AL-SR-1992-0005

AD-A257 612

ARMSTRONG LABORATORY

THE PROCEEDINGS OF THE 1990 HYPOBARIC DECOMPRESSION SICKNESS WORKSHOP

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Edited by

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June 1992

Final Special Report for Period 16-18 October 1990

Approved for public release; distribution is unlimited.

92-29697

AIR FORCE SYSTEMS COMMAND
BROOKS AIR FORCE BASE, TEXAS 78235-5000

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The Office of Public Affairs has reviewed this report, and it is releasable to the National Technical Information Service, where it will be available to the general public, including foreign nationals.

This report has been reviewed and is approved for publication.

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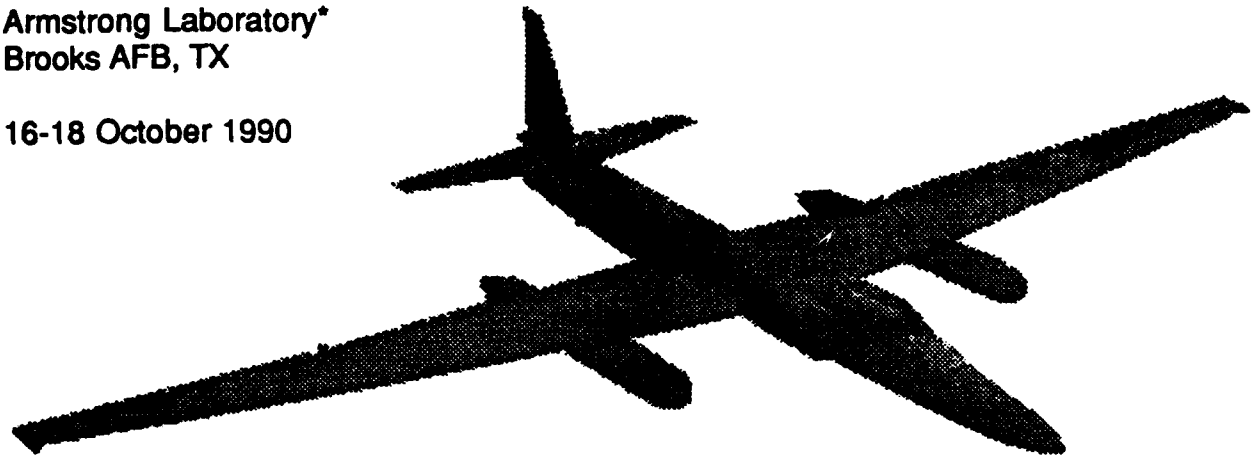
HYPOBARIC DECOMPRESSION SICKNESS

Proceedings of a Workshop
held at

Armstrong Laboratory*
Brooks AFB, TX

16-18 October 1990



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USAF Armstrong Laboratory
NASA Johnson Space Center
USAF Office of Scientific Research

*formerly the USAF School of Aerospace Medicine

Accession For
NTIS GRA&I ☒
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Justification
By
Distribution/
Availability Codes
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A-1

REPORT DOCUMENTATION PAGE

Form Approved
OMB No. 0704-0188

Public reporting burden for this collection of information is estimated to average 1 hour per response, including the time for reviewing instructions, searching existing data sources, gathering and maintaining the data needed, and completing and reviewing the collection of information. Send comments regarding this burden estimate or any other aspect of this collection of information, including suggestions for reducing this burden, to Washington Headquarters Services, Directorate for Information Operations and Reports, 1215 Jefferson Davis Highway, Suite 1204, Arlington, VA 22202-4302, and to the Office of Management and Budget, Paperwork Reduction Project (0704-0188), Washington, DC 20503.

1. AGENCY USE ONLY *(Leave blank)*	2. REPORT DATE	3. REPORT TYPE AND DATES COVERED
	June 1992	Final 16-18 October 1990

4. TITLE AND SUBTITLE

The Proceedings of the 1990 Hypobaric Decompression Sickness Workshop

5. FUNDING NUMBERS

PE - 62202F
PR - 7930
TA - 18
WU - 01

6. AUTHOR(S)

Andrew A. Pilmanis

7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES)

Armstrong Laboratory
Crew Systems Directorate
Crew Technology Division
Brooks Air Force Base, TX 78235-5000

8. PERFORMING ORGANIZATION REPORT NUMBER

AL-SR-1992-0005

9. SPONSORING / MONITORING AGENCY NAME(S) AND ADDRESS(ES)

10. SPONSORING / MONITORING AGENCY REPORT NUMBER

11. SUPPLEMENTARY NOTES

12a. DISTRIBUTION / AVAILABILITY STATEMENT

Approved for public release; distribution is unlimited.

12b. DISTRIBUTION CODE

13. ABSTRACT *(Maximum 200 words)*

Decompression sickness (DCS) is the clinical condition resulting from evolved inert gas bubbles in tissues caused by a reduction of environmental pressure. It is recognized that DCS continues to be an operational limitation in both aviation and space activities. Furthermore, it is expected that the crews of the next generation of military aircraft will be exposed to even higher altitudes.

In order to document the current understanding of altitude decompression sickness and ascertain the operational significance of this disease, a workshop was held at the USAF Armstrong Laboratory (AL) (formerly USAF School of Aerospace Medicine), Brooks Air Force Base, Texas, on 16-18 October 1990. The meeting was sponsored by: (1) USAF AL, (2) NASA Johnson Space Center, and (3) AF Office of Scientific Research, and was attended by over 50 participants representing the Department of Defense (DOD), NASA, and university researchers.

The reported incidence of altitude DCS is likely inaccurate due to potential career consequences. Efforts toward reporting with impunity were recommended. It was emphasized that DCS should be viewed as an occupational illness in the same way we view other physiological responses to environmental stress.

14. SUBJECT TERMS		15. NUMBER OF PAGES
Altitude chamber training flights	Hyper/hypobaric pressure	600
Bends	Nitrogen bubbles	16. PRICE CODE
Decompression sickness	Noninvasive bubble detection	
	Operational DCS incidence	

17. SECURITY CLASSIFICATION OF REPORT	18. SECURITY CLASSIFICATION OF THIS PAGE	19. SECURITY CLASSIFICATION OF ABSTRACT	20. LIMITATION OF ABSTRACT
Unclassified	Unclassified	Unclassified	UL

ACKNOWLEDGMENTS

We are indebted to the USAF Office of Scientific Research, USAF Armstrong Laboratory (formerly the USAF School of Aerospace Medicine), and NASA Johnson Space Center for providing the funding that made this workshop possible.

Special appreciation is extended to Senior Airman Keith Wilkerson, the workshop logistics coordinator, for his tireless enthusiasm and persistent efforts in the organization, execution, and follow-up of the workshop. Airman Wilkerson's professionalism and highly motivated technical assistance were critical to the successful completion of these proceedings.

The editorial assistance of Toniann Derion was invaluable in the document preparation and is greatfully acknowledged. Appreciation is also extended to Capt Terrell E. Scoggins, James T. Webb, and Barbara J. Stegmann for their support and editorial assistance in the preparation of this document.

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PREFACE

Decompression sickness (DCS) is the clinical condition resulting from evolved inert gas bubbles in tissues caused by a reduction of environmental pressure. Originally described in 1670 by Robert Boyle, this disease can occur under both hyper- and hypobaric conditions such as in diving, caisson work, aviation and space operations. Altitude DCS has been studied for over 50 years. During the last two decades, major advances in understanding decompression sickness have been made. The pathophysiology of DCS is still elusive, but has been clarified. Non-invasive bubble detection has provided a semi-objective means of studying DCS in humans. The physics and physiology of gas bubble formation and growth have been extensively studied. There is now more concern over long-term central nervous system (CNS) damage. Modeling of DCS prediction and prevention has been extensively expanded with the advancements in computing capabilities.

An accurate accounting of operational DCS incidence in the USAF does not exist. The total reported number of DCS cases appears to be approximately 100 to 120 cases per year. The vast majority of these cases occur with altitude chamber training flights. However, there is a reluctance to report DCS because of career considerations. Large numbers of highly trained personnel are routinely exposed to altitudes that, in controlled chamber studies, produce high levels of bubbles and bends. Yet reports of DCS from the field are minimal to non-existent. Nevertheless, it is recognized that DCS continues to be an operational limitation in both aviation and space activities. It is expected that the crews of the next generation of military aircraft will be exposed to even higher altitudes than those of today.

In order to document the current understanding of altitude decompression sickness and ascertain the operational significance of this disease, a workshop was held at the USAF Armstrong Laboratory (AL) (formerly USAF School of Aerospace Medicine), Brooks AFB, Texas, on 16 to 18 October 1990. The meeting, sponsored by: (1) USAF AL, (2) NASA Johnson Space Center, and (3) AF Office of Scientific Research, was attended by over 50 participants representing the Department of Defense (DOD), NASA, and university researchers. The objectives of the workshop included:

1. reviewing the current understanding of the pathophysiology of DCS,
2. evaluating existing and proposed options for DCS prediction,
3. defining the problems of decompression in space,
4. documenting the current incidence of DCS in aviation,
5. discussing the "acceptable risk" of altitude DCS, and
6. listing areas of needed DCS research.

This meeting was conceived as a workshop, not a scientific symposium. The emphasis will be on open discussion and the exchange of information. The papers are short and were viewed as reviews of topics rather than original scientific papers. Their purpose was to set a basis for discussion and to update the group on the current thinking in the area of Decompression Sickness. Because the forum resulted in much discussion, not limited to scientific fact only, we tolerated opinions, conjecture, etc. It was a meeting for throwing ideas out on the table.

EXECUTIVE SUMMARY

PATHOPHYSIOLOGY OF DCS

The workshop was opened with a historical perspective of altitude decompression sickness. It was emphasized that although early researchers did excellent work with inadequate equipment, their results should not be ignored. There are specific differences between hypobaric and hyperbaric DCS, but the disease is basically the same in both environments and altitude DCS should be viewed as part of an overall condition resulting from changes in the atmospheric pressure continuum. DCS involves many pathophysiological processes occurring both in parallel and in series, and it follows dose-response characteristics rather than all-or-none thresholds.

The current conceptions on bubble physics and dynamics were reviewed. The concept of bubble nuclei is still unclear, but was defined as "a collection of gas molecules that remain together even without supersaturation." Viscous adhesion in the normal motion of joints can result in sufficient cavitation to cause a "vacuum phenomena," which, in turn, may contribute to DCS bubble precipitation. The DCS bubbles should be viewed as dynamic, rather than static. New bubbles are growing while old bubbles are shrinking. A video tape of in-vivo bubbles in human subjects at altitude as recorded by the latest Echo Imaging technique was shown to the meeting. Clear bubble imaging in the right heart is now possible.

The cardiopulmonary effects of bubbles are currently the focus of research because of the potentially severe consequences of such emboli. Most of the documented altitude DCS deaths are considered the result of severe cardiopulmonary bubbles. Pulmonary vascular resistance and right-sided pressures increase with an increasing load of gas bubbles, which can lead to circulatory collapse and death and can also theoretically cause right-to-left shunting of bubbles resulting in cerebral arterial gas embolism. However, some recent human altitude DCS research in the US Navy did not find evidence of right-to-left shunting in subjects with patent foramen ovale. The concern over the presence of patent foramen ovale in flyers and astronauts may have been exaggerated. More work is needed.

The 19 recorded altitude DCS deaths were reviewed. Except for one, all of these deaths occurred prior to the initiation of routine oxygen prebreathing and hyperbaric therapy. The one exception is a case history published in 1988, and was fatal despite hyperbaric therapy.

Of great concern in the diving field is the recent accumulation of data on chronic CNS pathology linked to diving. Diffuse spinal cord degeneration, often asymptomatic, has been well documented. Damage to the eyes and the brain has also been documented. Comparable studies in the altitude field have not been done. The treatment of altitude DCS with standard hyperbaric oxygen treatment tables is well established and highly successful. The origin of the extensive USAF Hyperbaric Medicine Program was historically linked to the need for altitude DCS therapy

capability. The USAF has treated over 650 cases of altitude DCS with hyperbaric oxygen since 1977.

PREDICTION AND PREVENTION OF ALTITUDE DCS

Decompression modeling was reviewed. The overall objective of decompression tables or computers in the diving field is to prevent or reduce DCS injury. Today, the majority of divers wear decompression computers for real-time decompression guidance. Classic Haldanian diffusion/perfusion techniques used for diving tables are inadequate for altitude decompression risk assessment.

For 50 years, guidelines for safe altitude exposures have been developed by costly time-consuming studies, each specific to a unique exposure scenario. The application of such studies to new operational requirements is very difficult, often requiring new studies or "best guess approximations." Therefore, the development of an altitude decompression computer is long overdue and is currently underway in the USAF. The primary problem facing such a development is the lack of a "standard" altitude decompression algorithm. The model being developed will include Haldanian theory, bubble dynamics, and the application of maximum likelihood statistics. This model will then be incorporated into appropriate hardware and will provide both real-time and predictive DCS risk assessment capability. Models must evolve empirically. Thus, both operational and research DCS databases are required for this development. It was repeatedly pointed out that the operational DCS incidence numbers may be grossly inaccurate due to aircrew and chamber operator reporting problems.

Denitrogenation or prebreathing is standard practice in both aviation and space operations for DCS risk reduction. Prebreathing has been most effective in the reduction of the serious DCS symptoms, less so with bends pain. Prebreathe times vary among operational situations and need more standardization. Hard data on the effect of interrupted prebreathe and on denitrogenation/ renitrogenation processes with repetitive prebreathe/altitude exposure cycles are lacking. Denitrogenation is enhanced by increased temperature, immersion, negative pressure breathing and supine posture. For example, immersion in 37^{o}C water can accelerate denitrogenation by almost 50%. The inverse of these conditions tends to slow denitrogenation.

DECOMPRESSION IN SPACE

The second day of the Workshop began with a discussion of the problems of decompression in space. The history and current practice of NASA's extravehicular activity (EVA) decompression procedures were reviewed. The current Shuttle procedure uses a combination of oxygen prebreathing and 10.2 psia stage decompression based on an "R-value" (ratio of tissue PN_2 over ambient pressure) of 1.65 and the 360 minute half-time tissue compartment. For these conditions, ground-based NASA and USAF research predict an incidence of 23% mild DCS symptoms,

and 5-10% DCS symptoms severe enough to result in a mission abort. However, to date no reported DCS has occurred in the Shuttle program.

This apparent discrepancy may have two possible explanations. From the astronaut perspective, admitting to DCS symptoms is "not exactly a career-enhancing move." Second, weightlessness may improve denitrogenation and reduce DCS. Due to the very large number of projected EVA missions required to construct and maintain Space Station Freedom, there is a NASA recommendation to change the "R-value" to 1.40 resulting in a more conservative decompression schedule. There is also support in the European Space Agency (ESA) for the 1.40 value. The ESA has proposed a 7.3 psia EVA suit.

Because of the potentially severe consequences of DCS in space, hyperbaric treatment capability has been designed into the Space Station. The design calls for a 2.8 ata hyperbaric chamber. This facility would not be limited to DCS; it could also be used to treat air embolism and ebullism.

THE INCIDENCE OF ALTITUDE DCS

In the next session, a series of papers were presented documenting the DCS incidence and DCS reporting problems in the operational environment. Reports were presented on USAF operations including high altitude reconnaissance, high altitude parachuting, flight training in unpressurized aircraft, altitude chamber training operations, and Vietnam War high altitude operations. US Army flight and chamber operations, US Navy flight and chamber operations, RAF experience, and US civilian experience were also covered. The largest number of reported DCS cases occur in DOD altitude chamber training (approximately 120/year). The incidence, however, is small (1-3 cases/1000 exposures). In contrast, the Europeans reported essentially no DCS problems in their chamber training.

In USAF aircraft operations, 18 DCS cases were officially reported in 1989, more than double the number in previous years. Two possible explanations were postulated. A 1989 change in USAF Regulations permitting a waiver process for Type II DCS was thought to have encouraged more reporting. The 1988 published account of an altitude DCS fatality may also have contributed. However, it was emphasized repeatedly that the official reports represent a subset of the unknown true incidence. Despite the regulation change, there is a strong reluctance on the part of both chamber and flight personnel in the USAF to report DCS. There are multiple reasons for this reluctance, but the primary cause is career protection.

Perhaps the single most important conclusion of the workshop was that aviators must be allowed to report DCS with impunity. It was emphasized that DCS should be viewed as an occupational illness in the same way we view other physiological responses to environmental stress, such as hypoxia. Why should an aviator who

responds to decompression in the expected manner be penalized? If the treatment is successful, why ground a healthy aviator?

THE CLINICAL MANIFESTATIONS OF DCS

The results of a recent workshop in England on the classification of DCS manifestations were reported and discussed. The current Type I/Type II classification is inconsistently defined and arbitrarily applied resulting in treatment variations and making multicenter trials and database comparison almost impossible. The workshop recommended a new classification scheme based on specific description of the disease. For example, a case might be described as "acute relapsing neurological decompression illness," or "acute spontaneously resolving cutaneous decompression illness."

It was also emphasized that thorough neurological exams are crucial to DCS diagnosis, classification and treatment. Concern was expressed that proper patient examination is too often ignored with cases of altitude DCS. Some of the problems with the inaccuracy of DCS databases can be traced to improper and inadequate patient examination. Since hyperbaric treatment procedures are defined by the specific diagnosis, doing a complete but expeditious baseline examination is mandatory. Since DCS is a very dynamic disease, follow-up examinations during the course of therapy are also required. Specifics of DCS patient examination were reviewed.

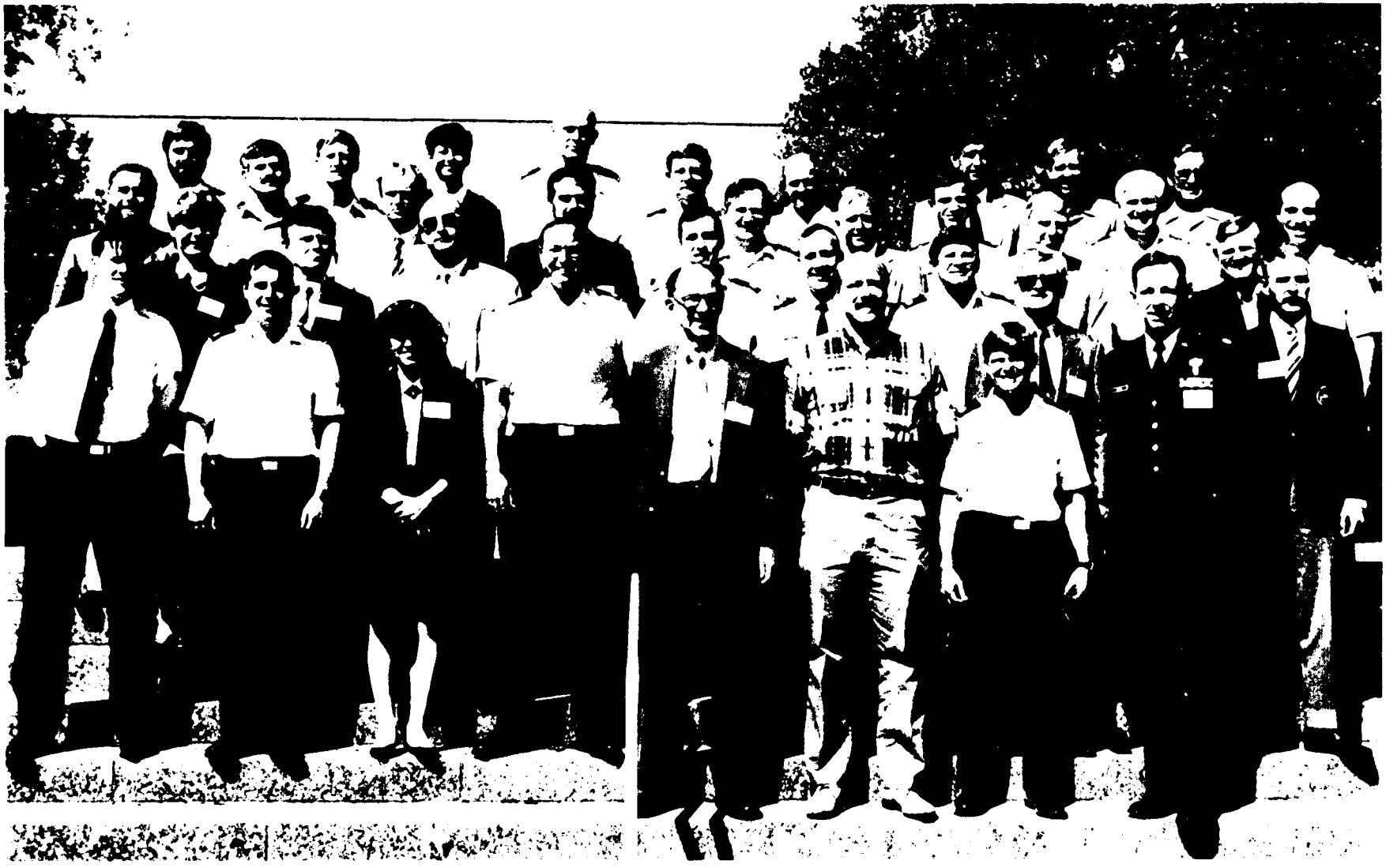
ACCEPTABLE RISK

A panel discussion followed on "acceptable risk" of altitude DCS. It was pointed out that a definition of acceptable risk varies with the mission and ultimately must be decided by the operational people responsible for that mission. It is the responsibility of the investigators to equip these field managers with the best available research information to enable them to make informed and rational decisions. In turn, the investigators must have access to accurate and complete feedback from the field in order to frame the research in proper perspective.

CONCLUSIONS

The 1990 Hypobaric Decompression Sickness Workshop provided a forum for a thorough review and in-depth discussion of the evolved gas problems associated with human exposure to hypobaric environments. Altitude DCS is a potentially hazardous condition that is treated by oxygen prebreathing and hyperbaric therapy. DCS is a potential hazard in the space program, and will continue to affect EVA operations in future efforts. An altitude decompression model and an altitude decompression computer need to be developed to permit real-time and predictive

DCS risk assessment. The reported incidence of altitude DCS is likely inaccurate due to potential career consequences associated with the reporting of DCS. Efforts toward reporting with impunity were recommended. The current classification of DCS manifestations was considered arbitrary and inaccurate. A new method of classification was recommended. The need for improvements in the examination of DCS patients was stressed. What level of DCS risk is acceptable varies and is defined by mission requirements. Such decision-making should be based on information provided by controlled laboratory research.



1990 Hypobaric Decompression Sickness Workshop

Left to right: Front row - K. Wilkerson, R. Bisson, T. Derion, G. Kemper, H. Van Liew, J. Gilbert, J. Garrett, J. Dixon; second row - J. Francis, B. Stegmann, R. Hamilton, Jr., J. Wenzel, B. Butler, P. Gardetto, G. Wolf, A. Parmet, J. Waligora, W. White, Ulf Balldin; third row - W. Norfleet, R. Harding, T. Scoggins, R. Olson, R. Russell, R. Sherman, W. Workman, R. Weien, D. Flannigan, J. Webb, F. Rudge, A. Pilmanis, R. Vann, J. Clark, P. Sheffield, R. Bason and J. Bagian.

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SCHOOL OF AEROSPACE MEDICINE WELCOMING ADDRESS

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It is my pleasure to welcome you to the USAF School of Aerospace Medicine. I am pleased that the school and its staff could be the sponsor for this workshop. Let me share with you a thought that I have. There are a lot of people who have important responsible positions, positions in which they make decisions relative to every major direction and expenditure of funds, who sometimes do not understand the groundwork or the background or the underlying scientific bases that support a lot of the systems that we have. I am sure most of you understand that a lot of our current systems are based on work that was done more than a decade ago. If we were to ask some of our operators about enhancements of systems, they would say, "well what is wrong with the current system or subsystem?"

Over the last year and a half I have heard people talk about the human in space, the military astronaut, in both positive and negative terms. They have talked about it being something we ought to be working on, and talked about it as something we should not be wasting our time on because it will not happen, and then six months later talked about it being something we ought to be working on, and back and forth. I am convinced that people who expressed those sentiments are expressing their view of what the current thinking is about that kind of direction.

I take a much longer view. I take a view longer than the next five years, and maybe the next ten years. I take a view of a decade and a half to two decades. I have a very strong feeling that the kind of issues you are going to address in this conference, the physiological challenges, the research that needs to be done, will be very important. We should understand, as a group, the value of pursuing research, and the full understanding of these workshop issues. It is very important to keep in focus the value of the discussions that are going to occur.

I am not dissuaded by a two star who tells me we should not be looking at issues such as these. In six months, he will tell me I should be. I have experienced that. So I do not want you to be dissuaded by occasional rumors asking, "why are we doing this?" I think it is important that we look at the topic of this seminar that Dr. Pilmanis has put together because I think this knowledge will be a very strong and important basis for future capabilities. Should future requirements be identified and committed to, we will be ready. We will not be limited by what we have only learned up to a point.

I am excited about what the challenges are for the conference. I am excited that we are committed to it. I am excited that we can do that here in this organization. I see a

lot of familiar faces, some of them current and former employees. A few faces I do not recognize at this point, I am sorry I do not, but I am glad that you are here, nevertheless, to participate in the conference. I hope that you have a very good series of discussions, and that you come out with a clear understanding of what it is we are trying to do in this arena. So have a good couple of days. Thank you.

