AND ENDOCRINE FUNCTION/CONTROL

Basic Neurophysiology (Action Potentials)
7.1 Describe the basic physics of membrane potentials.
7.2 Describe the use for/purpose of the Nernst and Goldman equations.
7.3 Describe the determinants of a resting membrane potential.
7.4 Describe the contributions of the sodium and potassium diffusion potentials and the sodium/potassium pump in the determination of a resting potential.
7.5 Describe the three stages of an action potential.
7.6 Describe the functioning of the voltage-gated sodium and potassium channels.
7.7 Describe the positive-feedback aspects of the action potential.
7.8 Describe the threshold for the propagation of an action potential.
7.9 Describe the all-or-nothing principle of action potentials.
7.10 Describe the mechanism for restoring the sodium/potassium concentrations following action potentials.
7.11 Describe the effects of nerve cell myelination and conduction speed.
7.12 Identify the effect of acidosis/alkalosis and hypoxia on synaptic transmissions.

**Hearing**
7.13 Describe the functional anatomy of the cochlea.
7.14 Identify the function of the cochlea in determining sound characteristics.
7.15 Describe the function of the organ of Corti in the acquisition of sound waves.
7.16 Describe the central nervous components of the sense of hearing.
7.17 Identify the two types of acquired deafness and their causes.

**Endocrine Physiology**
7.18 List the nine primary endocrine glands.

State the gland of origin and basic function of each (7.19 – 7.30) of the following hormones:

- 7.19 Growth Hormone
- 7.20 Adrenocorticotropicin
- 7.21 Thyroid-stimulating Hormone
- 7.22 Antidiuretic Hormone
- 7.23 Cortisol
- 7.24 Aldosterone
- 7.25 Thyroxine
- 7.26 Calcitonin
- 7.27 Insulin
- 7.28 Glucagon
- 7.29 Parathormone
- 7.30 Epinephrine and norepinephrine
7.31 Describe the basic mechanism by which hormones affect target tissues.
7.32 Describe the second messenger mechanisms for mediating hormonal functions.
7.33 Describe the relationship between the pituitary gland and the hypothalamus.
7.34 Describe the formation and excretion of the thyroid hormones and their effects on the tissues of the body.
7.35 Describe the formation and excretion of the adrenocortical hormones and their effects on the tissues of the body.
7.36 Discuss the relationship between Insulin, glucagon, and diabetes mellitus.
7.37 Describe the hormonal control effects of calcium levels in the body.

UNIT 8 - THE PHYSIOLOGY OF VISION AND THE VESTIBULAR SYSTEM

Anatomy and Physiology of Vision
8.1 State the function of the cornea.
8.2 State the function of the lens.
8.3 State the function of the retina.
8.4 State the function of the optic disk.
8.5 State the function of the cone cells.
8.6 State the function of the rod cells.
8.7 List the physiological steps in the process of the dark adaptation.
8.8 List the effects of glare and retinal bleaching.
8.9 Describe the term refractive index and how it relates to light passing through a lens.
8.10 Describe the function of concave and convex lenses.
8.11 Describe the optical principles of the eye that allow for near and distant vision.
8.12 Describe the fluid systems of the eye.
8.13 Describe the 10 cellular layers of the retina.
8.14 Describe the anatomical structures of the photoreceptors.
8.15 Describe the steps in the photochemical process of vision.
8.16 Describe the six types of cells that make up the retinal neural network.
8.17 Describe elements of the post-receptor neurophysiology of the retina.
8.18 Describe the primary neural pathway from the retina to the visual cortex.
8.19 Describe the function of the dorsal lateral geniculate nucleus in the neurophysiology of vision.
8.20 Describe the function of the visual cortex in the neurophysiology of vision.
8.21 Describe the function of the occipital cortex in the neurophysiology of vision.
8.22 Describe the color detection mechanism.
8.23 Describe the three types of eye movements and their neuromuscular control mechanisms.
8.24 Describe the mechanisms for visual accommodation.
8.25 List physiological/psychological concerns specific to the Focal Mode of visual processing.
8.26 List physiological/psychological concerns specific to the Ambient Mode of visual processing.
8.27 Describe the prevalence of color vision defects in males and females.

Anatomy and Physiology of the Vestibular System
8.28 Describe the anatomical components of the middle ear.
8.29 Describe the functional anatomy of the vestibular apparatus.
8.30 Describe the mechanism for sensory production of the saccule and utricle.
8.31 Describe the mechanism for sensory function of the semi-circular canals.
8.32 Describe vestibular reflex actions.
8.33 Describe the practical relationship between the semicircular canals and orientation.
8.34 Describe the practical relationship between the otolith organs and orientation.

Motion Sickness
8.35 Describe the physiologic mechanisms and symptoms associated with motion sickness.
8.36 Identify methods to prevent and treat motion sickness.

UNIT 9 – SPATIAL ORIENTATION AND SITUATIONAL AWARENESS

Visual Illusions

State the potential disorientation factors associated with each (9.1 – 9.19) of the following:

9.1 Empty field myopia.
9.2 Terrain size constancy.
9.3 Runway landing illusions.
9.4 Absent focal cues.
9.5 Whiteout approach.
9.6 Autokinesis.
9.7 Reduced optical flow.
9.8 Linear vection.
9.9 Angular vection.
9.10 Concentric vection.
9.11 False horizons.
9.12 Perspective illusion.
9.13 Black hole illusion.
Vestibular Illusions

9.15 Coriolis cross-coupling.
9.16 Somatogyral illusions.
9.17 Somatogravic illusions.
9.18 Inversion illusion.
9.19 G-excess illusion.

Spatial Disorientation and Situational Awareness

9.20 State the definition of Spatial Disorientation.
9.21 Describe the cognitive causes of Type I (Unrecognized) disorientation.
9.22 Describe the cognitive causes of Type II (Recognized) disorientation.
9.23 Describe the cognitive causes of Type III (Incapacitating) disorientation.
9.24 Describe the importance of focal dominance in preventing/recovering from spatial disorientation.
9.25 Describe factors that aid in maintaining spatial orientation.
9.26 List the steps that can be taken to recover from spatial disorientation.
9.27 Define situational awareness.
9.28 Describe the effects of task saturation on situational awareness.
9.29 Describe the effects of distraction on situational awareness.
9.30 Describe the effects of channelized attention on situational awareness.
9.31 Describe the effects of losing situational awareness on performance.
9.32 Identify the visual techniques used to determine an object’s relative distance.

UNIT 10 - OPERATIONAL PHYSIOLOGY

Stress and Stressors

Describe the potential effects of the following drugs on crewmember performance (Objectives 10.1 – 10.16):

10.1 Antihistamines
10.2 Antihypertensive
10.3 Anticholinergics
10.4 Analgesics
10.5 Antibiotics
10.6 Thiazides
10.7 Thyroid medication
10.8 Amphetamines
10.9 Modafinil
10.10 Zolpidem
10.11 Triazolam
10.12 Zaleplon
10.13 Temazepam
10.14 Anabolic steroids
10.15 Growth hormone
10.16 Melatonin

10.17 Describe the acute and post-consumption residual effects of alcohol on crewmember performance.
10.18 Describe the negative effects of smoking and chewing tobacco products on crewmember performance.
10.19 Describe the physiological need for proper diet and nutrition as they relate to human performance.
10.20 Describe the adverse impact of dehydration on crewmember performance.
10.21 Describe the recommended strategic use of caffeine to enhance aircrew performance.
10.22 List the symptoms of excess stress.
10.23 List examples of physiological stressors.
10.24 List examples of physical stressors.
10.25 List examples of psychological stressors.
10.26 List examples of mission-related stressors.
10.27 List methods of combating stress in the flying environment.

