FEATURE

Unexplained Physiological Episodes

A Pilot's Perspective

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What goes through a pilot's mind when he has exhausted his checklist with no improvement to his current condition? A pilot's pathway to safety is his checklist—a series of simple, linear steps that bring the comfort of years of knowledge to the cockpit environment during panic and crisis. Pilots who experienced aircraft emergencies when a checklist failed recall the surge of adrenaline and pounding heart-thumping accompanying the panicked thought of "What now?"

Now imagine the emergency your checklist failed to address is physiological. Imagine the visceral fear as you feel increasingly dizzy and light-headed with your vision darkening and limbs going numb. Combine the fear with the psychological

panic that ensues when you reach that last step of your procedure, and you only feel worse. Mentally put yourself in this moment, and you will understand what numerous fighter pilots in the past decade have called an unidentified physiological event (UPE). Listen to their stories, and you will hear them describe an environment where nothing they were taught worked to save them.[1]

The High-Performance Fighter Environment

High-performance aircraft were built to train and fight. At the advent of fighter aviation, "high-performance" aircraft flight envelopes stayed well within the human physiological envelope.[2] One hundred years later, the F-22 joined the ranks of fighter aircraft with an unaugmented flight envelope that well exceeds the human limits in g-force and altitude.[3] Only flight control limiters prevent the inconvenient F-22 "passenger" from routinely outperforming himself with the pull of a stick. Yet despite the meteoric advancements in aircraft performance during the past 100-plus years, hypotheses about human performance in flight remain largely unchanged.[4] Put simply, aerospace physiology has not kept up with high-performance aircraft. As a result, even our most modern fighter aircraft feature life support systems designed against an oversimplified set of assumptions: Our systems were designed to defeat hypoxia and decompression sickness; therefore an "excellent" system was one that delivered maximum oxygen and maximum pressure to the pilot.[5] Some of these assumptions are proving grossly inaccurate.

The ideal life support system for flight is one that provides the concentration and flow rate of gases that the human demands in a given situation—no more and no less. Too much pressure creates resistance a pilot must forcefully breathe against and too high a concentration of any molecule in a gas concentration forces the pilot's body to compensate for the nonideal mixture.[6] These two statements seem obvious, yet the breathing and pressurization systems found in today's high-performance aircraft are not designed to meet that criteria.

Current State of Physiological Incidents

As a result, UPEs are on the rise. As of the drafting of this article, UPEs have become a primary concern for both the USAF and US Navy (USN). Both the USAF and the USN deputy chiefs of staff testified before Congress in February 2018 and answered a request to explain their plan to solve this problem for their respective services.[7] Since 2007, the F-22, F-35, F/A-18, F-15, F-16, T-6, and T-45 have each reported *at least* 10 UPEs that are not explained by classic physiological training.[8] The F/A-18, operated both on and off aircraft carriers by the US and foreign governments, has reported more than 603 UPEs in the same pe-

riod of time.[9] UPEs are reported in benign phases of flight, and even on the ground before takeoff, making classic hypoxia and decompression sickness diagnoses essentially impossible.

Rates Across the Department of Defense

The UPE rates are likely due to a combination of increased awareness and reporting, aircraft part failures due to fleet age, or design flaws. In some cases, the prevailing cause is more obvious than in others. Newer aircraft like the F-35 and F-22 are being examined for design flaws that create inherently poor breathing environments. Older aircraft like the F-15, F-16, and A-10 are being studied for maintenance breakdowns.[10] In the middle, aircraft like the T-6, T-45, and F/A-18E are reaching the mature point in their life cycles when design errors should have been found, and major maintenance breakdowns have not yet surfaced.[11] In all cases, however, the DOD increased awareness and reporting—some of which was intentional, and some surfaced when fighter pilots started appearing on television refusing to fly—makes it difficult to discern exactly how significant this decade's UPE spike is. One thing is certain, however. These UPEs are aircraft-agnostic, oxygen system-agnostic, engine-agnostic, and even flight envelope-agnostic. They are occurring in every type of high-performance aircraft in the DOD, and they are occurring at every point in the flight envelope from the ground to the highest fastest corners.[12] In fact, the only aircraft that seem immune from these UPE spikes are our heavy transportation aircraft where pilots and passengers alike breathe pressurized ambient cockpit air with no life support gear. This fact alone is information worth digesting.

