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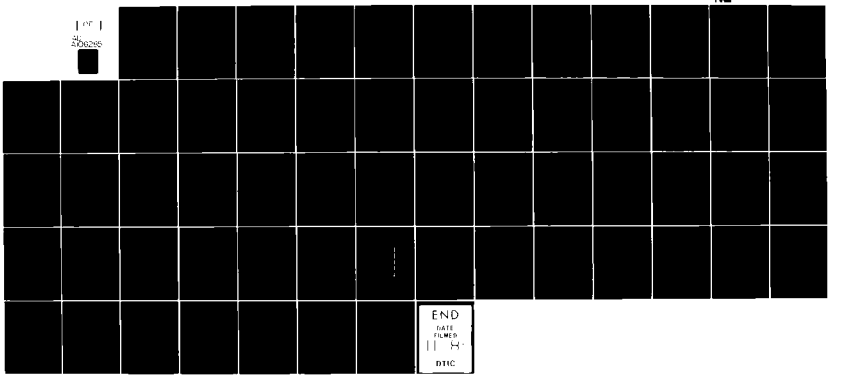

DECOMPRESSION SICKNESS IN AEROSPACE MEDICINE: THE DEVELOPMENT OF AN ONBOARD TREATMENT FACILITY FOR THE SHUTTLE-ORBITER SPACECRAFT

by

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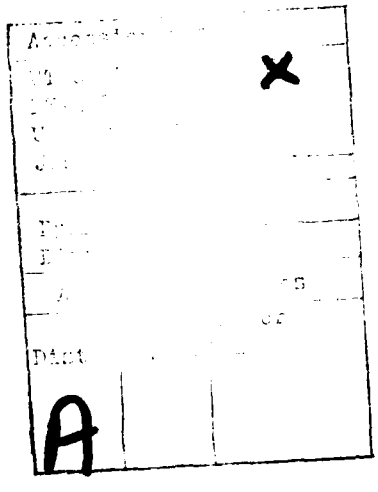
ABSTRACT

Altitude decompression sickness is a pathological disorder that can occur when aviators and astronauts are exposed to lower barometric pressure of altitude. Prompt care is available to most pilots within minutes to a few hours. For astronauts in orbit this capability is not available. This report develops an on-board recomprocession with an established treatment protocol.



PREFACE

With the introduction of the Shuttle-orbiter concept into the space program, new mission capabilities were also introduced that will once again expand the opportunities for man to extend himself further into the unknown. While advances in technology provide the capabilities for these ventures, man. structurely and physiologically, remains essentially the same and all the sophisticated hardware that will propel him to those outer limits will do little to change the physical capacity of this human explorer. Since he will continue to reach beyond his familiar environment, he must then develop the necessary supportive measure that will assist him in overcoming his physical inadequacies and provide himself with the capability of maintaining his physical integrity when encountering the insults of foreign environments.

In regard to this space mission, Dr. Joseph Degioanni of Flight Medicine at the Johnson Space Center was given the task of developing and providing the medical support equipment for the future missions of the Shuttle-orbiter spacecraft. Realizing the advances associated with this new concept of a re-usable spacecraft, his task was not without challenge. I would like to express my sincerest appreciation to Dr. Degioanni for allowing me the opportunity to develop the treatment method for on board decompression sickness for the Shuttle-orbiter spacecraft.

I wish also to express my appreciation to James Schlosser of the intra and extra vehicular systems division at the Johnson Space

Center for his expert engineering assistance in the modification on the Personnel Rescue Sphere and extend personal thanks to members of his staff.

Special appreciation is also extended to Dr. James Adams of the crew systems division of the School of Aerospace Medicine, Brooks Air Force Base, Texas, for his kindness in granting me the privilege of working with him in those studies that were invaluable to my project and for the encouragement he provided throughout my research.

I would also like to acknowledge the members of the library staff at the Strughold Aeromedical Library at Brooks Air Force Base for providing me material that may not have been so easily obtainable without their kind assistance.

Very special appreciation is also extended to my wife and children for their loving encouragement and understanding during this period of study.

Finally, I wish to express my appreciation to a number of associates whose diverse contributions were of enormous value. Included are the following: Dr. Michael Berry, B. Gen. Robert McIver, Col. Jeff Davis, Col. Jared Dunn, Gene Dixon, James Waligora, and David Horrigan.

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TABLE OF CONTENTS

	Page
Introduction	1
History of Decompression Sickness	2
Pathogenesis	3
Classification Based on Symptomatology	7
Predisposing Factors	10
Prevention	13
Treatment	17
Decompression Sickness in Space Operations	24
Treatment Facility For The Shuttle-Orbiter	30
Inflight Treatment of Decompression Sickness Aboard The Shuttle-Orbiter	39
Conclusion	42
Epilogue	44
Appendix A	45
Appendix B	47
Bibliography	48

LIST OF FIGURES

Figure		Page
1.	U.S. Navy Hyperbaric treatment table 5 for decompression sickness	19
2.	U.S. Navy Hyperbaric treatment table 6 for decompression sickness	20
3.	Flow sheet for disposition of patients with decompression sickness	23
4.	Results of a study to determine add on times after interrupted denitrogenation process	26
5.	Results of studies using a 34 minute add on time after a one minute interruption of denitrogenation	27
6.	Results of a study to associate bubble formation with the development of bends	29
7.	View of personnel rescue sphere designed for the Shuttle-orbiter.	33
8.	View of the reinforced version of the personnel rescue sphere	35
9.	View of the treatment configuration of the personnel rescue sphere	37
10.	Proposed treatment schedule for the modified personnel rescue sphere.	40

Introduction: Altitude decompression sickness (DCS) is a pathological disorder that can occur when aviators and astronauts are exposed to the lower barometric pressure of altitude. The symptoms can be mild or severe with resulting mild tolerable pain, total incapacitation, or even death. When the condition occurs immediate therapy is required. This consists of the immediate application of 100% oxygen and descent to lower altitudes.

In the case of pilots of aircraft who develop the disorder the flight is terminated and the aircraft landed as soon as possible so as to effect a appropriate therapy. With increased familiarization by the flight surgeon with the condition and the availability of hyperbaric treatment facilities, the prognosis for those developing decompression sickness is excellent (37).

Prompt and expeditions primary care and hyperbaric oxygen therapy is available to most pilots within a few minutes to a few hours. For the astronaut in orbit or on an interplanetary mission this capability is not available and failure to receive appropriate care, should the condition develop, could be catastrophic. Because of this potential the need for an on-board recompression capability with an established treatment protocol is apparent. The facility would have to meet the weight and space constraints of the space craft and a treatment schedule developed that would be compatible with those limitations and still provide and adequate form of therapy.

Recent studies, which involved the oxygen and pressure differential included in the environment of the Shuttle-orbiter (1,2),

revealed a significant potential for the development of decompression sickness in astronauts during extra vehicular activities. This situation, combined with other factors involved in future mission requirements, could result in a significant increase in the incidence of DCS during the space missions. Even with a thorough knowledge of the manifestations of decompression sickness and its management, the development of an onboard treatment facility for the spacecraft would be an exciting challenge.

History of Decompression Sickness: Early in the nineteenth century, with advent of compressed air and the development of diving suits, cassion workers began experiencing a series of symptoms that occasionally resulted in death (13). They soon learned the symptoms would occur during or upon their return to the surface after diving and then upon returning to the increased pressures of depth the symptoms would subside. It was not until several years had passed that the medical profession dismissed the idea that this disorder was caused by the damp and cold of diving and realized that increased pressures may cause a greater quantity of gases to be absorbed in the body fluids and tissues. When the pressure is then rapidly reduced, the gases would come out of solution and form bubbles in the intra- and extra-vascular spaces of the body. Fryer noted (13) that Paul Bert, one of the greatest contributors to the knowledge of the effects of reduced atmospheric pressures, in 1878 felt that such did occur particularly if the evolved gas formation overwhelmed the lungs' ability to excrete the gases. Bert was never able to demonstrate such a condition associated with the lowered barometric pressure of altitude and it wasn't until Armstrong, in

1939, (3) demonstrated intravascular bubbles in a rabbit exposed to an altitude of 38,000 feet that we recognized decompression as a possible hazard to high altitude and space flights.

Armstrong coined the term aeroembolism (3) to describe the disorder associated with decompression, but this term should probably be reserved for those conditions in which there is embolization of the arterial circulation by evolved gases. Decompression sickness is the more accepted term (37) for the disease that occurs with the presence of evolved gas in the body tissues as a result of reduced barometric pressures. "The Bends," a term that is frequently used interchangeably with DCS, has now been accepted as a manifestation of decompression sickness and is limited to the symptoms of musculo-skeletal pain. Barotrauma is a term that is used to describe the effects of lowered barometric pressure on trapped body gases. These diseases include barosinusitis, barotitis media, pulmonary barotrauma, pneumothoras, aerodontalgia, and gastrointestinal gas pain (37) and when included with decompression sickness should more appropriately fall under the heading of dysbarism. Decompression sickness, as it is described above, will be the most appropriate term of the purpose of this paper.

Pathogenesis: The signs and symptoms of decompression sickness apparently occur as a result of inert gas bubbles that form when individuals are exposed to reduced environmental pressures (13, 21).

Henry's Law states that the concentration of gas dissolved in a liquid is directly proportional to the pressure in which the liquid is exposed. If the environmental pressure is sufficiently reduced those gases dissolved in the body tissues and fluids would evolve to form

bubbles. Should the pressure differential occur rapidly and is of a great magnitude the evolution of gas bubbles would also occur very rapidly, and if the lungs' capacity to eliminate these evolved gases is exceeded, bubbles of evolved gas would then begin producing symptoms of decompression sickness (13, 21). Another factor involved in the pathophysiology of DCS is the collection of gas bubbles in the tissues very poor in blood supply, such as fatty tissue, periosteum, ligaments, tendons, and cartilage. Since these tissues probably contain a greater portion of the dissolved nitrogen, bubbles would easily form here and due to the limited blood supply would not be carried as readily to the lungs and would tend to collect in these areas (1, 23). This situation could result in distortion of the nerve endings in those tissues (19) and cause pain and would, further, tend to impinge upon the micro-capillaries, interrupting blood flow causing tissue hypoxia and edema (7). The gradual release of these trapped gases may contribute to the latent effects of DCS even after return to normal environmental pressures (1, 8, 37). If the bubbles remain relatively stationary for any period of time, some authorities feel a stabilization of the bubble occurs as a result of fibrin formation (1) over the surface of the bubble. With a minimal re-exposure to altitude these stabilized bubbles would tend to produce greater symptoms at lesser altitude as a result of the relative stability of the bubble (36).