**Desynchronosis and Fatigue**
10.28 Define fatigue and recognize differences between fatigue and boredom.
10.29 List factors that suggest performance is or is likely to be impaired by fatigue.
10.30 Describe circadian rhythms and how they predict alertness.
10.31 Define REM and non-REM sleep.
10.32 Describe a typical sleep cycle.
10.33 Describe the causes of acute and chronic fatigue.
10.34 State the effects of circadian desynchronosis (jet lag) on performance.
10.35 State the effects of fatigue on performance.
10.36 Describe accepted fatigue countermeasures for aviators.
10.37 Describe anti-fatigue strategies for situations involving sleep restrictions.

**Lasers and Laser Eye Protection**
10.38 Define “laser” and state the characteristics of a laser beam.
10.39 Describe the differences between pulsed and continuous wave lasers and the implications for laser eye damage, hazard distances, and protective devices.
10.40 State what wavelengths comprise the following bands of the electromagnetic spectrum: UV, visible, near and far IR and what portion compromises the most significant optical hazard.

10.41 Describe the basic levels of laser systems classification as they pertain to laser systems safety.

10.42 Describe laser eye protection requirements for personnel who may be exposed to laser energy during military operations.

10.43 Describe the relationship between optical density, Maximum Permissible Exposure, and Nominal Ocular Hazard Distance as they pertain to laser eye protection and laser safety.

10.44 Describe the differences between specular and diffuse reflective hazards as they relate to laser range safety.

10.45 State what wavelengths are absorbed by the cornea, lens, and retina and which are considered to be the most dangerous to the eye.

**Night Vision Devices**

10.46 State the typical peak spectral response (wavelength) range of 3rd Generation aviation Night Vision Goggles using gallium arsenide photocathodes (example: AN/AVS-9).

10.47 State the difference between the electromagnetic energy sources for FLIR and NVG sensor imaging.

10.48 State the three major environmental factors influencing NVD performance.

10.49 State the three primary terrain factors that need to be examined for proper NVG mission planning.

10.50 Describe the effects on NVG performance during inadvertent IMC.

10.51 Describe the techniques used to reduce the likelihood of spatial disorientation when using NVGs.

10.52 Describe the basic features of GEN III (AN/AVS-9) NVGs and how the components interact to provide optimal viewing across the operating range.

10.53 Describe the impact NVDs have on visual performance and perception compared to normal daytime vision.

**UNIT 11 - PHYSIOLOGY OF RESPIRATION**

**Pulmonary Anatomy and Physiology**

11.1 Describe the muscular actions associated with pulmonary ventilation.

11.2 Describe the pressures that are responsible for moving air in and out of the lungs.

11.3 Describe the effects of surface tension and surfactant on pulmonary ventilation.

11.4 Describe the functions of the respiratory passageways.

11.5 Describe the function of the three circulatory systems of the lungs.
11.6 Describe pulmonary artery, pulmonary capillary and pulmonary venous pressures.
11.7 Describe the blood volume of the lungs.
11.8 Describe factors that affect blood flow through the lungs.
11.9 Describe the three hydrostatic pressure zones of the lungs.
11.10 Describe the effects of exercise on pulmonary vascular resistance.
11.11 Describe factors that affect the fluid dynamics of the pulmonary capillaries.
11.12 Describe the causes and effects of pulmonary edema.
11.13 Describe the functions of the pleural fluids.

Neural Control of Respiration
11.14 Describe the role of the respiratory centers in the neural control of respiration.
11.15 Describe location and injury of the respiratory centers in the neural control of respiration.
11.16 Describe location and injury implications of the neural control of respiration.
11.17 Describe the effects of altered CO₂ on respiratory control and renal response.
11.18 Describe the role of the peripheral chemoreceptor system in respiratory control.
11.19 Describe factors that affect respiratory control during exercise.

Lung Volumes and Gaseous Composition
11.20 Describe the pulmonary volumes and capacities.
11.21 Describe alveolar ventilation and the associated the resultant air dead spaces.

Gaseous Diffusion in the Lungs
11.22 Describe the physical principles that effect gaseous diffusion.
11.23 Describe the composition of alveolar air and the factors that control this composition.
11.24 Describe the anatomical structure of the respiratory membrane.
11.25 Describe the factors affecting the rate of gaseous diffusion through the respiratory membrane.
11.26 Describe the ventilation-perfusion ratio.

Hemoglobin and Oxygen Transport
11.27 Describe the factors that determine the pO₂ and pCO₂ of the pulmonary blood and of the tissues.
11.28 Describe the percentages of oxygen transported by the red blood cells and the plasma of the blood.
11.29 Describe the oxygen-hemoglobin dissociation curve and the factors that can cause it to "shift".
11.30 Describe hemoglobin oxygen saturation at various pO₂ levels.
11.31 Describe the mechanisms by which CO$_2$ is transported by the blood.

**Hypoxia**

11.32 Recognize, from a list, the subjective and objective symptoms of hypoxia.
11.33 Discuss the relationship between the loss of atmospheric pressure and the onset of hypoxia.
11.34 State the times of useful consciousness at the following altitudes: 18,000 ft, 25,000 ft, 35,000 ft, and 43,000 ft.
11.35 List the five steps for the treatment of hypoxia.

**Hyperventilation**

11.36 List the causes of hyperventilation.
11.37 List the subjective and objective symptoms of hyperventilation.
11.38 List the five steps for treating hyperventilation.
11.39 State the steps for performing the Pause Breathing Method.

**UNIT 12 - SPACE PHYSIOLOGY**

**Basic Space Physiology and Adaptation**

12.1 Describe the effects on the cardiopulmonary system during spaceflight, particularly vital capacity, cardiovascular dysfunction and blood pressure.
12.2 Describe the effects on the musculoskeletal system during spaceflight, particularly effects on bone density, muscle strength and volume.
12.3 Describe the hematological effects of spaceflight, particularly effects on red blood cell volume / mass and plasma electrolyte levels.
12.4 Describe the effects on fluid, electrolyte and nutrition during space flight.
12.5 Describe the effects on renal and gastrointestinal (GI) physiology during space flight, particularly renal stones, urinary output changes and GI mobility.

**Space Motion Sickness (SMS)**

12.6 Describe the symptoms of SMS and distinguish the difference from ground motion sickness.
12.7 Describe the incidence and severity classification of SMS.
12.8 Differentiate between the theories of fluid shift, sensory conflict, Treisman’s, and otolith mass asymmetry hypothesis.
12.9 Describe countermeasures used and tested by the Russian space program.
12.10 Describe the effects on the proprioceptive system, the causes of those effects and the illusions that can occur.

12.11 Describe the effects on the visual system, the causes of those effects and the illusions that can occur.

12.12 Describe the effects on the vestibular system, the causes of those effects and the illusions that can occur.

12.13 Describe the effects human factors and stress can have on individuals experiencing space flight particularly stress states, environmental conditions, physiological changes and cognitive functions.

UNIT 13 - AVIATION, SPACE, AND ENVIRONMENTAL MEDICINE JOURNAL

Given the author(s), publication date and a short summary, describe the major findings and/or conclusions from empirical studies published in the latest three volumes (the last 3 years) of Aviation, Space, and Environmental Medicine.