Well-Publicized Examples

The DOD's UPEs have made headline news repeatedly since two F-22 pilots broke the ice on "60 Minutes" in May 2012.[13] For the first time in memorable history, Air Force fighter pilots refused to fly. The feeling among F-22 pilots at the time was akin to that of a community stricken by a series of crimes that all fell into the category of "unsolved."[14] Faith was broken between the Air Force's investigative bodies and the pilots who were told the aircraft was "fixed." Several iterations of fixes were added to the F-22 before the Air Force found one that seems to be working. Wholly missing from these trials, however, was any effective communication to the pilots regarding the reasons behind the fixes and the current state of the aerospace medical science. Looking back six years later, the group of doctors and physiologists working the F-22 case had pieced together much of the cutting-edge knowledge that is changing our training and checklists today but

instead of keeping the pilots informed of these discoveries, Air Force leadership instead levied new procedures and added new system components with little explanation of why.

One pilot recalls violating his "mask-up" checklist repeatedly during the heart of the Air Force's hunt for cockpit contaminants, and he broke the rule to survive.[15] The Air Force inserted a carbon filter into the breathing air delivery line to the pilot to filter contaminants that were not present. In doing so, they restricted a flow rate that soon was proven to be already overly restricted to begin with. "I would fight as long as I could with my mask up, trying to adhere to the rule as long as I could stand it," he recalls. "Then I would turn around from the fight so air hungry that I had to rip my mask off my face just to catch my breath. Once I had recovered, I put my mask on and tried again."[16]

While the F-22 was the first to make the news, it was hardly the only aircraft dealing with similar unexplained crashes and emergency landings. The F/A-18 community had been chasing contamination in their oxygen systems since 2007, and despite never finding any elements of significance, their search remained focused on one potential problem to the exclusion of others.[17] It was not until 2017 when their UPE count hit 500 incidents for a single type of aircraft that the USN opened its aperture to other potential problems.[18] In April 2017, the USN reported that more than half its F/A-18s were unable to fly due to UPE investigations.[19] Within a month, the T-45 fleet was grounded as well.[20] Public news sources began reporting in October 2017 that the F-35 fleet was cancelling flights due to UPEs.[21] In February 2018, the T-6A, the USAF's primary flight trainer, was grounded as a rash of UPEs peaked with a set of nine incidents within a 48-hour period.[22]

What does a typical UPE look like? Each UPE contains some details unique to the aircraft and environment, but they share common elements as well. The example that follows illustrates a typical UPE for this era. In 2015, an F-15C pilot was flying a routine training mission on a clear day when he noticed he felt less than 100 percent. Typical of the culture of the time, the pilot chose to continue the mission rather than confess his symptoms—breathing problems were for F-22s, and the F-15C had a liquid oxygen delivery system, largely thought to be immune to malfunctions.[23] Shortly thereafter, the pilot felt his hypoxia symptoms—tingling, dizziness, and a lack of concentration—and chose to return to base and initiate his emergency checklist. He pushed his regulators to maximum flow and concentration, felt the pressure at his mask, and took a breath. He did not feel better. Assuming his regulator had failed, the pilot then pulled his "green ring" to release a direct flow of emergency oxygen to the mask—the last step in the checklist—the step a decade of training had promised was a guaranteed solu-

tion.[24] He did not feel better. At this moment the emergency became a UPE. The pilot's checklist and knowledge were both exhausted, and he felt he had no choice but to sprint back to base before he passed out from hypoxia. He landed the jet almost incapacitated and did not feel normal again until several minutes after he exited the aircraft.[25] An initial maintenance examination of the aircraft found nothing to explain the pilot's symptoms, nor did the current state of physiological training. Hence, this emergency was unexplained.

Put simply, the objective of this article is to prevent harrowing incidents like the one above from happening again. Through education, communication, and a simple "do no harm" inflight technique, this article hopes to mitigate future UPEs.

High-Performance Aviation Physiology

> *Man must rise above the Earth—to the top of the atmosphere and beyond—for only thus will he fully understand the world in which he lives.*
>
> *—Socrates*

A necessary first step in understanding the nature of current high-performance physiological troubles is gaining a basic knowledge of the various systems at play. A jet pilot represents a complex physiological system that is constantly attempting to maintain a normal physiological state. Any change to typical bodily homeostasis will likely result in some manner of response on an autonomic level. Thus, the simple act of keeping a person alive in a high-altitude environment requires alterations to the respiratory environment, which must then elicit a bodily response. As a result, the very life support *systems* meant to satisfy respiratory requirements could, in fact, create unfavorable consequences because of the complex interactions between these two sometimes constantly varying systems. The importance of approaching these issues from a system-system interaction perspective cannot be overstated, as time and again a "properly functioning aircraft" and a healthy well-functioning pilot combine to create an unexplainable physiological episode.