CREW TECHNOLOGY DIVISION WELCOMING ADDRESS

Carter Alexander, PhD
Chief
Crew Technology Division
Brooks Air Force Base, Texas 78235-5301 USA

It's an honor to appear before this workshop today and to add my welcome to that expressed by Dr. Schwender.

I speak to you this morning from the United States Air Force Academy, where I'm enjoying a one year sabbatical assignment as distinguished visiting professor of biology. It gives me a great sense of pride to know that through my efforts in teaching the fundamentals of human physiology to the Cadet Wing, I have a hand, albeit in a small way, in contributing to the development of our future leadership.

As I reflect on the objectives of this workshop I realize just how deeply committed I am to the belief that the human is an essential element of earth based systems, and that neither the human nor the system can reach its full potential in an environment in which the other is excluded. Just as our engineers are providing the technology and the know-how to operate our flying machines further and further from the surface of the earth, we as physiologists are meeting the challenge to provide effective protective equipment as a means of support to keep our men and women in these machines.

In accepting this challenge to operate at higher altitudes and reduced barometric pressures we have also accepted the reality that manned operations in this unnatural and hostile environment are not without risk. Through either ignorance of the fundamental principles involved, or through failure of our systems, operation in this environment carries the very real risk of decompression sickness in our aerospace crews. The inevitability of failure in ourselves and our system is often further complicated by the political and economic pressures of cost and schedule.

I'm especially proud to be part of the team that is willing to accept this risk as an undeniable component of manned operations, yet at the same time realize and accept the obligation to do all within our means to reduce it.

This workshop is designed to identify the shortfalls in our knowledge concerning altitude DCS. The overriding objective is to reduce the risk to humans operating in the aerospace environment through a more complete understanding of the etiology and progression of decompression sickness in aerospace operations.

I'm confident as well that with the thoughtful and spontaneous discussion that is sure to characterize this distinguished group, ideas will emerge which will become the basis for improving reliability of our crew protective systems.

It's my personal belief that even the potential pressures which oftentimes drive compromises to effective protective schemes can be lessened as our understanding of the physiology of the human in the aerospace environment is advanced.

I applaud the efforts of Andy Pilmanis and his very capable research staff in conceiving, organizing and conducting this workshop. Many of you in attendance are very dear friends, and all are respected colleagues. Each of you has played a significant role and made lasting contributions to advancing our knowledge of this aspect of applied physiology.

I wish it were possible to greet each of you personally and to experience the unique opportunities that this workshop will provide. I do wish you the very best of success in the coming days. I realize that you give your most precious of gifts, that being your time, to come to Brooks to collectively work on this project. I salute your efforts and I'm confident of a positive outcome.

WORKSHOP ISSUES AND SCOPE

Andrew A. Pilmanis, PhD
Chief
High Altitude Protection Function
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1. What is the importance of hypobaric decompression sickness (DCS)? Is there mission compromise? If so, when and where? Have prebreathing and hyperbaric therapy solved the problem?

For example, below are references of recently published cases,

1. Fatal Pulmonary Decompression Sickness: A Case Report
   NEUBAUER JC, DIXON JP, HERNDON CM. Aviat. Space Environ. Med. 1988; 59:1181-4

2. A Case of Decompression Sickness in a Commercial Pilot
   WOLF CW, PETZEL DH, SEIDL G, BURGHUBER OC. Aviat. Space Environ. Med. 1989; 60:990-3

3. Probable Bends at 14,000 Feet: A Case Report
   VOGE VM. Aviat. Space Environ. Med. 1989; 60:1102-3

All three had mission compromise. One was a fatality. One resulted in unconsciousness of a pilot at altitude. One reported severe pain, severe enough to give up the controls of the aircraft. These are current events.

2. What pathology is associated with altitude DCS? How long do untreated bubbles last and where? How serious can acute DCS injury be? Are there long-term sequelae with altitude DCS? If so, under what circumstances?

3. Is the TRUE incidence of altitude DCS known? Is there reluctance to report it? If so, why? How can this be corrected? Should reporting and treatment have impunity?

We do not have accurate databases. Databases cannot be compared to each other because of this reluctance to report. There have been recent attempts to correct this problem. For example,

USAF Regulation 160-43
(Medical Examination and Medical Standards) has been changed.

Chapter 7: Medical Standards for Flying Classes II and III

Date of Revision: 7 April 1989

Page 87: "f. Decompression Sickness with neurological involvement or history thereof. Consideration for waiver may be given following medical evaluation by an Air Force hyperbaric qualified physician."

The purpose of the regulation change was to remove the penalty associated with reporting DCS, and the penalty of being grounded. Is it working?

Decompression is accompanied by a risk of DCS. Currently, there appears to be a general reluctance to report DCS. This trend results in no medical treatment in an unknown number of DCS cases. Hyperbaric therapy of altitude DCS is almost 100% effective and properly treated divers or aviators do not appear to have long-term sequelae. Untreated divers run a risk of chronic spinal, cerebral, and bone injury. These risk factors have not been studied in aviators. Is there mission compromise with unreported DCS? Unreported DCS results in inaccurate databases which also leads to misleading information dissemination and reinforcement of the status quo, thus perpetuation of the existing situation.

Accurate reporting could result if aviators could return to work with impunity. DCS treatment would become a routine aspect of their work. Accurate databases would provide the required information for improving life support, and decreasing DCS incidence.

Suggested motives for reluctance to report altitude DCS:

1. Mission protection
2. Career protection
3. Inability to detect and diagnose
4. Perception of unimportance
5. Treatment inconvenience
6. Peer pressure
7. Distrust of medical involvement
8. Concern of impact on operational aspects

4. There appear to be a number of DCS reporting discrepancies. Why?

- USAFSAM research databases vs. USAF operations

- USAF vs. RAF

- USAF vs. USN

- NASA/USAF research data vs. EVA experience

5. How does HYPObaric DCS differ from HYPERbaric DCS? Should they be treated as separate conditions? How do the risk factors vary?

There is a tendency to transfer information from one field to the other; sometimes it is valid, sometimes it is not. Some of the differences are listed below.

HYPObaric vs. HYPERbaric:

* Gas density and composition

* Less absolute amount of N_2 with altitude

* Bubble dynamics

* Inherent recompression with altitude

* Saturation is point of origin with altitude, i.e., equivalent of upward excursion for saturation diving

* Less severe symptomatology with altitude

Figure 1 illustrates the operational flight/dive profile differences. With altitude, the mission part is during the decompression, whereas with diving, decompression starts after the mission is completed. That is an important difference. Inherent in altitude exposure is recompression back to ground level. Thus, there is partial treatment inherent with every exposure. That is quite different from the diving situation.

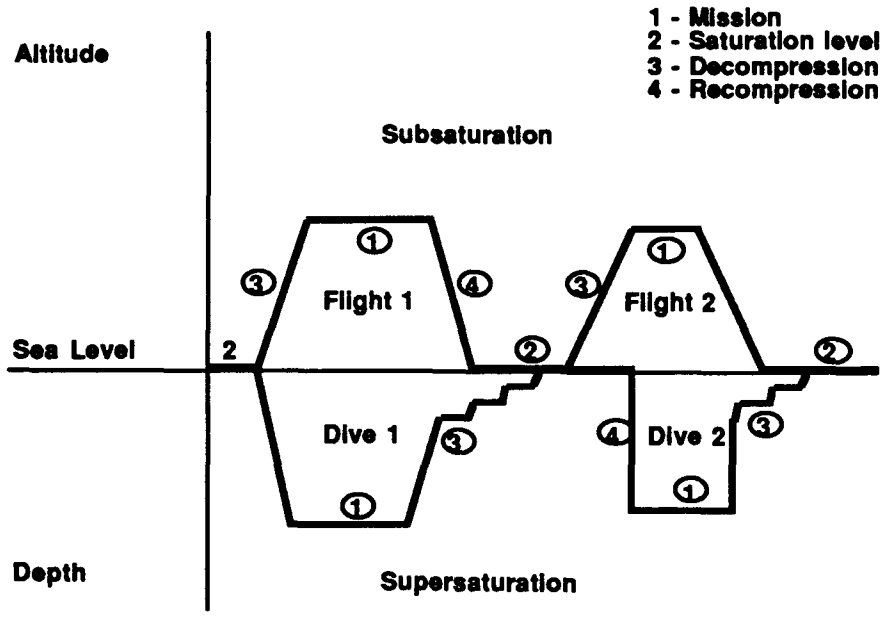

Figure 1. Flight/dive profile comparison.

Figure 2 shows the most analogous type of dive to flight profile. A saturation dive with upward excursions. That is a very unusual diving situation.

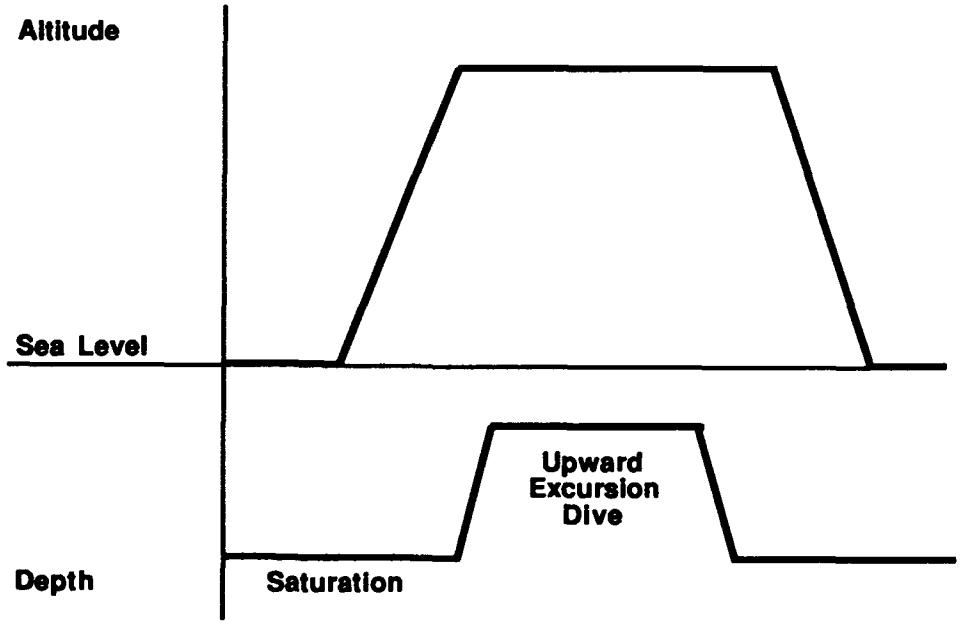

Figure 2. Saturation profiles.

The severity of symptomatology is another area that seems to be different. Altitude DCS is generally milder than diving DCS. However, fatalities have occurred with altitude DCS. Cerebral and spinal involvement does occur, probably more frequently than what is recorded.

6. Have recent technological and research advances with Doppler/Echo-Imaging bubble detection systems modified our view of altitude DCS? In what way? Has this penetrated to the operational level?

A short video tape was played illustrating noninvasive *in vivo* bubble imaging in a human subject at simulated altitude in a chamber. The video showed bubbles moving through the right heart. Figure 3 shows a photograph of one of these images. The resolution in Figure 3 is greatly reduced from the resolution of the video.

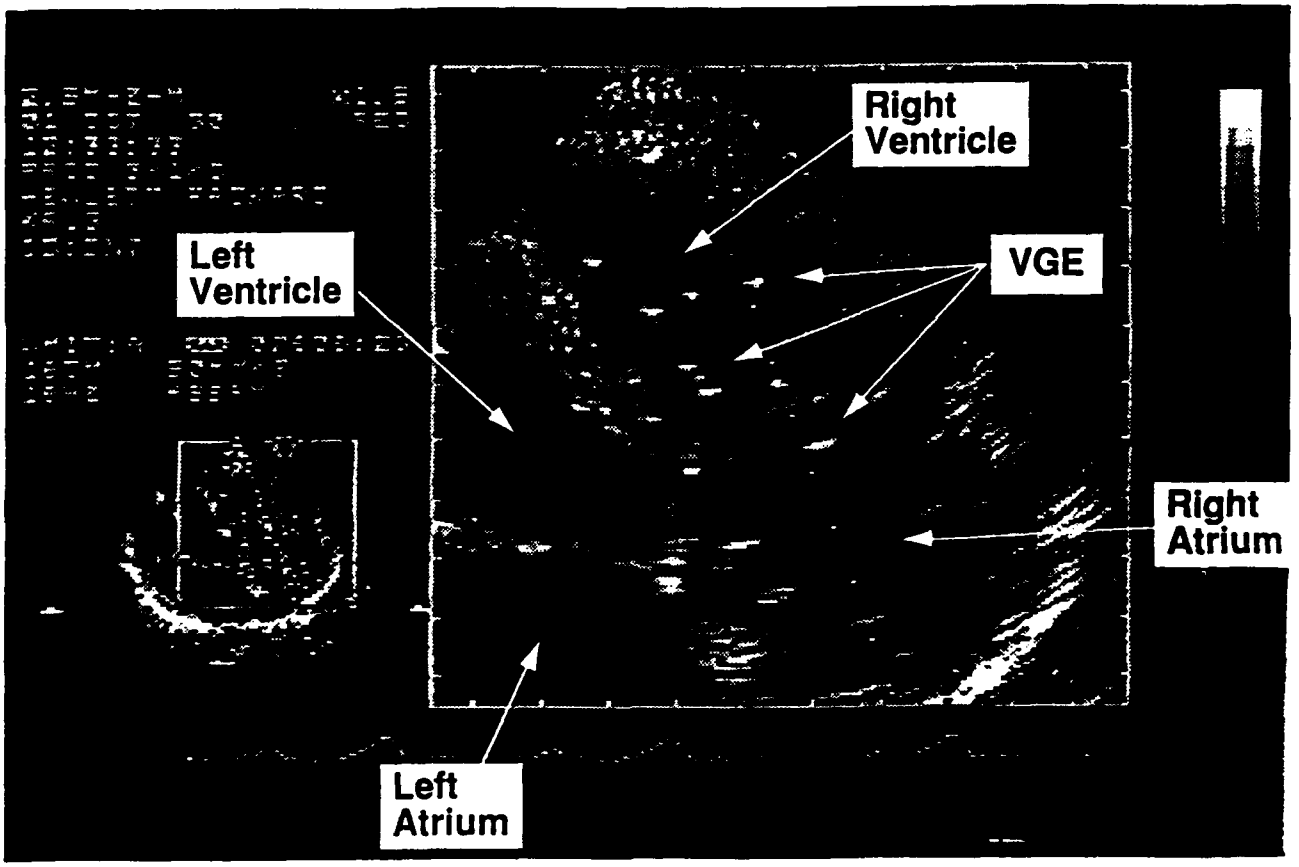

Figure 3. Venous gas emboli in the human heart.

This is "state-of-the-art" technology in bubble detection. The Doppler has been around for a long time. However, with this kind of imagery there is a shift toward visual rather than audio data. Finding methods for bubble quantification and bubble sizing is a major challenge.

7. Are the clinical manifestations of DCS universal in definition? If not, why? Should the terms "Type I, Type II" be dropped in favor of specific medical diagnoses?

8. Altitude DCS prediction and prevention capability can be modeled and computerized. Is such a risk analysis potentially useful? How would it be used? How can it be validated?

At AL, the development of an altitude decompression computer has been initiated. The objectives of such a computer are as follows:

a. To provide "cockpit or pressure suit" readout of DCS risk under constantly changing conditions.

b. To provide the capability for evaluating various options in high altitude mission planning.

c. To provide altitude chamber crew safety and manning control.

d. To provide desktop DCS risk analysis capability.

The major problems associated with this development center around the available "DCS databases." DCS is a very subjective disease. When does "DCS" become a clinical entity? Some manifestations that today define DCS were ignored in the 1940s. Thus, attempting to compare databases from 50 years of DCS research is a very difficult task. DCS reporting in the operational setting is an even greater problem because of mission and career considerations.

9. How serious is the EVA decompression problem in the space program? Figure 4 shows the dilemma NASA faces.

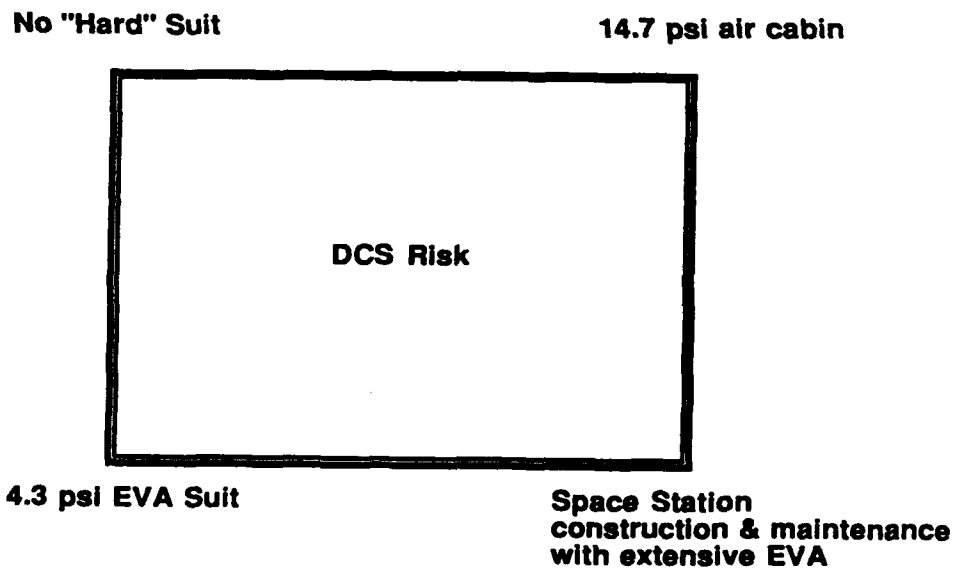

Figure 4. Space program dilemma.

10. What is "acceptable DCS risk"? To whom? Under what conditions?

11. Are the current prebreathing requirements adequate? What are they based on? Which ones should be reviewed?

12. How successful are the current altitude DCS treatment procedures? Are modifications justified?

13. Is the "Patent Foramen Ovale" issue of serious concern? Under what circumstances is it important?

14. Are current ground level intervals between flights acceptable and safe? If not, which operations are not? Why not?

1990 Hypobaric Decompression Sickness Workshop

Session One: DECOMPRESSION SICKNESS

Richard D. Vann, PhD, Chairman

ORIGINS AND EVOLUTION OF PATHOPHYSIOLOGICAL CONCEPT

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A special gain in looking back in a scientific field is in learning how very much smarter past investigators were than we may now seem to be, with our massive assets of all prior information, instrumentation developments, concepts and discussions. The continuous flow from earliest contributions of measurement, physical analyses, mathematics and imagination now finds its way into newly purchased computers which can carry out measurement or even analysis while the investigator sleeps. Isaac Newton said this more kindly, in a 1676 letter to Robert Hooke - - - -

"If I have seen farther than others, it is because I have stood on the shoulders of giants" (1).

My aim for this conference introduction will be to link the evolution of major pathophysiologic concepts of hypobaric decompression sickness to the evolution of practical human activities which generate it. Since history begins in the past, but includes the present and goes on forever into the future, this introduction will offer predictions for subsequent appraisal.

An important source of clues for early contributions remains the collection of papers and references provided by Fulton (2) following the intensive altitude decompression research activity of World War II.

THE EARLIEST GIANTS

The undisputed beginning, with Robert Boyle in 1659, arose from his invention of an effective "evacuation pump." Using his pump in various experiments led to his discovery that cold-blooded animals exposed to reduced pressure appeared to be agonized, whereas animals enclosed in a sealed air compartment at atmospheric pressure succumbed quietly over a few hours (3, 4). It was in a viper, "furiously tortured in our Exhausted Receiver," that he "once observed" a "conspicuous bubble moving to and in the waterish humour of one of its eyes."

Boyle's investigations were many and thoughtful in the attempt to separate what he clearly saw as concurrent different effects of reduced air pressure. The astounding quality of his deductive query, out of simple visual observation, endures in the example from his 1670 paper in Philosophical Transactions (4):

"Note, that the two foregoing Experiments were made with an Eye cast upon the inquiry, that I thought might be made; Whether, and how far the destructive operation of our Engin upon the included Animal, might be imputed to this, that upon the withdrawing of the Air, besides the removal of what the Airs presence contributes to life, the little Bubbles generated upon the absence of Air in the Bloud juyces, and soft parts of the Body, may by their Vast number, and their conspiring distension, variously streighten in some places, and stretch in others, the Vessels, especially the smaller ones, that convey the Bloud and Nourishment; and so by choaking up some passages, and vitiating the figure of others, disturb or hinder the due circulation of the Bloud? Not to mention the pains that such distensions may cause in some Nerves, and membranous parts, which by irritating some of them into convulsions may hasten the death of Animals, and destroy them sooner by occasion of that irritation, than they would be destroyed by the bare absence or loss of what the Air is necessary to supply them with. And to show how this production of Bubbles reaches even to very minute parts of the Body I shall add on this occasion (hoping that I have not prevented myself on any other) what may seem somewhat strange, what I once observed in a Viper, furiously tortured in our Exhausted Receiver, namely, that it had manifestly a conspicuous Bubble moving to and fro in the waterish humour of one of its Eyes."

Hoppe-Seyler and Bert

It was nearly 200 years later, in 1857, that Hoppe-Seyler reported seeing "bubbles" in animals exposed to 50-mmHg pressure (equivalent to 62,000 feet altitude)(5). Bert's equivalent animal exposures, reported in 1878, failed to find bubbles, and, as his own subject, he breathed oxygen while reducing chamber pressure to the equivalent of 29,000 feet altitude, experiencing no symptoms (6). Practical interest in hypobaric states, at this period more than one hundred years ago, involved the slow ascents of mountaineering. Diving activity was extremely limited.

Hill and Greenwood, Haldane

Soon after 1900, the vigorous and imaginative investigations of human hyperbaric (compressed air) exposures related to expanding caisson work and air diving led to progressive understanding of decompression sickness. This expanded and gradually contributed to physiological awareness that similar phenomena might also occur in decompression to reduced atmospheric pressure. However, the blending of research and concept in undersea and hypobaric (altitude) physiology progressed very slowly, requiring World War I and subsequent advances of aviation before recognition of the reality of "altitude decompression sickness" as a medical problem.

The occurrence of bubbles in animals exposed to hypobaric states in small chambers was reaffirmed (7), and the systematic deductive and empirical contributions

of J. S. Haldane and his associates brought lasting order to a basic concept of inert gas exchange between respired gas and body tissues in diving and diving decompression (8).

GIANTS OF RECOGNITION OF THE CLINICAL ENTITY IN AVIATION

Henderson

The technical emergence of controllable aviation, superseding balloon flight, and the combat use of aircraft in a great war stimulated Yandell Henderson, one of the giants of respiratory physiology of his era, to derive in 1917 a prediction of bubble development in human flight higher than 20,000 feet. He included recognition that the slow ascent and the limited altitude then practical made the occurrence of bubbles unlikely (9).

Barcroft, Douglas, Kendall and Margaria

It was only during the 1930 decade that inventive advances in aviation technology, ultimately leading to compressed air breathing by aircraft engines (the supercharger), aroused specific physiologic interest in altitude decompression by giants of British physiology. In 1931, the first human to experience hypobaric bends was Margaria, in "stepping exercise," at 30,000 feet altitude equivalent (10). The observation was temporarily passed over, to be reaffirmed later. Of special modern significance, related to an apparent and important paradox of present aerospace EVA decompression studies at the NASA Johnson Space Center (11), is that Margaria's exercise was performed by repetitive one leg stepping, and his knee pain developed in the nonexercising leg. In the modern arm exercise studies at NASA, hypobaric decompression symptoms develop, not in the exercising arms, but in the non-exercising legs of standing subjects (11, 12). These 60-year-old and continued observations have special significance to the role of exercise in precipitating decompression sickness.

Armstrong

Armstrong's important influences through hypobaric decompression experiments demonstrating bubbles in blood and tissues in animals, including goats, were fortuitously magnified by his broader lifetime role as an imaginative military aviation medical officer (13). In that continuing responsible role, he recognized and communicated the significance of decompression sickness as an entity in developing aviation. His influence stimulated fertile minds of undersea and respiratory physiology, prior to the expanded research endeavors of World War II.

Boothby and Lovelace

Two fertile minds were the physiologist Boothby and collaborating physician-scientist Lovelace. It was their discovery in 1938 of the occurrence of "spinal paralysis" in a human subject at an altitude chamber equivalent of 35,000 feet that exposed the potential severity of decompression sickness in human flight (14). This ended argument, just in time for World War II and the tremendous technologic expansion of military aviation.

While the phenomenon of hypobaric decompression sickness was from then on accepted and respected, the therapeutic management of its multiple clinical expressions has been even until now considerably neglected.

THE GIANTS OF WORLD WAR II

The wartime requirements for advance in military aircraft altitude ceiling and rate of attainment, of even greater urgency than improvement in diving, brought officially together the "Subcommittee on Decompression Sickness" of the U.S. National Research Council - charged with study of decompression sickness encountered in aviation. The giants of this United States-based "Subcommittee," having close liaisons within the United Kingdom, rapidly had their role expanded to include continuous direct laboratory research, collaboration in research planning, and active communication in the field, to a degree not often encountered before or since.