The presence of gas nuclei may enhance the development of bubbles that give rise to decompression sickness (23). Gas nuclei have been defined as minute collections of gas differing only from bubbles in size, the difference being arbitrary (37). The importance of gas

nuclei in DCS lies in the fact that they can develop prior to decompression (21, 23) and their presence enhances the formation of larger bubbles more readily during exposure. Also they probably occur, in part, during exercise which makes physical activity during decompression a predisposing factor to the development of the disease (13, 27). The nuclei are believed to form during cavitation of tissue spaces (19, 21), hydrostatic pressure differentials (23), turbulent blood flow in damaged tissue (13), and as a result of CO_2 formation during muscle contraction (21). It is possible that persons with recent injuries may be more susceptible to DCS (37).

Although bubbles are composed of a mixture of nitrogen, carbon dioxide, oxygen, and water vapor, nitrogen is the major component of the gas bubble of DCS. The relatively high solubility of nitrogen combined with its inertness gives rise to this situation. Since the formation of intra-vascular bubbles occurs as a result of pressure reduction, they have a tendency to develop in the venous capillary system (21). The higher arterial pressure is not conducive to bubble formation (37). When bubbles do form, there is an interruption of the venous outflow that produces a venous congestion, hypoxia, and edema. This can also give rise to the fibrin formation as previously mentioned. When this occurs a foreign body response may occur with subsequent platelet aggregation (1, 17, 37), sludging of red blood cells, rouleux formation (17), thrombo cytopenia (21), formation of lipid emboli (8, 21) and elevated serum enzymes (37). There remains some question as to the exact mechanism involved in the production of these responses since mechanism does not appear to be present in all individuals

stricken with DCS (7).

The mechanisms and effects discussed above are a representative group of some of the causes of the symptoms of decompression sickness. These conditions could produce minor symptoms or they could produce a fulminating pathological condition resulting in the more serious or major form of the disease.

Another aspect of the disease and representing, usually, the major type of DCS would be the occurrence of bubbles on the arterial side of the cardiovascular system. This could occur when the degree of bubble formation on the venous side overhwelms the pulmonary vasculature (1) and the bubbles shunt to the arterial side (21) causing serious central nervous system injury as a result of aeroembolism. Many studies have been conducted to determine the actual mechanism involved in the passing of air emboli from the venous circulation to the arterial system (2, 6, 21), and that question remains in the minds of many authorities today. Another, somewhat related, serious consequence of massive bubble formation is the bubble buildup within the pulmonary arterial system. This condition produces tachypnea, severe pulmonary hypertension (38), and if allowed to persist by continued exposure, acute circulatory collapse and possibly death.

Any one or all of the conditions described in the pathogenesis of DCS can occur giving rise to a variety of symptoms. Indeed, the nascent bubble has been described as an "adroit dissembler capable of not only deranging rheology and morphology, both of vessels and formed elements in blood, but able to assume various guises to mask prime pathogenecity" (3). While symptomotology may be misleading a history

of exposure to decreased barometric pressures (altitude) combined with the presence of relatively well established predisposing factors should alert the flight surgeon to giving this disorder prime consideration in his differential diagnoses.

Classification based in symptomatology: Using the British categorization, which is widely accepted, decompression sickness can be classified as type I and type II based on presenting and/or subsequent symptoms. Type I DCS is usually considered a minor form of the disorder and includes pain only "bends" and cutaneous manifestations (6, 37). Type II is considered the major or more serious form and is comprised of pulmonary involvement, "chokes," neurological manifestations, vasomotor, and circulatory collapse and shock (13, 37).

"Bends" is a term used to describe the symptoms of joint pain (13), deep bone pain and muscle aching (37). The pain can occur in any joint or all joints and can migrate from one joint to another (12). Occasionally the pain can involve the entire limb or spread up and down the limb from the affected joint (37, 13). While beginning as a mild aching, the pain may progress to becoming severe and incapacitating (6, 7). These symptoms can occur at altitude or may occur after descent to ground level with 90% of them occurring within 12 hours of exposure, with a delay of occurrence rarely exceeding 24 hours (37).

The cutaneous manifestations of DCS, included in type I, probably develop when bubbles collect in the superficial lymphatics or produce subcutaneous emphysema that is followed by rashes, erythema, itching, and formications (37). This condition is considered minor and usually subsides upon or before reaching ground level from altitude.

Urticaria-like edema may also appear as an "orange peel" effect of the edema (24). Mottling and marbling of the skin can also occur and is usually considered a part of the minor manifestations of the disorder but should it occur with the major or type II symptoms it may represent and impending circulatory collapse (37).

Type I or bends pain only represents the most frequently occurring manifestation of decompression sickness (6, 7, 13, 37) and is the only condition of DCS that may not require hyperbaric therapy. The symptoms of type I DCS usually subside during descent but can persist or recur with the subsequent development of severe or type II DCS (7).

Type II or the major symptoms of DCS, consisting of pulmonary involvement or chokes, neurological manifestations, and vasomotor or circulatory collapse, represent the smaller percentage of cases but by far the most serious. These symptoms may be preceded by type I symptoms or can occur as the only presenting symptoms (7, 37). The patient could develop type I symptoms and recover during descent then develop type II symptoms several hours following return to ground level (6, 7). A re-exposure to even minimal altitude after a short ground time following minor symptoms, could result in type II symptoms (23, 36).

The pulmonary manifestations or chokes probably develop when bubbles backup in the pulmonary vasculature distending the sensitive structures of the lung causing trauma and, perhaps, rupture of some of the tissues producing the tussive effect (21). This condition would include the classic triad of symptoms of dry, non-productive cough, dyspnea, and sub-sternal chest pain or discomfort (37). Some investi-

gators feel these symptoms could also be due to multiple micro-emboli of platelet aggregates (1) created by the presence of stabilized bubbles previously described. These symptoms could be heralding a very serious situation since they are occasionally followed by acute pulmonary hypertension and circulatory collapse (38). It should be noted the symptoms may be delayed, usually occurring in the first two hours but may be as long as 12 hours.

Neurological symptoms occur as a relatively prominent manifestation of decompression sickness. Davis (7) found these symptoms in 34% cases requiring hyperbaric therapy, and as the only presenting symptom in 14% of cases.

The symptoms of neurologic involvement in decompression sickness could cover any symptom of neurological pathology and its characteristic would depend on the site involved (12). The symptoms may consist of any combination of paresthesias, disorientation, dysphasia, visual disturbances, migraine-like headache, vertigo, nausea and vomiting, lethargy, generalized or localized weakness, fatigue (3), coma, convulsions (8), and death (12, 37). The mechanism involved in the production in these conditions or symptoms is probably the direct effect of the formation of gas bubbles in the central nervous system, both extravascular and intravascular (6, 7, 8, 21). The gas bubbles could also evolve outside the central nervous system and embolize (8, 21) causing an occlusive condition with resultant ischemia, edema (8), and perhaps infarction. The lipid content of the central nervous system is high which could be a rich source of nitrogen (1) and could give rise to extra-vascular bubble formation causing a tissue insult and extra-vascular compression on the vessels suppling the brain and nervous system (8, 12). Vasomotor

collapse probably comes about with the occurrence of both, central nervous system and circulatory disturbances (8, 12, 37).

While categorizing the symptoms of DCS into type I and type II classifications it should be realized that one does not necessarily follow the other. Indeed, type II can develop without any of the symptoms in type I appearing. The symptoms of type I and type II can occur at altitude, resolve during descent, then after an asymptomatic period at ground level the symptoms of type II DCS appear (7, 8). The classification is a convenient and useful method of determining the most appropriate approach to managing the disease and has proven successful in that regard (7). One must be certain, however, not to overlook any subtle symptom of type II DCS and proceed to manage only type I (24). Some investigators (1, 21) are not convinced that symptoms of type I and type II represent a different stage of the disease since they obviously represent a different pathophysiological process. Two distinct entities, indeed, remain a possibility (19, 20).

Predisposing factors: It is generally agreed that DCS occurs as a result of bubble formation and that any factor that would contribute to that process would also serve to enhance the development of decompression sickness. It is also accepted (6, 7, 19, 20) that there exists an individual variation in the propensity for the disorder suggesting a physical "fitness" attribute. Nitrogen has a great affinity for adipose tissue and, consequently, fatty tissue is rich in nitrogen content. With the relatively limited blood supply in that tissue obesity has to be implicated as a major predisposing factor to DCS. Although the actual risk is not known there appears to be an increased

incidence of DCS associated with obesity (4). Age appears, also, to increase the incidence of decompression sickness, but the actual reason is not known (4). Recovery from DCS with compression therapy is more difficult and recurrence after therapy in the older person is more likely (13, 39). Circulatory factors may well play a role.

There is no evidence to support the suggestion that old injuries predispose to the development of limb bends (37), however, pain occurring at recent injury sites has been reported as has pain in chronically infusing knee problems (13, 39).

Exercise at altitude is a profound contributing factor to the development of DCS (1, 6, 7, 21). The factor is of great importance to the aviator and astronaut since exercise at altitude is common place in both these occupations, particularly the astronaut during the extra vehicular activities. Exercise playing such a major part in the disease is a result of several factors. Nitrogen washout periods, to be discussed later, probably does little in eliminating the dissolved gases in fatty tissue, ligaments, tendons and cartilage (19, 14). At altitude this gas evolves to form the extra vascular bubbles previously discussed. This evolved gas would probably remain relatively well localized until acted upon by forces such as contracting muscles, active tendons, and ligaments cartilagenous stresses that would occur with exercise (12, 21). This combined with changes such as increased cardiac output and increased CO_2, would have a tendency to encourage diffusion of nitrogen into the intra-vascular compartment (19, 21). CO_2 development from metabolism may also play a direct role in bubble formation (21). Muscle contraction with resultant forces on ligaments, tendons and

cartilage surfaces may cause cavitation effect and surface-to-surface pressure differentials producing the physical properties to further exaggerate bubble formation (21, 23).

The altitude or degree of reduced barometric pressure in which an individual is exposed is an important factor in the incidence of DCS. In studies conducted by Haldane, the English physiologist, it was found by reducing the barometric pressure by one-half, bubble formation could be demonstrated as could the symptoms of decompression sickness. The comparable altitude would be 18,000 feet (5488 meters). Fryer's (14) report of a case in 1964 supported Haldane's and was, in fact, the lowest altitude case of decompression sickness reported, occurring at 18,500 feet (5640 meters). The case was of a serious nature and the individual had not previously been exposed to greater than sea levelpressure. Davis (8) reported two cases of severe neurological decompression sickness with associated seizures occurring after exposure to 19,000 feet (5794 meters) and 28,000 (8536 meters).