Defining the Pilot Respiratory Environment

Description of the Environment

At its most basic level, the challenge of operating in the high-altitude regime is simply a function of pressure. This is driven by the nature of the atmosphere itself and is then compounded by introducing high-gravitational (high-g) forces into the mix during fighter maneuvering. Understanding these problems can inform a reflective study on the development of life support systems over time.

Earth's atmosphere at almost all altitudes is composed mostly of nitrogen (78 percent) and oxygen (21 percent).[26] A common misconception is that there is a lower percentage of oxygen at high altitudes. Instead, thin air at altitude is the result of a drop in the number of molecules of oxygen present in any given volume of air due to the decrease in atmospheric pressure at high altitudes. At sea level, a person breathing air will have an amount of oxygen in their lungs that is described by the term P_aO_2, which is the pressure of oxygen in lung alveoli. This is sometimes referred to as the *partial pressure of oxygen* with a value of 103 mm Hg (mercury) at sea level. As altitude increases, and the pressure of the air drops, aviators must breathe a higher percentage of oxygen to maintain 103 mm H_g P_aO_2.[27] Put another way, the intent of supplemental oxygen is to keep an aviator's respiratory system breathing at sea-level equivalents.

An additional and unique challenge of the high-performance fighter environment is increased gravitational forces, where blood pools in the lower extremities, and internal organs—specifically the lungs—are also compressed. The most obvious hazard of these forces is a g-induced loss of consciousness caused by a loss of blood (e.g., oxygen) in the brain, but the gravitational effects on the lungs are also concerning.

Life Support Systems

Based on the above hazards of high-performance flight, an appropriate life-support system must provide: a pressurized cockpit to minimize hypobaric conditions, supplemental oxygen to provide sufficient P_aO_2, and some manner of assistance to the pilot in resisting gz forces over time.

To minimize hypobaric conditions, the cabin pressurization schedule typically used by modern fighter aircraft holds cabin altitude below a 15,000 foot cabin altitude for the majority of their tactical time with a preponderance of that time spent at an 8,000 foot cabin altitude.[28] USAF regulatory guidance limits cabin altitude to a 25,000 foot maximum without a pressure suit to mitigate the risk of decompression sickness.[29]

With those cabin altitudes in mind, a fighter breathing system must be able to provide supplemental oxygen on a regular basis to satisfy sustained flight up to a 25,000 foot cabin and provide emergency protection up to the maximum aircraft altitude in case the pressurization system fails. Physiologically, this means delivering a percentage of O_2 (oxygen gas) such that P_aO_2 is 100–03 mm Hg (sea-level equivalent) at all sustained altitudes (less than 25,000 foot cabin). In an emergency decompression scenario, sea-level (SL) equivalent P_aO_2 can be maintained up to an aircraft altitude of 33,700 foot by breathing 100 percent oxygen. Above that point, 100 percent oxygen must be administered under positive pressure—

pressure breathing for altitude (PBA)—to make up for the extremely low atmospheric pressure of the breathing environment, although in practice PBA typically begins at 40,000 foot.[30]

The manner in which military aircraft provide oxygen support generally fall into three different categories: gaseous oxygen, liquid oxygen (LOX), and an onboard oxygen generation system (OBOGS). Gaseous oxygen and LOX systems are well-known and have been used for many years without substantial functional deficiencies. However, aircraft maintainers were required to service these systems periodically with ground-based stores that created logistical challenges in maintaining these systems.

The advent of OBOGS seemed to be the panacea for those logistical hurdles as OBOGS can produce oxygen on board the aircraft continuously. This production eliminates logistical hurdles, allows long flight durations, and eliminates any battle damage fire/explosion considerations. OBOGS consist of two or more cylinders (sieves) of a crystalline substance called *zeolite*. When a zeolite sieve is pressurized, typically by sending high pressure engine bleed air into the sieve, the zeolite structure absorbs nitrogen but allows oxygen and argon to pass through as the product gas, which is approximately 90–95 percent oxygen. The nitrogen-saturated zeolite can be purged simply by depressurizing the sieve, making the process reversible and cyclical. Thus, using two or more alternating zeolite beds allows for a almost continuous supply of high-oxygen concentration air for the aircrew.[31] New fighter aircraft (F-15E, F-16 Block 50+, F-18E/F, T-6, F-22, F-35, and others) have been designed with an OBOGS while some aircraft, which previously used LOX, have been retrofitted with OBOGS for logistical reasons (F-16, F-18C/D, T-45, AV-8, and others.)[32]