It is practical here only to remind you of a few of the extensive critical observations and conceptual awarenesses that evolved from such individuals into what has remained the mainstream of understanding over the 50 years since World War II and through to the present. Their experiments, analyses and concepts have been regenerated and restated or adapted by so many of us that their origins tend to be obscured by each new repetition of experiment or interpretation.

Some examples are of special relevance to this Conference:

Newton Harvey, a physicist who by necessity of the times became an expert physiologist, intensively studied by experiment and mathematical analysis the initiation, growth and resolution of gas "bubbles" in physical systems, in biological fluids, and in animals. He knew already that tolerance to hypobaric decompression at rest was greater than in exercise, and therefore with his associates concentrated animal studies (e.g., in cats) on the separation of circulatory, respiratory and mechanical factors in exercise (15, 16). Experiments examined stretch, passive movement, and electrically-induced contractile exercise in hypobaric decompression, to learn that venous gas bubbles were found only in the contractile exercise. Repeated experiments revealed that venous gas bubbles tended not to appear in animals at rest. In physical systems he demonstrated the extreme tolerance of "denucleated liquid" to gaseous supersaturation (even to greater than 100

atmospheres) without bubble formation. He conceived therefore that decompression sickness development could occur only because gas spaces always exist in the living animal. He provided the mathematics of initial bubble growth from such preexisting "nuclear" gas spaces. He stated the obvious conclusion of "delta P," that whenever an excess gas pressure existed above the sum of bubble ambient pressure forces, bubble growth is inevitable. And much more (17, 18, 19).

Lesle Nims (20) provided detailed elaborations of in-vivo factors of gas phase growth which included the mathematics of gas phase growth theory, the importance of duration of exposure to an excess gas pressure, the implications of "rate of production" of altitude decompression sickness, and investigations of the pathophysiologic origins of the pain symptoms postulated by Boyle 300 years before.

Hardin Jones was the master respiratory physicist/physiologist who clearly demonstrated that the rate constants of gas exchange between atmosphere and tissues for different gases were identical and determined by circulation alone, without a detectable diffusion component, over the 8-fold range of molecular weight of helium, nitrogen, argon, krypton and xenon. He distinguished among decompression sickness-relevant factors of rate constants, gas solubility, and molecular weight-dependence in perfusion and diffusion (21, 22). He recognized that his advanced, isotopic measurements of inert gas washout, defining major conglomerate components of tissue perfusion, left behind the then, and as yet undefinable, other conglomerations of multiple small foci of other tissues most likely related to the actual symptoms of decompression sickness. His intricate mathematical physiologic analysis indicated that resting muscle and fat have comparable time constants, and exercising muscle itself should not be the source of the venous gas bubbles induced by exercise at high altitude. He also pointed to indirect consequences of exercise as potential factors.

J. R. Bateman and A. R. Behnke were pioneers of what came to be called "preoxygenation" in delaying onset of bends symptoms. Behnke's extensive measurements of nitrogen elimination and effects of oxygen breathing on hypobaric bends development (23, 24) were expanded by others (2). Bateman (25) recognized early that the simplified Haldanian concept of tolerable supersaturation was practical but not reasonable and provided a lucid theoretical and mathematical analysis of the relations of "tissue gas exchange" and gas phase growth in decompression and the influence of oxygen breathing. These analyses have retained their rational character through a long period of inattention. Both Behnke and Bateman astutely emphasized study of delay rather than prevention of decompression sickness, considering the inevitability of bubble formation pointed out by Harvey. Bateman called the period prior to overt symptoms in human beings the "Silent Phase of Decompression Sickness"; Behnke used the term "Silent Bubbles." Doppler acoustics technology has now affirmed this repeatedly ignored early awareness. "Noisy Bubbles" are now beginning to attract the serious attention they should have had from the original deductions of their presence. A culmination of efforts by the Subcommittee on Decompression Sickness was the

systematic empirical evaluation of preoxygenation in thousands of altitude chamber exposures, by the many dedicated physiologists cited elsewhere (2).

EVOLUTION FROM EARLY CONCEPTS TO THE PRESENT

Out of these extensive early investigations and intensive communications of thought came the beginning of concepts relevant to both undersea and aerospace decompression. The technologic transition from past to present involves as much the remembering and assembling of the exceptionally clear vision of predecessors as it does the uncovering of new principles.

The final purpose of this summary is therefore a reminder that large recent technologic advances in offshore and military diving, and in aviation and aerospace activity, have increased the need for a blending of hypobaric, isobaric and hyperbaric research and applications. Examples of selected early concepts relevant to present and future research activity are cited below.

Patterns of Decompression Sickness - The Evolution of Understanding

It is evident that especially the giants of World War II considered that the disorders of decompression sickness could develop at many sites, in many forms, in any combination and to any degree (2), as indicated in Figure 1. Present efforts proposed to integrate such understanding in terms of a family of "Gas Lesion Diseases" (26) have emphasized that:

"Decompression Sicknesses, except for Pulmonary Barotrauma, should be considered a generalized systemic process of gas phase expansion, which may become severe enough in the right microanatomical locations to be recognized. Effects can simultaneously go unrecognized and tolerated in many different locations elsewhere."

"Decompression Sickness, except for Pulmonary Barotrauma, should therefore not be considered a single location, 'yes or no' event, in either undersea or aerospace activity."

"It should be considered a 'Dose-Response,' not a 'Threshold,' phenomenon at each involved tissue site. It can be considered a diffuse continuum of graded degrees of pathophysiologic events and effects, in many scattered locations, each of which has its own stress-effect consequences."

"Symptoms and signs of overt decompression sickness should be considered as resulting from multiple pathologic factors."

"Differential gradations of decompression sickness effects, as for example into Type I (e.g., Pain) and Type II (e.g., Neurological), are operationally practical indices of locus or "importance," but should not be considered sensible descriptors of the fundamental processes or their severity."

ASYMPTOMATIC BUBBLE FORMATION - INTRAVASCULAR, SOFT TISSUE,

CUTANEOUS - ITCHING, BUBBLE FORMATION, LESIONS ("SKIN BENDS"),

LIGAMENTOUS, FIBROUS TISSUES - "BENDS". PAIN, TENDERNESS,

PULMONARY - "CHOKES". SUBSTERNAL DISTRESS, COUGHING,

SHALLOW RESPIRATION, PROFOUND HYPOXEMIA, SYNCOPE , AND SHOCK,

CARDIOVASCULAR - HYPOTENSION, LOW URINE OUTPUT,

INCREASED VASCULAR PERMEABILITY (?), CELL AGGREGATION (?),

NEUROLOGICAL - SPINAL MORE THAN CEREBRAL OR MEDULLARY.

DIPLOPIA, SCOTOMATA, LABYRINTHINE DYSFUNCTION, DEAFNESS,

CORD LESIONS, SENSORY LOSS, PARALYSIS, BONE - ASEPTIC NECROSIS,

BLOOD - PLATELET AGGREGATIONS, COMPLEMENT CHANGES,

EYE - ASYMPTOMATIC BUBBLES IN ANY COMPARTMENT.

Figure 1. Patterns of decompression sickness (any combination, any site, any degree).

Factors In Altitude Decompression Sickness Generation

The major combination of factors involved in generation of altitude or other decompression sickness, relating to rates and degrees of gas phase development, were well cited by both Newton Harvey (16) and Hardin Jones (21), as graphically summarized in Figure 2 (27). The summary figure indicates the early awareness that inert gas exchange between atmosphere and myriad micro-volumes of different tissues is a function of local blood flow and gas solubility; exchange between microtissue and gas phase is a function of gas molecular weight and solubility (diffusivity); initiation of gas phase growth was conceived as counteracted by surface forces of a gas-liquid interface.

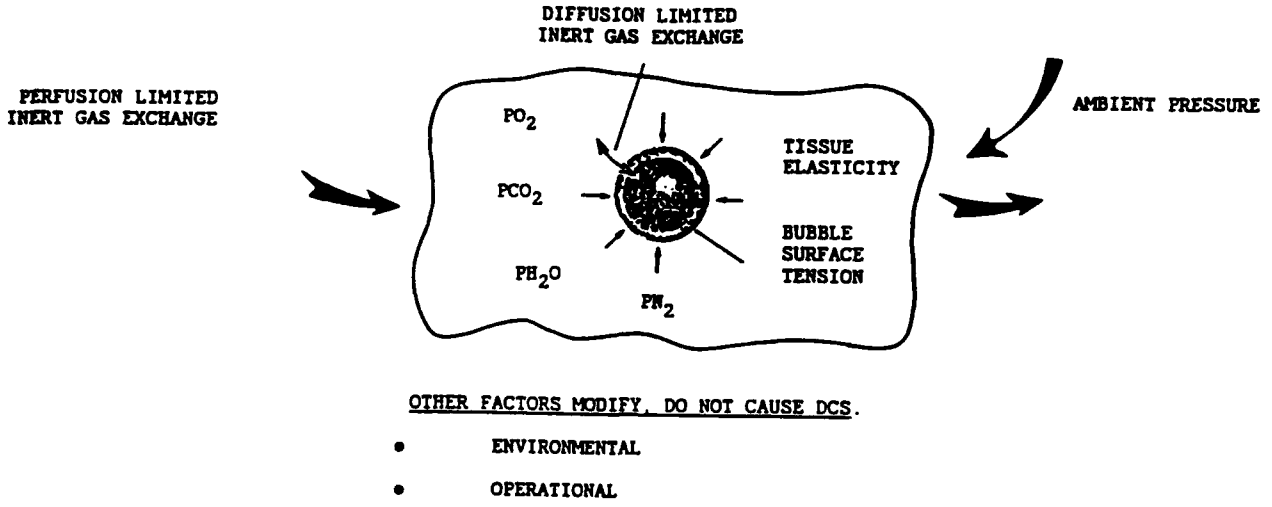

Figure 2. Factors in DCS generation.

Circumstances for Development of Gas Lesion Diseases

New situations for occurrence of Altitude Decompression Sickness and other Gas Lesion Diseases have evolved over past decades and now exist as important problems for prevention and treatment (Figure 3). However, in each new situation the principles of physics and physiology remain the same, and the human target has remained unchanged over millennia.

O DIVING AND FLYING
O FLYING AND DIVING
O AEROSPACE OPERATIONS

REPETITIVE DECOMPRESSION IN EVA
DECOMPRESSION FROM PROLONGED HYPOBARIA
FLUCTUATING DECOMPRESSION
EXTREME DECOMPRESSION

O DIVING AT HIGH ALTITUDE
O DIVING AND DRIVING - TO HIGHER ALTITUDE
(THE SWISS CONNECTION)

Figure 3. <u>Recent history - altitude/aerospace DCS ongoing expansion of circumstances</u> from past to present.

Concepts derived during or from past investigations, even without present unanimity of acceptance, provide the bases for refinement of research and operational development. Figure 4 summarizes examples of such present concepts, derived from cited past investigators, and activity used in a current integrated decompression analysis system (28). The concepts examples derive variously from deductive reasoning, empirical measurement, from obvious fact, or from common sense.

- O ANY EXCESS GAS PRESSURE (P) WILL PRESENT A POTENTIAL FOR SYMPTOMATIC DCS.
- O GAS PHASE NUCLEI EXIST NORMALLY IN TISSUES BEFORE AND DURING DECOMPRESSION.
- O SYMPTOMATIC EXPRESSIONS RELATE TO GAS PHASE EXPANSION, NOT TO P OR RATIO OF P TO AMBIENT PRESSURE.
- O A "SYMPTOMATICALLY TOLERABLE" DEGREE OF GAS PHASE EXPANSION EXISTS.
- O IF A "PHYSICALLY TOLERABLE" EXCESS P EXISTS, IT MAY REPRESENT THE P NECESSARY TO FORCE INITIAL EXPANSION OF EXISTING NORMAL GAS PHASE.
- O DCS TAKES TIME TO DEVELOP. INTEGRATED DURATIONS OF EXPOSURE TO EXCESS P DETERMINE DEGREE OF GAS PHASE EXPANSION AND SYMPTOM GENERATION.
- O APPARENT DIFFERENCES IN TOLERABLE P BETWEEN "FAST" AND "SLOW" TISSUES ARE THE RESULT OF DIFFERENT DURATIONS OF INTEGRATED P EXPOSURE.
- O IF A "SYMPTOMATICALLY TOLERABLE" P EXISTS, IT IS SMALLER FOR ALTITUDE THAN HAS BEEN ASSUMED AND CONVENTIONALLY USED IN DIVING DECOMPRESSION TABLE DEVELOPMENT.

Figure 4. "The past is prologue" present concepts of decompression sickness from past history.

PREDICTION OF FORWARD HISTORY

Paraphrasing the "Philosopher" Samuel Goldwyn,

"PREDICTION IS DANGEROUS -
ESPECIALLY WHEN IT DEALS WITH THE FUTURE."

Nevertheless, this Introduction must predict that large improvement in understanding, operational applications and safety can be expected along several broad paths such as indicated in Figure 5.

- O SENSIBLE OPERATIONAL USE OF HYPOBARIA AND HYPOBARIC OXYGEN CAN FACILITATE NITROGEN ELIMINATION FROM SLOWLY PERFUSED TISSUES.
- O SIGNIFICANT SUCCESS IN OTHER PHYSIOLOGIC OR PHARMACOLOGIC ACCELERATIONS OF NITROGEN ELIMINATION IS UNLIKELY.
- O MAXIMAL EXTENSION OF TISSUE OXYGEN TOLERANCE IS MOST CRITICAL.
- O EVOLUTION OF SPECIFIC HYPEROXIC METHODS IS DESIRABLE FOR THERAPY OF DCS GENERATED IN HYPOBARIC ACTIVITY.
- O DELINEATION OF THE "CONTINUUM" OF AEROSPACE AND UNDERSEA DECOMPRESSION THEORY, BIOPHYSICAL, AND PATHOPHYSIOLOGIC BASES IS FUNDAMENTAL.

Figure 5. Potential for future physiologic improvements in altitude DCS prevention/therapy.

Essentially all large successes in safety and effectiveness are here predicted to be oxygen-related. Favorite areas of discussion at conferences, such as other means of significant and controlled acceleration of inert gas from tissues, are predicted as unlikely. The largest and most fundamental mark on future history should result from establishing the physical, physiologic and pathologic continua of hypobaric (aerospace) and hyperbaric (diving) decompression. A continuum surely does exist (30), as indicated in Figure 6, and only the pressure range of their occurrence should distinguish the biophysical initial events in aerospace and undersea decompression sicknesses. The final consequences should prove to be matters of degree.

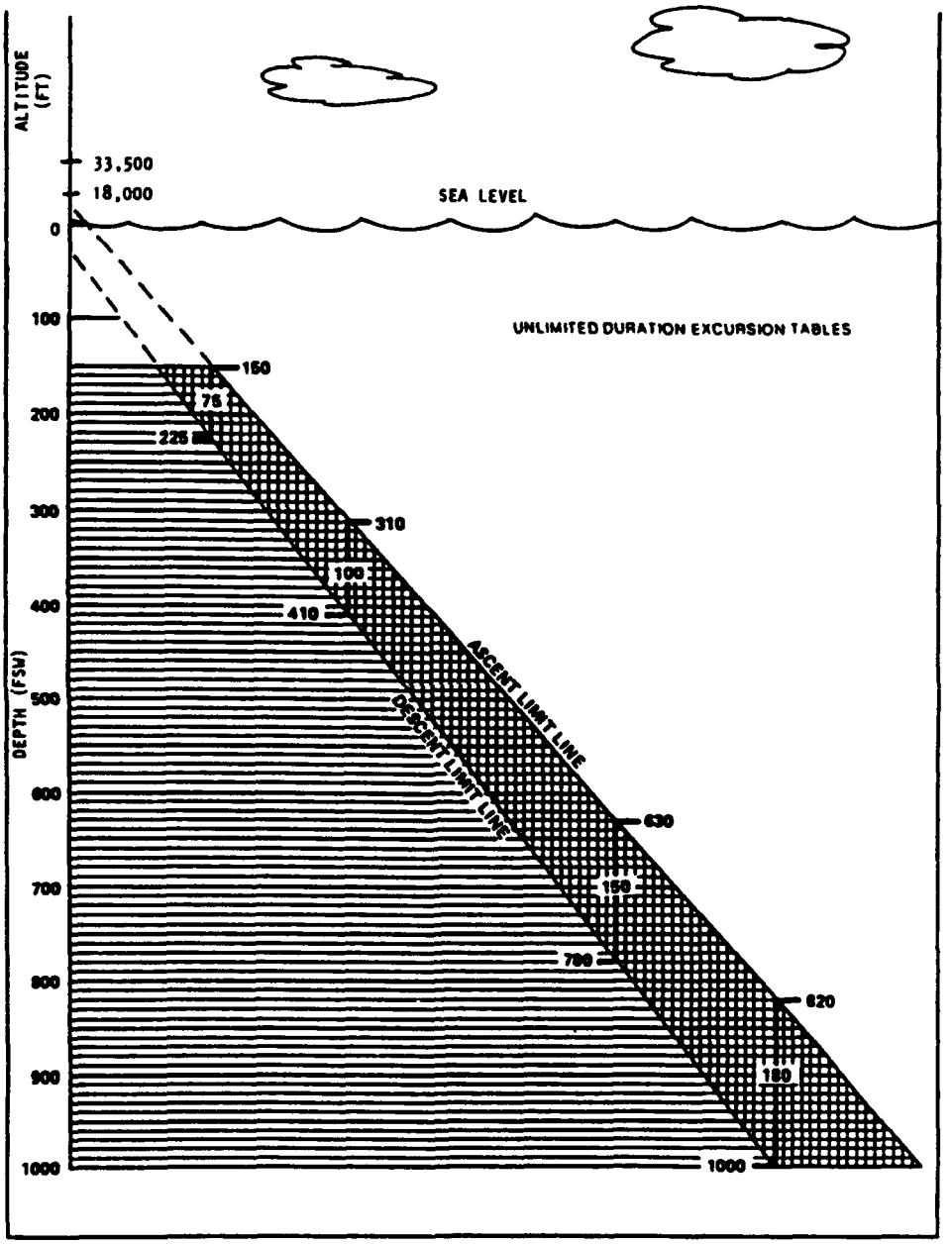

Figure 6. An hyperbaric/hypobaric "continuum" must exist for nitrogen saturation decompression. The U.S. Navy helium-oxygen undersea continuum requires a nitrogen counterpart extending from shallow water through the water-atmosphere interface to high altitude.

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BUBBLE DYNAMICS

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Some issues about gas diffusion in living tissue and exchanges between a bubble and its surroundings can be studied empirically, as with 25-ml subcutaneous gas pockets on the backs of unanesthetized rats (1,2). Extrapolations from such studies to the small, inaccessible bubbles that cause the trouble in decompression sickness can be accomplished with mathematical formulations (3-6). If there is a communications gap between these diffusion-based types of information and their practical application for prevention and cure of decompression sickness, it may be because of such complicating issues as the variability that is inherent in living tissue, uncertainties about the nature of decompression bubbles and gas nuclei, and other matters that we have not even become aware of yet. Also, decompression table calculations are complex by nature so that the basic assumptions, about diffusion or about other fundamentals, tend to get lost in the mathematical development. This communication will start with review of diffusion matters and progress to conjectures about the origin of decompression bubbles; contrasts will be drawn between the hypobaric and hyperbaric situations.

Diffusion Fundamentals

Figure 1 is an attempt to illustrate one of the fundamentals of diffusion and bubble behavior: that there are important interrelationships between partial pressures of the different gases, bubble volume, and the total pressures. The arrows are meant to correspond to permeation coefficients. When a particular gas has a big permeation coefficient, it is easy for that gas to enter or leave the bubble. Oxygen and carbon dioxide permeate easily, so they have negligible partial pressure differences between inside and outside of the bubble ("gradients"). The N_2 is less permeable. As indicated in Figure 1, the total pressure inside a bubble is expected to be equal to the ambient pressure that surrounds a person plus any additional pressure due to surface forces or elasticity of tissue. Oxygen and CO_2 inside the bubble are low; in particular, O_2 is low because of metabolic removal of O_2 by living cells. Low O_2 and CO_2 in the bubble conspire to make bubble N_2 high, whereas dissolved N_2 in the tissue is set by N_2 level in the lungs where high O_2 makes N_2 low. The resulting gradient of partial pressure for N_2 promotes outward diffusion of N_2 and bubble shrinkage. Exit of N_2 tends to leave O_2 and CO_2 behind in the bubble, but excesses of these gases rapidly diffuse out because they permeate so easily. These relationships are the bases of what is known as the "oxygen window" or "inherent unsaturation" (7); the principle can be demonstrated to work as predicted with special preparations such as rat gas pockets (8). However, it is worth noting that the assumptions are for a standard tissue

with unremarkable blood perfusion and metabolism; it is conceivable that the particular tissues which give trouble in decompression sickness may be complex or idiosyncratic, so that the oxygen window idea may not apply so simply as described here.

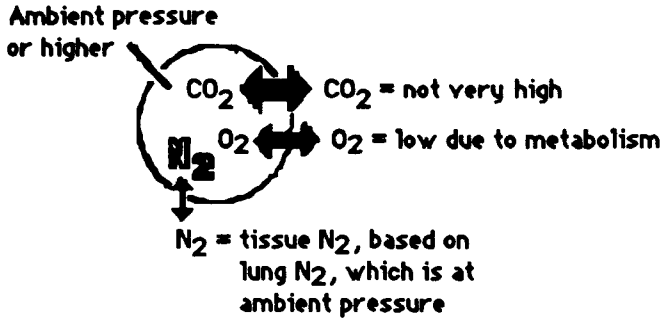

Figure 1. Diagram of the interrelations between permeation coefficients for different gases (indicated by the arrows), the partial pressures of the gases, and the ambient pressure. See text for explanation. The partial pressure difference for nitrogen between inside and outside of a bubble due to these relationships is known as the "oxygen window."

In diving work, N_2 is very high and in a person at normal atmospheric pressure, N_2 is 80 or 90% of all gas present. However, in hypobaric situations, total pressure is lower so partial pressure of nitrogen (PN_2) is lower. The partial pressures of O_2 and CO_2 in tissue are nearly independent of hyperbaric or hypobaric conditions; appropriate levels of O_2 and CO_2 are necessary for life. Therefore, O_2, CO_2 and also water vapor are appreciable fractions of the gas in the hypobaric bubble; it follows that they are more influential in bubble dynamics.

When ambient pressure decreases, as in ascent to altitude, PN_2 in the tissue stays high until washout removes dissolved N_2, whereas total pressure and PN_2 in the bubble decrease in synchrony with decrease of ambient pressure. The N_2 partial pressure outside the bubble exceeds PN_2 inside temporarily, so N_2 diffuses in and the bubble grows.

Consider the picture in Figure 1 for the case of a person breathing pure oxygen. If N_2 outside a bubble is zero or low, the tendency for N_2 to diffuse out is much greater than when there is N_2 outside to furnish a back pressure. If a person breathes some other gas instead of O_2, say a helium-oxygen mixture, outside N_2 will also go to zero, and N_2 will diffuse out rapidly. However, the helium will diffuse in, and there could actually be a volume increase if helium gets in faster than N_2 gets out (9,10).

Changes of PO_2 or PCO_2 in the tissue will be reflected by changes both in volume of the bubble and in PN_2 inside as a consequence of changes of PO_2 or PCO_2

inside; in addition, decompression brings on a short-lived volume change which is due to a readjustment of O_2 and CO_2 (8). Figure 2 illustrates this readjustment with some concrete numbers for a bubble decompressing to altitude. The CO_2 and O_2 are both given as 6% of the total pressure and also 6% of the total volume before decompression. After decompression, partial pressures of O_2 and CO_2 in tissue do not change appreciably -- as mentioned before, living tissue depends on fairly constant levels of O_2 and CO_2. However, the decrease of total pressure makes the O_2 and CO_2 a larger fraction of the total volume. Before decompression, O_2 and CO_2 accounted for 12% of the total -- afterward they account for 24%. If the decompression were instantaneous, O_2 and CO_2 would still be 6% of the total volume immediately after decompression but partial pressures would be only 3 kPa. Because of their high permeation coefficients, the two gases will rapidly diffuse into the bubble until they are 6 kPa. Therefore there is a step increase in volume which amounts to 12 % of the bubble volume in this example. It is seen that decompression causes the bubble to grow for three reasons -- physical decompression by Boyle's law, a short-term entrance of O_2 and CO_2, and a longer-term entrance of N_2 from tissues which became super- saturated by the decompression. The O_2 and CO_2 effect would be negligible at depth, but becomes appreciable in altitude decompressions.