A higher risk of DCS can be expected with increasing altitudes, particularly above 18,000 feet.

Recent studies at the U.S. Air Force Academy (36) revealed the development of DCS upon re-exposure to minimal altitude. The cadets had been exposed to the routine physiological training altitude of 35,000 feet. Following the training they returned to ground level of approximately 4,500 feet. All were apparently asymptomatic and after a few hours (less than 12) they returned to the campus of the academy, an altitude of approximately 7,000 feet. Several cases of DCS occurred in the next 12 hour period requiring recompression therapy. The in-

vestigators felt this was a result of unresolved bubbles that formed without producing symptoms during the first ascent, remained during descent, become stabilized as a result of fibrin formation over the surface, and then re-expanded during the second ascent to the relatively low altitude at the campus (36). While the exact mechanism is not known this study lends support to the observation by some workers that re-exposure to even minimal altitudes after a short ground time can be a major predisposing factor to DCS (1, 6, 10, 36), and must be considered a serious risk factor.

Duration at altitude must also be considered a risk factor since the longer exposure would tend to augment the previously discussed predisposing factors, particularly exercise.

While the occurrence of altitude decompression sickness following scuba diving is rare (15), it should be considered a potential hazard and recommended sea level time precautions before flying should be followed (11).

Exposures to cold temperatures appears to increase the risk of DCS though the exact function is not known (14).

Hypoxia, implicated as a possible risk factor in DCS (14), could produce a variety of other serious hazards, whether DCS occurs or not, but DCS must be considered with every occurrence.

Prevention: Prevention of DCS probably begins with the physical examination of the individual who plans to be exposed to decreased barometric pressures (39). Several factors that should be studies during the exam are mentioned in the section on predisposing factors. Obesity contributes significantly to the incidence of DCS (7, 39) and should be

disqualifying for these occupations requiring exposure to reduced barometric pressures (39). Body build probably has no effect providing the excess is not fat. Age is a factor as previously mentioned, and limitations with age must be considered (39). The periodic physical examinations required for aviators and astronauts are strict, and those meeting those standards will qualify based on our present knowledge regarding physical fitness and risk to exposure to altitude (39).

There is some indication that females may have a greater propensity for the development of the disorder (1, 10, 36) and those who develop it tend to be more difficult to manage (10). There are no studies to support this, but cases have occurred that suggest the situation exists. While eliminating females from programs involving altitude exposure may influence the incidence of DCS, other preventative measure such as limiting the duration and extent of exposure would seem more practical.

Being aware of the factors that contribute to the incidence of altitude DCS, it would seem appropriate, from a preventive medicine standpoint, to merely place limits on those excesses that produce the hazards. This can, to some extent, be accomplished but with realization, of course, that man will continue to increase his exposure to greater hazards as advances in technology continue to provide the capability to do so.

Two of the most important preventive measures that can be taken when the situation will not allow the limitation of the predisposing factors is the nitrogen washout and cabin and suit presurization (3, 13, 29, 37). Denitrogenation or nitrogen washout is a

process of eliminating as much as possible the nitrogen absorbed in the body tissues by breathing 100% oxygen prior to exposure to decompression. This process which is accomplished, or should be (1), at ground level pressure (14.7 psi), with the oxygen being provided by tight fitting mask, displaces the nitrogen from the tissues by "washing out" with increasing concentrations of oxygen. The process should continue long enough to eliminate a sufficient amount of the nitrogen so that exposure to altitude would not produce an overwhelming amount of evolved gas. A longer pre-breathe period should be required for higher altitudes of greater duration. Other predisposing factors that may be present should also be considered with the pre-breathe requirement.

The U.S. Air Force has now tailored their pre-breathe requirement to the various types of missions anticipated by the aviators. Pilots of fighter aircraft, for example, not expecting altitudes greater than 25,000 feet for a period not longer than 2 hours with minimal exercise can do well with only 30 minutes denitrogenation. Astronauts, planning extra vehicular activities, must ascend from a pressure of 14.7 psi in the Shuttle-orbiter to 33,000 to 35,000 feet, or 3.7 psi suit pressure and expect moderate to severe exercise for two and one half hours (41). Based on recent studies including various periods of denitrogenation, (1, 2, 30, 31) a 4 hour pre-breathe period should be considered. While that period will not completely clear the resolved nitrogen the suit pressure should maintain the evolution of gases low enough to be eliminated adequately and prevent decompression sickness (13).

In regard to the predisposition of re-exposure to DCS, adequate ground times after exposure to altitude is recommended and

that period should be determined by the altitude and duration at that altitude previously experienced. This may vary from 2-3 hours up to 24 hours (7, 36, 37).

Individual selection for the various aircrew duties has been accomplished, in part, based on susceptibility to decompression sickness. During WW II this was done by exposing candidates to various altitudes for varing periods of time, then selecting the air crew members for assignment based on their response to the studies (13). This process is still being carried out with periodic altitude chamber exposure requirements by aircrew members. This process can in most cases serve to detect, early, the potential of an individual developing DCS (1, 10).

Another area of prevention that has been suggested in regard to identifying susceptibility would be to quantify the actual pre-breathe requirement on an individual basis (1). Recently the rate of bubble formation has been graded during exposure to altitude (1, 2) by the use of a Doppler device placed over the pulmonary artery (1). This technique, which can detect bubbles greater than 50 microns in diameter, also shows promise in predicting the development of bends prior to the appearance of symptoms (1, 2). The period required for each individual to pre-breathe could be determined by measuring the rate of bubble formation against the pre-breathe periods (1, 2, 22).

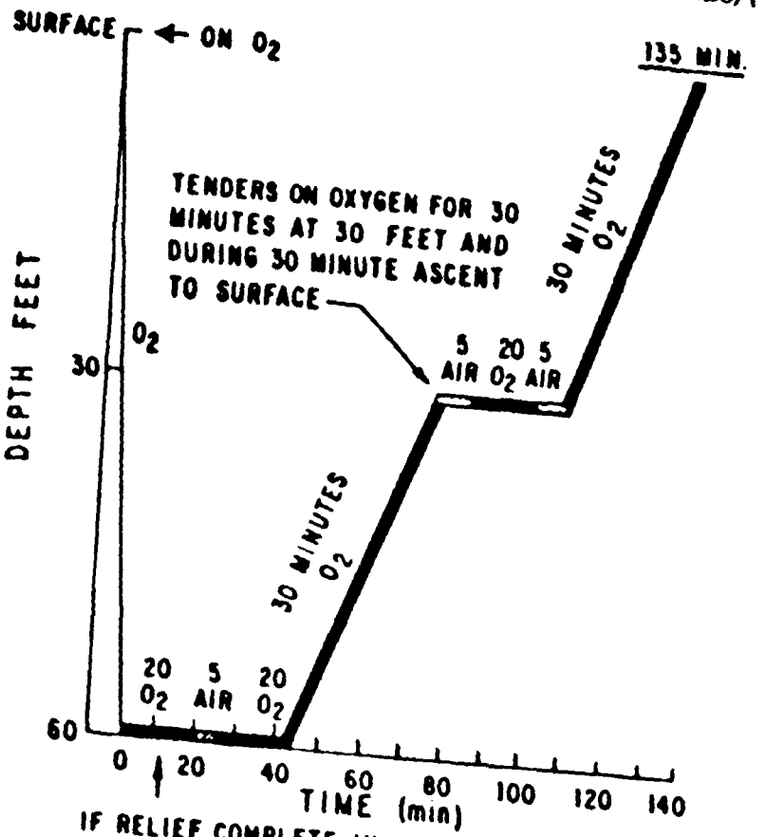
While the mechanisms of DCS remain unclear many of the risk factors have been apparent and by exercising those preventive measures outlined, and avoiding the risks as much as the mission would allow, has resulted in a relatively low incidence of all types of decompression sickness with the majority being type I (28, 37).

Treatment: The treatment of DCS must be directed at reducing the bubble size and enhacing the resolution of evolved gases (6, 6, 37). The urgency of the disorder can be supported by the previously discussed points in the pathophysiology, i.e. a bubble obstructing blood flow can result in ischemia of that part being supplied and if continued can result in irreversible tissue damage, particularly tissue of the central nervous system (8, 21, 37). Unresolved bubbles can become more stabilized with time by fibrin formation over the surface and tend to be more difficult to reduce in size. Platelet aggregates may be associated with the stabilized bubble and when the gas has resolved the non gaseous materials become a potential hazard as emboli (1, 20). These factors as well as others outlined makes it imperative to take those steps first, to halt the process of DCS and then to initiate therapy to resolve those pathological conditions present. Hyperbaric oxygenation (HBO) is the only definitive treatment that can provide those factors necessary to reverse the process of DCS and can also assist in alternating some of the conditions produced by the disease (1, 7, 18). The pressure exerted by the hyperbaric environment will reduce the size of the bubble based on Boyle's law, which states that at constant temperature the volume of gas is inversely proportional to the pressure. The reduction of the size of the bubble will relieve pain that has occurred from its mechanical effects and will also allow re-establishment of blood flow previously obstructed (37). This pressure effect may also cause the bubble to collapse or assist in the resolution of the gas into the tissues (1, 18, 41). The 100% O_2 provided in combination with the increased barometric pressure will produce an increased

tissue oxygen gradient causing nitrogen to diffuse from the bubble further reducing its size (1). The O_2 increases the amount of resolved nitrogen to be eliminated by the lungs by the increased O_2 gradient at the pulmonary level. This will prevent the reformation of bubbles during ascent from the chamber (1, 41). The O_2 combined with the hyperbaric pressure hyperoxygenates the tissues reducing hypoxia previously caused by bubble obstruction, cerebral edema, and ischemia (8, 18, 37). This combination may also assist in correcting any pH abnormalities that may have occurred as a result of the hypoxia. Hyperbaric oxygenation therapy (HBO) is provided in the U.S. Air Force (18) by placing the patient in a standard U.S. Navy double lock hypo-hyperbaric chamber and providing O_2 by an air tight aviator's mask. While several treatment tables are available which provide the depth, time at depth, and air mixture, the U.S. Air Force has found the Navy treatment tables 5 and 6 the most effective in the treatment of altitude DCS (7, 18). See Figures 1 and 2. These tables recommend treatment schedules which provide a descent to 60 feet of sea water pressure 2.8 atmospheres absolute (Ata) in both table 5 and 6. Table 5 is used if treating bends pain only or type I DCS: table 6 is used for all other conditions. If table 5 is being used and symptoms of type I subside before 10 minutes at 60 feet of sea water pressure (FSW) or 2.8 Ata then the treatment is carried out as indicated for that schedule. If the symptoms do not subside then the schedule for table 6 is followed. As mentioned, table 6 is used for type II DCS. Prior to 1965 compressed air therapy at 165 FSW was used for most of the cases of DCS and consisted of 4 different schedules that could be used depending on the type