OBOGS systems all follow the same basic principles to produce an oxygen-enriched gas. However, the same cannot be said about the delivery mechanism of that gas to the pilot, which varies in many ways that will be briefly summarized here. In general, there are two different methods of delivery: continuous flow and pressure demand. In a continuous-flow system (F-18, T-45), breathing gas is continually delivered to the pilot through his mask whether he is breathing or not.[33] In contrast, pressure-demand systems only provide breathing air in response to aviator inhalation through a regulator. This method allows for flexibility in the delivery of breathing gas, enabling gas dilution (termed *airmix*), and pressure breathing (for g or altitude). The engineering trade space created by these systems have led to their use on virtually all USAF aircraft although implementation varies in two distinct ways.

First, is whether or not the regulator provides airmix, whereby cockpit air is mixed with the output of the OBOGS or LOX system to provide the pilot with

an appropriate percentage of oxygen to maintain 100–103mm H_g P_aO_2. This mixture was the standard method of delivery in older aircraft (F-15, F-16, and A-10).[34] In contrast, many newer airframes do not provide airmix in the classic sense (that is, through a dilution regulator) but rather vary oxygen content by controlling OBOGS cycle times.[35] As a result, the F-18, F-22, F-35, and T-6 all provide significantly higher percentages of O2 to aircrew than their predecessors (with F-18 being 90–95 percent continuously). A representative delivery schedule for these systems can be seen in figure 1 below, with oxygen content beginning in the 50–60 percent range (well above the SL equivalent) and increasing to 90–95 percent in an 11,000 foot cabin. It should be noted that because of OBOGS cycling, the actual oxygen output of the OBOGS varies in a sinusoidal nature, represented by the large width of the oxygen content range. The effects of a continuously varying oxygen content on a pilot are unknown.

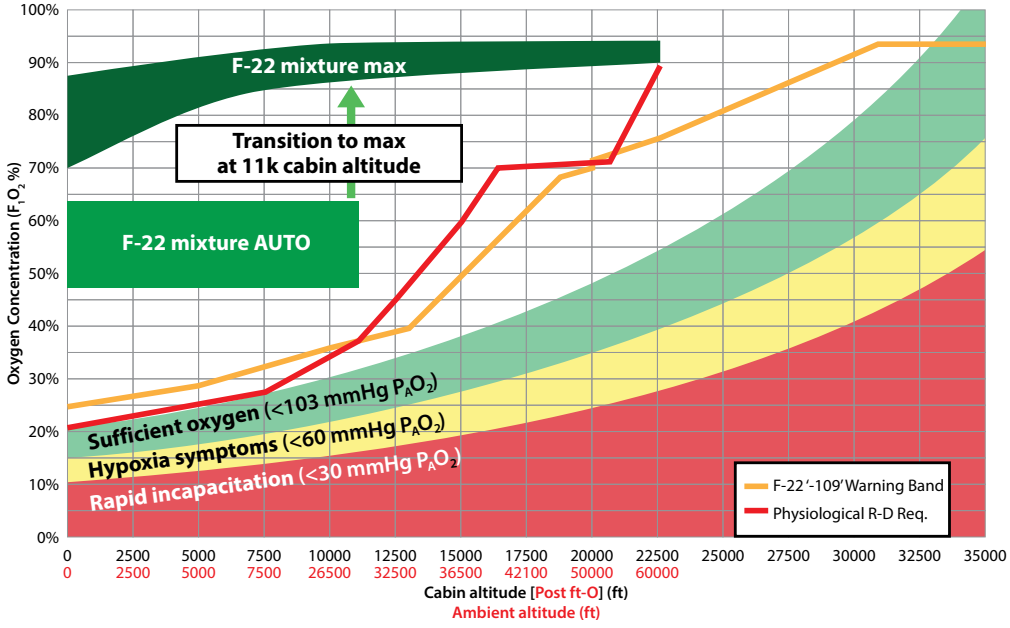

Figure 1. Figure shows the F-22 Oxygen Delivery Schedule with oxygen content beginning in the 50–60 percent range and increasing to 90–95 percent in an 11,000 foot cabin.