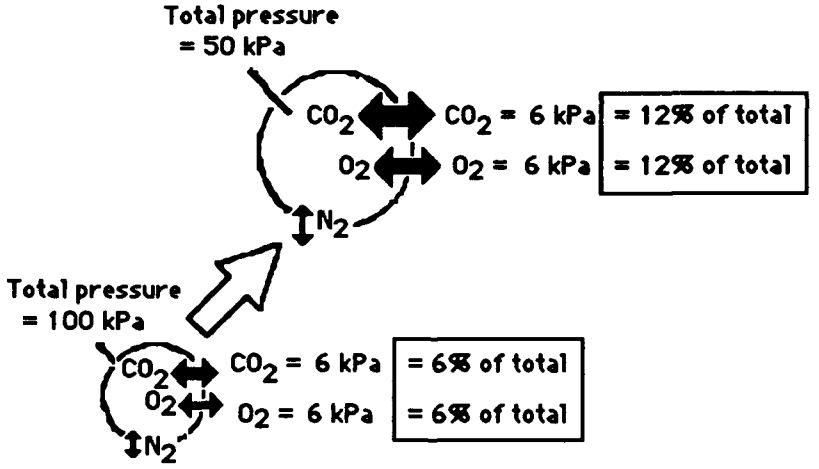

Figure 2. Diagram of a bubble which is subjected to decompression from one atmosphere (below) to a half atmosphere (above). A readjustment of O_2 and CO_2 results in a 12% increase of bubble volume.

Gas Nuclei

There are many issues about decompression sickness bubbles which are not as clear as the matters discussed above. A major problem is how bubbles originate. If surface tension at the interface between bubble and the surroundings behaves in the manner that is usually taught, a small spherical bubble should have much higher

pressure inside than a large bubble because the pressure due to surface tension is inversely proportional to the radius. It follows that small bubbles should be absorbed rapidly because of what can be called "surface tension squeeze." It might be logical to infer that bubbles cannot be small because if they were they would be squeezed away. Further logical thought might lead to the patently incorrect conclusion that it is impossible to have decompression sickness because if there cannot be small bubbles, there cannot be big ones. How can big bubbles exist if there are no baby bubbles from which they can grow? This logical problem has led to the popularity of the idea of "gas nuclei" or "bubble formation centers."

Do nuclei really exist, and if so, what are their characteristics? Data about the forces required to form bubbles *in vitro* can be interpreted to mean that little force is needed in minimally-clean preparations, but that when more precautions are made to clean the system, more force is required (11). This comparison suggests that a normally-copious supply of nuclei can be diminished by cleaning efforts. However, it need not apply to living creatures, which may have their own biological mechanisms for destroying or creating nuclei. Hemmingsen (12) reports that particles which ordinarily cause bubbling in water, presumably because they contain nuclei, do not bubble after being ingested by *Tetrahymena*. Apparently these small aquatic animals deactivate the nuclei in some way. On the other hand, Walsby (13) presents a very interesting description of how certain bacteria generate gas-filled vesicles in their protoplasm by aligning hydrophobic proteins into a definite structure. The evidence about characteristics of nuclei in gelatin is quite convincing (14,15), and experiments designed to demonstrate crushing of nuclei in animals seem to work, thus providing evidence that there are nuclei to crush (16), but it seems prudent to maintain an open mind about the nature of nuclei in living creatures.

A simple definition of a nucleus is that it is a persistent small quantity of gas that can grow to become a *bona fide* bubble. Simulations of bubble growth with a computer program led to the conclusion that the transformation from nucleus to bubble is an explosive positive-feedback process (17). Figure 3 shows the proposed positive-feedback loop. During a decompression, if nitrogen partial pressure inside the nucleus becomes lower than its counterpart outside (box 1), there is inward diffusion of N_2 (box 2), which increases the radius (box 3), which decreases the pressure due to surface tension (box 4), which lowers the total pressure inside (box 5), which lowers the PN_2 inside (box 6) thus further increasing the diffusion gradient (box 1 again), so more N_2 enters and the radius becomes larger, and so on. The loop continues action until the bubble radius is so large that pressure due to surface tension is negligible. This loop is analogous to lighting gasoline with a match -- a small influence has a big effect. It means that the transformation of nuclei into bubbles tends to be an all-or-none phenomenon.

The increased importance of O_2 and CO_2 in hypobaric decompressions could be reflected by an impact on the loop. The shaded arrow at the right in Figure 3 is intended to suggest that small changes of tissue O_2 or CO_2 could change the volume of the nucleus and set off the feedback loop by creating a gradient for N_2 where there

was not one before. For example, if CO_2 increased in the tissue, CO_2 entrance into the nucleus would change the radius. Even a small effect might be large enough to start the explosive loop action, with lowering of the PN_2 inside and entrance of significant amounts of N_2 to form a *bona fide* bubble.

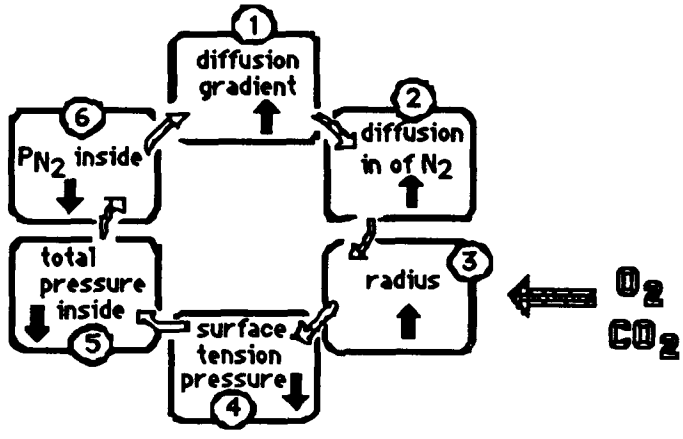

Figure 3. Positive-feedback loop which is thought to operate when nuclei are transformed into bubbles. (Redrawn from (17)).

Simulations of Bubble Dynamics

Figure 4 shows results of a computer program which solves diffusion equations to provide simulations of the growth and decay of decompression bubbles (18). The dashed barometric pressure trace falls from two atmospheres to one atmosphere (decompression from one to a half atmosphere would look much the same). When the barometric pressure changes, the partial pressure inside the bubble changes but PN_2 in tissue lags, so there is a time which can be called a "crossover" when a bubble can grow. The curve labeled "radius" shows that a pre-existing bubble first decreases in size due to the oxygen window, then enlarges due to the crossover, and then shrinks after the crossover is finished.

The left panel of Figure 5 shows a nucleus during a similar decompression. The PN_2 inside is higher than it would be in a bubble because of the pressure due to surface tension engendered by the small radius of the nucleus. During the decompression, PN_2 inside the nucleus falls but a crossover is narrowly avoided. If a crossover had occurred right at the angle at the end of decompression, the positive feedback transformation of nucleus into bubble would have occurred. The right panel of Figure 5 shows that a crossover does occur when the nucleus is slightly bigger than the one in the left panel. The gas-phase PN_2 falls drastically as the radius of the bubble grows explosively.

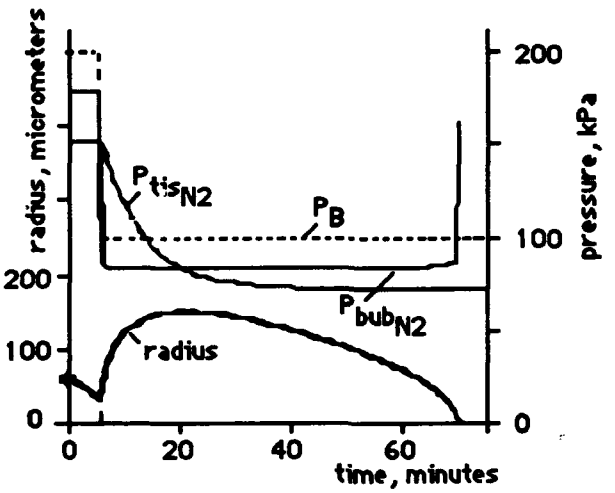

Figure 4. Simulation of growth and decay of a pre-existing bubble in the body when it is decompressed. (Redrawn from (18)).

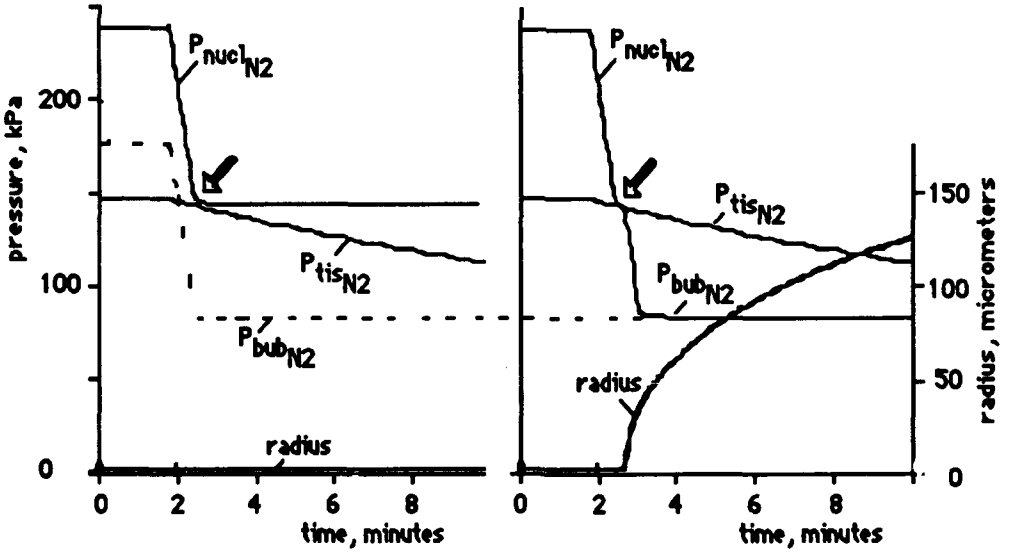

Figure 5. Simulation of the behavior of nuclei during decompression when transformation to a bubble is avoided (left panel) and when transformation occurs (right panel). (Redrawn from (17)).

Simulations such as those in Figure 5 indicate that the transformation is most likely during decompression, when tissue PN_2 is nearest to nucleus PN_2; yet it is a common finding that pain or damage is not noticed until later (19). There are reports that the more stressful the decompression, the less lag there is between decompression and symptom appearance, suggesting that the delay in less stressful situations is a period in which transformed nuclei grow to damage-producing size.

A simple generalization is, that to prevent a nucleus from transforming into a bubble or to keep a bubble from growing, one should keep PN_2 in the nucleus or bubble as high as possible and keep PN_2 in tissue as low as possible. One of the conclusions that arose from application of the bubble simulations to decompression sickness in divers is that slow ascent rates can keep PN_2 in the gas-phase high, so that the nucleus transformation process may be avoided. The left panel of Figure 6 shows how a slow ascent can make transformation from nucleus to bubble unlikely by causing nitrogen in the nucleus to fall slowly while the washout lowers the tissue nitrogen. The right panel of Figure 6 shows why slow ascents probably do not help in hypobaric decompressions. According to one series of experiments (20), the relevant tissue in hypobaric decompressions has a very slow washout (a halftime of 360 minutes). If so, the tissue trace would be essentially flat over reasonable time frames, so the PN_2 in nucleus and tissue would come close whenever the ascent finally ended. It would take an ascent that lasts for hours to have the kind of beneficial effect shown on the left panel.

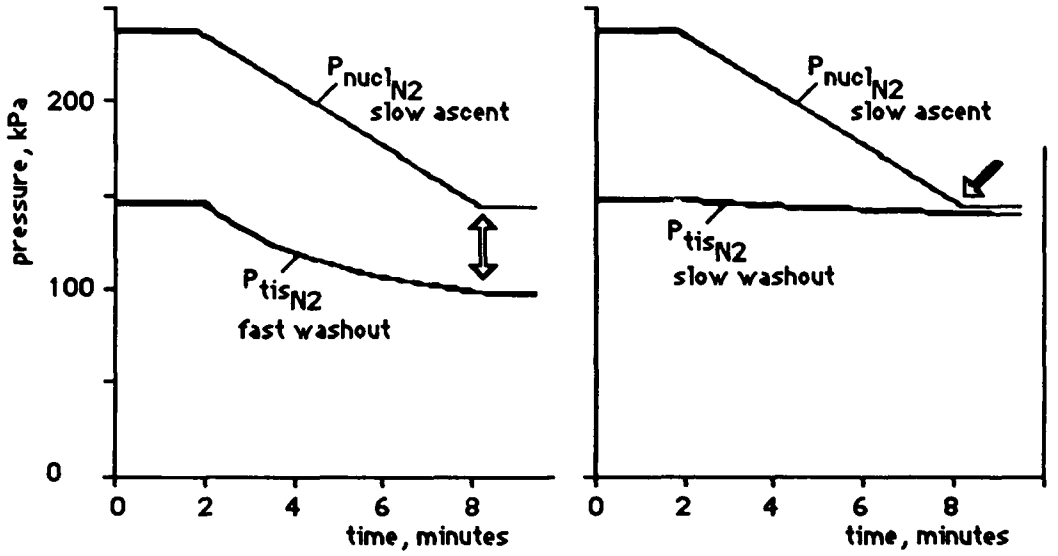

Figure 6. When washout is reasonably rapid, slow ascent may keep PN_2 in a nucleus above PN_2 in the tissue so that transformation of nuclei to bubbles is unlikely (left panel), but when tissue washout is slow, as in hypobaric decompressions, a slow ascent does not have a beneficial effect (right panel).

Uncertainties About Nuclei and Bubbles

Figure 7 imposes a structure on some questions about the origin of the bubbles which cause pain or damage in decompression sickness. The left column lists some possibilities for so-called "primordia"; these may be spherical nuclei, or nuclei of some other shape, or the origins of bubbles may be something else altogether. The primordia may give rise to one or more harmless intermediate forms, as shown by the column in the middle. The figure shows the possibility that a spherical nucleus could give rise to one or more of the four intermediate forms, but the other two kinds of primordia could do so also -- additional arrows are not shown to simplify the diagram. Finally, the intermediate forms presumably give rise to damaging forms, and again the possibilities are manifold. The damage-causing agents may be bubbles blocking blood vessels, as in pulmonary emboli, or a critical volume of gas in bubble form, or some other matter, such as changes in blood elements which are only secondary to bubbles. If intermediate forms do not exist, arrows should point from primordia to damaging forms. The figure shows a daunting array of uncertainties and presents a challenge for research workers to devise convincing experiments which will allow some of the possibilites to be ruled out.

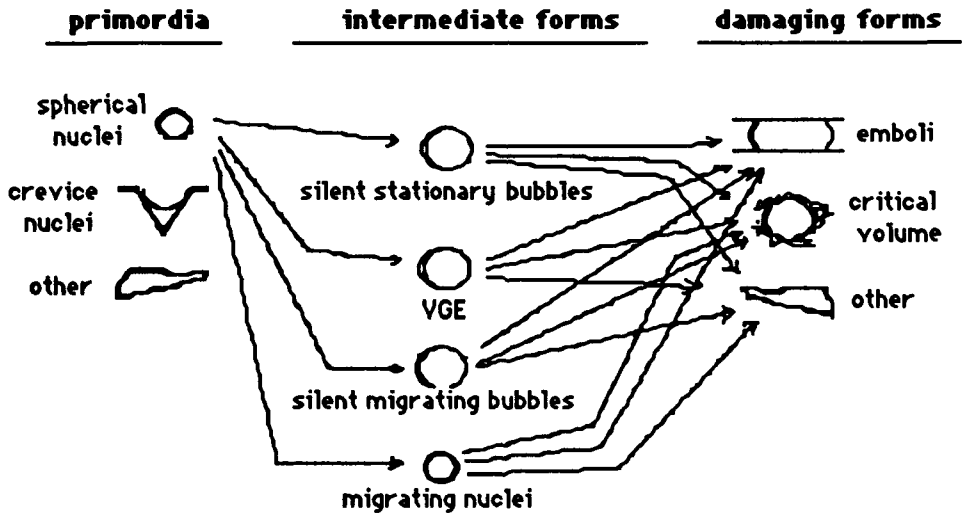

Figure 7. Chart showing some of the uncertainties about the process of formation of damage-causing bubbles in decompression sickness.

Figure 8, a whimsical picture of a decompression bubble wondering about itself, is a guide for partial summary of issues discussed above. Small bubbles should be absorbed by the surface tension effect and by the oxygen window, yet bubbles do arise, presumably from something small; the proposed positive-feedback loop indicates that nuclei move explosively through the small stage to become bubbles that are large enough to be immune to surface tension effects. The origin of bubbles

remains a matter for debate, or better, for imaginative experimentation. The concept of the oxygen window suggests that bubbles should be absorbed fairly rapidly after the decompression is over, at least in tissues which have a reasonable amount of blood perfusion, yet there are reports which suggest that bubbles persist for surprisingly long times in the body. Venous gas emboli migrate, but they seem to be harmless; do harmful bubbles originate *in situ* or arise from migrating forms? What is happening in the lag time between decompression and the appearance of signs and symptoms? It is hoped that future research will allow us, first, to decide which of the matters discussed here are simply conjectures and which correspond to real phenomena, and second, to manipulate the real phenomena in ways that elucidate their characteristics, and most to be hoped, to manipulate the real phenomena in ways that help to prevent or cure decompression sickness.

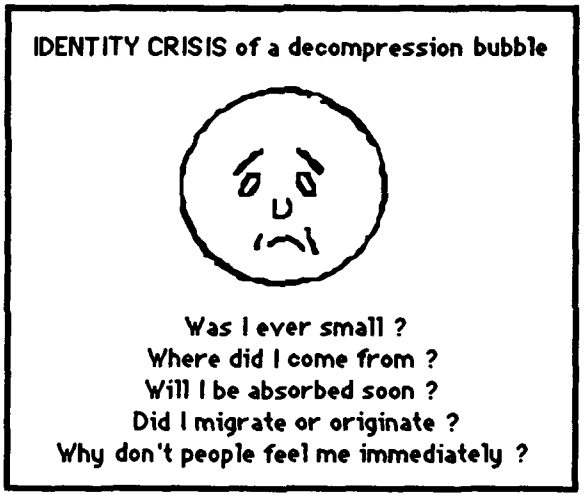

Figure 8. Some important questions about decompression sickness bubbles; see text.

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DR. VANN: Let us kick off the discussion of both the historical aspects and the behavior and nature of bubbles by looking at the specific DCS symptoms, not classifications such as Type I and Type II.

Let us start with what may be a reasonably simple, although obviously not too simple, problem. That is pain only decompression sickness. What I would like to ask is relevant to Behnke's observations in the 1940s, that if you expose the same individual to both altitude decompression and decompression after diving, he generally got pain in the same locations.

What differences would you expect, if any, between hypobaric decompression sickness and hyperbaric decompression sickness? Are the mechanisms the same, are you dealing with just fewer gas molecules so you can treat the hypobaric situation more easily, or is there some fundamental difference that we should be keyed into and we need to talk about today?

Is there any fundamental difference between a given bubble that is formed after decompression from 150 feet of sea water, and one formed from decompressing to 30,000 feet of altitude?

DR. LAMBERTSEN: I would like to help start the discussion. I think what we need to do is first consider bends pain. Historically people have thought about this for a long time. Long ago Boyle asked, "what is making this animal agonize?"

Many individuals have studied pain as a process. What is pain? Pain is chemistry. You would not hurt if there was not chemistry going on at the nerve ending. So if you will start with that, knowing that no matter what it is that makes you hurt, the ultimate cause of that hurt is a chemical reaction at the nerve ending, i.e., the release of substances which stimulate the nerve.

Furthermore, it does not have to be one thing. The concept that has been generated over these many decades is that there would be no such pain if there was not some formation of a gas phase. Granted, it has been said that maybe there is not. Let us just say that and then put that topic aside. Suppose there has to be a gas stage to create pain. Pain can come from multiple consequences of that gas phase. Chemical changes can come from multiple consequences of the gas phase formation. It can be hypoxia locally. It can be interference with blood flow for other reasons, locally. It can be rupture of tissues. All of these things were mentioned 350 years ago.

We could imagine that the bubble differences that Dr. Van Liew was talking about, namely the generation of a bubble volume or the size of a gas phase or a rate

of development of the gas phase, would modify the rate of development of the symptom. Also, it should not be just pain that we talk about. Again, people have attempted for years to answer this question.

It has been pointed out that if you have a disturbance in a region where that disturbance makes a difference, then you will probably have a symptom in that region. If you have a pain in an exercising muscle, it probably will not hurt. If you have pain in a ligament, which that exercising muscle is interfering with the circulation of, it may hurt in that ligament or tendon. If you have pain in your behind, in the buttock, in the fat pad, you will not have pain because it does not hurt. In that location, there is no phenomenon going on that makes masses of gas lesions make any difference. So I do not think we want to just talk about symptoms but, more importantly, discuss the systemic process. If we only talk about pain in an elbow, and ignore the bubbles Dr. Pilmanis showed which were accompanied by no pain in that elbow, you are making a mistake. I think you have to look at this process as though that symptom was not the important thing, but the disease was the important thing.

I hope this gets us back, first to the history and second to the systemic aspects of DCS. We are dealing with a multiplicity of processes, not symptoms. These processes do not necessarily produce symptoms. We should be dissecting these physiologic processes, not the symptoms.

DR. VANN: Let me address another question to Dr. Van Liew. We are dealing with gas nuclei here, and a particular difference between a hyperbaric exposure as opposed to a hypobaric exposure is that you have an initial compression phase. You showed that the compression has a tendency to shrink those gas nuclei and to make them go away. Would you then possibly be dealing with a different population of nuclei after a hyperbaric decompression than you are after a hypobaric decompression?

DR. VAN LIEW: Others have shown that nuclei tend to go away by being squeezed. My work is all theoretical. In order to begin talking about nuclei at all, I had to say that I did not know what one was, and then I had to ascribe some characteristics to it. I did the simplest possible thing; I just said a nucleus was a spherical gas quantity that would not get any smaller than a certain size.

One could go ahead and make rules that would say that nuclei have other characteristics, such as being squeezed and going away. If you tell me the rules for the squeeze then I can put that into my mathematical system and work it out. I think the gist of my talk today was that we really know very little about even whether or not nuclei exist in animals, or what the nature of nuclei are. It is easy enough to model something if you make up the rules for it, but we really do not know what rules we should use.

DR. BUTLER: The concept of bubble nuclei makes good sense to me. The first question I have is, how do we stabilize? Then, how are these nuclei so stable? If

they are gas pockets, surface tension has to come into play, unless there is no surface effect. The only way I can see that you could have no surface effect is to have some sort of hard shell. A hard shell composed of some blood products could generate over time.

With decompression, large gas gradients exist. This hard shell could be permeable, but the permeability may be a lot different than you would expect just with naked blood. With either decompression to altitude or decompression from hyperbaria, you have newly imposed large gas gradients. Could these gradients result in rupture of the shell? Could you then apply your equations for growth? Could this approach also fit into some of Dr. Vann's work involving a hyperbaric pre-exposure? The pre-exposure could rupture the nuclei, resulting in definition of the factors for predicting growth.

Also, could bacteria models define mechanisms for breaking the shell, activating the nuclei, and then falling victim to your equations for growth?

DR. VAN LIEW: Yes, the answer is yes, yes. All those things could be. If your premises are true, then what you get out is true. But we do not know about your premises. Is a nucleus made of something hard, is it something in a crevice, is it something that has surfactant materials from the blood? I do not see any good reason for opting for one or another of those possibilities at this time.

DR. BUTLER: Your equation would depend on there being gas initially present?

DR. VAN LIEW: Right. All I have got going for me is a model and a model is a statement that says, "if the following are true, it follows that...." I think that it is a worthwhile exercise because it gives you a framework to use.

DR. FRANCIS: There are attractive reasons for believing that the pain type of decompression illness relates to static tissue bubbles, as opposed to embolic ones.

I recall the experiments of Piccard, back in the 1940's. He took a liter of water, probably in a rather dirty container, and allowed this beaker of water to saturate with its ambient gas under two different conditions. The first condition was at one atmosphere. Having allowed this liquid to saturate, he then decompressed it to one-fifth of an atmosphere pressure and watched for gas to evolve from the water. In a second experiment, the liquid was compressed to five atmospheres, allowed to saturate, and then decompressed to one atmosphere.

He noticed that although the volume of gas released in the two experiments was the same, the rate of gas release was different in the two conditions. From the hyperbaric exposure the gas was evolved over a period of a few seconds, whereas from the hypobaric exposure the gas took many minutes to evolve from water. I wonder whether this, with reference to static tissue bubbles in decompression illness,

has relevance. If you are looking for gas to evolve in a tissue, you are dealing with a dynamic situation of tissue gas washout. If it is going to take a long time for bubbles to form, then there is much more time for the tissue to get rid of the gas. This is the gas that would form the bubble that is going to cause the problem.

I wonder if you would care to comment on this. Are we dealing with different dynamics for hypobaric compared with hyperbaric decompression?

DR. VAN LIEW: I think the experiment you cite suggests we are dealing with different dynamics. My ideas about the transformation of nuclei of bubbles would suggest that both of them transform, that things transform at the time of decompression in both cases, but that then it took the bubbles longer to grow in the hypobaric situation because there was less gas available. The amount dissolved in the water is less.

DR. FRANCIS: There were fewer bubbles available to form a stable bubble.

DR. VAN LIEW: Right.

DR. VANN: Do your simulations show that it does take longer?

DR. VAN LIEW: No, it does not, and I do not understand why yet. So that is work for future study.

DR. WENZEL: If you have the five-powered amount of gas in the water, and you compress it to the five-powered pressure, then you have the same size of gas bubbles. So I do not see why then the hypobaric bubble should form slower.