FIGURE 1

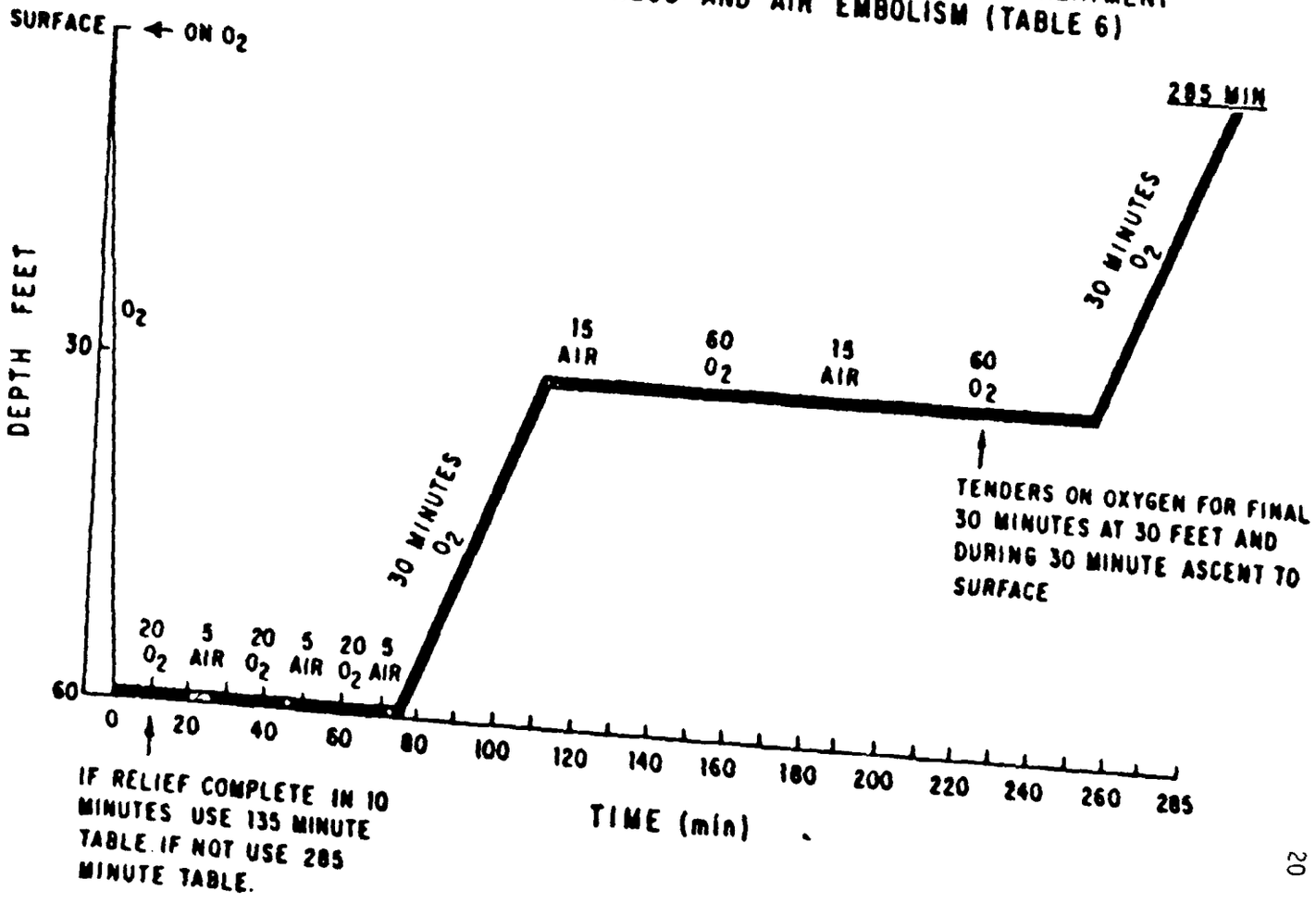
MINIMAL COMPRESSION, OXYGEN BREATHING METHOD FOR TREATMENT OF DECOMPRESSION SICKNESS ("PAIN ONLY" BENDS) (TABLE 5)



IF RELIEF COMPLETE IN 10 MINUTES USE 135 MINUTE TABLE. IF NOT USE 285 MINUTE TABLE.

FIGURE 2

MINIMAL COMPRESSION OXYGEN BREATHING METHOD FOR TREATMENT OF DECOMPRESSION SICKNESS AND AIR EMBOLISM (TABLE 6)



of DCS and response to initial recompression phase (7, 26). Treatment with hyperbaric oxygen using U.S. Navy treatment tables 5 and 6 has been used since 1965 with excellent results (7). Oxygen toxicity is a concern in the use of oxygen at increased partial pressures; indeed, the use of O_2 below 60FSW or 2.8 Ata is not recommended because of the possibility of that condition. The toxicity, a result of the interruption of cellular metabolism, can produce central nervous system disorders such as convulsions and pulmonary damage (25). This condition can be avoided by calculating the cumulative effects of hyperbaric oxygen using a formula divised by Wright and Lambersten at the University of Pennsylvania, the UPTD (Unit Pulmonary Toxicity Dose), and by not exceeding the recommended level (21, 42). See Appendix A. During treatment of mild DCS it is recommended not to exceed a UPTD of 615 and for more severe cases not to exceed 1424 (42). Using the standard table 6 based on calculations a total UPTD of 645.65 would be reached, well below the oxygen toxicity level. Additional time would be available if needed (42).

Another factor associated with DCS that requires therapy is hemo-concentration (21, 17). While it is not known if this occurs as a result of, or is a contributing factor to, DCS; but studies have shown it to exist in a significant number of cases (37, 7). Because of this, fluid therapy is recommended prior to HBO and perhaps during, to reduce red blood cell sludging that may occur interfering with O_2 transferred to tissues (21, 17). I.V. isotonic fluids, Lactated Ringer's Solution, or, in more severe cases, a colloid solution such as low molecular weight dextran is recommended (37). Corticosteroids

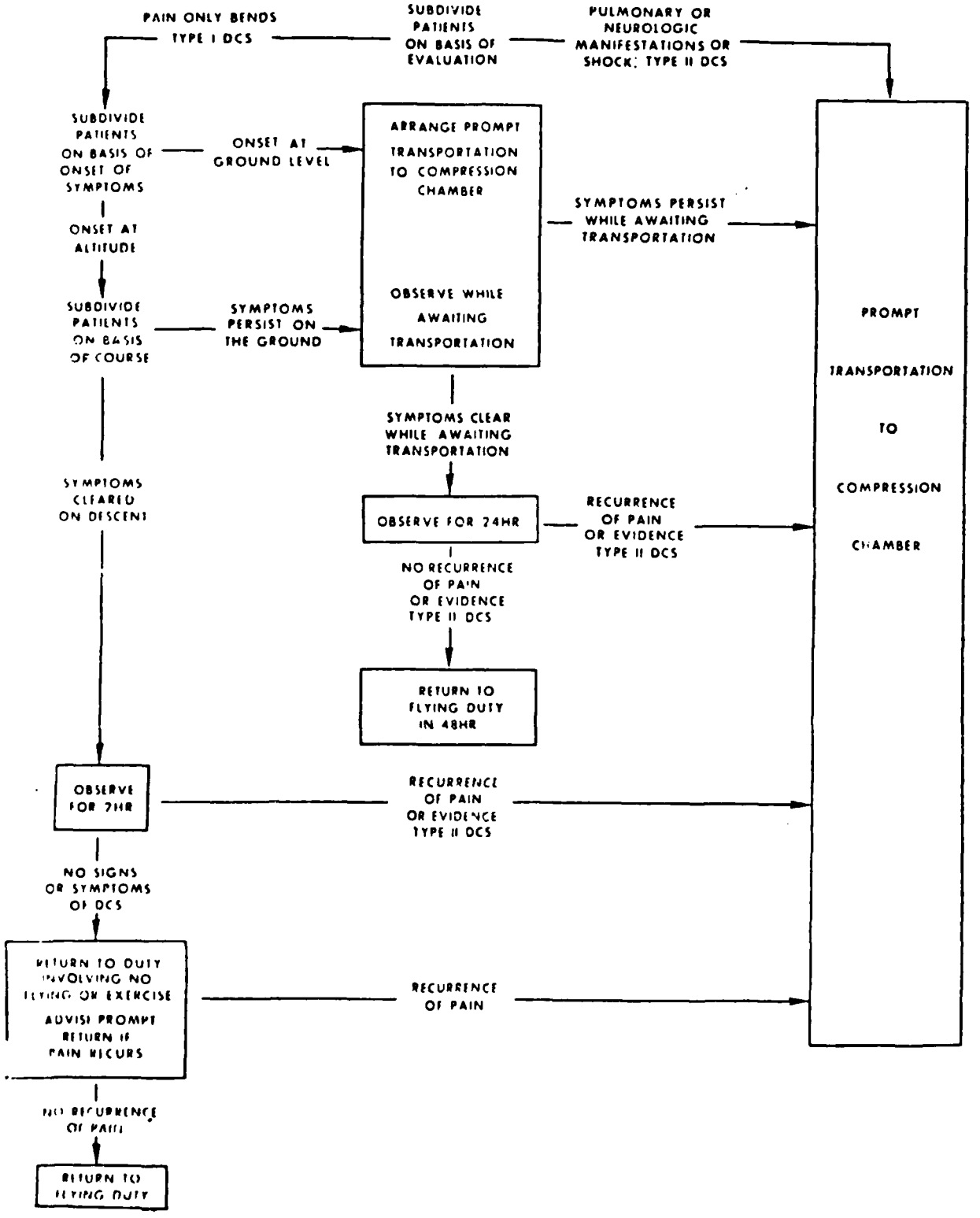
may be used as initial or primary therapy prior to HBO in cases with suspected cerebral edema (7, 37). The use of heparin, aspirin, and other anticoagulants have been evaluated but further studies are needed to determine efficacy (37).

Prompt institution of compression therapy is essential to the favorable outcome of cases of DCS. If any symptom of DCS develops at altitude, immediate descent to ground level while breathing 100% O_2 is imperative (37). The majority of symptoms of DCS developing at altitude will subside during or upon reaching ground level (7, 33, 34). Those whose symptoms were of type I or bends pain only but subsided upon reaching ground level should be observed at rest for 2 hours. If symptom free after 2 hours, returning to duties not involving flying or altitude exposure may be considered. Flying or altitude should not be resumed for 24 hours. If the symptoms recur then the patient should be transported and treated with compression therapy (7, 37). Type II DCS developing at altitude or after return to ground level should receive prompt HBO (5, 6, 7).