(Source: USAF Scientific Advisory Board, *Report on Aircraft Oxygen Generation*, 45)

The final critical difference is the presence or absence of a plenum in the breathing line. A plenum is a storage container of gas (air or O_2) that can provide a fill-in source of breathing gas during a system shutdown, interruption, or breath-

ing demand surpassing the available supply. For LOX aircraft utilizing a dilution regulator, the cockpit essentially functions as a plenum. However, because most OBOGS aircraft do not use a dilution system, a dedicated plenum should be required, but implementation varies greatly from airframe to airframe. Plenum volumes range from 262L (F-15E), to 250 cu in (A-10, F-16, T-6), to 97 cu in (F-18), to 0 cu in (F-22). The smaller plenum volumes, particularly in OBOGS aircraft, mean the complicated human system is directly connected to OBOGS without any buffer to account for system abnormalities on either side. A summary table of these OBOGS systems in USAF and USN aircraft can be found in the US Air Force Scientific Advisory Board's *Report on Aircraft Oxygen Generation*: https://apps.dtic.mil/dtic/tr/fulltext/u2/a567568.pdf.[36]

Possible Undesirable Outcomes

Outcomes, as discussed in this article, refer to the physical condition in an aviator that is most closely responsible for the symptoms they experienced. This does not refer to the start of a chain of incidents that lead to symptoms but rather the final step in that chain. Possible outcomes linked to UPEs include a lack of oxygen in the brain termed *hypoxia* (hypoxic, histotoxic, hypemic, or stagnant), too much or too little cerebral CO_2 (hypercapnia or hypocapnia), or nitrogen bubbles in the bloodstream (decompression sickness). These conditions can result in similar symptoms, are well discussed in other literature, and are important to identify to treat the aviator medically. However, the mechanism that caused the outcome to occur carries greater significance in determining how UPEs happen.

Potential Causal Mechanisms

Mechanisms represent the means by which a certain outcome occurs. For example, in the case of a system malfunction that results in increased breathing resistance, then hyperventilation resulting in hypocapnia (outcome), the ventilation issues (increased resistance and hyperventilation) represent the mechanisms of the system with the system malfunction being the trigger event that started the negative physiological chain. While, in this example, correcting the trigger condition may seem to be the solution, if the trigger is not readily apparent (as is the case in most of today's incidents), a solid understanding of the various mechanisms can be a valuable analysis and risk-reduction tool.

Ventilation Issues

The human respiratory system, from a control system design perspective, represents perhaps the greatest compensation device in existence. Human autonomic

respiration manages P_aO_2 and P_aCO_2 levels, blood pH, and a variety of factors through careful control of the rate and depth of respiration. However, this compensation device is finely-tuned to its normal respiratory environment, which typically consists of near sea-level pressure, standard oxygen levels in the air, few pollutants, and no restrictions to inhalation or exhalation (for a healthy individual). These normal conditions represent a series of assumptions made by the body about its environment and define the area to which it should continually strive to compensate back to. As such, when exposed to a demonstrably new environment for a short period of time (e.g., high-altitude, or a fighter life support system), the body is unable to change its stated assumptions (acclimatization) and instead continues to search for its original condition set. This is a critical fact to remember when considering the various ventilation (e.g., respiratory) mechanisms that can result in negative outcomes.

Hyperventilation as a mechanism is defined as an increase in the rate and/or depth of breathing, such that minute ventilation (volume of gas exchanged in the lungs each minute) is increased above normal. Of the outcomes discussed above, hyperventilation most readily leads to hypocapnia in a properly functioning respiratory system as the increased ventilation results in additional CO_2 elimination without any change in the body's production of CO_2. However, hyperventilation does not always lead to hypocapnia, depending on the severity of the hyperventilation, individual body physiology, or an already compromised respiratory system. In the latter case, a person might not be able to increase minute ventilation above normal due to blockages or restrictions in the respiratory tract as these conditions typically lend themselves to CO_2 retention due to the lack of ventilated lung tissue available for CO_2 exchange. There are multiple different well-documented causes for hyperventilation that all make logical sense when viewed from a system compensation perspective. These include hypoxia, breathing restriction, increased thermal stress, psychological stress, and hyperoxia.[37]

It *should* go without saying that hyperventilation is a natural compensation technique used by the body to reachieve its normal state. Despite this fact, there still exists a strong tendency to equate hyperventilation with a lack of mental or emotional control, which results in a prejudice against admitting to hyperventilation inflight and hampers investigative efforts. It is incumbent upon each member of the aviation community to divorce hyperventilation as a medical mechanism from the classic image of a panicked individual breathing into a paper bag. An aviator experiencing hyperventilation is a *natural human reaction* to external stressors for that person's physiology on that day and should be viewed through a critical, rather than visceral, lens.