DR. FRANCIS: Piccard did actually answer that. He calculated the number of molecules of gas that were necessary to form a stable bubble, i.e., one that would not collapse in on itself. And he came to the conclusion that there were six or seven times more molecules required to form a stable bubble in the hypobaric decompression compared with the hyperbaric decompression. It seems you are dealing with the probability that these molecules of gas will assemble at one given point and form a bubble. Maybe that is how this happens, but given that as an assumption, bubble formation is more likely in the hyperbaric decompression compared with the hypobaric decompression. It will eventually happen under both circumstances to the same extent, but it happens more rapidly in the hyperbaric decompression because of this phenomenon.

DR. WENZEL: The amount of surface tension, as a contribution, remains the same in both cases?

DR. FRANCIS: I think he made that assumption, yes.

DR. HAMILTON: With regard to bubble stability, the lung surfactant was discovered because bubbles were found to be stable. Normally a bubble will

disappear. They were stable because there was a material on the bubble that did away with the surfactant squeeze. In experiments with lung surfactant, you have a material that is on the surface that is not acting as a surfactant when it is spread out. When it is compressed it becomes a surfactant and has a very strong effect. I do not see any reason, if you shrink a bubble small enough in some other tissue than the lung, that there might not be enough material in the fluid to again form more surfactant, such that at some point there would essentially be zero surfactant squeeze effect. You can see it very clearly in a lung extract.

DR. VAN LIEW: My understanding of the lung extract is that the surfactant that is there is attracted to a gas fluid interface, but it does not make the surface tension go to zero. It tends to lower it. It lowers even more when the bubble is getting smaller than when it is getting bigger, so it is a material that allows the surface tension to be a variable, but it does not take it all the way to zero.

However, it seems quite reasonable that if there is some material around a bubble, and the bubble gets smaller and smaller, that material would come to a state at which it would resist any further change. It would be poking on itself so hard that it would not change. In that case, the surfactant would have changed from being a surfactant to being a rigid substance.

DR. LAMBERTSEN: It is important again to point out that the work that Harvey did with his physical and animal studies of nucleation included the mathematics of conversion from nucleus to bubble and a discussion of the role of substances of surfactant character. The mathematics and concepts are spelled out in his papers, item by item. We should crosslink that very careful multiyear study with the present activity and concepts.

PHYSIOLOGY OF DECOMPRESSION SICKNESS

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Bubble Formation

Bubbles form as a result of supersaturation. Supersaturation is the excess gas tension and water vapor pressure over the absolute pressure,

$$\text{Supersaturation} = Pg + Pv - Pa \qquad (1)$$

where Pg is dissolved gas tension, Pv is vapor pressure, and Pa is the absolute pressure. The probability that bubbles will form increases with the supersaturation.

Bubbles can form *de novo*, from nothing, or from pre-existing gas nuclei. As *de novo* bubble formation requires gaseous supersaturations of over 100 atm (1,2) while bubbles in animals and humans are detected after less than 0.5 atm of decompression (3-5), *in-vivo* bubbles would appear to originate from gas nuclei.

As equation (1) indicates, supersaturation can result from a reduction in the absolute pressure (Pa) as well as from excess gas tension. In a hanging water column, for example, hydrostatic tension reduces the absolute pressure at the top of the column, and the resulting bubble formation limits the height to which water can be lifted by a suction pump (6).

During the negative pressure phase of a sound wave, the absolute pressure becomes less than zero and causes acoustic cavitation (7). Negative pressure and cavitation occur due to viscous adhesion in the lubricant between a journal and bearing (8). Viscous adhesion generates negative pressure in any liquid between moving surfaces. The pressure becomes more negative with the closeness of the surfaces and their relative velocity and can exceed hundreds of negative atmospheres (9,10). These pressures may be sufficient to cause *de novo* bubble formation as the lowest negative pressure that has been sustained before cavitation is -270 ata (11).

Vacuum Phenomena and Decompression Sickness

Viscous adhesion and bubble formation are the cause of the cracking sounds which occur in joints when their articular surfaces are pulled apart (12,13). Cracking

occurs when vapor-filled bubbles collapse. These bubbles can persist and expand, however, if a joint is put in traction. The expanded bubbles are known as "vacuum phenomena" and are readily detected by x-ray and CT-scan.

Vacuum phenomena are found in the fingers, wrists, elbows, shoulders, spine, sacroilliac joint, illium, symphis pubis, hips, and knees (14). Figure 1 shows a typical vacuum phenomenon of the hip in a one-year-old girl (15). A spontaneous vacuum phenomenon, visible without traction, is present on the left. On the right, the volume of the void increases when traction is applied.

Figure 1. A vacuum phenomenon in the hip of a one-year-old girl (15). (a), spontaneous and (b), with traction.

The response of vacuum phenomena to decompression is suggested in Figure 2. On the left, vacuum phenomena were produced by traction of the wrist (16) while, on the right, similar voids were found after decompression to altitude (17).

Gas in a joint space, however, be it a vacuum phenomenon or the result of decompression, does not cause pain, and vacuum phenomena *per se* are not suggested as the cause of decompression sickness. Rather, the hypothesis is that

viscous adhesion at the interfaces of moving surfaces generates both vacuum phenomena and the gas nuclei which grow into the bubbles that cause DCS.

Figure 2. (a) Vacuum phenomena of the wrist produced by traction (16). (b) Gas spaces in the wrist of a subject at 35,000 feet (17).

Bubbles and pain were statistically correlated in radiographs such as that in Figure 3 of a subject with moderately severe pain at 35,000 feet (17). Pain was associated with gas lesions present as discrete round bubbles in periarticular tissues (Site A) and as fine longitudinal streaking suggesting a distribution along tendon and muscle bundles (Site B) (18).

Table 1 presents the results of 85 similar exposures (18). When one of a subject's knees became painful, both knees were radiographed. Discrete bubbles and longitudinal streaking were highly correlated with pain at p-values of less than 0.00001. There was no correlation between pain and gas in the joint space.

Table 1. The Results of 85 Radiographic Studies of Both Knees at 35,000 Feet When Pain was Present in One Knee Only (18).

$p << 0.00001$	DCS	No-DCS
Bubbles	49	3
No Bubbles	8	25

$p < 0.0001$	DCS	No-DCS
Streaking	48	9
No Streaking	9	19

$p = n.s.$	DCS	No-DCS
Gas in Joint	57	28
No Gas in Joint	0	0

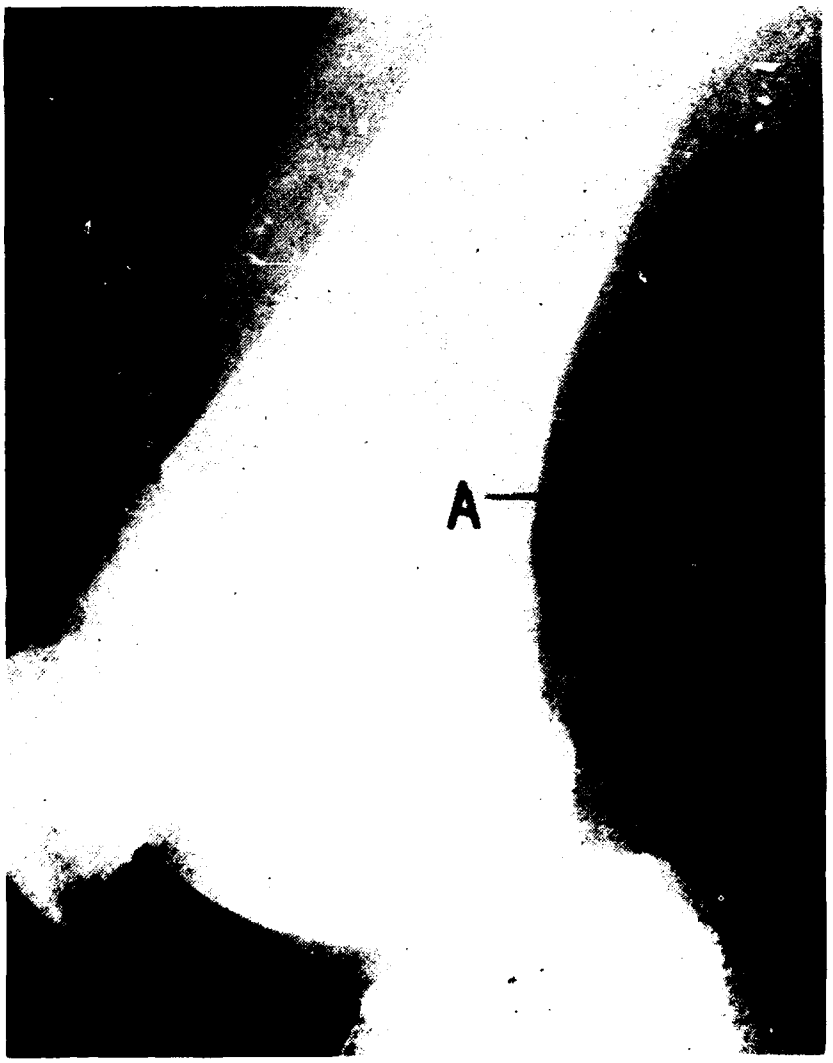

Figure 3. The knee of a subject with moderately severe bends after 3 sets of 5 deep knee-bends during 10 minutes at 35,000 feet (17). (A) A collection of discrete and irregular bubbles. (B) A wavy streak of gas probably along a fascial plane or tendon.

Vacuum phenomena of the spine are found within disks, facet joints, vertebrae, and the spinal canal itself (14). Figure 4 is a CT-scan of a 52-year-old man with chronic back pain and gas in the spinal canal (19). Spinal vacuum phenomena are diagnostic of degeneration and become more common with advancing age (20). Perhaps increased bubble formation with age is related to the parallel increase in DCS risk (21).

Figure 4. A CT-scan demonstrating a gaseous void within the spinal canal at the L3 level (19).

During decompression, a spinal vacuum phenomenon would grow and, like a herniated disk, might impinge on a nerve root. This explanation would not account, however, for the voids and punctate hemorrhages observed in the white matter of animals and humans with spinal DCS (22). The origin of these bubbles, referred to as "autochthonous" or *in situ*, is uncertain, but viscous adhesion during shearing motion between cord fibers is a possible explanation.

Vacuum phenomena may be only rarely related to decompression sickness, but their existence demonstrates that bubbles are routinely present in humans at sea level. If the viscous adhesion that generates vacuum phenomena also produces the gas nuclei which cause DCS, then increased motion during exercise would be expected to generate more nuclei and greater DCS risk. Exercise before decompression is associated with increased bubble formation in controlled animal studies (23,24) while anecdotal reports link weightlifting and long-distance bicycle racing with increased DCS risk in humans (25,26). This relationship is relevant to the potential use of exercise for countering the physical deconditioning which occurs during life in microgravity. Conversely, reduced physical stress in microgravity might decrease the production of gas nuclei and lower the decompression risk in space which might explain the apparent absence of DCS in space as opposed to a 20-30% incidence of symptoms during similar ground-based exposures (27). Ground-based investigations of the effects on DCS risk of both bed rest and heavy exercise appear warranted.

Bubble Formation in Blood

Bubbles are routinely detected in the blood and can have significant consequences which are discussed elsewhere in this workshop. Bubbles are so common in the blood during decompression that they are often assumed to form there. This has been a difficult question to resolve experimentally because blood is easily contaminated with air during handling. The problem can be circumvented, however, by isolating blood within its natural containment system, a blood vessel. This method was first used in 1774 by Erasmus Darwin (28). We have repeated and extended Darwin's studies (29).

Blood-filled sections of inferior venae cavae were isolated between two sutures and removed from freshly sacrificed rats, rabbits, and dogs. When immersed in saline and decompressed to an altitude of 60-75,000 feet, no bubbles were observed in the isolated blood. This observation is contrary to the appearance of venous bubbles in humans and animals at altitudes above 12,000 feet (3).

In a second study, a section of rat vena cava was isolated between two sutures but was not removed from the animal. Thus, the isolated vessel remained connected to the heart and to the distal vena cava. With its chest open, the rat was compressed to 120 ata on air and left for eight hours to allow blood in the vena cava to saturate with air by diffusion. Upon rapid decompression to ground level, there were no bubbles in the isolated vessel, but both the heart and distal vena cava contained bubbles.

These results imply that bubbles do not form in blood which is isolated from the circulation, but their presence in unisolated blood suggests they might form in tissue. Bubbles expanding in tissue during decompression could enter the circulation through ruptured capillaries and expand centrally in dead animals or be carried centrally by the venous circulation in live animals. The best evidence linking peripheral bubble formation, vascular disruption, and central venous bubbles is from Lambertsen's studies

of cutaneous counterdiffusion in which supersaturation of the skin led to bubble formation, subcutaneous bruising, and large volumes of venous gas emboli (30).

If this scenario is correct, arterial bubbles would not occur unless, as discussed by Butler and Garrett in this workshop, venous bubbles were arterialized through the heart or lungs. An unphysiological exception to this rule, known as the "arterial paradox," appears to occur when decompression is faster than the circulation rate, and tissue bubbles expand retrograde into arterial vessels (31).

If bubbles form extravascularly in tissue, they do not appear to form in all tissues. Using implanted arterial and venous Doppler probes, Powell and Spencer (32) studied bubble formation in the kidney and brain of sheep rapidly decompressed from hyperbaric exposure. Bubbles were not observed in the venous blood of these organs unless arterial bubbles were detected first. If bubble formation is a result of viscous adhesion, bubbles would not be expected to form in organs such as these which lack moving surfaces.

The Oxygen Window

Bubbles grow by the inward diffusion of nitrogen from tissue, but oxygen, carbon dioxide, and water vapor play an important role at altitude in establishing the nitrogen gradients. Figure 5 shows the gases in the lung, tissue, and a bubble at 20,000 feet. The height of the bars represents the sum of all gases. In the lung and bubble, this sum equals the barometric pressure by Dalton's law. The subject is breathing oxygen, and there is no nitrogen in his lungs. Carbon dioxide and water vapor are the only other gases.

In tissue, the metabolic conversion of O_2 to CO_2 reduces the tissue oxygen tension to below its level in the lung, but the CO_2 tension rises only slightly because CO_2 is some 20 times more soluble than oxygen. The excess nitrogen in tissue can leave by two paths. It can be carried to the lungs by perfusion as discussed shortly, or it can diffuse into the bubble causing it to expand.

In the bubble, O_2, CO_2, and water vapor are controlled to their tissue levels while N_2 makes up the remaining partial pressure. If the altitude is increased to 30,000 feet, the O_2, CO_2, and water vapor pressures remain nearly unchanged, but the PN_2 is reduced. The reduced PN_2 increases the N_2 gradient between the tissue and bubble leading to faster bubble growth. At altitudes somewhere above 42,000 feet, equilibrium between gases in the bubble and tissue is impossible, and the bubble grows without bound.

Exercise at altitude is well known to increase DCS risk. In a study reported by Cook (33), exercise had the effect of an extra 5,000 feet of decompression. Figure 5 illustrates a possible explanation for this phenomenon. Viscous adhesion around a

bubble during exercise might reduce the pressure in the bubble. As would additional decompression, this reduced bubble pressure would increase the N_2 gradient responsible for bubble growth.

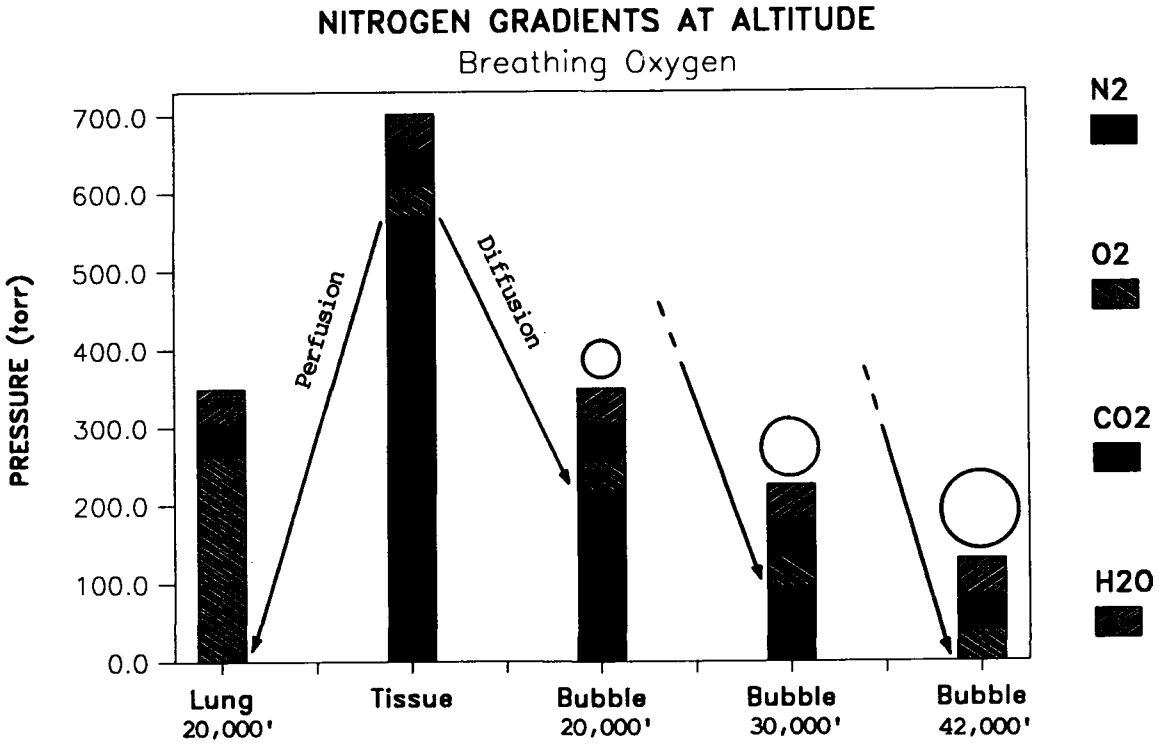

Figure 5. Nitrogen gradients between the lung, tissue, and a bubble at altitudes of 20,000, 30,000, and 42,000 feet. The subject is breathing oxygen.

Figure 6 shows the N_2 gradients while the bubble is resolving after recompression to sea level and to two atmospheres. Again, the subject is breathing oxygen. At sea level, the total gas pressure in the lung is 1 ata. As at altitude, nitrogen in tissue is carried to the lung by the circulation. This is the basis for pre-breathing oxygen before decompression.

In the bubble, O_2, CO_2, and water vapor are controlled to their tissue levels -- a total of about 130 torr -- and the remaining partial pressure -- about 640 torr -- is N_2. The gradient between N_2 in the bubble and in tissue is known as the "oxygen window." Since the PN_2 in the bubble exceeds its tissue tension, N_2 diffuses back into tissue, and the bubble resolves. If the subject is compressed to 2 ata, the O_2, CO_2, and

water vapor pressures do not change, but the N_2 for resolving the bubble increases by a factor of two. This is the basis for treating decompression sickness with hyperbaric oxygen.

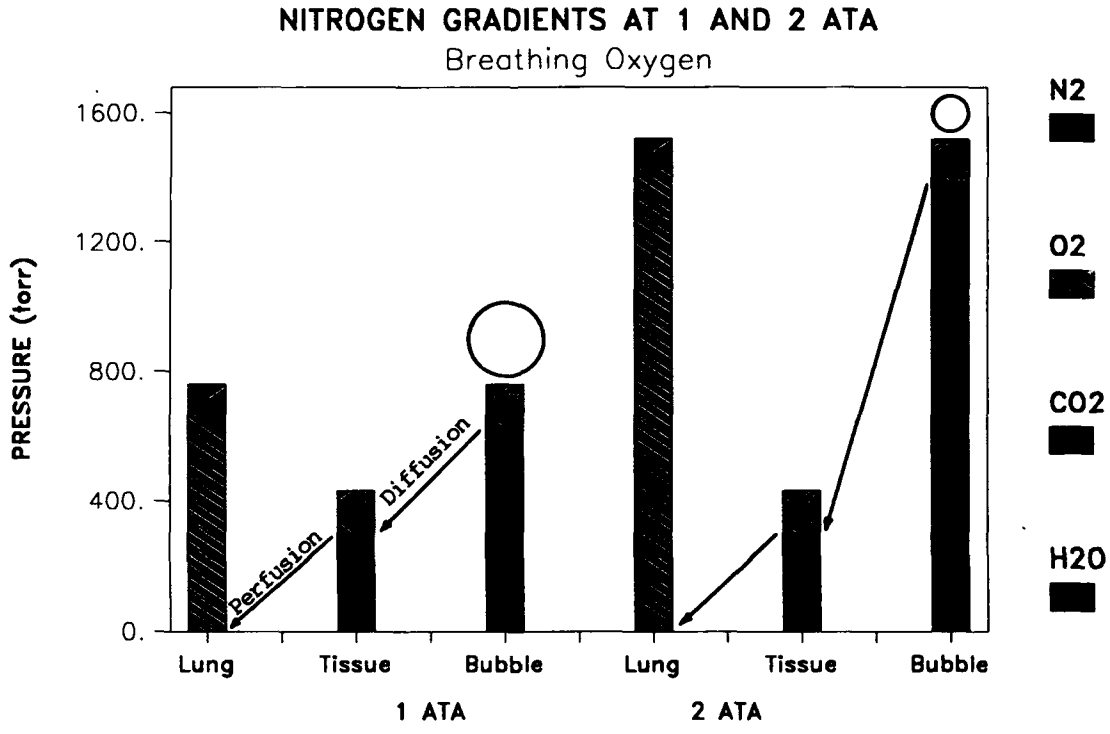

Figure 6. Nitrogen gradients between the lung, tissue, and a bubble at 1 and 2 ata. The subject is breathing oxygen.

Bubble to Tissue Nitrogen Exchange

Diffusion and perfusion are in series when a bubble is present (Figure 6), and N_2 in a bubble must diffuse back into tissue before it can be removed by blood flow. Thus, N_2 in a bubble is eliminated more slowly than dissolved N_2. Figure 7 shows Van Liew's simulations of the concentration gradients of oxygen, helium (He), and N_2 around a dissolving bubble (34). Oxygen has the steepest gradient because it is metabolically consumed. The He and N_2 gradients extend further into tissue because they are eliminated only by perfusion. Nitrogen has a steeper gradient than He because N_2 is less diffusible than He.

Tissue to Blood Nitrogen Exchange

Diffusion gradients become negligible several millimeters away from a bubble (Figure 7), and diffusion distances between capillaries are so short in most tissues that intercapillary domains are essentially well-stirred. Nitrogen exchange in these domains can be considered to be perfusion-limited. Diffusion is more likely to affect N_2 exchange on a regional level such as illustrated in Figure 8. Figure 8a shows N_2 diffusing between adjacent well-stirred tissue regions that are perfused at different rates or have different inert gas solubilities (35), and Figure 8b shows N_2 diffusing between adjacent arterial and venous vessels (36). Arteriovenous diffusion shunting allows N_2 to by-pass a tissue in which the intercapillary domains are otherwise perfusion-limited. Nitrogen exchange in such a tissue would be slower than expected on basis of perfusion alone. This may be the reason that the N_2 exchange halftimes in decompression models must be so very long.

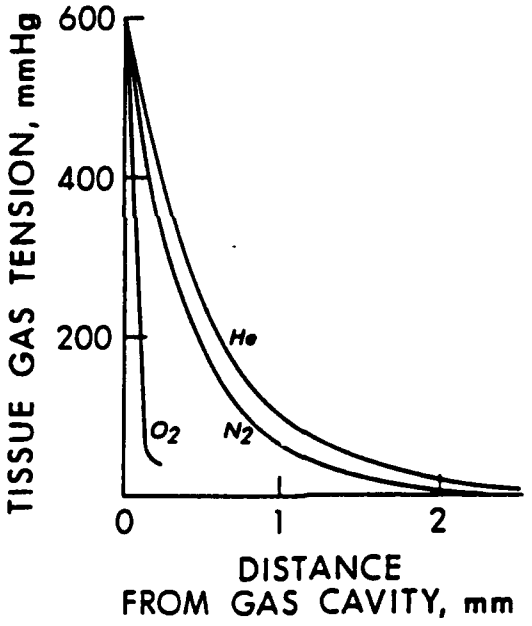

Figure 7. Simulations of the concentration gradients of oxygen, nitrogen, and helium around a gas cavity in tissue (34).