Frequently when DCS develops landing at a site where HBO is available is not always feasible. When this occurs several steps can be taken to provide primary care and to effect a transfer to a facility with HBO capabilities (37). Figure #3 is a flow sheet adopted by the U.S. Air Force and recommended for use by physicians dealing with DCS. While waiting transport to the appropriate facility 100% O_2 should be maintained by a snug fitting O_2 mask and appropriate I.V. fluids begun (7, 37). The decision to transport and treat can easily be determined by the flow sheet or by consulting with a flight surgeon familiar with

FIGURE 3

FLOW CHART FOR DISPOSITION OF PATIENTS WITH DECOMPRESSION SICKNESS



the diagnosis and treatment of DCS. Should any doubt arise as to treating with HBO or not, one should always take all the steps necessary to treat and without delay.

Decompression Sickness in Space Operations: Since space operations involve exposure to pressure differentials of altitude and potentially, greater differentials than aviators, the possibility of astronauts experiencing DCS is obvious. While the Shuttle-orbiter space craft will be pressurized to ground level, that is 14.7 psi, astronauts during EVA will be experiencing suit pressures of 3.5 - 3.7 psi or the equivalent of ascending from ground level to 33,000 - 35,000 feet (41).

The cabin atmosphere will consist of approximately 80% nitrogen and 20% oxygen. The cabin pressure being 14.7 psi will provide combined partial pressures of these gases equating, or nearly so, earth's sea level atmosphere. This condition plus those physical stresses and presure differentials that will be involved with the extra vehicular activities (EVA) will provide many of the factors frequently associated with the occurrence of DCS. The physical exercise will exceed that usually experienced by crew members of aircraft under normal operations.

While no experience is available to determine the incidence of DCS under these conditions some studies have been conducted using the proposed environmental profiles of the Shuttle-orbiter and associated EVA's, and would indicate an expected incidence similar to that experienced with the U.S. Air Force air and chamber operations (2). A working session on altitude decompression sickness was conducted at the Johnson Space Center, Houston, Texas, on February 8, 1979, and at that time current data on crew protection were reviewed. The major concern, while

other aspects were considered, was the prebreathe or the nitrogen wash-out period (2, 22, 41). The present Shuttle EVA profile will require the initial prebreathe period to begin without the complete pressure suit at cabin pressure of 14.7 psi. After a designated period, not yet decided upon, the astronaut will then don the upper half of his space suit (EMU). This will require the interruption of oxygen breathing, and presents the problem of renitrogenation (5, 29, 36, 39). Cooke in his studies in 1975 investigated the effects of the interrupted pre-breathe periods at 1, 2, and 3 hours for 5 and 10 minutes in duration and found 10% limb bends upon exposure to altitude (41). The astronaut chamber training for Apollo and Skylab was discussed at the meeting and it was pointed out that a minimum of 3 hours prebreathe was required before being depressurized to suit pressure. One case of bends and possibly two occurred during this training and was attributed to breaks in the prebreathe period (41). Adams (2) presented his finding of a study to determine the compensating "add on" times following a break in the denitrogenation process and has subsequently recommended a 34 minute add on time after a one minute break, the time now proposed to don the suit. See Figure 4. During his studies, Adams (1, 2) used Doppler Bubble detector placed over the pulmonary artery of his subject and from past similar experiments was able to predict the onset of bends by the degree of bubble formation detected. Of 7 subjects exposed to the Shuttle EVA profile using the one minute interruption after 3 hours denitrogenation then another 34 minute add on time, one developed bends and 2 others developed bubbles without symptoms. See Figure 5. Adams pointed out that preliminary studies, using just the 3 hour prebreathe,

FIGURE 4

RESULTS OF A STUDY TO DETERMINE "ADD ON" TIMES AFTER INTERRUPTED DENITROGENATION PROCESS

BREAK DURATION (MIN)	BREATHING TIME (HRS) 0.5	1.0	2.0	3.0
	ADD-ON TIME (MIN)			
1.0	9.0 ± 0.9[2] n[1] = 3	17.4 ± 6.9 n = 5	22.2 ± 10.2 n = 6	34.0 ± 8.7 n = 3
2.5	11.7 ± 1.6 n = 3	17.3 ± 6.8 n = 4	27.0 ± 8.1 n = 5	39.5 ± 7.3 n = 7
5.0	14.0 ± 4.0 n = 3	24.9 ± 7.7 n = 4	39.7 ± 7.7 n = 5	52.2 ± 19.9 n = 5
10.0	16.9 ± 2.8 n = 4	32.3 ± 6.8 n = 3	46.3 ± 1.1 n = 2	

1. n represents the number of subjects in each study.

2. The add-on times plus the standard deviation were determined by that time required to return to the same expired nitrogen level that existed just prior to the break in the pre-breathe.

Note: Nitrogen content of expired air was measured by a mass spectrometer before and after each prebreathe period with 100% O_2.

FIGURE 5

EXPERIMENTAL (PRELIMINARY RESULTS)

RESULTS OF A STUDY USING 34 MINUTE ADD ON TIME AFTER ONE MINUTE INTERRUPTION OF DENITROGENATION

BENDS & BUBBLE FORMATION AFTER 3 HOUR DENITROGENATION WITH 1-MIN AIR INTERRUPTION PLUS 34 MIN ADDITIONAL DENITROGENATION, BREATHING GAS AT 33,000' WAS 92% O_2 + 8% N_2

SUBJECT NUMBER	MAXIMUM BUBBLE GRADE	STAGES OF GRADE	TIME OF ONSET OF GRADE OR BENDS	SITE
1	2	2-1	37-76	Left & Right Arms
2	1	None	54	Left Leg
3	0	-	-	-
4	0	-	-	-
5	4	4 Bends	69-77	Right Knee
6	Not Exposed			
7	Not Exposed			
8	4	2-4	37-47	Right Leg
9	Not Exposed			
10	Not Exposed			
11	Not Exposed			
12	0	-	-	-
13	Not Exposed			

Note: Study is to determine the incidence of bends and/or related bubble formation so as to quantify the appropriate "add on" time after an interruption of denitrogenation occurs. Bubble grade is based on the intensity of bubble formation as detected by a Doppler placed over the pulmonary artery. Graded 1 thru 4. Site indicates that part of the anatomy exercised prior to bubble detection or bends onset (1, 2, 41).

had resulted in 5 cases of limb bends out of 13 trials. See Figure 6. Three out of 17 inside observers developed limb bends during these runs. Since the O_2 delivered during the Shuttle mission will not be 100% oxygen, the subjects were required to prebreathe 95% O_2 and 92% O_2 while at altitude. This would represent the worst possible situation in the variations of percentages of O_2 delivered during the flight (41). Adams plans to continue these exposure studies (1).

A preparation time involving the doning of the EMU, 3 hours denitrogenation, plus 34 minutes of additional prebreathe after the one minute break, has been proposed (5, 29, 39). There remains a desire, due to time allotments and the discomfort of the denitrogenation process, on the part of Crew Systems Division at NASA JSC, to denitrogenate for just 3 hours (41). Other investigators feel the 4 hour prebreathe period would be more acceptable (1, 7, 10, 33, 34). Studies are continuing, and more data will be presented.

Members at them meeting concluded that more work needed to be done based on their findings and felt there was a need to develop an appropriate treatment plan for DCS aboard the Shuttle-orbiter (9, 41).

A similar concern occurred with the Apollo flights when McIver felt that exposures to the pressure differentials that were planned for the Appollo missions would present a significant incidence of DCS, and a treatment protocol was needed. The environmental profiles of the Apollo missions were somewhat different from the Shuttle environment. The cabin pressure was 5 psi with a 60% N_2 and 40% O_2 initially at lift off. The occurrence of DCS was possible before or after orbit due to the depressurization from ground level to cabin level. The

FIGURE 6

CONTROL

RESULTS OF A STUDY TO ASSOCIATE BUBBLE FORMATION WITH THE DEVELOPMENT OF BANDS

BENDS & BUBBLE FORMATION AFTER 3 HOUR DENITROGENATION WITHOUT BREAK AND AT AN ALTITUDE OF 33,000'

WHILE BREATHING 92% O_2 + 8% N_2

SUBJECT NUMBER	MAXIMUM BUBBLE GRADE	STAGES OF GRADE	TIME (IN MIN.) OF ONSET OF EACH GRADE OR BENDS	SITE
1	0	-	-	-
2	4	1-3-4	38-46-61	Left Leg
3	0	-	-	-
4	0	-	-	-
5	4	1-4	68-100	Right Arm
6	3	2-3 Bends	47-64-77	Right & Left Leg
7	0	-	-	-
8	4	3-4 Bends	17-25-93	Right Knee
9	0	Bends	103	Right Knee
10	3	3 Bends	50-79	Right Knee
11	2	2 Bends	92-95	Right Knee
12	0	-	-	-
13	0	-	-	-

Note: Study was to determine relationship of bubble grade to the development of decompression sickness. Bubble grade is based on the intensity of bubble formation as detected by a Doppler placed over the pulmonary artery. Graded 1 thru 4. Site indicates that part of the anatomy exercised prior to bubble detection of bends onset (1,2, 41).

cabin gas mixture over a period of several days gradually shifted to pure O_2. After passing the phase of depressurization the astronaut was next faced with the exposure to 3.5 psi suit pressure of EVA, another potential for the development of DCS. Realizing the essential inability to abort the mission and de-orbit to obtain HBO, McIver conducted studies by treating DCS with maximum pressures available in the Apollo space craft. The suit pressure had a maximum limit of 4.5 psi and that combined with the cabin pressure would provide 9.5 psi absolute (psia). By using that method and providing 100% O_2 for 4 hours McIver felt that 75% or more of cases of DCS could be treated adequately by this method (29, 31, 33).