References
Aviation, Space, and Environmental Medicine. Last 3 years/volumes.
Appendix 11: Organizations of Potential Interest to USAF Aerospace Physiologists

Aerospace Medical Association (AsMA)
http://www.asma.org/

From their Bylaws:
VISION, MISSION, AND GOALS
A. Vision: The international leader in aviation, space, and environmental medicine.
B. Mission: Apply and advance scientific knowledge to promote and enhance health, safety, and performance of those involved in aerospace and related activities.
C. Definition: As used in this document, aerospace medicine is the multidisciplinary application of professional and scientific knowledge, training, and research to promote and maintain the health, well-being, safety, and performance of those involved in aerospace activities.

D. Goals:
(1) Provide governance of the Association to maintain a sound financial structure and ensure continuity of the Association.
(2) Provide opportunities for education and promote research.
(3) Provide members opportunities for professional growth and development.
(4) Represent the discipline of aerospace medicine to professional, commercial, and governmental organizations and advocate policies and standards.

Publication: Aviation, Space and Environmental Medicine
Certification in Aerospace Physiology is available by examination

Aerospace Physiology Society (AsPS)
http://aspsociety.org/

From their website:

As a Constituent Organization of AsMA the, the AsPS:
- Has similar objectives and purposes to those of the Aerospace Medical Association,
- Supports these purposes through local meetings, acquaintanceship, and discussion of matters relating to aviation, space, or undersea medicine, or their allied sciences, and
- Increases the value of this Association to their members, and help maintain and increase membership.

Aerospace Physiology Society (AsPS) Objectives and Purpose:
1. To encourage, promote, and advance the science and practice of aerospace physiology by:
Establishing and maintaining cooperation between aerospace physiology and other scientific disciplines,
stimulating and accomplishing physiological investigation, and
studying and disseminating pertinent knowledge and information through teaching and participation in scientific and technical meetings.

2. To enhance the professional stature of Aerospace Physiologists and associated disciplines within the Aerospace Medical Association, and

3. To provide a single unified voice within the Aerospace Medical Association (AsMA) to present the views of the Society.

**To achieve these objectives, the Society:**

1. Participates in the world’s largest and most comprehensive single annual scientific meeting dedicated to aviation, space and environmental medicine as a constituent organization of the Aerospace Medical Association.

2. Recognizes outstanding leadership, research and educational achievements and research contributions with four annual awards.

3. Promotes and participates in the administration of the Aerospace Medical Association’s Aerospace Physiology Certification Examination increasing the visibility of and interest in aerospace physiology, as well as promoting a standard of academic achievement amongst physiologists.

4. Provides an opportunity for meaningful professional dialogue and easy exchange of information with scientists and engineers in the field of aerospace medicine with an interest in aerospace physiology, worldwide.

5. Performs a public awareness service for the field of aerospace physiology through information on the Society’s website and social media.

Human Factors and Ergonomics Society (HFES)

From their website:

The Human Factors and Ergonomics Society is dedicated to the betterment of humankind through the scientific inquiry into and application of those principles that relate to the interface of humans with their natural, residential, recreational, and vocational environments and the procedures, practices, and design considerations that increase a human’s performance and safety at those interfaces.

Publication: *Human Factors: The Journal of the Human Factors and Ergonomics Society*

Certification in Human Factors is available by examination
Undersea and Hyperbaric Medical Society (UHMS)
http://uhms.org/
From their Constitution:

This Society shall be international in scope. Its primary purposes shall be:
1. to provide a forum for professional scientific communication among individuals and groups involved in basic and applied studies concerned with life sciences and human factors aspects of the undersea environment and hyperbaric medicine.
2. to promote cooperation between the life sciences and other disciplines concerned with undersea activity and hyperbaric medicine.
3. to develop and promote educational activities and other programs, which improve the scientific knowledge of matters related to undersea and hyperbaric environments and the accepted applications of hyperbaric oxygen therapy for the membership, as well as physicians and allied health professionals, divers, diver technicians and the public at large.
4. to provide a source of information and support in the clinical practice of hyperbaric medicine and to stay abreast of legislative, legal, and regulatory changes in the field.
5. to provide a means by which hyperbaric facility directors/owners will have an opportunity to request an accreditation survey of their facility for safety, staffing and verifying the adequacy of the professional medical application of hyperbaric therapy.

Publication:  Undersea and Hyperbaric Medicine Journal

Certification in Hyperbaric Technology is available by examination

SAFE Association
http://safeassociation.com/
From their website:

Objective: The primary objective of the SAFE Association is to stimulate research and development in the fields of safety and survival and to disseminate pertinent information to concerned individuals in government and industry. In addition, the objective is to establish and maintain a meaningful relationship between the SAFE Association and the scientific communities related to safety and survival.

The SAFE Association is dedicated to the preservation of human life. It provides a common meeting ground for the sharing of problems, ideas, and information.
SAFE, a non-profit professional association headquartered in Oregon, maintains chapters throughout the world. It boasts an international group of members.

Membership is not restricted by academic background, experience, or specialty. SAFE members represent the fields of engineering, psychology, medicine, physiology, management, education, industrial safety, survival training, fire and rescue, human factors, equipment design, and the many subfields associated with the design and operation of aircraft, automobiles, buses, trucks, trains, spacecraft, and watercraft. Individual and corporate members include equipment manufacturers, college professors and students, airline flight attendants, government officials, pilots, and military life support specialists. This broad representation provides a unique meeting ground for basic and applied scientists, the design engineer, the government representative, the training specialist, and the ultimate user/operator to discuss and solve problems in safety and survival.

Publication: SAFE Journal; Symposium Proceedings; SAFE News

American College of Sports Medicine (ACSM)
http://www.acsm.org/AM/Template.cfm?Section=About_ACSM
From their website:
Mission: The American College of Sports Medicine promotes and integrates scientific research, education, and practical applications of sports medicine and exercise science to maintain and enhance physical performance, fitness, health, and quality of life.

Publication: Sports Medicine Bulletin

Certification in several sub-specialties is available by examination

Society for Human Performance in Extreme Environments (HPEE)
http://www.hpee.org
From their website:
The Society for Human Performance in Extreme Environments (HPEE) was created to inspire and facilitate collaboration between researchers, practitioners, and other professionals to improve human safety and performance in extremely risky and challenging settings.

Publication: Journal of Human Performance in Extreme Environments (http://docs.lib.purdue.edu/jhpee/)
Board of Certified Safety Professionals
http://www.bcsp.org/bcsp/index.php?option=com_frontpage&Itemid=1

The principal purposes of the BCSP, as more fully set forth in its Articles of Incorporation, are to:

A. Establish the minimum academic and experience requirements necessary to receive certification as a Certified Safety Professional, the designation of Associate Safety Professional, or other such designations established pursuant to resolution by the Board of Directors.

B. Determine the qualifications of applicants and arrange, control, and conduct investigations and examinations to verify the qualifications of candidates for certificates to be issued by the BCSP.

C. Grant and issue to qualified applicants a certificate and maintain a directory of the holders of all valid certificates.

D. Establish requirements for the continuance of certification. The BCSP also has such powers as are now or may hereafter be granted by the General Not-For-Profit Act of the State of Illinois and determine compliance of certificate holders with approved requirements.

E. Represent its certificate holders in communication and, where appropriate, in negotiations with public and private agencies, groups, and individuals with respect to matters of common interest; and it will inform employers, specifiers, public officials, the public, and engineering and related practitioners of the benefits of certification.

Certified Safety Professional is available by evaluation examination.