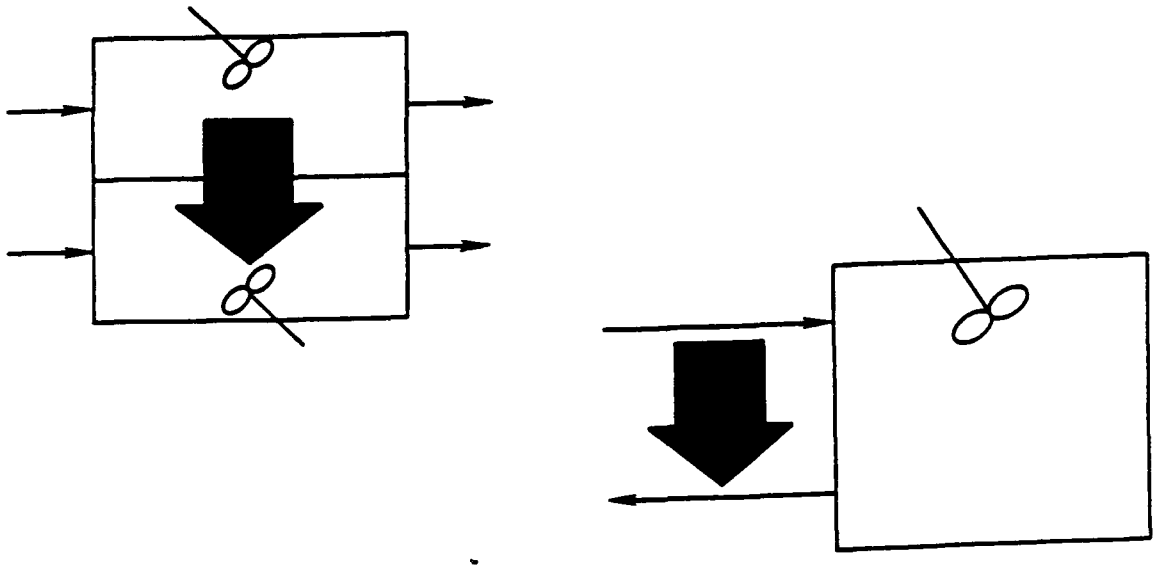

Figure 8. (a) Inter-tissue diffusion between perfusion-limited (well-stirred) tissues. (b) Arteriovenous diffusion shunting around a perfusion-limited tissue.

Exercise and Microgravity Effects

If the exchange of inert gas is, to a large extent, perfusion-limited, then factors which affect perfusion also will affect N_2 exchange. We are investigating how such factors influence respiratory N_2 elimination during 3.5 hours of O_2 breathing and the subsequent risk of decompression sickness at 30,000 feet (37,38). Figure 9 shows N_2 elimination curves from one subject under conditions of supine rest, 6° head-down rest, and seated work at 25 watts. Nitrogen elimination was greater in the head-down position than in the supine position but greatest during work.

Current studies are comparing 25 watts of supine work to rest with head-down tilt, immersion in thermoneutral (35°C) water, and continuous infusion of the vasodilator sodium nitroprusside (nipride). The resting studies attempt to simulate the acute effects of microgravity. As Figure 10 shows, however, none of these simulations eliminates N_2 as effectively as light work.

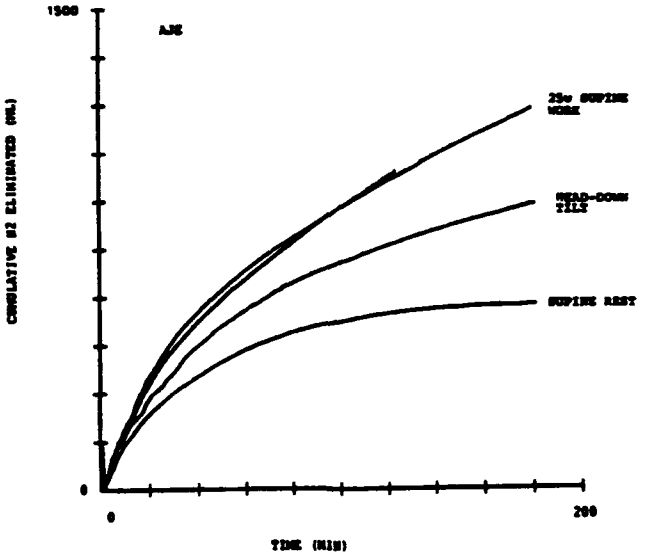

Figure 9. Whole-body nitrogen elimination through the lungs measured in an oxygen breathing subject during supine rest, head-down rest (6° tilt), or 25 watts of seated arm and leg work.

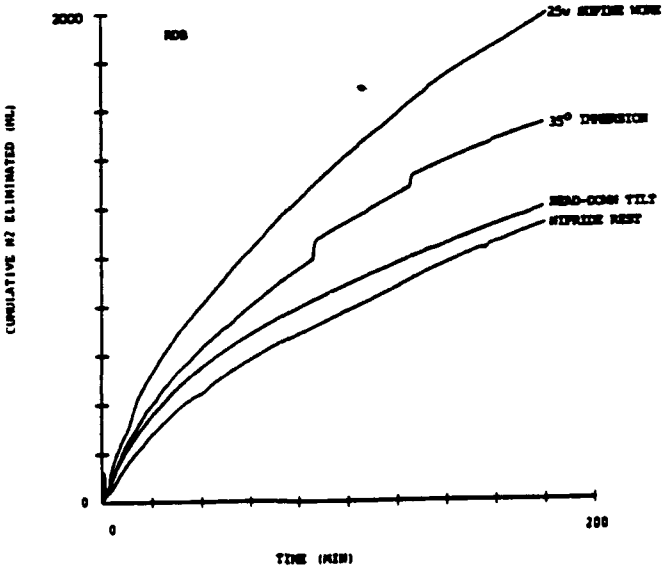

Figure 10. Whole-body nitrogen elimination through the lungs measured in an oxygen breathing subject during continuous infusion of the vasodilator sodium nitroprusside (nipride), head-down rest (6° tilt), immersion in thermoneutral (35°C) water, or 25 watts of supine arm and leg work.

Of all the pre-breathe conditions tested, only work has had a statistically significant effect on DCS incidence (Table 2). While this is consistent with the observed N_2 elimination, the specific correlation between DCS and eliminated N_2 is poor suggesting the tissues responsible for pain-only symptoms contain little N_2 compared with other tissues. The best correlation between DCS and N_2 elimination was during the last 30 minutes of measurement where bends-free subjects eliminated 70% more N_2 than subjects with bends (Figure 11). Thus, tissues responsible for pain-only symptoms appear to be among the slowest in the body to eliminate N_2.

Table 2. The Effect of 25 Watts of Work During 3.5 Hours of Oxygen Pre-breathing on DCS Incidence at 30,000 Feet.

p = 0.001	DCS	No-DCS	% DCS
Seated & Supine Rest	13	10	57%
Seated & Supine Work	1	16	6%

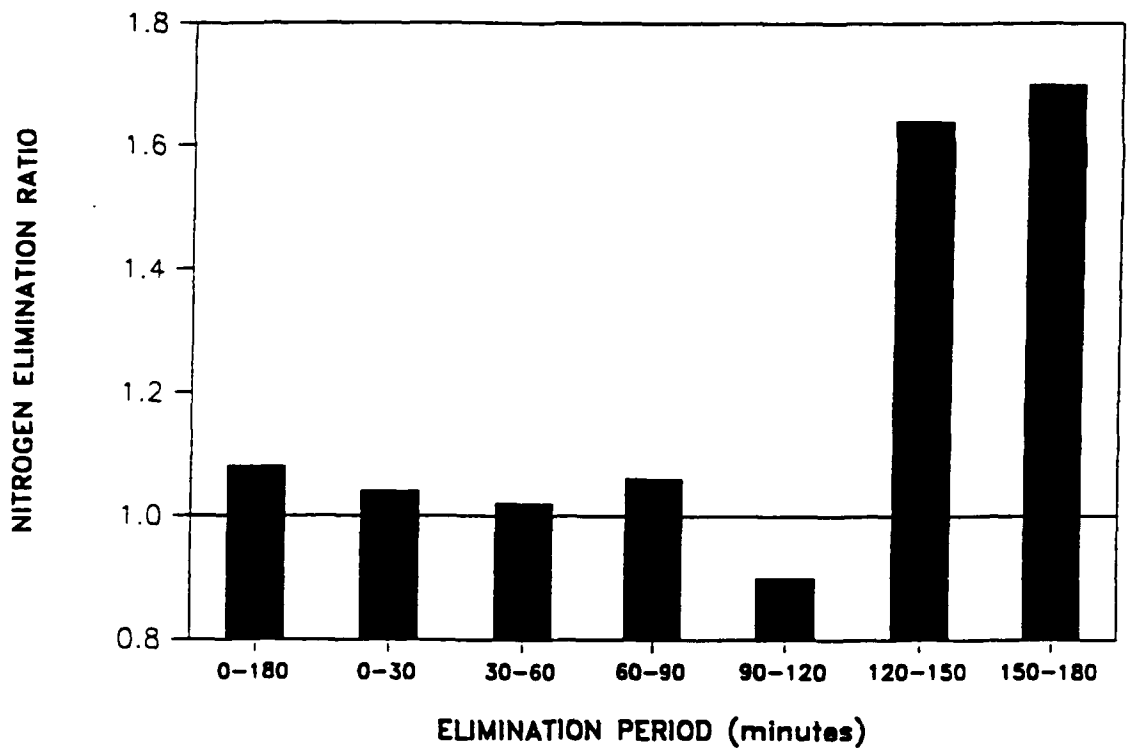

Figure 11. The ratio of nitrogen eliminated by subjects who did not develop DCS and subjects who did develop DCS. Ratios are shown for total nitrogen elimination over 180 minutes and during 30-minute intervals.

Conclusion

While there are many uncertainties concerning the physiology of decompression, it is becoming apparent that bubbles in tissue are a normal occurrence and that factors which influence both bubble formation and perfusion are important determinants of DCS risk.

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CARDIOPULMONARY EFFECTS OF DECOMPRESSION BUBBLES: Physiology of Bubbles in the Pulmonary and Systemic Circulation

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Supported in part by NASA Grant NAG 9-215

INTRODUCTION

This report represents an overview of the cardiovascular effects of decompression bubbles. Although the more common occurrence of decompression sickness (DCS) relates to limb pain, especially in or around the joints, the detection of circulating venous bubbles is often reported. These intravascular bubbles pose the more serious threat of adverse cardiopulmonary impact. The cardiovascular effects of DCS bubbles are represented by a myriad of symptoms ranging from local blood flow abnormalities, due to mechanical blockage of the involved vessels, to complex neurogenic effects or complete circulatory collapse. The scope and magnitude of the problem are directly related to the degree of decompression (pressure/time relationship) and hence the amount of gas bubble formation. This report examines the origin of DCS bubbles, their distribution throughout the body to the effective target organs and the resultant cardiovascular effects.

Origin of Decompression Bubbles

DCS results from the evolution of dissolved gas bubbles that are formed in the tissues and blood when the surrounding pressure is reduced to a point where supersaturation occurs. Altitude decompression from one atmosphere to reduced ambient pressure releases bubbles more slowly than occurs with decompression from increased ambient pressures (34), even at the same absolute ratio of pressure change (68).

Theories of bubble formation are described by two principal mechanisms. The first involves *de novo* formation which requires supersaturations in excess of 120 atmospheres, while the other consists of bubble formation from gas nuclei which are preexisting and require supersaturations of fractions of an atmosphere (77). Numerous factors influence the degree of bubble formation such as exercise, pressure, and time criteria as well as individual susceptibilities including age, fat content, etc. (1, 34). Bubbles may be formed extracellularly such as in the eye, synovial, amniotic and cerebral spinal fluids (9) or directly within the interstitial spaces (34). Lymphatic

bubbles (9) represent an especially interesting and often overlooked category that may impact directly at the site of formation or cause obstruction to lymph drainage from nearby tissues.

Intravascular bubbles formed after decompression have been reported as early as 1857, and their prevalence in the veins rather than the arteries was originally described by Bert (7). With the development of Doppler ultrasound, precordial detection of venous bubbles is now widespread and is routinely reported even with asymptomatic decompressions, as originally referred to as "Silent Bubbles" by Behnke et al. (6). The occurrence of venous bubbles prior to arterial detection is widely accepted (6, 38, 39, 48, 61, 74) although contradictory reports have appeared in the literature (59). Additional confusion exists concerning the distribution of intravascular bubbles considering the abundant evidence of their ability to pass through the systemic (35, 76) and pulmonary circulation (11, 16, 19, 20, 63, 71).

Target Organs of Decompression Bubbles

Lungs:

An obvious trend emerges from the numerous reports of venous bubble formation after decompression suggesting that a relationship exists between the extent of decompression and the amount of gas phase formation. There is a resultant dose/response relationship between size or number of bubbles and symptoms (34). The consequences of these bubbles include hematological, neurological, biochemical, surface, mechanical, fluid balance and hemodynamic changes affecting either the specific target organ or the whole organism (14, 49, 52, 53, 67, 78).

Venous bubbles are carried into the right heart and then circulated into the lungs (Figure 1). The adverse effects of DCS bubbles obstructing the pulmonary circulation are widely recognized (6, 8, 9, 28, 52, 53). Mechanical obstruction and vasoconstriction cause elevations in pulmonary artery pressure (Ppa), right ventricular pressure (70), and pulmonary vascular resistance (PVR). The vasoconstriction is due to both direct vascular reflexes and local mediator release (27, 82). Active pulmonary vasoconstriction likely occurs when small muscular arteries (100-500 micron diameter) are obstructed by bubbles. Pulmonary vasoconstrictor mediators are released from aggregated platelets and leucocytes. Experimental studies in which venous air emboli (VAE) have been infused have shown that the amount of gas infused results in a linear dose/response increase in PAP (Figure 2) (19). Subsequent increases in PVR, right ventricular and central venous pressures impose greater stress on the right heart often culminating in a decrease in cardiac output and arterial blood pressure if sufficient gas volumes are present (Figures 3, 4, 5) (19). Left ventricular end-diastolic pressures are usually unchanged after VAE (Figure 6) (19, 24). The occurrence of right ventricular failure has been attributed to myocardial ischemia as a result of diminished coronary artery blood flow due to both the decrease in aortic diastolic pressure and the significantly increased right ventricular pressure (50). With excessive volumes of

pulmonary vascular gas, resultant ischemic type changes can occur. Recent evidence indicates that the subsequent reperfusion that occurs after blood flow is returned to normal may exacerbate the injury, possibly due to accumulation of lysolipids (15), arachidonic acid metabolites, or oxygen radicals.

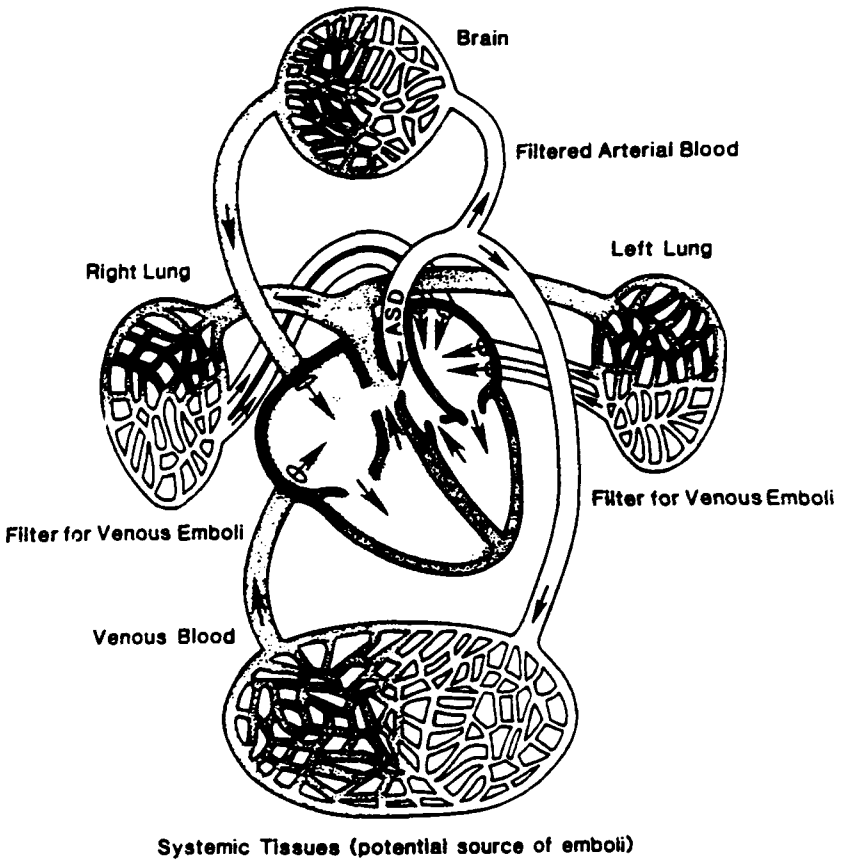

Figure 1: Schematic of the cardiopulmonary system demonstrating the peripheral tissues as the likely source of DCS venous bubbles that are carried through the right atrium, right ventricle and into the lungs where they are normally filtered. Also depicted is an intracardiac septal defect (ASD) which, if present, is a possible route for bubbles to enter the systemic circulation. The main pulmonary artery before it branches is the common site for precordial Doppler ultrasonic monitoring for DCS bubbles. (Redrawn from Hills, Decompression Sickness. Vol. 1. Wiley, Chichester, 1977.)

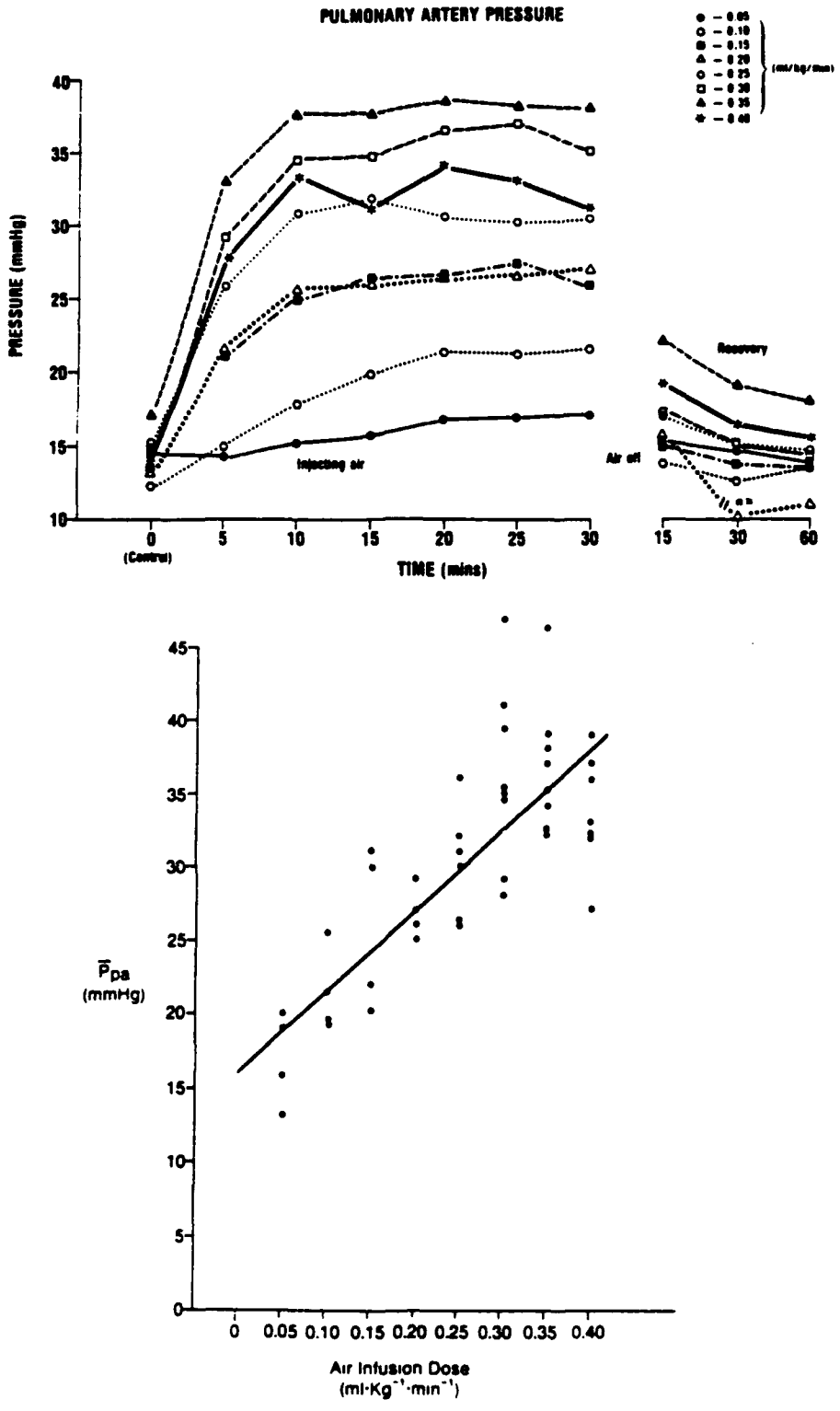

Figure 2: Top. Pulmonary artery pressure changes with 30-min continuous air infusion into right atria of dogs. Sixty-min recovery follows air infusion. Doses ranged from 0.05 ml kg^{-1} min^{-1} to 0.40 ml kg^{-1} min^{-1}. Response to 0.40 ml kg^{-1} min^{-1} is decreased from lower doses as arterial blood pressure in these animals (Fig. 4) is also decreased due to the large volume of gas. Bottom. Dose response relationship between air infusion dose and pulmonary artery pressure (Ppa). (r = 0.93) (From: Butler and Hills, J. Appl. Physiol. 59:543-547, 1985)

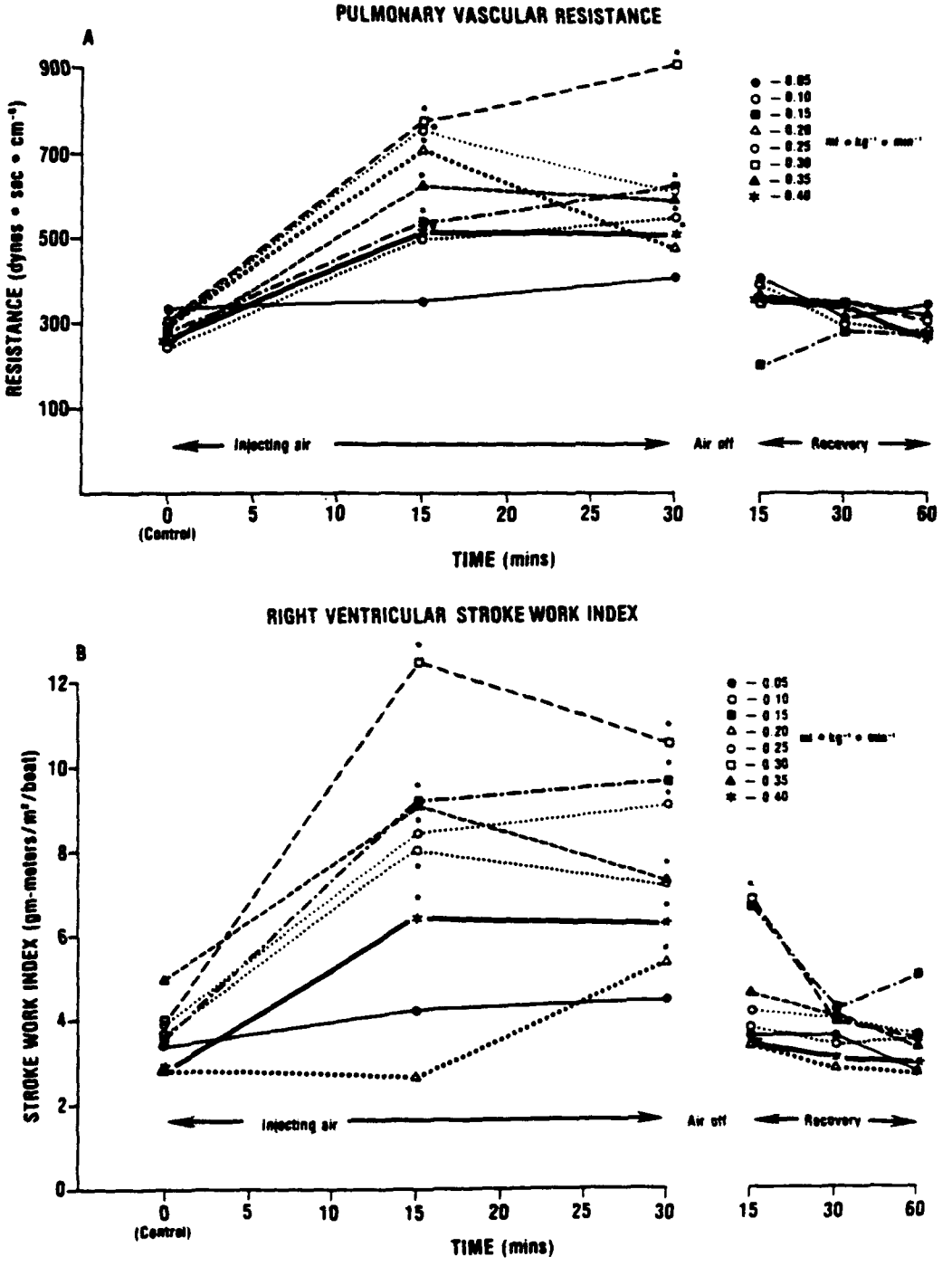

Figure 3: <u>Top</u>. Pulmonary vascular resistance changes with venous gas infusions. Doses described in Figure 2.
<u>Bottom</u>. Right ventricular stroke work index with venous gas infusions.