Treatment Facility For The Shuttle-Orbiter: The environmental profile design of the Shuttle will provide protection to the crew from DCS during lift off and during normal internal operation of the missions. With the exception of an extremely rare instance of an accidental decompression of the cabin, DCS will not present a problem to the crew members operating in the shuttle craft. However, the astronaut performing duties during extra vehicular activities (EVA) is exposed to risk of DCS, and in view of the pressure differential 14.7 psi to 3.7 psi, his risk may be greater than those crew members of the Apollo spacecraft (1, 5, 10). In addition to the previously discussed predisposing factor, the astronaut involved with EVA's during Shuttle flights may be faced with any of the following (9):

1. Interrupted denitrogenation schedule
2. Inadequate nitrogen purge from the space suit
3. A contingency EVA with an inadequate prebreathe period

4. A strenuous or plolonged EVA in a susceptible individual

These possible situations combined with an awareness of 10% incidence of DCS in general and with studies, using the Shuttle profiles, indicating possibly a greater incidence, the need for a treatment method and a treatment schedule was obvious (1, 2, 41). Such a facility would have to meet the weight and space constraints of the Shuttle craft but be capable of providing adequate treatment for enough of the cases as to narrow the risk of failure within an acceptable margin of safety (9).

Utilization of the suit pressure, which would provide a maximum of 4.5 psi, combined with the cabin pressure of 14.7 psi would provide a total pressure of 19.2 psi absolute. This was considered since it would provide 10 psi over McIver's treatment procedure for the Apollo. That pressure, combined with 100% O_2 for 4 hours would, based on McIver's data, treat the majority of cases not responding by the return cabin pressure from the EVA. But it was felt that the 4.5 psi of the suit pressure would not improve the management over just remaining at cabin altitude for 4 hours with 100% O_2. Any appreciable improvement over that treatment would require an overpressure at or near the standard treatment table's requirement of 41.1 psia or 60 feet of sea water pressure (31, 34, 33, 30).

The altitude pressure bag designed and tested for use by the Air Force in 1943 held some promise and was investigated (16). The bag was equipped with communication outlets, electrically heated suit circuits, and a demand type oxygen regulator. Unfortunately, it was designed to provide only 4 psi above ambient pressure, less than the

EMU emergency pressure. Interestingly, however, studies using the bag in DCS did reveal relief of bends immediately upon pressurization. The bends pain returned, however, if pressure was reduced while still at altitude (15).

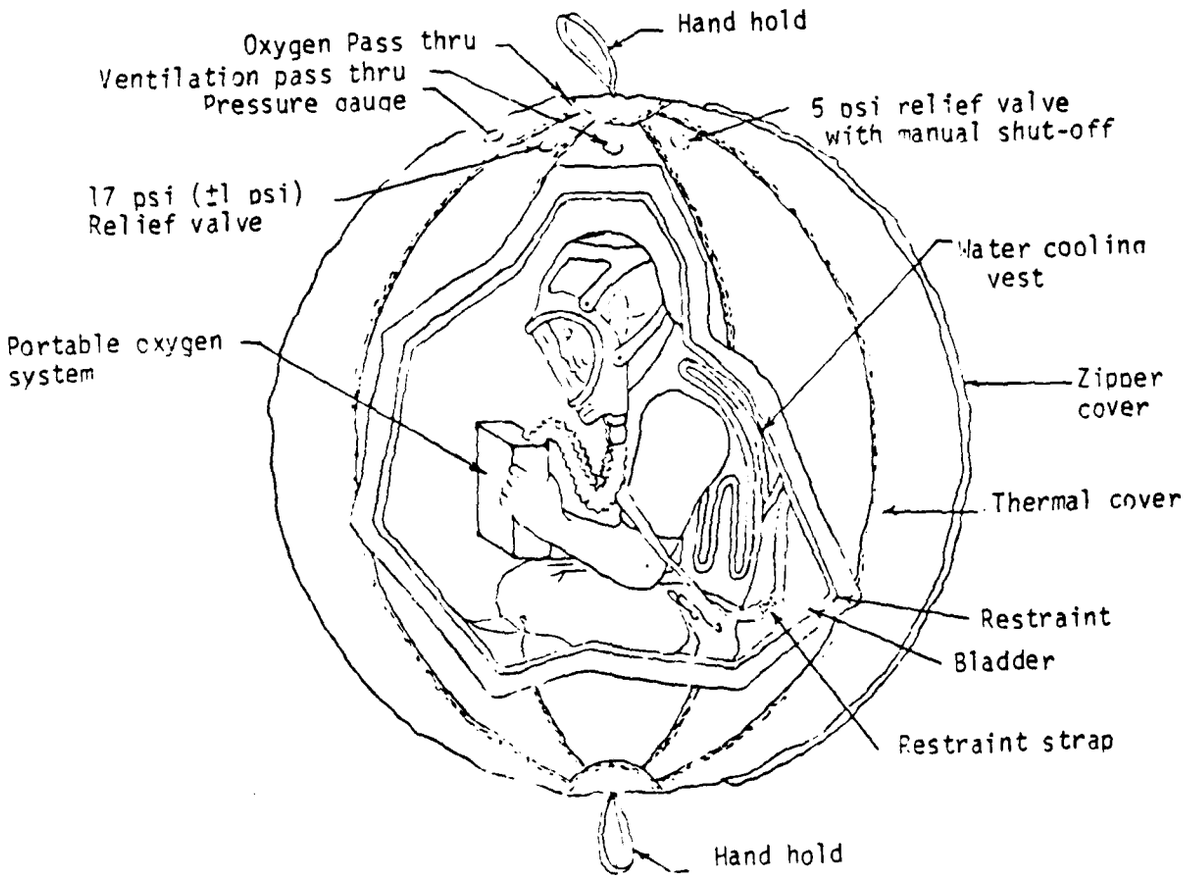
Other portable hyperbaric chambers were also investigated and found to be prohibitive as to weight and size (10).

The personnel rescue sphere (PRS), a device that is designed for the transfer of crew members from a disabled orbiter to a rescue vehicle, was examined as a possibility (10).

The PRS (35) is a 3-layer collapsible sphere 34 inches (1 meter) in diameter and weighing 11 pounds. See Figure 7. The inner lining is constructed of an airtight polyurethane impregnated nylon material. That layer is covered with the re-enforcement layer of Kevlar material manufactured by DuPont with a tensile strength of 92.2 psi. These two structures are then covered with a protective thermal layer of anti-micromeorite material. The sphere is presently designed for pressures of 5 psi over ambient pressure with a safety margin of 7.5 psi above that, giving a total maximum of 12.5 psi above ambient. It is equipped with 2 relief valves, a 5 psi valve that can be closed and a 17 psi relief valve. Medical monitoring and communication outlets are provided as is O_2 supply and compressed air supply connectors. There is a viewing port or window and the method of entry is through an air tight polyurethane ziplock in the bladder layer and zipper at the heat shield layer. Hand holds are provided in the interior as well as the exterior (35).

While the PRS is designed specifically as a pressure chamber and also offers the advantage of already being scheduled to be aboard the Shuttle, several disadvantages to its use for treatment were

FIGURE 7



PERSONNEL RESCUE ENCLOSURE/RESCUE CONFIGURATION

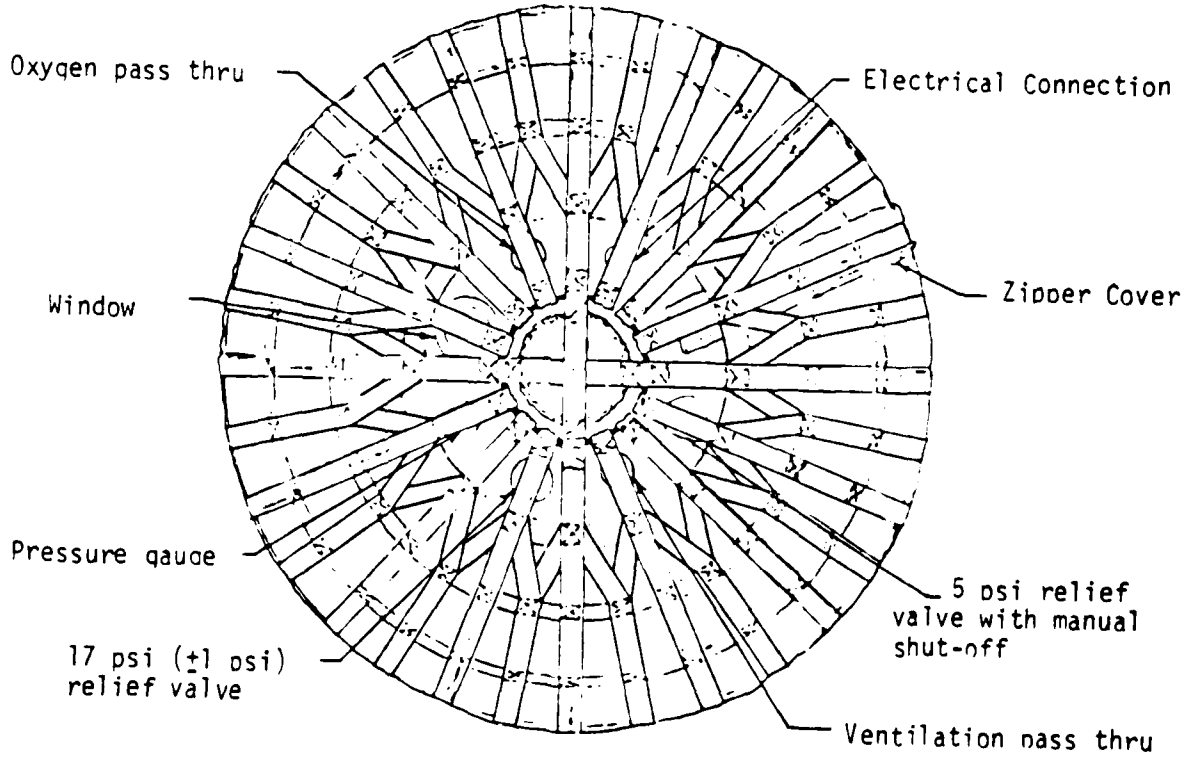
apparent. The size will only provide a 34 inch diameter space for treatment and the present engineering design would not allow pressure in excess of 5 psi above ambient. The pressure restrictions are based on the weakness at seamlines and the viewing window or port (35, 9).

Recent studies at NASA/JSC using the 0 gravity aircraft revealed the sphere to be relatively comfortable with adequate capability to move all limbs and to assume a comfortable position during weightlessness. The size of the sphere and position assumed in the sphere does not appear to offer any interference with normal body functions and physiology, particularly in a weightless state (35).

The engineers of the Crew Protection Branch at NASA/JSC has, based on recommendations, designed a net or exoskeleton of Kevlar, the same restraining material of the middle layer of the sphere (35). The net is made of 2 inch strips of Kevlar sewn 3 inches apart. It will cover the entire sphere re-enforcing the seamlines and the port structure. See Figure 8. With this in place the pressure can be increased to 17±1 psi above ambient with a safety margin of 25.5±1 psi above ambient (35).