Extreme Physiology and Medicine
http://www.extremephysiolmed.com/

From the website:

*Extreme Physiology & Medicine* is a peer-reviewed open access, online journal focussing on integrative human physiology under conditions of physiological stress, including that exerted by extreme environments, exercise and certain clinical conditions.
Appendix 12: Contributors’ Biographies

1. Michelle Aaron, Lt Col, USAF, OD, Ph.D.
   Dr. Michelle Aaron is a clinical and research optometrist. She has worked for the Air Force Research Laboratory (AFRL) on cell-laser interactions and at the USAF School of Aerospace Medicine as a research optometrist. Dr. Aaron is a 1996 graduate of the University of California, Berkeley, School of Optometry and received her Doctorate of Philosophy in Physiological Optics from the University of Houston, College of Optometry.

2. Charles G. Acron, CMSgt, USAF
   CMSgt Charles G. Acron is the Chief Enlisted Manager of the 9th Physiological Support Squadron, Beale AFB California. With over 23 years in the Aerospace & Operational Career Field he has experience in every facet of the business, including Undergraduate Flying Training, centrifuge operations with over 100 rides, U-2 High Altitude Reconnaissance Mission Support both stateside and deployment operations, hyperbaric dive operations and High Altitude Airdrop Mission Support. He also was vital in the establishment of the first Human Performance Training Team at Misawa AB, Japan.

3. William B. Albery, Ph.D.
   Dr. Bill Albery is a 36-year civilian in the U.S. Air Force and technically manages the Wright-Patterson AFB centrifuge facility as biomedical engineer and deputy branch chief for the Air Force Research Laboratory. He has a B.S. in systems engineering from Wright State University (1971), an M.S. in biomedical engineering from Ohio State University (1976), and a Ph.D. in biomedical sciences from Wright State (1987). Bill received the Paul Bert Award from the Aerospace Physiology Society of the Aerospace Medical Association (2004) and serves as an Assistant Clinical Professor in the School of Medicine at Wright State.

4. Quentin D. Bagby, Lt Col, USAF, BSC
   Quentin "Q" Bagby's 29 years of experience in the AETC undergraduate flying training arena include serving as Executive-Level Courseware Developer and Program Manager. After graduating as a Distinguished Graduate from both Commissioned Officer Training and Aerospace Physiology Officer’s courses, he became an aerospace physiologist in 2000 at Moody AFB. He helped usher in a new era of flying training with the T-6A Texan II and was directly involved in developing the Airsickness Management Program, from its inception in the mid-1980s to the program of today.

5. James Barnaba, M.S.
   James Barnaba is the Chief Engineer/Technical Advisor, Human Systems Division, Agile Combat Support Directorate, Air Force Life Cycle Management Center (AFLCMC/WNU), Air Force Materiel Command (AFMC), Wright-Patterson AFB OH. He is responsible for providing advanced performance, survival, and force protection capabilities to U.S. and allied air, ground, and naval forces through design, development, production, and sustainment of human-centered systems, including aircrew life support, egress, survival, nuclear/chemical/biological defense, aeromedical equipment, medical information, aeromedical evacuation equipment, testing/certification, Air Force uniforms, and aircraft mishap analysis. He has held various positions in acquisition program offices and in the Engineering Directorate at AFLCMC, providing his expertise to over 60 programs across 35 platforms. Prior to his current assignment, Mr. Barnaba was the Supervising Chief Engineer for the 641st Aeronautical Systems Squadron. He was responsible for the technical leadership of head mounted technology acquisitions, to include the Joint Helmet Mounted Cueing System, Panoramic Night Vision Goggle, and Night Vision Cueing and Display; as well as the Universal Armament Interface and the A-10 Operational Flight Program software development programs. He also worked research and development efforts with AFRL, DoD, and Industry to address technology needs for lightweight night imaging and increased aircraft wiring bandwidth; supported the F-35 Head Mounted Display System, the A-10 Head Mounted Integrated Targeting, and the Modular Aircrew Common Helmet Programs as a Subject Matter Expert; and was Chairman and U.S. Head of Delegation to the NATO Aircraft/Aircrew Integration Panel.
6. **Neal Baumgartner, Ph.D.**
Dr. Neal Baumgartner is an exercise physiologist currently serving in AETC to improve and maintain optimal physical training of USAF battlefield airmen. He also serves as subject matter expert to HQ Air Force for the Air Force Fitness Program, including research and development of the new 2010 fitness test standards. Dr. Baumgartner served as an active duty aerospace physiologist retiring in 2001. He has a B.S. degree in biology from the USAF Academy and M.S. and Ph.D. degrees in exercise physiology from the University of New Mexico and the University of Texas at Austin, respectively.

7. **Eric Samuel Blacher, MD, MPH**
Dr. Blacher earned his undergraduate degree in Theater from Middlebury College in Middlebury, Vermont (2005), medical degree from St. George's University School of Medicine, Grenada, West Indies (2011), and completed Family Medicine residency at Columbia University in New York, New York (2014). He is board certified in Family Medicine, and is currently a full-time Clinical Assistant Professor in Family Medicine at the University of Texas Medical Branch. Dr. Blacher has collaborated on numerous projects with the University of Texas Medical Branch Aerospace Medicine Residency and NASA Johnson Space Center. Dr. Blacher is also a flight surgeon in the United States Air Force Reserve.

8. **James W. Brinkley**
Mr. Brinkley is the former director of the Human Effectiveness Directorate of the Air Force Research Laboratory. Since graduating from Ohio State University in 1958, he performed research on impact acceleration effects and protection technologies. He is the author of 75 journal articles, technical reports, and numerous book chapters on impact acceleration, windblast, and protection technologies. He is a Fellow of Aerospace Medical Association. His numerous awards include the Association’s Eric Liljencrantz Award and the John Paul Stapp Award for his accomplishments in biodynamics, and induction into the International Safety and Health Hall of Fame of the National Research Council.

9. **Mary T. Brueggemeyer, Col, USAF, MC, SFS**
Col Brueggemeyer received an M.D. degree from the University of Louisville in 1992. She completed residency training in general surgery and served as staff surgeon at Dyess AFB, TX, from 1997 – 1999. Dr. Brueggemeyer entered aerospace medicine in 2001 and has served as Flight Medicine Flight Commander, Moody AFB, GA, from 2001 – 2004; Director of the Department of Instructional Programs, Defense Medical Readiness Training Institute at Ft. Sam Houston, TX, from 2004 – 2006; and Commander, 355 Aerospace Medicine Squadron, Davis-Monthan AFB, AZ, from 2006 – 2008. She received an MPH in 2009 and completed a Residency in Aerospace Medicine at USAFSAM in 2010.

10. **John R. Buhrman, M.S.**
Mr. Buhrman is a biomedical engineer with the Biomechanics Branch of the Human Effectiveness Directorate of the Air Force Research Laboratory at Wright-Patterson AFB. His M.S. degree is from Wright State University. Mr. Buhrman has conducted research in the areas of paraplegic gait modeling and human biodynamic response to impact acceleration, the effects of neck loading due to weighted helmet systems, and the evaluation of spinal injury risk during aircraft ejection. He leads the Performance and Safety Team of the Biomechanics Branch, administrates the AFRL Biodynamics Data Bank, and is a member of the Wright Site Institutional Review Board.

11. **Tarah L. Castleberry, DO, MPH**
Dr. Castleberry earned her B.S. in Biology from Grand Canyon University in Phoenix, AZ (1994) and DO from Kirksville College of Osteopathic Medicine in Kirksville, MO (1998). She completed a transitional/family medicine internship at the University of Alabama, Birmingham (1999) followed by an MPH at Johns Hopkins Bloomberg School of Public Health (2000). She joined the Navy and completed its Aerospace Medicine residency program at the Naval Aerospace Medical Institute, Pensacola, FL (2002), then served as a U.S. Naval flight surgeon through 2007. She completed Family Medicine Training at the Mayo Clinic Arizona (2009) and went on to work for Wyle as a
contracted flight surgeon at NASA, supporting astronaut training in Russia and ISS mission support through 2012. She currently serves as the program director for the Aerospace Medicine and General Preventive Medicine residency programs at the University of Texas Medical Branch in Galveston, TX, and is a flight surgeon for Virgin Galactic.