Hypoventilation as a mechanism is defined as a decrease in the rate and/or depth of breathing such that minute ventilation is reduced. Typically, this mechanism leads to an outcome of hypercapnia, as the reduced ventilation leads to CO_2 retention. In the medical community, most conditions that lead to hypoventilation revolve around some kind of respiratory disease (COPD, asthma, etc.) that limits the body's ability to effectively exchange gases.[38] From an aviation perspective, the closest analogous scenario is when a pilot's lung volume is decreased or restricted in some manner, either by an additional medical mechanism or by tight-fitting life support gear. Additionally, increased breathing resistance has been shown in multiple studies to lead to hypoventilation in a large group of aviators.[39] From a compensation perspective, this represents a pilot subconsciously trying to avoid increased resistance by decreasing minute volume, a different but also observed compensation technique than that discussed previously regarding *hyper*ventilation.

Ventilation (V)/perfusion (Q) mismatch is another potential mechanism that could lead to blood gas imbalances as an outcome. The ratio of V (air breathed into the lungs) to Q (blood flowing through the lungs) typically remains balanced at a value of about 0.8 under normal conditions. What this means physically is that there is almost the same amount of oxygenated air brought into the lungs to provide O_2 and remove CO_2 as there is venous blood to soak up O_2 and provide CO_2 for the lungs to off-gas. In a well-functioning respiratory system, if one factor changes (e.g., increased perfusion during exercise) the other changes as well to maintain the V/Q balance (e.g., increased respiration during exercise). If one of the factors changes without a response from the other (due to respiratory disease or impairment, changes to blood flow patterns, etc.) the body's blood gas ratio can become out of balance, leading to hypoxia, hypercapnia, or hypocapnia depending on the type of mismatch (i.e., high or low V/Q). These mismatches can occur throughout the lung, but can also happen locally (i.e., only in a specific region). This is especially true under G, where the lower lung can be shunted (V/Q=0), and the upper lung over-ventilated as seen in figures 2 and 3.[40] While history would suggest these localized ratio changes do not create negative outcomes independently, they could combine with other mechanisms to foil the body's natural compensation ability.

Breathing Resistance and Work of Breathing

Work of breathing (WoB) as a mechanism is defined as an *increase* in the amount of physical work required by the pilot to execute a breath and includes both inspiratory and expiratory work. It is typically not a mechanism that leads directly to an outcome but instead leads to another mechanism such as hyper - or

hypo-ventilation, depending on the individual. However, it does represent an important mechanism that is potentially one step closer to the trigger issue, and thus one step closer to fixing problems in the future.

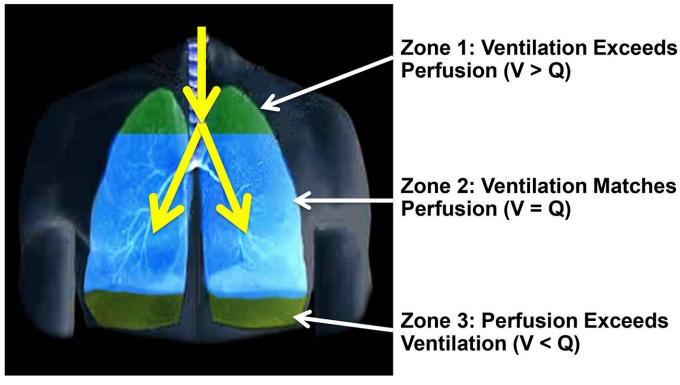

Figure 2. Figure shows how perfusion exceeds ventilation in the typical localized V/Q ratios.

(Source: Dr. Gregg A. Bendrick, *Atelectasis in High-Performance Aircrew*, slide 22, Powerpoint presentation)

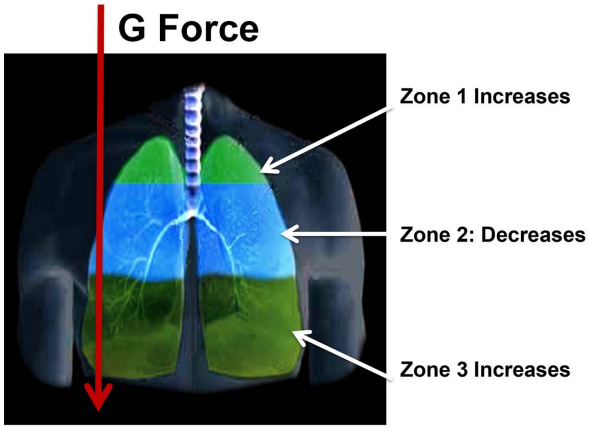

Figure 3: Figure shows the effects of high g-forces on V/Q ratio. Figures 2–3 demonstrate how the lower lung can be shunted (V/Q=0) and the upper lung over-ventilated.