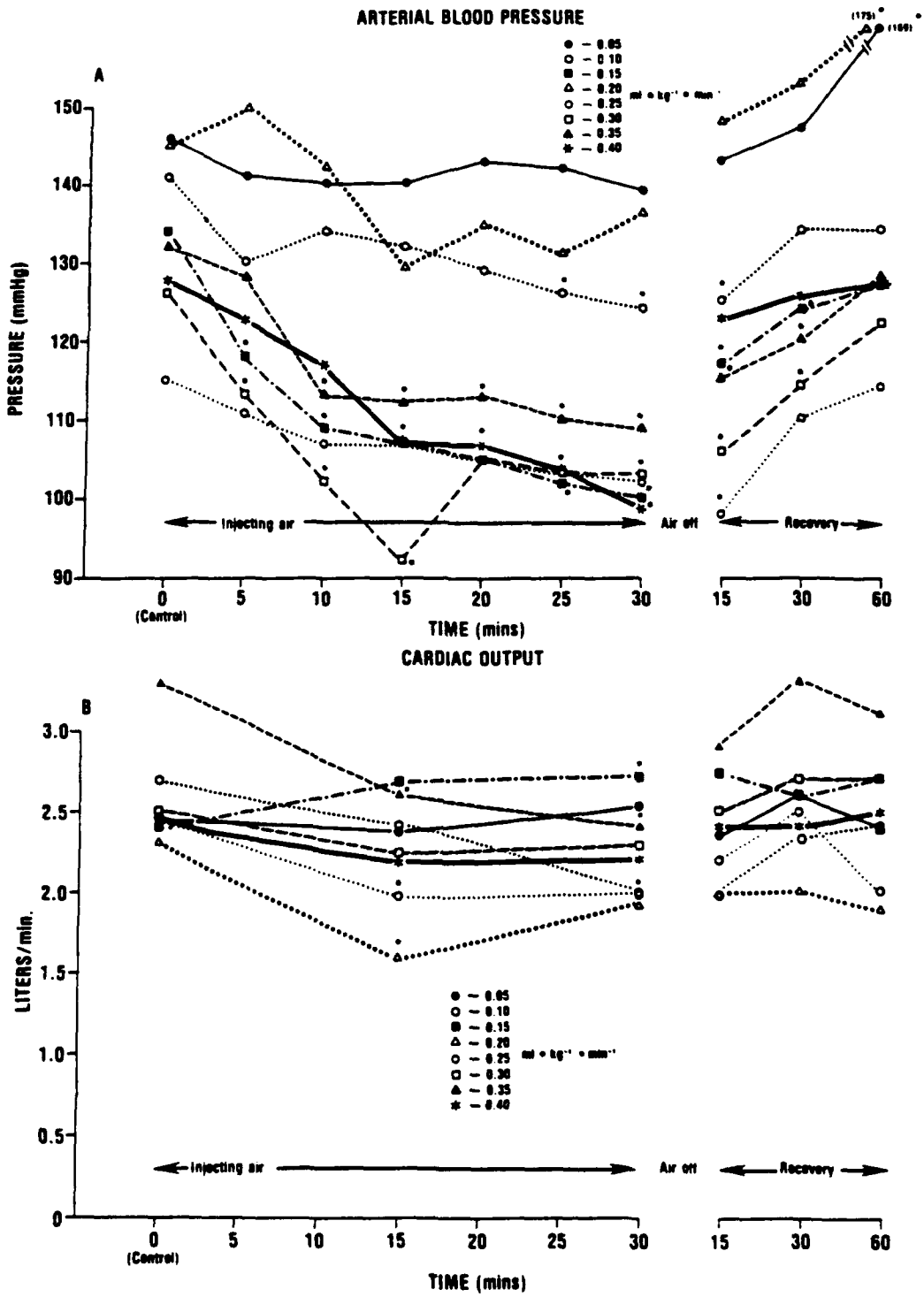

Figure 4: <u>Top</u>. **Arterial blood pressure (mean) with venous gas infusions.**
<u>Bottom</u>. **Cardiac output changes with venous gas infusions.**

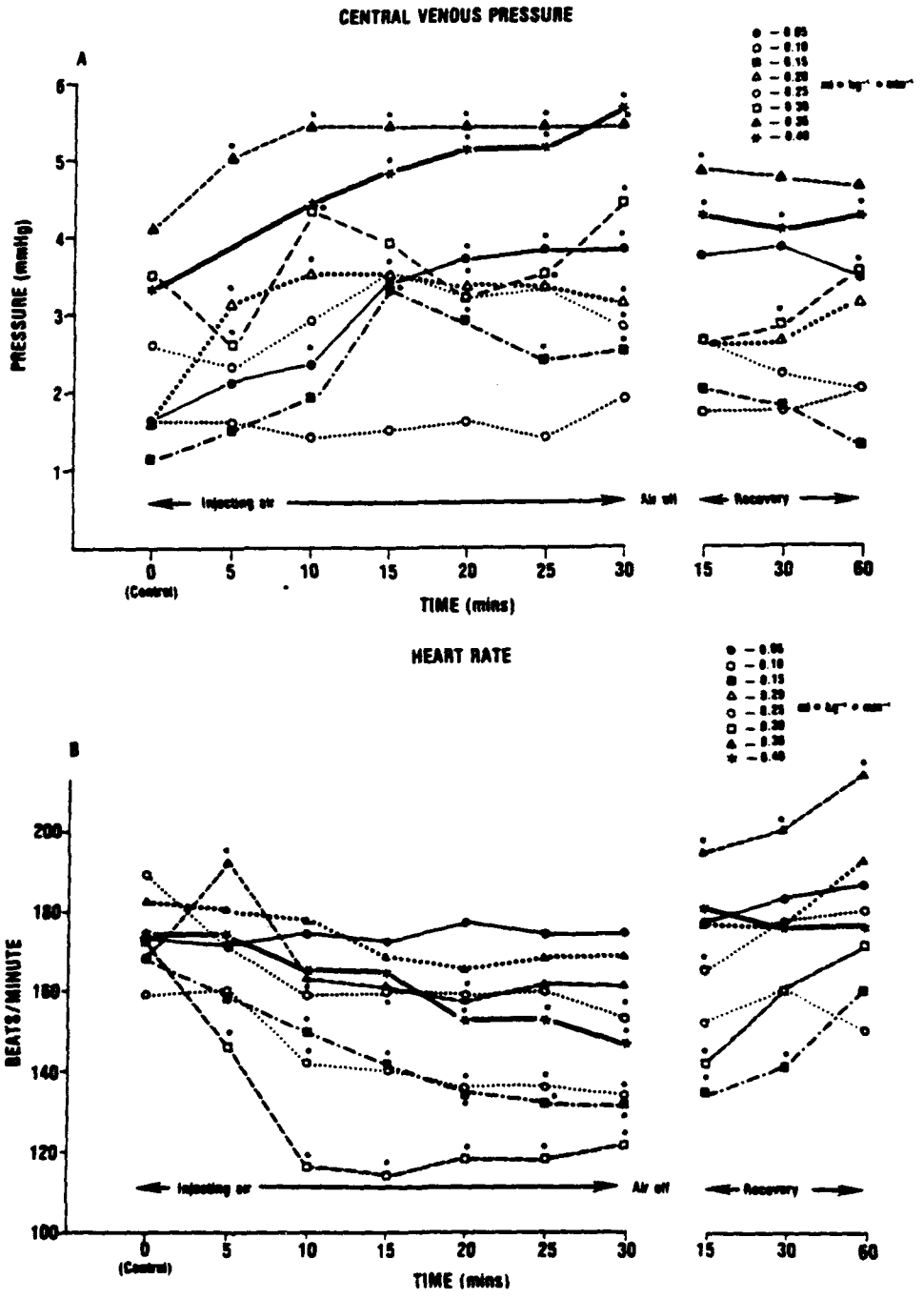

Figure 5: <u>Top</u>. **Central venous pressure changes with venous gas infusion.**
<u>Bottom</u>. **Heart rate changes with venous gas infusions.**

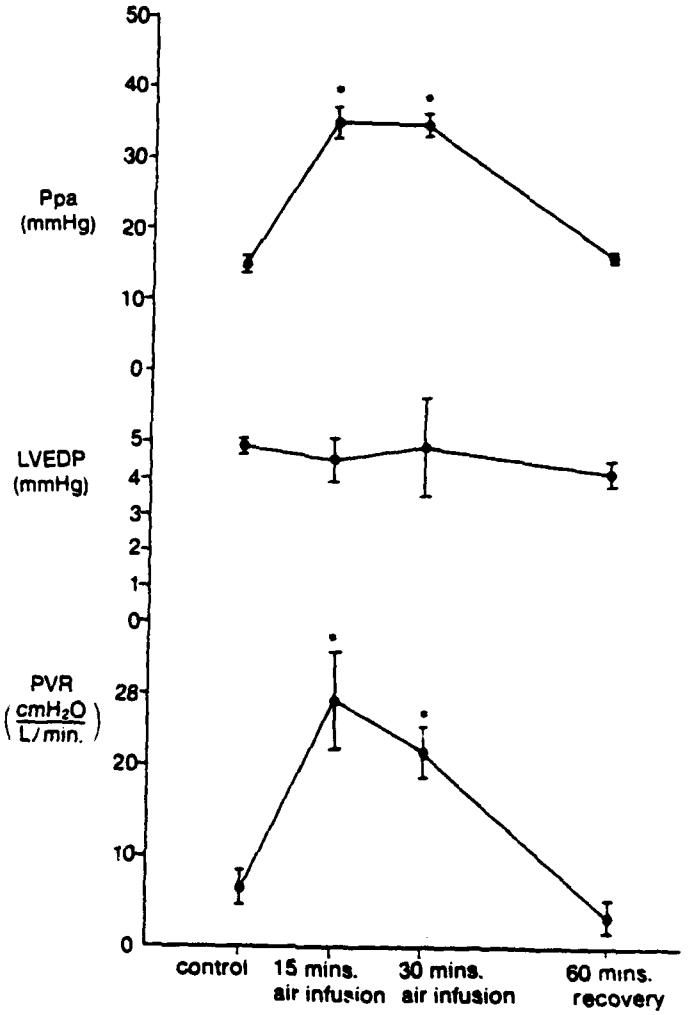

Figure 6: Pulmonary artery pressure (Ppa), left ventricular end-diastolic pressure (LVEDP) and pulmonary vascular resistance (PVR) with venous gas infusion in dogs (0.25 ml kg^{-1} min^{-1}). Note no change in LVEDP.

Additional consequences of venous bubbles circulating into the pulmonary vasculature involve reflex events including vasoconstriction, bronchoconstriction, pulmonary function abnormalities (e.g., compliance change) and "chokes." Chokes, also referred to as respiratory DCS, is actually a descriptive misnomer in that the subject is not choking but experiencing substernal stress that ultimately becomes paroxysmal coughing which can lead to faintness, cyanosis and ultimately vasodepressor syncope (38). The onset of symptoms is usually later than the occurrence of common bends symptoms. Fulton reported that chokes usually occur after 45-60 min at altitude (35,000 ft) in aviators, although some exceptions were reported with symptoms not occurring until 2.5 hr. Onset times after hyperbaric decompression have been reported as long as several hours following a dive (42). Respiratory symptoms

such as tachypnea, dyspnea or chest tightness may occur in addition to mottled skin, focal neurological manifestations and evidence of pulmonary congestion (38). Early reports have suggested that chokes result from gas formation in the mucous membrane of the bronchopulmonary passage while others have suggested that pulmonary vascular bubbles cause the reflex symptomatology (3). Chokes have also been reported following venous air infusions in dogs (8). Others have suggested that the etiology includes pulmonary vasoconstriction, pulmonary hypertension or mediator stimulated bronchospasm (34). Alternatively, stimulation of interstitial J-type receptors, by pulmonary vascular congestion (65) or swelling of the interstitial space with edema fluid might offer additional theories as to the etiology (8, 60). Mills et al. (62) and Armstrong et al. (2) reported the stimulation of pulmonary irritant and J receptors after pulmonary embolism in cats. In a similar fashion, increases in right ventricular pressure due to embolism of the lungs would likely stimulate pulmonary artery baroreceptors (12), and irritant receptors (72), as well as J receptors (65). In more severe cases of DCS or VAE, where large volumes of venous gas circulate into the lungs, the resultant increase in central venous pressure (19) will decrease the amount of lymph drainage from the interstitial space (13, 56), allowing edema fluid to accumulate. This may attenuate the reflex response. In recent studies, Atkins et al. (4) reported pulmonary interstitial edema, not alveolar, in respiratory decompression sickness in sheep, which they proposed as a pure model applicable to chokes and that resulted from massive amounts of venous bubbles. The latent period between DCS and chokes may well be the time necessary to accumulate a sufficient pulmonary vascular gas load (8). A humorally mediated response is an equally attractive mechanism for causing chokes because histamine, serotonin, prostaglandin and bradykinin are released after pulmonary embolism and may stimulate pulmonary J receptors.

A common event after VAE or DCS is a change in the normal fluid balance mechanism in the lungs which results in accumulation of edema fluid in the interstitial spaces surrounding the alveoli (8, 13, 24). The degree of pulmonary edema is shown to be related to the dose of venous gas as well as to the length of time that the bubbles obstruct the blood flow. Some investigators have suggested that pulmonary bubbles cause a change in the permeability of the microvascular membrane to protein and water (64) which is reversible after the gas infusions are terminated. This premise is consistent with reports of decompression-induced pulmonary edema where no change was observed in left ventricular end-diastolic pressure (24), negating a hydrostatic pressure mechanism for the fluid shift. Alternatively, others demonstrated no change in the permeability of the pulmonary microvascular membrane to protein molecules after VAE in experimental dogs (13). They subsequently found that lymph flow from cannulated lung lymph vessels was actually decreased in the face of increasing pulmonary perfusion pressures, due to VAE, since the central venous pressure was elevated and outflow drainage was obstructed (56).

The humoral response to VAE or DCS, which involves the release of mediators such as serotonin, histamine, kinins, prostaglandins, lymphokienes, thromboxane A_2, etc., may have specific action locally within the lung vasculature, or systemically (34, 67, 81). Additional cellular interactions with the bubbles involve the surface active

molecules secreted by the lungs that are effective at the air:blood interface (18). Other compounding influences may occur with different breathing gases (17) or redistribution of blood flow with resultant hypoxemia that occurs after embolization.

A final consideration relating to the lungs as the target organ for venous bubbles is the large quantity of gas overloading the vascular filtering mechanism and spillover of bubbles into the arteries occurs (Figure 7). Filtration thresholds of the pulmonary circulation are reported experimentally (16, 19) and can change with oxygen damage (17), pharmacologicals (16) and anesthetic gases (22, 54). Butler and Katz (20) have described the conditions whereby venous bubbles cross the pulmonary capillaries in dogs when perfusion pressures are in excess of 50 mmHg. An alternative mechanism might be that the pulmonary hypertension following VAE or DCS opens pulmonary arteriovenous shunts and allows spillover later in the course of the symptoms (8, 16, 63). The cardiovascular consequences of spillover depend upon the subsequent target organ of the newly arterialized bubbles. The role of transpulmonary passage of venous bubbles following DCS in the etiology of Type II DCS is likely; however, the extent is presently unclear.

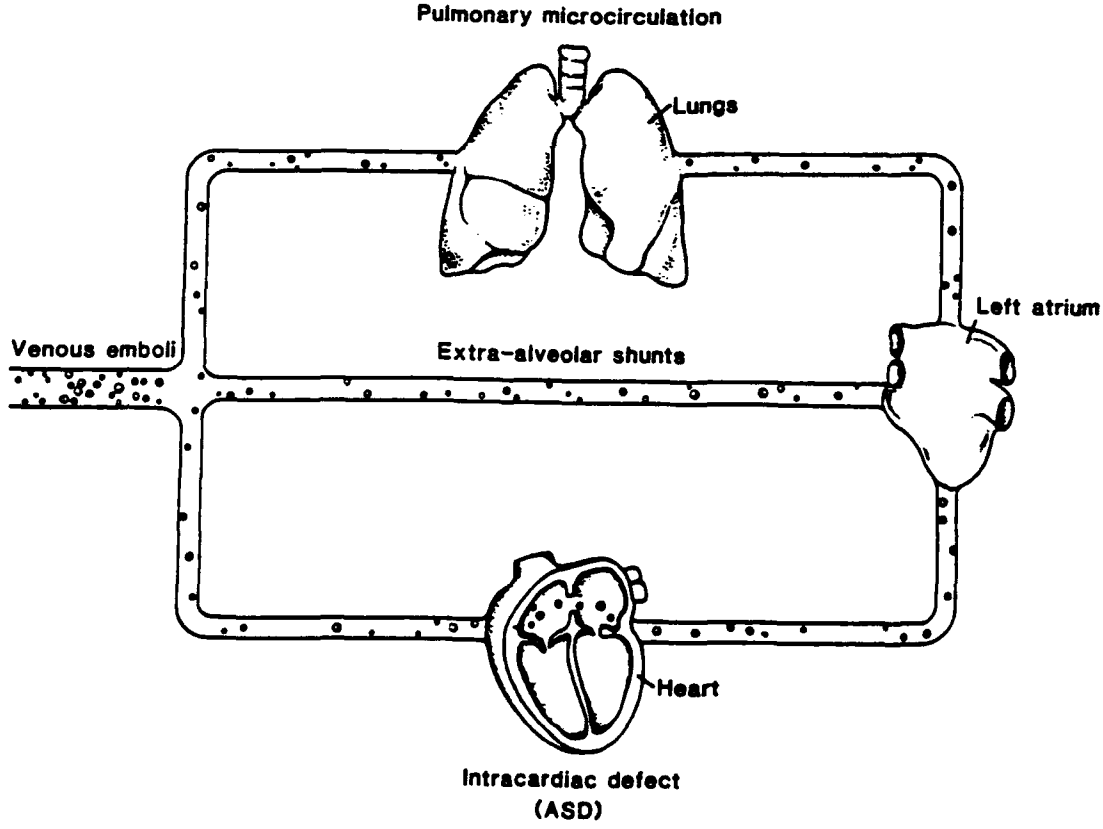

Figure 7. **Three possible routes for DCS or venous bubbles to cross the lung barrier and enter the systemic arteries. The upper route depicts the pulmonary microcirculation. The middle route depicts large arterio-venous shunts in the lungs and the lower route depicts intracardiac defects. An atrial septal defect (ASD) is indicated here.**

This spillover phenomena should not be confused with passage of venous bubbles into the arteries via an atrial septal defect (ASD) in the heart which reportedly occurs in up to 25% of the adult population (44). In these situations, it should be stressed that the amount of venous gas produced by the decompression must be sufficient to cause a reversal of the normal pressure gradient between the left and right atria so as to cause the blood flow and entrained bubbles to cross into the left side of the heart. Because many routine decompressions may not result in the release of sufficient quantities of venous gas bubbles, it follows that symptoms of arterial air embolism are fewer than would be predicted from the reported incidence of ASD's.

Heart:

Bubbles entering the pulmonary veins are carried into the left heart and subsequently into the coronary, cerebral or systemic circulation. The occurrence of arterial bubbles following decompression has been reported to cause an increase in systemic vascular resistance, probably due to vascular obstruction (8). Coronary artery bubbles result in a decrease in stroke work which is attributable to myocardial ischemia (33) usually accompanied by varying degrees of heart block and electrocardiographic changes including prolongation of QRS, as well as ST and T wave changes (30). Residual ischemic electrocardiographic changes are reported even after the gas bubbles are cleared from the coronary arteries (33). Histological evidence of focal necrosis is likely to be associated with the decrease in ventricular function after coronary air embolism. Eguchi & Bosher (33) reported that slow injections of 0.5 to 1.5 $ml \cdot kg^{-1}$ of air into the left ventricles of dogs produced significant myocardial depression in both the right and left ventricles. The predominant influence occurred in the right ventricle with the smaller doses and was equal between the two ventricles with the larger doses. When left ventricular air injections were rapid, both ventricles demonstrated marked depression. Goldfarb and Bahnson (40) expressed the myocardial depression after coronary air injection in terms of ventricular power to evaluate cardiac work per minute (Figure 8). In their studies left ventricular effects dominated over right. Additional effects of coronary air injections include depression of arterial pressures and slowing of the heart rate (40). Fulton (38) reported significant bradycardia with explosive hypobaric DCS and resultant coronary air embolism in dogs, which was preventable with bilateral vagotomy.

The lethal volume of pulmonary venous gas has been reported to be 1.0 ml in 100% of experimental dogs, while 0.5 ml proved lethal in 50% of the test animals (40). Irreversible myocardial damage was also observed in some of these dogs after coronary artery injections of air of volumes as small as 0.1 to 0.2 ml (< 0.01 $ml \cdot kg^{-1}$) total dose. Residual bubbles were observed up to 8 hours following injection. Other ischemic injuries to the heart may follow after reperfusion occurs once the bubbles are cleared, which can lead to edema formation, accumulation of arachidonic acid metabolites and subsequent microvascular permeability changes.

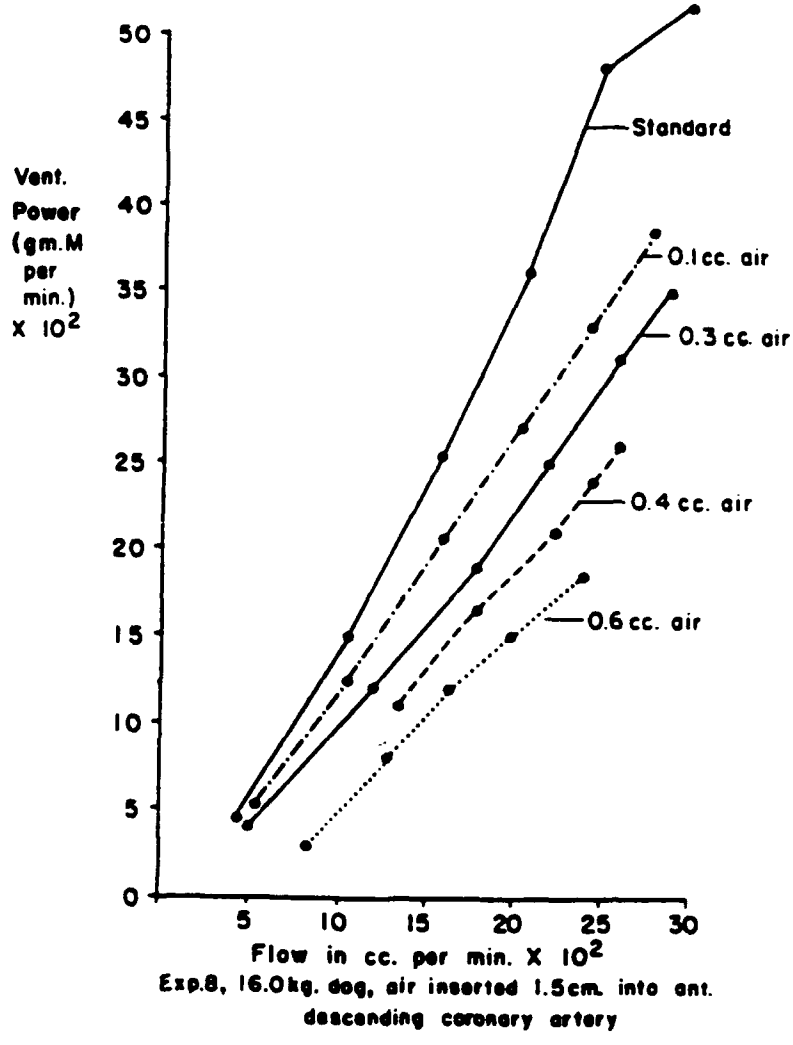

Figure 8: Ventricular power function with increasing dose of coronary air injections into dogs. (From Goldfarb and Bahnson, J. Thorac. Cardiovasc. Surg. 46:368-378, 1963.)

Brain:

Bubbles in cerebral vessels reportedly behave the same as in other arterial vessels where temporary occlusion occurs at the arteriolar level and is followed by segmental spasm (25). This symptomatology was followed within minutes by a marked dilation, not only of the involved vessels but of the downstream microcirculation and venules (25). A subsequent hyperemia was observed, but was not attributable to that normally associated with temporary circulatory arrest where replenishment of the oxygen debt is provided (37). Duft et al. (29) suggested that the vascular dilation following gas embolism was due to direct injury of the endothelial lining of the vessel wall.

Passage of cerebral gas bubbles through the capillaries and into the veins has been reported experimentally (76) and has been suggested as a possible explanation for why up to 60% of divers with cerebral DCS recover before recompression therapy (47, 75). This postulation has led to much evidence and speculation that neurological dysfunction following cerebral air embolism is related more to the post embolic effects on endothelial cells and blood components than to the bubbles and their associated transient obstruction of blood flow (45, 46, 66, 81). Helps et al. (47) reported a decrease in cerebral blood flow and sensorimotor cortex function in rabbits embolized with 25 µl air into the carotid artery. Such a response could not be explained by transient occlusion or blood pressure fluctuations and was not of an autoregulatory nature due to vessel caliber changes. These authors raised the issue of recompression treatment alone with cerebral gas embolism and how this might be effective in clearing the arteriolar and capillary bubbles (41) but still account for a significant failure rate (41, 55, 58) due to secondary effects on cerebral blood flow and cellular dysfunction (31).

Changes in brain microvascular permeability have been reported following DCS (26, 43) and gas embolism (10, 66). Lehtosalo et al. (57) reported that the mechanism for protein extravasation with DCS is via vesicular transfer. They found no ultrastructural evidence for mechanical rupture or damage to the microvascular endothelium and concluded that protein permeability after DCS is not of major significance. They did report occasional perivascular edema.