12. **Tony Chao, M.S.**
   Mr. Chao received his B.S. in exercise science from the University of Houston in 2010 and his M.S. in exercise physiology from Texas A&M University in 2012. He is currently working on his Ph.D. in rehabilitation science at the University of Texas Medical Branch in Galveston, Texas. His current research is in burn injury induced metabolic dysregulation in humans utilizing labeled isotope tracers and investigating novel pharmacological therapies to treat the stress response.

13. **Eric Chase, Maj, USAF, BSC, M.S.**
   Capt Eric Chase earned a B.S. in physiological sciences from the University of Arizona in 1998. He entered graduate school in 2001 at the University of Washington Medical School to study physiology and biophysics. After earning an M.S. degree in 2004, he moved to a village in Africa to teach with the Peace Corps. He returned in 2007 and entered Commissioned Officer Training in May of 2008 and became an aerospace and operational physiologist with the U.S. Air Force later that year. He is currently assigned to Air Force Special Operations Command and has been certified in several phases of SCUBA diving since arriving on the Florida coast.

14. **Natacha Chough, MD, MPH.**
   Dr. Chough earned her undergraduate degree in cell and molecular biology from the University of Washington in Seattle, WA (2001), medical degree from the University of Michigan in Ann Arbor, MI (2010), completed Emergency Medicine residency at Stanford University in CA (2013), and Aerospace Medicine residency at the University of Texas Medical Branch in Galveston, TX (2015). She is Board-certified in Emergency Medicine and board eligible in Aerospace Medicine. Natacha has held various positions at NASA, including Planetary Protection Biologist for the Mars Exploration Rovers, Spirit and Opportunity, and is currently a full-time flight surgeon. She also served a full tour in the United States Peace Corps as a Community Health Volunteer in Turkmenistan (2003 – 2005). She is a Fellow of the Academy of Wilderness Medicine, with special interests in wilderness medicine and international travel.

15. **Elizabeth Combs, Capt, USAF, BSC, M.S.**
   Capt Combs is the Officer in Charge of Acceleration Operations at the centrifuge in Joint Base San Antonio. Previously she was the Chief of Human Performance Enhancement at Elmendorf AFB, AK where she was responsible for in-flight oxygen monitoring for the two F-22 squadrons. In 2007 she received her B.S. in Physiology from Michigan State University as well as her commission as a Lieutenant. She received her M.S. in Disaster Medicine and Management from Philadelphia University. Prior assignments as an aerospace and operational physiologist include Laughlin AFB, TX, and Kunsan AB, South Korea.

16. **Anthony R. Cosentino, 1Lt, USAF, BSC, B.S.**
   Anthony "Coz" Cosentino earned a B.S in aeronautics from Embry Riddle Aeronautical University in 2010 and is currently working on his M.S. in Exercise Science/Human Performance. He has over 10 years of experience in Aerospace and Operational Physiology. He served 7 years as an Aerospace Physiology Craftsman and High Altitude Airdrop Mission Support (HAAMS) technician at Randolph AFB, TX, and Andrews AFB, MD. Prior to his commission, he deployed with Special Operations Forces in support of Operation Enduring Freedom, Afghanistan. He is currently assigned to Little Rock AFB, Arkansas.

17. **Jennifer L. Davis, B.S.**
   Ms. Davis is a mechanical engineering student from Miami University in Oxford, OH, working as a summer hire for the Human Effectiveness Directorate of AFRL. She was a research assistant on several biomechanics research efforts supporting the Biomechanics Branch. Her work included conducting tests involving human subject research to ascertain comfort and fatigue over extended
periods of time. She was also involved in compiling pilot ejection injury statistics and conducting impact injury literature searches as part of her completion of a B.S. degree in mechanical engineering.

18. Steve Dawson, Lt Col, USAF, BSC, M.S.
Steve “Lenny” Dawson currently serves as Chief, Human Factors and Risk Management Division, USAFE Safety, Ramstein AB. He began his career in 1988 as an aircrew life support technician, and moved on to become a technical school instructor and CDC writer. In 2000, he was commissioned an Aerospace Physiologist, assigned to Euro-NATO Joint Jet Pilot Training at Sheppard AFB. He returned briefly to his life support roots in 2004 when he was assigned as a program manager/physiology consultant at the aircrew life support equipment program office at Brooks City-Base, TX, followed by an assignment at the USAF School of Aerospace Medicine. He transferred to Randolph AFB in 2007, where he taught the USAF NVG Academic Instructor Course. In 2009, he moved down the street to work on the AETC staff and facilitated moving the Aerospace and Physiology program from higher headquarters to the new lead command, while also managing USAF Initial Flying Training Curriculum. In 2011, he transferred to Laughlin AFB where he served as Aerospace and Operational Physiology Flight Commander and interim Squadron Commander, 47th Medical Operations Squadron, Laughlin AFB. He has an M.S. in Biomedical Sciences from Colorado State University. He married his wife, Cynthia, in 1990 and they have two sons, Steven Jr and Curtis.

19. James Denniston
As the Assistant Technical Director of the 9th Physiological Support Squadron, Mr. James Denniston provides crucial direction in representing the interests of the 9th Reconnaissance Wing’s intelligence, surveillance and reconnaissance (ISR) community to the American public and to those in all levels of government. He serves as an adviser to the Technical Director and the Squadron Commander on all issues regarding high altitude aircrew flight equipment, quality assurance, and unit security management for the officer, enlisted and civilian force. Mr. Denniston also serves as a fully qualified Aerospace and Operational Physiologist to instruct aircrew in the areas of human performance in high altitude flight. He is certified in hyperbaric technology through the National Board of Dive and Hyperbaric Medicine and has worked as a hyperbaric technologist for four years at numerous medical centers in Florida and Michigan before becoming a Department of Defense civilian employee at the 9th Physiological Support Squadron.

20. Lee Diekmann, MSgt, USAF (Ret)
MSgt Diekmann was the Superintendent of Hyperbaric Medicine Branch, Wilford Hall Medical Center, Lackland AFB, TX, prior to his retirement. During his first assignment in aerospace physiology at Beale AFB, CA, he became the supervisor for launch and recovery of U-2 and SR-71 aircraft. During his next assignment at Vance AFB, OK, he became the lead instructor in motion sickness training for the 71st FTW. He conducted student training using biofeedback to increase their relaxation and skills and improve their ability to avoid motion sickness. His following assignment at Brooks City-Base involved supervision of Aerospace Physiology Apprentice and Craftsman courses.