(Source: Bendrick, *Atelectasis in High-Performance Aircrew*, slide 24)

Increased WoB is generally caused by increased breathing resistance or impedance. As the work required to breathe increases, most humans will begin to compensate for the increased work, either through increased or decreased ventilation which, depending on the person, will result in either hyper - *or* hypo-ventilation

and possibly an ensuing outcome. During inhalation, any resistance present inline in the breathing system itself, whether from system connectors, small diameter hoses, contamination filters, a lack of a large plenum (breathing reserve), or OBOGS cycling issues, will necessarily cause an increase in WoB for that breath. Similarly, any physical restrictions on the pilot's chest/abdomen (tight-fitting survival gear, G-suits, equipment vests, etc.) will resist the chest's expansion during inhalation and increase the amount of work required to take that breath. During exhalation, various factors such as exhalation valve cracking pressure, safety pressure, and constant flow pressure (if applicable) can also affect the exhalation resistance and increase WoB.

In most aircraft today, although the literature asserts WoB must be minimized in totality,[41] impedance issues have typically been outprioritized by hypoxia (both histotoxic and hypoxic) and DCS concerns or discounted completely, resulting in safety pressure, small connectors, filters limiting gas flow to the pilot, and physical impediments to normal respiration (tight gear, survival vests, exposure suits, etc).

Hyperoxia

Hyperoxia as a mechanism is defined as a P_aO_2 of greater than normal (>100-103 mmHG). On the surface, this seems counterintuitive as oxygen is typically thought to "only be beneficial" due to its necessary life-sustaining properties. However, breathing excessive quantities of O2 can have multiple effects leading to other mechanisms or outcomes, including atelectasis, delayed otitic barotrauma (ear blocks), hyperventilation, reduced cerebral blood flow, hypo or hypercapnia (depending on the individual), and oxidative stress.[42] With such far-reaching effects, O2 delivery to pilots should be carefully controlled to provide both adequate O2 to prevent hypobaric hypoxia while simultaneously avoiding the effects discussed above. Unfortunately, this is not the case in the majority of our fighter aircraft today.

Atelectasis

Atelectasis is a physiological mechanism wherein lung alveoli collapse and remain closed due to a lack of gaseous pressure within the alveoli itself. In general, atelectasis in an aviation environment has been attributed to a combination of a high O_2 concentration, high g-forces, and wearing restrictive gear (i.e., a G-suit).[43] In these cases, g-forces and tight gear create a large low V/Q area in the lower lung, where high O_2 concentration air is rapidly absorbed *in totality*, thereby collapsing the alveoli due to a drop in total pressure.[44] Collapsed alveoli no longer ventilate, creating a shunt, until the alveoli are reopened. In essence, the result of

atelectasis is a temporary reduction in functional lung capacity. Studies performed since the 1950s to quantify this reduction, have uncovered several "knowns" about atelectasis. First, O_2 concentrations above 50–60 percent, flight gear that either restricts chest expansion or compresses the diaphragm, and high-g forces put pilots at risk of developing significant atelectasis in flight. Second, the respiratory effects of atelectasis are *not* insignificant and can reduce lung volumes by 30 percent as a mean value, with possible individual variation above that. Third, these effects are not purely transient as some level of atelectasis will be present until a forced full lung expansion is performed to clear it. In practice, if an aviator is exposed to risk factors which create atelectasis in the first place, he will likely be unable to fully re-expand his lungs in flight and/or prevent a reoccurrence of atelectasis formation later in the sortie. When these factors are combined with the status of current aircraft life support systems and AFE, which almost perfectly match atelectasis risk factors, it seems likely that many current fighter aircrew members execute at least part of their tactical maneuvering with some level of reduced lung function due to atelectasis.

Bringing it All Together: Interactions of Potential Causal Mechanisms

With the above "knowns" in mind, consider an aviator who is stepping to fly in a current fighter type aircraft. This means her aircraft's OBOGS produces an excessive and constantly varying amount of oxygen in the breathing gas for most of the flight envelope, and she is likely wearing a large amount of aircrew flight equipment (AFE) (a dry suit, harness, survival vest, partial pressure suit, full-coverage G-suit) that will restrict and constrain lung expansion along multiple axes. These factors, combined with the high-g environment (well above the levels tested in the above studies), will create atelectasis that will likely persist or reoccur for the duration of the flight, resulting in an approximate 30-percent reduction in lung volume and an associated shunt of unoxygenated blood. The body will likely attempt to compensate for this reduction but will be challenged to do so because of and complicated by interactions with other mechanisms.