This pressure capability combined with a cabin pressure of 14.7 psi will provide a hyperbaric chamber with a capability of 31.7 psi absolute or 2.2 atmospheres. Pressurization of the sphere will be provided by a 24 volt electrically operated aircraft air compressor. Cabin ambient air will be compressed and delivered at a rate of 6 cubic feet per minute (cfm) through the ventilation pass through. The 5 psi relief valve will be closed and the 17±1 psi valve will be open. This open loop system will provide air at cabin temperature and will

FIGURE 8



TOP VIEW

REINFORCED CONFIGURATION

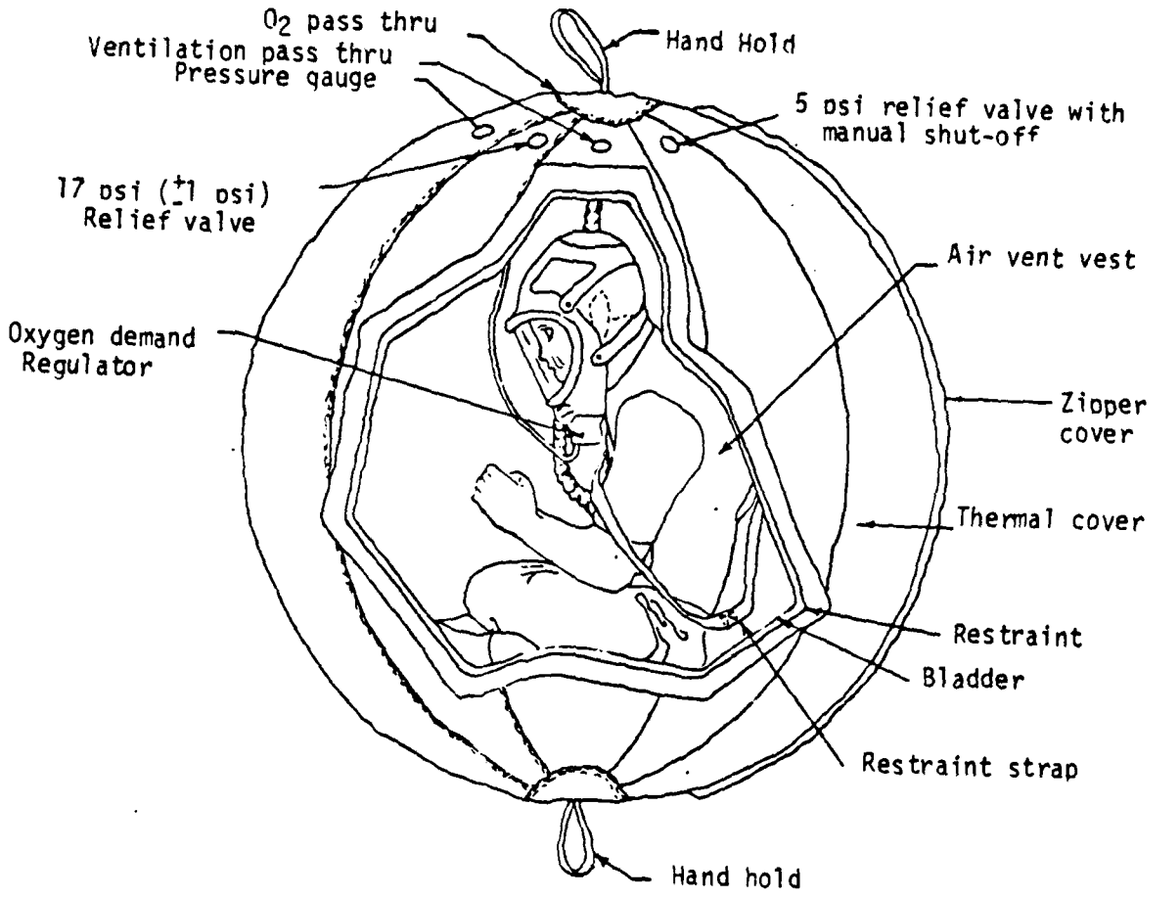
ventilate off the expired gases within (35). The delivery of 6 cubic feet per minute (cfm) of ambient air will handle 650 BTU's of generated heat per hour (40). The oxygen will be delivered by a snug fitting firefighter's mask that can be removed as necessary by the wearer (35).

Crew members placed in the sphere will be provided a perforated vest for cooling while enclosed. See Figure 9. An individual under the normal physical activity expected within the sphere will generate approximately 400 BTU per hour. The 6 cubic feet/minute (cfm) of air should provide adequate cooling (40). Communication can be maintained and monitoring of heart rate, respiratory rate, blood pressure, and temperature will be accomplished through the biomedical monitoring systems provided.

Since the modification design will not provide pressure capabilities safety in excess of 31.7 psi or 2.2 Ata, the standard treatment tables 5 and 6 cannot be used; therefore, a treatment schedule must be designed with these modifications.

Prior to 1965 DCS cases treated with hyperbaric therapy were treated with compressed air to pressures of 6 Ata. While the results were good with compressed air therapy, the use of oxygen with lowered pressures, using tables 5 and 6 (see Figures 1 and 2), has improved the management of the disease (7). It is relatively well accepted that oxygen and increased barometric pressure both play a role in the resolution of bubbles in DCS (3, 7, 8, 10, 18, 37). Both these factors probably also contribute greatly in re-oxygenation of hypoxic tissues with subsequent reduction of ischemia and edema (7, 8, 37). Most recent studies of the treatment of DCS have involved the use of 2.8

FIGURE 9



PERSONNEL RESCUE ENCLOSURE/TREATMENT CONFIGURATION

Ata of tables 5 and 6 (7, 8, 17), compressed air at 6.0 Ata (42), and those studies in simulated space flight using 9.5 psi or 0.65 Ata (29, 33, 34). The two former methods have had excellent results with numerous successful cases recorded. While McIver's studies represent a few cases, the therapy at 9.5 psi appears to have the capability of treating the majority of cases of DCS that could occur at altitude (33, 34). It is further recognized that more than 90% of cases (5, 6, 7, 10, 34) occurring at altitude will respond by returning to sea level pressure of 14.7 psi and breathing 100% O_2 by mask. Since further compression would tend to reduce bubble size (17, 23) and, with 100% O_2, increase the O_2 partial pressure of blood and tissues (18), then the additional 17 psi of pressure should be an important adjunct to ground level pressure treatment. While at 2.2 Ata the patient would be provided intermittant 100% O_2. The standard tables 5 and 6 call for O_2 for 20 minutes then air breathing for 5 minutes with step by step decreasing depths for varying periods depending on the table being used. See Figure 1 and 2. The purpose of 2 tables with varying periods of treatment is to assure the maximum treatment for the type of DCS being managed without over treating and needlessly exposing the patient to oxygen toxicity. Table 5 is designed for the treatment of minor or type I DCS and by calculating the UPTD (unit pulmonary toxic dose), table 5 would produce a UPTD of 336. Table 6, recommended for major or type II DCS, would produce a UPTD of approximately 646. With a UPTD of 1425 being selected and recommended as the upper limit for theraputic O_2 exposure, both tables proved adequate therapy for DCS and remain well below the level of O_2 toxicity (42).

Inflight Treatment of Decompression Sickness Aboard The Shuttle-Orbiter: The proposed treatment table using the pressure atainable in the PRS will call for a descent to 2.2 Ata (31.7 psi or 47 FSW) over a period of 10 minutes while breathing 100% O_2 then maintain the 100% O_2 for 30 minutes before interrupting the O_2 to breathe ambient air for 5 minutes. 100% O_2 is resumed for 30 minutes then again interrupted by 5 minutes of air. This process is continued for a total of 150 minutes at which time a slow ascent to ground level pressure is accomplished over a period of 30 minutes while breathing 100% O_2. See Figure 10. The total treatment period will require 180 minutes assuring an adequate O_2 supply from the orbitor supply system and will only produce a UPTD of slightly less than 443.2.

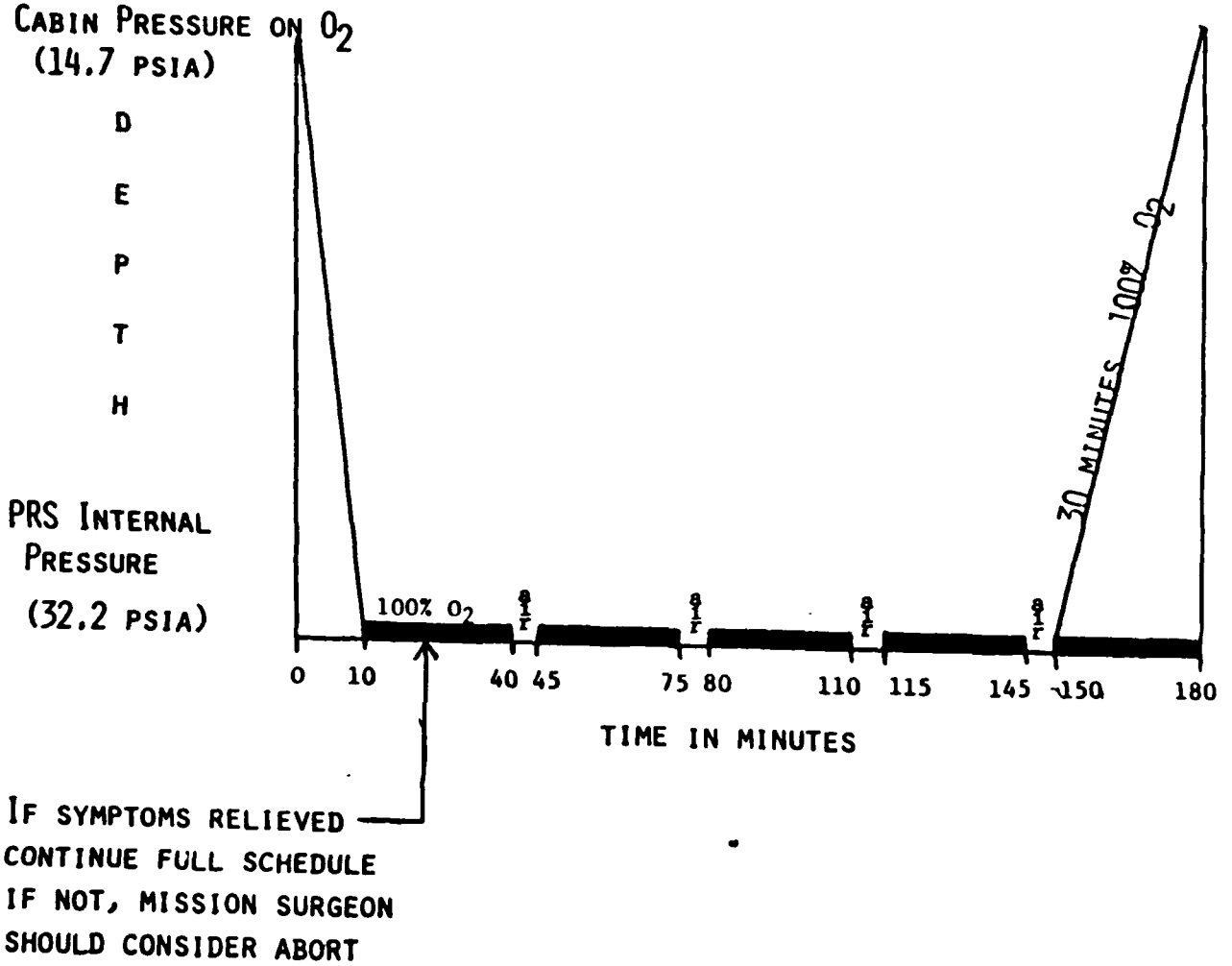
It is felt by several authorities on DCS the proposed treatment plan will provide adequate therapy for those cases of DCS one can expect during space flight missions with only rare exception. The extremely rare or difficult case could be maintained or stabilized in the PRS until decisions to abort the mission could be made (1, 5, 10, 32).