21. James Dreibelbis, Capt, USAF, BSC, M.S.
Capt Jimmy Dreibelbis is an USAF Aerospace and Operational Physiologist currently serving as the OIC, Physiology Flight Operations, at Laughlin AFB, TX. He earned a B.S. in Exercise Physiology from West Virginia University in 2009 and M.S. in Exercise and Nutrition Sciences from SUNY University at Buffalo in 2013. His professional background centered on strength and conditioning fundamentals, fitness development, and nutritional concepts. He has worked in a strength specialist and exercise physiologist capacity with athletes ranging from high school through Division 1 collegiate to professional athletes. He is a member of the American College of Sports Medicine (ACSM) and National Strength and Conditioning Association (NSCA) as a Certified Strength and Conditioning Coach (CSCS).
22. **Stephen R. Emmerthal, Capt, USAF, BSC, Pharm. D**
Mr. Emmerthal received his 2 B.A.s from The University at Albany, New York, and subsequent PharmD from Wilkes University, Wilkes-Barre, Pennsylvania. He gained residency experience working extensively for CVS, The VA, Berwick Hospital, and Wilkes-Barre General Hospital. After graduation and licensing, Mr. Emmerthal attended Air Force Officer Training School. He was subsequently assigned to the 47th Medical Group at Laughlin Air Force Base in Del Rio, Texas, as a Clinical Pharmacist. Currently, he is the Chief of Pharmacy Services at Laughlin Air Force Base.

23. **William R. Ercoline, Lt Col, USAF (Ret), Ph.D.**
Bill is the San Antonio area manager for Wyle Integrated Science and Engineering Group. He is a former USAF C-130 pilot and AETC instructor pilot in the T-41, T-37, and T-38 aircraft. Bill was an assistant professor of physics at the USAF Academy from 1978 – 1982. Since 1988, he has conducted numerous research studies in the areas of spatial disorientation countermeasures and aviation-related human factors issues for AFRL at Brooks City-Base, TX, improving flight symbology for reducing pilot workload in low-visibility conditions. He recently developed flight training profiles for AETC’s ground-based SD trainers and provides teaching support to USAFSAM.

24. **Paul W. Fisher, Col, USAF (Ret), BSC, Ph.D.**
Colonel Paul Fisher is the Commander, AF Office of Scientific Research and formerly the Senior Military Professor, Biology Department, USAFA. He received his B.S. in biology from the University of Miami, M.S. in zoology from Montana State University, and Ph.D. in cell biology from Duke University. He served as an aerospace physiologist for the high-altitude reconnaissance and airdrop programs and an instructor at USAFSAM and led high-altitude and acceleration physiology and advanced oxygen systems research at AFRL. Colonel Fisher completed the Clinical Hyperbaric Physiology Fellowship and is a recipient of the Paul Bert Award for Aerospace Physiology Research. He is board certified in both aerospace physiology and hyperbaric technology.

25. **Eric E. Geiselman, M.A.**
Mr. Geiselman is a Senior Engineering Research Psychologist at the U.S. Air Force Research Laboratory’s 711th Human Performance Wing, in the Human Effectiveness Directorate where he performs visual display research. Mr. Geiselman spent 8 years as an airline pilot and eventually became a Crew Resource Management instructor. Mr. Geiselman holds an M.A. in Experimental Psychology from the University of Dayton and is pursuing a Doctorate in Systems Engineering at the Air Force Institute of Technology.

26. **Benjamin Johansen, DO, MPH.**
Dr. Johansen received his B.A. degree from Brigham Young University and his DO degree from the Arizona School of Osteopathic Medicine at the Glendale Arizona campus of Midwestern University. Following graduation from medical school Dr. Johansen completed a residency in Internal Medicine at Banner Good Samaritan Medical Center in Phoenix, Arizona (2014). He is currently an Aerospace Medicine Resident at The University of Texas Medical Branch, where he also earned a Master of Public Health Degree (2015). Since starting his Aerospace Medicine Residency he served as the Resident Medical Coordinator for the Wings Over Houston Air Show in charge of medical support for approximately 90,000 spectators over the three-day event. In his free time he enjoys traveling and competing in triathlons.

27. **Martin Johnson, B.S.**
Mr. Johnson received a B.S. in Electronics Engineering from Colorado Technical University. He is currently the Deputy Director of the Laser and Optics Research Center at the US Air Force Academy, a post he has held for the last 15 years. Mr. Johnson is a Certified Laser Safety Officer, and is currently involved in research into black silicon for more efficient solar power generation. His other research topics have included laser bio effects on the human eye and real-time holography for adaptive optics.
28. **David R. Jones, Col, USAF (Ret), MC, CFS**
Dr. Jones completed residency in aerospace medicine, later serving at Myrtle Beach AFB, Nha Trang AB, Torrejon AB, and Randolph AFB, logging nearly 2000 hours (286 combat) in 35 aircraft types. After completing psychiatry residency, he served at the Neuropsychiatry Branch, Clinical Sciences Division, USAFSAM. His military awards include the Legion of Merit (1-OLC), Bronze Star, and Air Medal (3-OLC). He was editor-in-chief of *Aviation, Space, and Environmental Medicine* from 1987 – 1996 and is currently Clinical Professor of Psychiatry at the USUHS. Dr. Jones is a member of the IAASAM and a Fellow of the ACPM, AsMA, APA, and AHFA.

29. **Christy Kayser-Cook, Lt Col, USAF, M.S.**
Lt Col Kayser-Cook received a B.S. in Space Physics from the USAF Academy and a M.S. in Aeronautical Science from Embry-Riddle University. She is currently an instructor of Physics at the U.S. Air Force Academy where she has taught for the past 5 years. She is an AF instructor pilot with over 3,000 hours in the KC-135 and the T-37. Additional assignments have included: Chief, Policy and Resources Division, Air Mobility Command Test and Evaluation Directorate; and Chief, Air Refueling Operations Division, 618th Tanker Airlift Control Center.

30. **William D. Kosnik, Ph.D.**
William Kosnik earned a Ph.D. from Simon Fraser University in Vancouver, Canada, and is an engineering psychologist for the Human Performance Optimization Division of the 711th HPW. He's a human factors engineering expert implementing human systems integration solutions within Air Force acquisition programs. Dr. Kosnik worked for 21 years as a vision scientist for AFRL's Directed Energy Optical Radiation Branch. He conducted force protection program research on directed energy effects for visual function and air crew performance, designed computational models of optical radiation visual effects, and developed laser-based optical technologies.

31. **Robert M. Lindberg, M.A.**
Robert M. Lindberg received his M.A. in management from Antioch University McGregor, and his B.S. in mathematics is from the University of Wisconsin. He is Chief of the Human Performance Optimization Division at the 711th Human Performance Wing, Human Performance Integration Directorate. He is responsible for the establishment of human systems integration within the AF. Robert has over 25 years of operational and acquisition experience in aerospace systems such as B-1, F-4, F-15, F-16, and F/A-18 E/F. His specialty areas are acquisition, aircraft engines, avionics, flight test, human systems integration, logistics, maintenance, manufacturing, and program management.

32. **Valerie E. Martindale, Lt Col, USAF (Ret), BSC, Ph.D.**
Following retirement from the USAF, Valerie Martindale is currently working as a consultant to Boos Allen Hamilton. Prior to retirement, she served the Human Performance Consultant, Air Force Human Systems Integration Office and Chair of the Air Force Human Performance Functional Area Working Group. Previously, she was Chief of the Human Performance Division on the Air Staff, Chief of Aerospace Physiology; Chief, International Human Factors for AFRL; and Chief of Altitude and Hyperbaric Research. Lt Col Martindale is a private pilot and board certified in aerospace physiology. She is a member of the Undersea and Hyperbaric Medical Society and the American Society for Cell Biology and is a Fellow of the Aerospace Medical Association.

33. **Ryan W. Maresh, Lt Col, USAF, BSC, CASP, Ph.D.**
Lt Col Maresh is currently the Aerospace and Operational Physiology Flight Commander at Laughlin AFB, TX. Prior to his current assignment, Lt Col Maresh completed two tours an Assistant Professor of Biology at the United States Air Force Academy. Previous assignments include Chief, Aerospace and Operational Physiology Branch, U.S. School of Aerospace Medicine at Brooks City-Base, TX, where he oversaw training and education of new officer and enlisted accessions into the 43A and 4M0 career fields, as well as the aerospace physiology training of several other medical specialties, and working with the high-altitude reconnaissance program at Beale AFB, CA. He received has M.S.
in Physiology and Ph.D. in Biomedical Sciences from Colorado State University. He is board certified in aerospace physiology and is an Associate Fellow of the Aerospace Medical Association.