First among these complications is the hyperoxia that partially caused the atelectasis in the first place. This hyperoxia causes the aviator to hyperventilate slightly to compensate for reduced CO_2 transport ability and reduces cerebral blood flow (possibly causing mild hyper or hypocapnia depending on her body chemistry). The high O_2 gas content will also likely prevent P_aO_2 from dropping because of the atelectasis produced shunt, making the shunt a latent condition that is essentially "lying in wait." Additionally, this hyperventilation will likely take the

form of increased breathing rate, not depth (due to the AFE), which would both prevent temporary reexpansion of areas of atelectasis and would exacerbate the V/Q mismatch caused by the shunt. If no additional demands are placed on the body, it will likely be successful compensating to maintain a physiological balance, as evidenced by the various atelectasis studies which found no serious medical outcomes from atelectasis, only pilot annoyance and discomfort.

However, now consider an additional mechanism in the form of increased WoB. Using current airframe examples, this could be due to safety/continuous pressure (F-18, T-45, F-22, and F-35), restrictive AFE (all depending on the mission), small hoses/connectors (all), ECS/OBOGS pressure transients (if insufficient plenum size), a lack of instantaneous flow capacity (all), or a simple aircraft malfunction (kinked or broken hose, stuck valve, etc.). The aviator's body now tries to compensate for WoB through ventilation changes (hyper or hypoventilation) while simultaneously compensating for the effects of high O_2, V/Q mismatch and shunt, and reduced lung function. And remember, not all of these mechanisms are static, with the WoB changing constantly due to the pilot's exertion and the oxygen output of her OBOGS also constantly changing with little or no plenum to soften the variance.

This is a significantly dynamic and complex problem for a human body to sort out. There likely exists some level of *physiological margin* for each individual on any given day that represents the maximum amount of compensation their system can perform. In the aviation world, this would be analogous to stall margin in a jet engine. A pilot's physiological margin can be overwhelmed *acutely* by a single event (e.g., rapid decompression) or *systemically* whereby the confluence of multiple different constantly varying factors in both the aircraft and human system stack on top of each other at the right time to overwhelm the pilot's compensation ability. Viewed through the prism of this theory, it should come as no surprise that the fighter aircraft of today continually take a "well-functioning" human system and a "well-functioning" aircraft system and create a UPE. As the various mechanisms described above interact with and change the body's compensation methods, they become out of sync, compound, and produce seemingly unexplainable results.

Conclusions: How Can This Help in the Air?

As complex the systemic and variable breathing problems discussed above are, the airborne solution for the pilot appears to be simple and most importantly is virtually the same regardless of which outcome is at play. From hypoxia to hypocapnia, our bodies are built to combat any breathing irregularity automatically, provided nothing is stopping us from doing so.[45] Therefore, our approach to solv-

ing these problems is simple: Do not try to diagnose physiological problems airborne. Instead, first ensure you have enough oxygen to breathe—hypobaric hypoxia is still the top threat. Once you have eliminated the oxygen variable, get to a safe place that mirrors the open-air breathing environment of Earth's surface (ideally somewhere below 10,000 foot), remove any impediment to your breathing (i.e., your mask, tight gear, etc.), and relax until you feel recovery begin. In other words, go to "Colorado," hold until you feel better,[46] then go home. Whether the initial problem was hypoxia, hypocapnia, work of breathing, atelectasis, hypercapnia, or a likely combination of factors, if a pilot can relax and breathe restriction-free open air at an earth-like mixture, she is working toward recovery.

Think back to the opening of this article to the pilots who reached the definitive end of their physiological checklists with no condition improvement. The procedure would allow pilots to cope with this UPE where their checklists do not hit a definitive end. This "do no harm approach" provides pilots with the tools to handle UPEs to their conclusion in a scenario where the underlying trigger, mechanism(s), and outcome are in doubt. The aerospace medicine field is rapidly progressing in its understanding of these issues, but substantial questions and unknowns still remain. Airmen should not fear these unknowns but rather embrace airborne procedures that possess a robustness in their triage of airborne symptoms until the many interactions between aviators—and the systems meant to keep them alive—are fully understood. ✪

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