Based on the information available in the literature concerning the management of decompression sickness and the relative inaccessability to an appropriate hyperbaric treatment facility during orbital flight, the following treatment protocol, using the modified PRS, is recommended:

1. Any crew member developing symptoms of DCS, type I or type II, should return to cabin pressure immediately, maintaining 100% O_2 during descent.
2. Those individuals with type I symptoms, pain only bends or cutaneous manifestations, whose symptoms subside during

FIGURE 10

PROPOSED TREATMENT SCHEDULE USING THE MODIFIED PRS



descent to cabin pressure should be observed at rest for for 4 hours. If symptom free throughout this period, then minimal activity may be resumed for 24 hours. EVA's should not be attempted for 48 hours.

3. If symptoms do not subside or if they recur at cabin pressure after resolution, then hyperbaric oxygen treatment should be initiated.
4. Anyone developing type II symptoms should be treated according to the treatment schedule.
5. While the crew surgeon at mission control will be assessing the situation and making decisions based on the severity of the condition, it is recommended that should symptoms not subside after 15 minutes of HBO treatment, a decision to abort should be considered.

In view of the frequently associated hemoconcentration (7, 17, 21, 37), fluid therapy should be added to the treatment regimen. It is recommended that approximately 500 cc of an electrolytically balanced formula be given orally prior to entering the PRS, and an additional 1000 cc of the fluid to be taken aboard, with 500 cc being taken at 90 minutes and 250 cc to 500 cc taken just prior to ascent (5, 10). A standard urine collection bag should be taken aboard and used as necessary.

Proposed studies involving the modified PRS: While it is felt by several authorities in the field of hyperbaric medicine that the proposed treatment protocol will provide adequate therapy for DCS occurring aboard the Shuttle-orbiter (5, 10, 32), several studies should

be conducted to determine, from collected data, the efficacy and feasibility of such a procedure. The following studies have been proposed.

1. Testing pressure limits of the PRS.
2. Testing the proposed treatment schedule by treating DCS in a standard chamber using the PRS, pressures, and the proposed treatment table.
3. Establish a physical tolerance index for an individual confined to the PRS.
4. Determine any interference factors that could occur as a result of the physical configuration assumed in the PRS.
5. Fluid and electrolyte studies in decompression sickness to establish standard tables for fluid and electrolyte replacement during treatment.

Conclusion: The increasing proficiency of technology continues to provide man with the mechanism to extend himself into environments foreign to his tolerance. When this occurs, his physiology will respond to make him more tolerant or will react in such a manner as to limit his presence in that environment. The notion that man is limited to anything frequently becomes a stimulus for innovation to lift the barriers. This action is not always without hazard so it becomes necessary for survival to avert peril. While prevention of those hazards would seem the most logical approach to protecting man, and indeed it is, hazards do not always become apparent until the trauma associated with them occurs.

Altitude decompression sickness has not posed a serious problem to aviation and space programs, but it has occurred often enough to require preventive measures and methods for its treatment should it

occur. Many of the predisposing factors are well known and by avoiding or eliminating these factors the incidence of DCS remains low. With the advent of space missions, some of the predisposing factors were augmented such as greater pressure differentials, more physical activity while exposed to lower pressures and longer duration of exposure. In spite of these situations to date only one unconfirmed case of DCS has occurred during the U.S. Space Missions. This excellent record can be attributed to an acute awareness of the factors predisposing to the disease, adequate prebreathe periods prior to exposure, excellent physical condition of the astronauts, and a relatively small number of exposures to the reduced pressure environments. However, preliminary studies using the Shuttle environmental and EVA profiles suggest a potential increase in incidence of DCS for those missions. The Shuttle era will bring new approaches to the space flights and because of its design will require many more and longer EVA's. As we pregress into the program, more individuals will have the opportunity to be involved, many requiring only a class III flying physical, hence, more susceptible people. A great deal of work is being done to determine adequate nitrogen washouts on an individual basis, determining susceptability level and exploring ways to detect the disease before symptoms develop. Newer treatment methods are also being explored, such as simple aspirin reducing the cohesiveness of platelets to retard emboli formation and over hydration with fluid and electrolytes.

While progress is being made in these areas, the condition will still occur and procedures to control or treat the disease must be present. While this proposed treatment facility for the Shuttle-orbiter

is by no means intended to become a standard for future missions, the capability does offer all these factors that can be made available with the present limitations. The treatment schedule does not meet all the standard requirements, but the physical factors involved, when compared with other techniques, show promise of providing the most adequate capability available at this time. Future testing will, indeed, find need for further modification but these modifications combined with the data collected during the testing will be invaluable information to be applied to the management of decompression sickness in future missions.

Epilogue: During a symposium on decompression sickness (41) conducted on April 12, 1979, at the Johnson Space Center, the proposed treatment facility for the Shuttle-orbiter was presented to members of NASA Staff. The proposal was received favorably and a study protocol is now being prepared to begin testing during the summer of 1979.

APPENDIX A

A Simplified Arithmetic Method for Calculating UPTD

It can be seen from the formula for calculating UPTD that at any constant PO_2 the UPTD is a linear function of the time of exposure to that PO_2. That is the formula:

$$UPTD = t \left(\frac{0.5}{P-0.5} \right)^{\frac{1}{-1.2}}$$

reduces to:

$$UPTD = k_p \, t$$

when P is held constant.

At any PO_2, then, there exists a factor, k_p, which when multiplied by the time of exposure to that PO_2 yields the UPTD for that exposure.

$$k_p = \left(\frac{0.5}{P-0.5} \right)^{\frac{1}{-1.2}}$$

The table on Appendix B is a list of these k_p factors for each 0.1 Ata PO_2 from 0.5 Ata to 5 Ata. To calculate the UPTD for a given exposure:

a. Convert the partial pressure of oxygen breathed at each depth to PO_2 in atmospheres.

b. Select the corresponding k_p from table on Appendix B.

c. Multiply the time of exposure at that PO_2 by the corresponding k_p to get the UPTD for that depth.

d. Add the UPTD's for each PO_2 in the complete exposure

together to get the total UPTD for the exposure.

Example:

If we wish to calculate the UPTD incurred by a treatment Table 6, we can construct the following table:

Time (minutes)	O_2 (%)	Depth (feet)	PO_2 (Ata)	UPTD (minutes)
60	100	60	2.8	214.02
15	21	60	.6	3.92
120 (60 + 60)	100	30	1.9	283.02
30	21	30	.4	0
30	100	45	2.4	91.26
30	100	15	1.5	53.45
		Total UPTD - - - - -		645.67 = 646

APPENDIX B

PO_2	k_p
.50	.00
.60	.26
.70	.47
.80	.65
.90	.83
1.00 --	-- 1.00
1.10	1.16
1.20	1.32
1.30	1.48
1.40	1.63
1.50 --	-- 1.78
1.60	1.93
1.70	2.07
1.80	2.22
1.90	2.36
2.00 --	-- 2.50
2.10	2.64
2.20	2.77
2.30	2.91
2.40	3.04
2.50 --	-- 3.17
2.60	3.31
2.70	3.44
2.80	3.57
2.90	3.70
3.00 --	-- 3.82
3.10	3.95
3.20	4.08
3.30	4.20
3.40	4.33
3.50 --	-- 4.45
3.60	4.57
3.70	4.70
3.80	4.82
3.90	4.94
4.00 --	-- 5.06
4.10	5.18
4.20	5.30
4.30	5.42
4.40	5.54
4.50 --	-- 5.66
4.60	5.77
4.70	5.89
4.80	6.01
4.90	6.21
5.00	6.24

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VITA

Kenneth R. Hart was born in Columbus, Kansas, on March 8, 1936, the son of Fern and Leslie Hart. He received his high school training at the Cherokee County Community High School in Columbus, Kansas, and in 1953 entered the U.S. Navy and served 4 years as a hospital corpsman. After his discharge, Dr. Hart entered Pittsburg State University at Pittsburg, Kansas as a premedical student. He completed his premedical training and was accepted at the Kansas City College of Osteopathic Medicine for his training in Medicine in 1961. In 1965 he received his medical degree and began a one-year rotating internship at Bashline Memorial Hospital, Grove City, Pennsylvania. Upon completion of the internship in 1966 Dr. Hart entered clinical practice as a family physician in Mercer County, Pennsylvania. In 1967 he was commissioned as a first lieutenant in the U.S. Air Force Medical Corps and graduated in December of 1967 from the School of Aerospace Medicine as a Flight Surgeon. He was assigned to the 910th TAC Clinic, 910th Special Operations Group, Youngstown, Ohio where he served as Chief of Aerospace Medicine and Clinic Commander. In 1973 he was promoted to major and reassigned to the U.S. Air Force Hospital, Patrick Air Force Base, Florida and Served as the Chief of Aerospace Medicine for the base and Cape Canaveral Air Force Station. During that assignment he was awarded the Meritorious Service Medal for his contributions to Aerospace Medicine by his service in support of the base and flight operations for Skylab II and Skylab III missions. He was promoted to lieutenant colonel in

1974 and in 1975 assigned to the U.S. Air Force Hospital, Mountain Home, Idaho, as the Hospital Commander and Director of Base Medical Services. In 1978 he was again awarded the Meritorious Service Medal with an Oak Cluster for his contributions to Aerospace Medicine. Colonel Hart began his training at the University of Texas School of Public Health in 1978 as Phase I of his residency in Aerospace Medicine.

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