34. Andy McKinley, Ph.D.
Dr. McKinley received a B.S. in biomedical engineering from Wright State University and completed a Ph.D. in human factors engineering. He works as a biomedical engineer at AFRL’s Biobehavioral Performance Branch located at Wright-Patterson AFB, OH. Mr. McKinley is the Human Effectiveness Team Lead for the AFRL Rotary-Wing Brownout Solution Program. He is also exploring noninvasive transcranial stimulation techniques to improve human cognitive performance. Dr. McKinley’s work has focused on modeling the effects of acceleration stress on physiologic and cognitive performance, use of multisensory displays for unmanned aerial systems, and use of eye metrics for monitoring human performance.

35. Richard L. McKinley, M.S.
Mr. McKinley is the principal acoustics engineer at AFRL’s 711 HPW at WPAFB, OH. He received degrees in biomedical engineering and communications and digital signal processing from Vanderbilt University and AFIT. Mr. McKinley has led development of high-performance hearing protection, establishment of noise exposure criteria, measurement of intense noise fields, enhancement of voice communication performance, and development of 3-D auditory displays. Mr. McKinley is the USAF voting member for three ANSI-accredited standards committees and has over 150 publications in acoustics and noise. In 1996, he was elected Fellow of the Acoustical Society of America.

36. George Miller, Maj, USAF (Ret), M.S.
George Miller is a research engineer with the 711th Human Performance Wing, Brooks City-Base, TX. He has a B.S. degree in chemical engineering from Auburn University, Auburn, AL, and an M.S. degree in chemical engineering from Ohio State University, Columbus, OH. His areas of expertise are on-board oxygen generating systems (OBOGS) and aircraft oxygen systems safety-of-flight testing. He has numerous publications on oxygen generation technology and holds six U.S. patents. He is the USAF Co-Chair of the DoD Oxygen Standardization Coordinating Group and a member of the SAFE Association and the American Institute of Chemical Engineers.

37. James C. Miller, Maj, USAF (Ret), Ph.D.
James C. Miller, Ph.D., CPE, has over 30 years of experience in the measurement and analysis of human physical and cognitive performance in military and civil aviation; highway, rail, and maritime transportation; and night and shift work. He focuses mainly on reducing the effects of fatigue on productivity and safety, especially in 24/7 and night operations. Prior to undertaking his doctoral studies in 1971, Dr. Miller served as a C-130E Hercules tactical transport pilot in Vietnam. He logged 699 hours of combat time and was awarded the Distinguished Flying Cross and the Air Medal with four Oak Leaf Clusters.

38. Craig Olson, Lt Col, USAF (Ret), BSc, M.S.
Craig is the President of Salus Education, LLC in San Antonio and an independent contractor working for the American Dietetic Association, Office of Scientific Affairs and Research. He is a former navigator with over 1,000 hours in the KC-135. He graduated from the USAF Dietetic Internship in 1993 and received an M.S. degree in nutrition from the University of California, Davis in 2002. Craig has conducted research related to the performance-enhancing properties of specific food constituents. Among Craig’s many assignments, he had the honor to serve as the commander of the Air Force’s largest nutritional medicine squadron.

39. James Pattarini, MD, MPH.
Dr. Pattarini was accepted into the combined Aerospace Medicine/Internal Medicine residency program at the University of Texas Medical Branch (UTMB) in March of 2010. After serving as Chief Resident of Internal Medicine from 2013 – 2014 he completed his training in aerospace medicine in 2015, and currently serves as Flight Surgeon to NASA via UTMB/Wyle, and Assistant Professor in the Department of Internal Medicine at UTMB in Galveston, TX. His educational background includes a B.S. in Evolutional Biology from Syracuse University (2002-2006), a MD from the State University of
New York at Buffalo, NY (2006-2010), and a MPH completed as a component of his aerospace medicine training at UTMB in 2013. James resides in Houston, TX with his wife Harita, a short drive from Johnson Space Center.

40. **Dwayne Porter, Lt Col, USAF, BSC, M.S.**
Lt Col Porter is an Aerospace and Operational Physiologist and the Human Factors Investigations Branch Chief within the Human Factors Division at the Air Force Safety Center where he provides Aerospace Medicine, Aerospace Physiology, and Operational Psychology support to Air Force aviation, ground, space, and weapons safety programs. He earned his B.S. in Human Biology from Brigham Young University in 1997 and an M.S. in Aviation Human Factors from Embry-Riddle Aeronautical University in 2005.

41. **Jeff Rabin, OD, M.S., Ph.D.**
Dr. Jeff Rabin is a tenured Professor of Optometry and Assistant Dean for Graduate Studies, Research and Assessment at the University of the Incarnate Word Rosenberg School of Optometry. He received all degrees from the University of California, Berkeley, School of Optometry. After retiring from the US Army, Dr. Rabin served as Chief, Visual Function Laboratory at the USAF School of Aerospace Medicine. His research interests include color vision, contrast sensitivity and visual electro-diagnosis.

42. **Sean Sarsfield, Maj, USAF, BSC**
Maj Sarsfield is an Aerospace and Operational Physiologist currently assigned as the Chief of Acceleration Training, USAFSAM Operating Location Brooks City Base, San Antonio TX. He earned a B.A. in Kinesiology from the University of Colorado (CU) in 2000. While at CU, he completed the ROTC program and was commissioned upon graduation. After two assignments in the aircraft maintenance officer field, he switched to Aerospace and Operational Physiology. Maj Sarsfield earned his M.S. Degree in Human Factors in Aerospace Systems from Embry Riddle Aeronautical University in 2005. He has experience in all phases of the USAF pilot training pipeline, with particular focus in the high-G/fighter aircraft specialty. He is a Certified Strength & Conditioning Specialist (CSCS) from the NSCA and holds an FAA Recreational Pilot certificate.

43. **Patrick Shorter, Maj, USAF, OD, Ph.D.**
Maj Patrick D. Shorter is the Chief of Visualization Operations, 711 Human Performance Wing, Warfighter Interface Division located at Wright-Patterson Air Force Base, OH. He provides expert input and analysis for research into the complex visual demands placed on Airmen to improve safety and warfighting capabilities. He earned his Bachelor of Science degree in Biology from the University of North Florida in 2000. In 2004, Dr. Shorter was awarded his Doctor of Optometry degree from Southern College of Optometry. He entered private practice in Jonesborough, TN, until 2010, when he joined the U.S. Air Force as a direct accession. He served as Optometry Operations Officer at the Wright-Patterson Air Force Base Medical Treatment Facility before earning his Ph.D. in Vision Science from The Ohio State University in 2015.

44. **Brian Stanley**
Brian Stanley received a B.S. in aeronautics from San Jose State University. Upon completion of Undergraduate Pilot Training, where he flew the T-37 and T-38, he then served in two Air National Guard units and flew the C-130 E/H aircraft. He is currently an airline pilot and has flown the Boeing 727, 757, and 767; the Fokker 100; and now the McDonnell Douglas MD80. He holds an FAA flight engineer certificate and an aircraft powerplant mechanic certificate. He is a Lieutenant Colonel and serves in the U.S. Air Force Reserves providing operational perspective to AFRL’s Biobehavioral Performance Branch.

45. **Richard Sumrall, Lt Col, USAF, MC, CFS**
Dr. Sumrall is the Medical Director for the Centrifuge and Altitude Chamber operations at Brooks City Base, TX. He has been a flight surgeon for 19 years with many units and deployments and over 1,000 flight hours as flight surgeon.
46. **Rahul Suresh, MD, M.S.**
Dr. Suresh earned his undergraduate degree in Biochemistry and Cell Biology from Rice University in Houston, Texas (2008), medical degree and Masters of Science degree in Clinical and Translational Research from the Mayo Medical School and Mayo Graduate School, respectively, in Rochester, Minnesota (2014). Rahul is currently a second year Internal Medicine and Aerospace Medicine resident at University of Texas Medical Branch (UTMB) in Galveston, Texas. Rahul's academic interests include the Visual Impairment in Intracranial Pressure Syndrome, management of diabetes during long-distance air travel and the cardiovascular effects of lower body negative pressure and its implications for attenuating effects of cephalad fluid shift in microgravity.

47. **Ronald C. Tutt, Maj, USAF (Ret), BSC**
Ronald C. Tutt, OD, MS, FAAO, is on the USAF School of Aerospace Medicine faculty and is a published vision scientist. He has extensive clinical and aeromedical experience covering over 20 years of service to include research of altitude/centrifuge effects following refractive surgery and low-altitude NVG hypoxia; development/integration programs such as night vision devices, laser eye protection, head-mounted cueing systems, and aircrew chem/bio systems; as well as management of USAF aviation vision correction programs (contact lenses and spectacles). Dual qualified as optometrist and aerospace physiologist, Dr. Tutt serves as consultant/lecturer for U.S./international flight surgeons, optometrists, and aerospace physiologists.

48. **Henning E. von Gierke, D.Eng.**
Dr. von Gierke was a researcher and technical leader at Wright-Patterson AFB in the areas of biodynamics, sound, and vibration. His 40 years of work led to the development of human tolerance criteria for vibration and shock that were used as the basis for a comprehensive set of ISO safety and performance consensus standards for vibration exposure. His studies are documented in over 160 publications on noise exposure and its effects on biodynamics of human exposure to impact, crash, and on vibration loads, on vestibular effects, and protection against hazardous force environments.

49. **James T. Webb, Maj, USAF (Ret), Ph.D.**
Dr. Webb is a former USAF command pilot with >3,900 hours in F-4D and C-141A aircraft. He received a Ph.D. from the University of Washington and became an Associate Professor of Biology at the USAF Academy in 1979. Later, he worked as an acceleration and altitude research scientist with USAFSAM at Brooks AFB, TX. After retirement, he served as a contracted senior scientist for AFRL, publishing 18, first-author, peer-reviewed research papers. Dr. Webb is board certified in aerospace physiology by the Aerospace Medical Association (AsMA), a Fellow of AsMA, recipient of AsMA’s Leverett Environmental Science Award in 1999, and was President of the Aerospace Medical Association from 2013 – 2014.

50. **Donald J. White, Col, USAF (Ret), BSC**
Colonel White’s operational career included experience in acquisition, research, and education. He served as faculty at USAFSAM, aircrew training, operational physiology, and operational safety. He was commander of an Airmanship Training Squadron, Aerospace Physiology Flight, and Aerospace Medicine Flight and served as the Biomedical Science Corps Associate Corps Chief for Aerospace Physiology. He participated on 14 Class A Safety Investigation Boards, including consultant to the Columbia Shuttle Accident Investigation Board and two Medical Incident Investigation Boards. Col White served two higher headquarters tours and was a HALO and Static Line Master Parachutist with over 4,000 parachute deployments.

51. **Mark T. White, M.S.**
Mark White was a USAF aerospace physiologist and Chief, AP Operations Element at Brooks City-Base, TX. He has a B.S. in exercise science (1994) and an M.S. in exercise physiology (1999). His professional background is in work-related physical ability testing, application of occupational physiology, and collegiate coursework curriculum development and instruction. He served as an educator at CSU Sacramento, a UC Davis researcher, and a private-business co-owner. He is a member of the American College of Sports Medicine (ACSM) and has held ACSM Exercise Specialist
and National Strength and Conditioning Association (NSCA) Certified Strength and Conditioning Coach certifications.

52. Dorian Williams, Maj, USAF, BSC
Major Williams is the Aerospace and Operational Physiology Curriculum Manager at HQ AETC. He is a former KC-135 Boom Operator with over 2,500 hrs and honors including USAF Top Enlisted Aviator and Top Instructor. In 2002, he was commissioned in the BSC and was the Distinguished Graduate of the USAF Aerospace Physiologist tech school. His educational and research background revolve around, aviation science, education, and religious philosophy. He has published in subject areas of human factors in remote aviation and adult learning methodologies. He has over 21 years of aviation experience serving as a human factors consultant in 122 Class A thru E flight mishaps. He is certificated in Aviation Safety, Advanced Project Management, and Airport Planning, Design, and Development. He is a member of the American Society for Training and Development, the Project Management Institute, and the Airport Executives Association. Maj Williams has worked and/or led hyperbaric and hypobaric operations, centrifuge, high altitude airdrop support, night vision, and completed the KC-135R Central Flight Instructor Course and the MQ-9 Sensor Operator Course.

53. C. Brad Wilson, Maj, USAF, M.F.S., Ph.D.
Major Brad Wilson is an assistant professor of biology at the U.S. Air Force Academy. His specialties include neurology, molecular biology, and organ systems physiology. He enlisted in the Air Force in 1988 and remained on active duty until separating in 1995. He completed his bachelor’s degree in psychology and was commissioned through OTS in 2002. In 2008, he received his master’s degree in forensic sciences from The George Washington University and served as the primary AFOSI forensic scientist for all Air Force bases in the Rocky Mountain region. In 2014, Major Wilson earned his Ph.D from Louisiana State University where he conducted research into the pathophysiology of post-traumatic stress disorder (PTSD) in the brain and other organs. He currently continues his research with the Clinical Research Lab at Keesler AFB, MS, where he is investigating the effects of rapid aeromedical evacuation, exercise, and toxoplasmosis on the progression of PTSD.

54. Andrew D. Woodrow, Lt Col, USAF (Ret), BSC
Lt Col Woodrow’s 32 years in operational and applied aerospace physiology and human factors has touched every aspect of the field; from SR-71/U-2/TR-1 support to clinical hyperbaric medicine he has made a career of understanding performance issues in extreme environments. He has expertise in underwater escape systems, extreme thermal stress, high altitude life support OT&E, and sustained fatigue trials, and he has personally participated in over 170 centrifuge runs to test and evaluate advanced crew systems. He has been adjunct faculty for graduate and undergraduate programs at King’s College London, Wright State University, and Embry-Riddle University. Lt Col Woodrow is a board certified hyperbaric technologist (CHT) and ergonomics manager (CEM), and is a Fellow of the Aerospace Medical Association and Member of the Royal Aeronautical Society. He has been consultant to 10 Class A aircraft mishap investigations and numerous Class B investigations, and has amassed over 350 flight hours in 14 aircraft types.

55. Steve Wright, OD, M.S.
Dr. Steve Wright is a clinical and research optometrist with the USAF School of Aerospace Medicine, Aerospace Ophthalmology Branch. Dr. Wright is a 1995 graduate of the Pacific University College of Optometry. He worked in private practice optometry for eight years before joining the branch in 2004. His research interests include color vision, stereopsis, refractive surgery and automated test development, which have resulted in over 20 publications and presentations.