Gross symptoms of rapidly injected cerebral air (1.5-3.0 $ml \cdot kg^{-1}$) in dogs include a marked increase in arterial blood pressure, which may subside over a few minutes, and various neurologic signs including nystagmus, loss of lid reflex, pupil dilation, flaccidity, opisthotonus, extensor rigidity and convulsions (37). Evans et al. (36) reported severe cardiac arrhythmias and acute hypertension after injection of air (2.0 $ml \cdot kg^{-1}$) into the vertebral artery of anesthetized cats. The authors attributed these symptoms to the autonomic nervous system and not to local or myocardial hypoxia. They concluded from the results of their study and others (36) that the neurologic dysfunction caused by cerebral air embolism may be a common mechanism of cardiac dysfunction in DCS.

Much of the earlier work on cerebral air embolism was conducted with air or nitrogen gas bubbles. With the DCS likely to occur in space applications during extravehicular activity, there is a possibility that the circulating bubbles will contain significant amounts of oxygen, due to the extensive 100% oxygen prebreathe protocols and oxygen required for ventilation in the space suit. Using oxygen injections instead of air, Fries et al. (37) found that cats demonstrated similar qualitative changes to those with air infusions; however, a greater tolerance was exhibited for equal volumes of oxygen. It could be argued that this greater tolerance to oxygen bubbles may preclude early or prolonged symptoms in humans following decompression with adequate oxygen prebreathe. This argument is speculative, however, and requires further study.

Other Factors/Considerations

Several other factors may influence the cardiopulmonary effects of DCS bubbles. They include, although not exclusively, body position, breathing gases, potential cardiovascular changes that occur in the microgravity of space and the effects of prolonged venous bubbling on the lungs.

The first of these factors involves the position of the subject and its effect on the distribution of arterial bubbles. Numerous reports recommend the placement of a patient in the head-down Trendelenburg Position as immediate adjunctive therapy for arterial air embolism. The rationale for this maneuver has been based upon the concept that the buoyant properties of bubbles will cause them to float away from dependent regions and hence spare the brain from the more serious consequences of cerebral embolization (30, 73, 76). An additional rationale has been based on the premise that the head-down position will cause cerebral venodilation and allow bubbles to pass through the microcirculation more easily. Although cerebral venous pressure may be elevated in this position, it is also known to decrease cerebral blood flow which may have adverse effects. Additionally, as is the case with most vascular beds, the site of obstruction by circulating bubbles is usually at the precapillary level, not at the venous level.

Van Allen et al. (76) reported that the tolerance to pulmonary venous infusions of air into dogs was greater if the animals were placed in a 90° vertical position. In this position the head-up animals demonstrated a greater incidence of neuromuscular-type symptoms, while those with the head-down, manifested primarily cardiovascular responses. Bagdonas et al. (5) reported that a 20° head-down position was favorable in minimizing cerebral air embolism from the ascending aorta, but that coronary air embolism may ensue from bubbles trapped elsewhere in the aorta.

Butler et al. (21) reported that the Trendelenburg Position (10-30°) was not effective in preventing left ventricular air bubbles from moving in the same direction as the blood flow and into the cerebral circulation of dogs. They further concluded, from separate in vitro studies using a simulated carotid artery preparation, that even in a 90° vertical position the buoyant properties of bubbles with diameters up to 0.4 cm were not sufficient to oppose the force of blood flow and prevent the distribution of bubbles into the brain. This situation is depicted in Figure 9. Dutka et al. (32) have recently reported that the head-down position after cerebral air embolism in dogs reduced recovery in cortical somatosensory evoked response even after recompression. In related studies, Polychronidis et al. (69) reported that prolonged head-down position (45°) in dogs after cerebral air embolism caused a further increase in intracranial pressure and blood brain barrier permeability even with hyperbaric therapy. These findings may be of relevance to DCS in space where body position and gravity are less of a factor. In this context it could be concluded that no particular position of an astronaut with circulating bubbles will necessarily prevent the distribution of the bubbles away from the direction of blood flow.

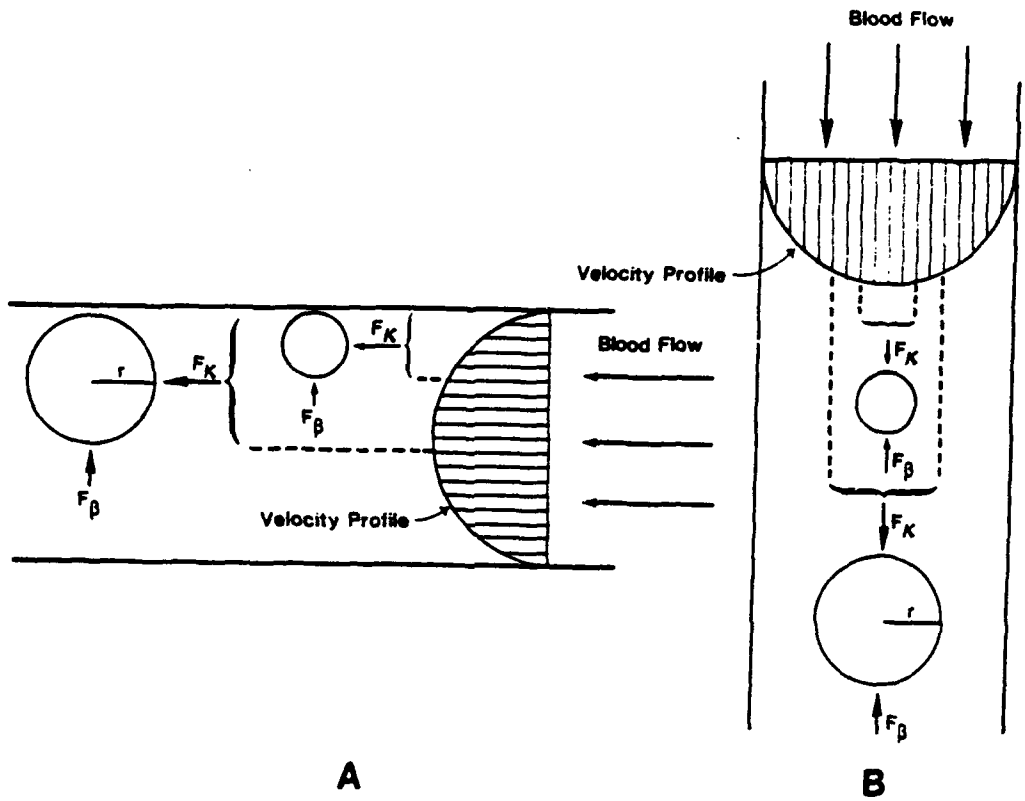

Figure 9: A. Schematic of large and small bubbles in a vessel. If the force of buoyance were to cause bubbles to rise to the upper regions of the vessel, the greater cross-sectional area of the larger bubbles results in a larger portion being affected by the blood velocity profile, hence bubble velocity increases with increasing diameter. This increase in blood velocity occurs in spite of any increase in drag that occurs at the top of the bubble.
B. Large and small bubbles in 90° angle. As the bubble radius increases, the force of the blood remains greater than the force of buoyance. The result is that the velocity of the bubble is decreased from that of the blood flow but does not reverse in direction. Thus in spite of body position, bubbles appear to always travel in the direction of the blood flow. F_B = force of buoyancy; F_K = force of blood flow; r = bubble radius. (From Butler et al., Ann. Thorac. Surg. 45:198-202, 1988.)

Additional factors that may influence the cardiovascular effects of DCS bubbles relate to the amount of venous gas produced by decompression, repetitive decompression, as well as different breathing gases (80). With extensive 100% oxygen prebreathing before decompression and during EVA activities in space, there is the obvious benefit of reducing the amount of nitrogen bubble formation (80). Other questions arise pertaining to the gas composition of bubbles that are formed and of the subsequent half-life of these bubbles with air or oxygen ventilation. Experimental studies of VAE in dogs have demonstrated that discrete gas bubbles continue to obstruct the pulmonary circulation for up to 43 ± 10.8 min. post infusion with air ventilation, yet this value is reduced to 19 ± 2.5 min. with 100% oxygen ventilation (Figure 10) (23). Elevated arterial levels of carbon dioxide (hypercapnia) or decreased inspired oxygen tensions (hypoxia) can also have profound cardiovascular influences alone, and their interactions with the effects of DCS bubbles are areas needing further clarification (79).

In space applications, the occurrence of body fluid shifts and their associated effects on the cardiopulmonary system, ventilation-perfusion ratios, and whole body nitrogen washout may influence to some degree the consequences associated with DCS bubbles. Another potential area of study relates to the effects of prolonged DCS bubbling (6-8 hr) on pulmonary function and the cardiovascular system.

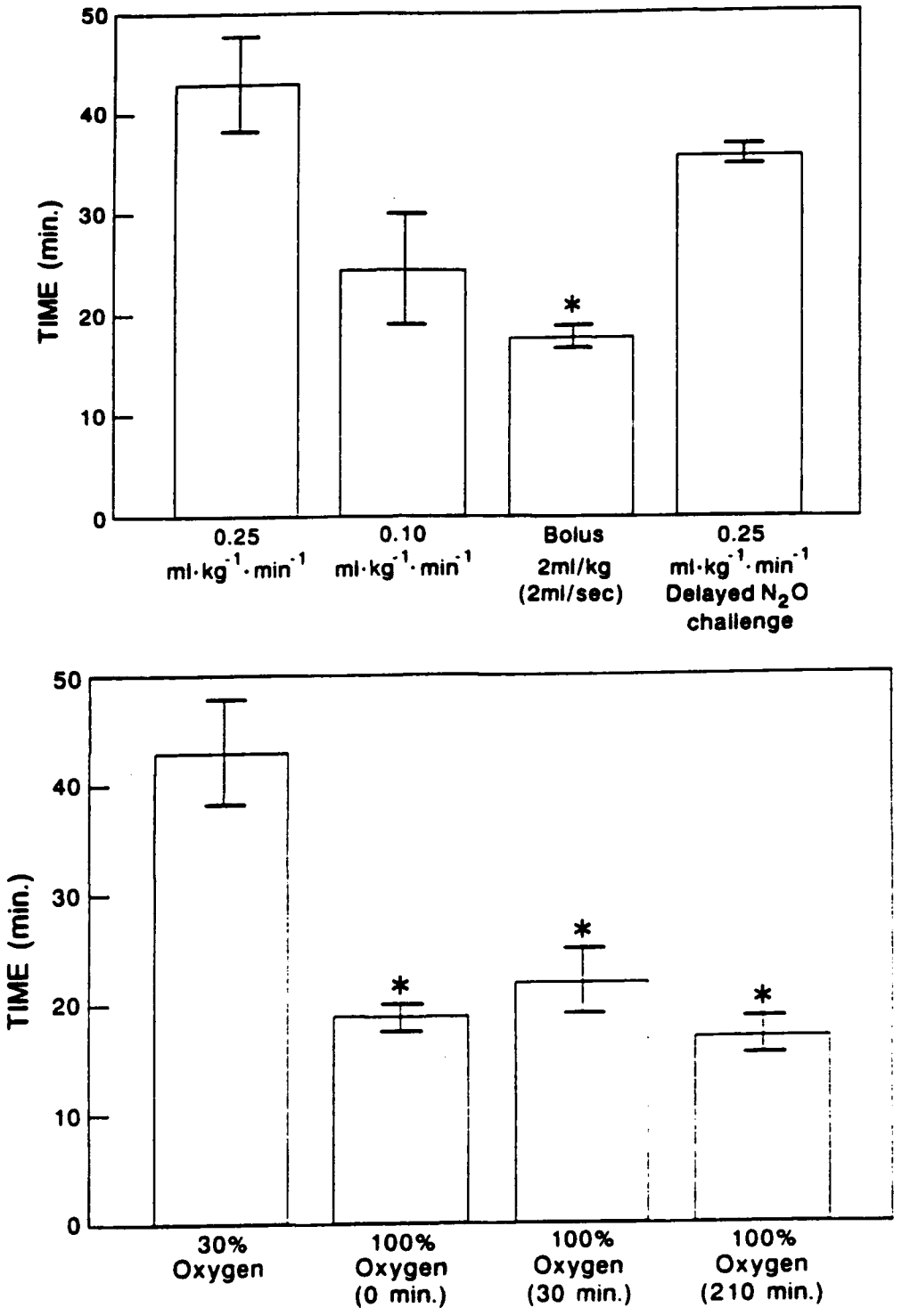

Figure 10: **Top. Mean bubble longevity times in the pulmonary vasculature after infusion into the right atrium of dogs. The bubbles were infused in three different doses. Residual bubbles were detected using the N_2O challenge technique (see ref.).**
Bottom. Mean bubble longevity times in the pulmonary vasculature with 100% oxygen prebreathing for 0, 30 or 210 min. versus 30% oxygen ventilation. Venous air dose was 0.25 ml · kg^{-1}· min^{-1}. Bubble longevity times were reduced ($p < 0.05$) regardless of the time of preoxygenation. (From Butler et al., Undersea Biomed. Res. 16:21-29, 1989.)

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DR. HAMILTON: In the first few graphs you showed the gas dose ranged from .05 to .40 ml/kg/min; the largest number was rarely the extreme. It was right in the middle on most of them.

DR. BUTLER: If you look at the third slide, you see that the mean arterial blood pressure and cardiac output were depressed. There was so much gas injected at that point that the animal was going into circulatory collapse. So when you say it was not the top number, it was because the pulmonary artery pressure was not the highest because the animal was going into collapse.

I should add one thing. The first two doses, 0.05 and the 0.10, and possibly even the third dose, gave us comparable values to those seen with Grade 4 Doppler bubbling. This compared well with Mike Powell's early work with sheep. It also compared well with another study with dogs at, I believe, NMRI.

DR. VAN LIEW: Regarding atrial septal defects, you indicated that you would not get much flow through unless the central venous pressure was high. However, during some very short time intervals of a normal cardiac cycle, or during a slightly abnormal cycle, such as with straining, could you get a little bit of flow through, and then conceivably you could pass a couple of bubbles that might end up in the brain?

DR. BUTLER: Certainly. However, it depends on the type of atrial septal defect. There are a variety of types, some of which have no normal flow between them, others which do have flow between them. In those situations in which you do not, in other words it is propatent, the potential is there for the flow to exist if you reverse the pressure gradient. For flow to occur, you have to raise central venous pressure. If the great rise in central venous pressure is occurring from the bubbles impinging on the lungs, you would have to have a great deal of gas bubbles trapped there. In your situation, where there may be some flow, yes, you would probably have a higher predicted factor for arterial symptoms with even routine decompressions.

COLONEL STORK: In reference to the increase in pulmonary vascular resistance across time, have you dissected how much of that is simply the physical presence of the bubble and how much is the reaction of the tissue?

DR. BUTLER: No, I haven't. Others have tried various blocking agents, either direct denervation or chemical blocking agents. Our knowledge of mediators has just mushroomed of late. I think there are a variety of mediators that we know more about now and thus we can selectively block their action. We can now address which of those factors is more important, whether it is a mechanical obstruction, or a mediator of a vasoreflective type, or a vasoreactive type. I think another very important question is that if there is an injury that occurs, does the lung then become activated, or is the lung then put in a presymptomatic state? Is the next decompression then going to impact

an activated lung? These questions can be addressed more specifically by looking at the mediator blocking agents that we know more about now.

DR. LAMBERTSEN: You are studying a graded sequence of different degrees of stress upon the lung with increasing amounts of venous gas embolism. That resembles the circumstance of different degrees of stress in an actual decompression circumstance. What is your judgment of this problem in the beginning detectable decompression sickness that we tend to encounter in aerospace activity? Do you think that the presence of venous bubbles is of any consequence at all? Is it something that we have to be concerned with?

DR. BUTLER: Let me preface my answer by saying I am going to only address circulating bubbles, not tissue bubbles. I think if the altitude exposure results in the release of venous bubbles, and with the subsequent return to normal atmospheric pressure you get any sort of relief of symptoms, or the bubbles themselves are absorbed, then there is probably not a concern. I think when we talk about repetitive exposures and for prolonged decompression bubbles, such as we see with space applications (in which bubbling may occur for hours), we then need to start looking at a cumulative effect or an activated circulatory response. This is different than the quick exposure situation. Until recently, most exposures in altitude or aviation literature have been of the short duration type. You cannot study the prolonged effect with such exposures.

DR. LAMBERTSEN: Thus, in relation to the concept of a systemic disease you can have the mild systemic disease which you can put up with, do not feel, does not hurt you very much, and you can ignore. Or you can have a more severe systemic disease which can produce even serious ramifications.

DR. BUTLER: Let me give you an example involving the pulmonary circulation. Let's say an individual bubbles for 4 hours or 6 hours, and fluid balance is pushed over until edema fluid is not really accumulating but is right at that threshold. Now you reimpose the decompression stress on the individual. You are giving a second insult of bubbles. At this point, you are liable to put that individual into a greater risk of edema formation or fluid balance change. The same could occur in other tissues. It may be that there is a cumulative effect and we do not see it until we get into the repetition situation.

DR. VAN LIEW: Are you implying that nothing happens unless there is an activation of mediators, an activation that got inactivated by natural mechanisms?

DR. BUTLER: I think that there is an impact from mediator release caused by circulating bubbles. It may be subsymptomatic. I am not saying that nothing happens if there is no symptom. The effect remains at a subsymptomatic level, or subeffective level. In other words, there is not enough response or not enough mediator accumulation or not enough basal active reflex to be able to actually detect an effect.

DR. VAN LIEW: Or that there are mechanisms for repairing any damage?

DR. BUTLER: Yes. Most of the mediators that we have talked about are cleared in a single pass through the pulmonary circulation, so you would have to have a continuum of effects.

THE ROLE OF PATENT FORAMEN OVALE IN ALTITUDE-INDUCED DECOMPRESSION SICKNESS

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ABSTRACT

The potential for a patent foramen ovale (PFO) appears to exist in 20-30% of the human population. Because pressure in the left atrium is generally greater than that in the right, if the foramen exists it is usually functionally closed. If the interatrial septum is not anatomically closed, right-to-left shunting can occur allowing venous blood to bypass the lung . This could result in altered gas exchange and bypass of the filtering function subserved by the lung. Except for slight arterial oxygen desaturation, there are generally no significant physiological effects in the PFO-positive individual. However, if there are emboli present in the venous circulation, right-to-left atrial shunting can potentially result in arterial embolism. There is evidence in the diving literature that documents the occurrence of venous gas emboli even after some asymptomatic dives. Altitude decompression can also produce venous gas emboli. This review summarizes what is known of the environmental conditions and physiological mechanisms that potentiate the right-to-left shunt of venous emboli induced by altitude decompression.

INTRODUCTION

A patent foramen ovale (PFO) is essential for fetal life since it allows blood to pass from the right heart to the left heart in order to bypass the collapsed lungs. PFO defined for this review refers to that vestigial atrial channel that is potentially patent but functionally closed in normal situations and persisting in the adult animal or human. The PFO condition is further defined to exclude frank septal defects.

At birth, when the umbilicus is occluded, systemic arterial pressure rises, pulmonary arterial pressure falls as the lungs inflate, and reversal of the interatrial pressure gradient causes closure of the valve of the fossa ovalis against the limbus of the fossa ovale (2-6). Usually within the first year of life, fibrous adhesions form to permanently isolate the right atrium from the left (anatomical closure). In 20-30% of the population, a potential patency remains. This so-called probe PFO usually causes no obvious clinical symptoms and the valve flap remains closed except for certain right-to-left atrial flows resulting from momentary pressure gradient reversals. It has been shown that right-to-left shunting can occur during quiet breathing without significant complications (7, 23, 31). More profound reversals occur upon release of a

Valsalva (4), during restricted inhalation (36), and during any situation that causes rapid and substantial venous return to the right heart (3). Other provocative maneuvers also include crying in the neonate (2), pulmonary hypertension (2, 13, 18), chronic obstructive pulmonary disease (2), and positive end-expiratory pressure (2, 3, 4).

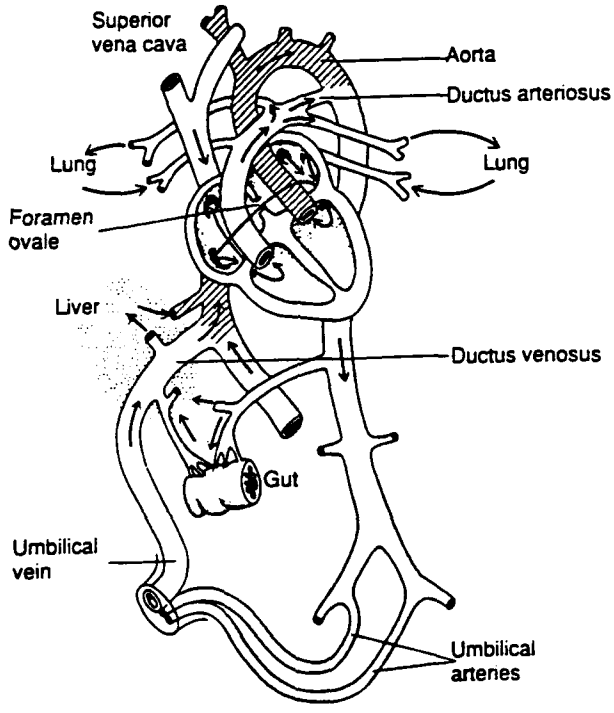

Figure 1. Organization of the fetal circulation (1).

The acute consequences of shunting range from insignificant to catastrophic depending upon the magnitude of shunt, the presence and location of arterial emboli, and the physiological sequelae to those emboli. This review will cover the following issues regarding PFO:

Human Incidence of PFO
Animal Incidence of PFO
Clinical Concerns Regarding PFO
Embolic Phenomena
Altitude Decompression Sickness
Modern Aerospace Environment
Technology Advancements
Areas Requiring Further Research
Potential Methodology

HUMAN INCIDENCE OF PFO

In a study of 965 human autopsy specimens, the incidence and size of PFO appeared to vary according to age but not by gender (2). Specimens in the first three decades of life showed an incidence of 34%, declining to 25% during the fourth through eighth decades. Measurement of the size of the patency was made using calibrated probes with the recognition that formalin fixation may have caused some shrinkage of the fibroelastic elements. The mean probe-determined diameter was 5 mm, with a range of 1 to 10 mm (2). These autopsy data were from normal human hearts with no known cardiac dysfunction prior to death.

In-vivo diagnosis can now be accurately determined using relatively non-invasive techniques (5-9, 23, 24, 28, 29). Contrast echocardiography combined with various respiratory maneuvers can detect shunting in asymptomatic subjects (5-8, 23, 24, 28, 31).

ANIMAL INCIDENCE OF PFO

Many placental mammals (pigs and cows for example) appear to have an incidence of PFO similar to that of humans (14, 15). Calves exhibit the type of PFO most often exhibited by humans, ostium secundum (15) and, like humans with PFO, are generally asymptomatic. Yucatan Miniature Swine exhibited an incidence of 8 PFOs out of 29 animals in a recent study, again without clinical symptomatology (14). Pigs, dogs, and calves have served as cardiovascular models for the study of septal defects (4, 14), arterial gas embolism (18) and congenital cardiac defects (15), respectively. The natural incidence of PFO elicited in these animals and the clinical symptomatology are somewhat analogous to the human condition.

CLINICAL CONCERNS IN REFERENCE TO PFO

Momentary right-to-left shunting due to PFO is generally of little significance in the otherwise normal individual. Lowered arterial oxygen saturation is generally transient and of little consequence. Physiological shunt, which occurs in the absence of PFO, is usually a minor contributor to arterial oxygen desaturation. The bronchial circulation and a few small thoracic vessels drain directly into the pulmonary veins which, with the thebesian circulation of the myocardium, slightly reduce arterial saturation. Normal distributions of ventilation and perfusion in the lung contribute to oxygen desaturation because a fraction of the pulmonary arterial blood is not reoxygenated. This physiological shunt is caused by the effect of gravity on the upright lung and is normally without clinical significance.

EMBOLIC PHENOMENA

Vascular surgery or breathing air at high barometric pressure may put those with persistent patency at risk from paradoxical embolization of foreign intravascular substances. (2, 3, 8, 9). Air, fat, tumors, and intravascular thrombi have all been found in the systemic circulation following these events (3, 4, 13, 18, 36). Arterial emboli can occlude critical vascular beds resulting in cerebral and myocardial ischemia (3, 18, 19). Permanent and diffuse ischemic damage can result from any type of arterial embolus (3, 35).

Air emboli may be introduced into the vascular system in surgical situations as well as from decompression from diving and flight (18). A decompression that creates a transthoracic pressure difference of approximately 80-100 mmHg is sufficient to tear the lungs (37, 38). The most severe decompression situation occurs when gas in the lung cannot escape or is restricted from escape by breath-holding, straining, swallowing, or restrictive breathing equipment characteristics (38). In addition to causing mediastinal emphysema and pneumothorax, air emboli occurring as a result of lung rupture can invade the systemic circulation (38). Transthoracic pressure differentials on the order of 80-100 mmHg, experienced only briefly, increase the likelihood of air embolism (38). Air embolism, though historically rare in altitude decompressions, may be of increasing concern as the potential for extreme decompression increases with cabins and breathing systems capable of greater differential pressures.

Fat emboli occurring as a result of trauma have been postulated to lodge in the lungs, cause pulmonary hypertension, elevate right heart pressures, and open the formerly nonfunctioning foramen ovale through which more emboli pass to potentially obstruct cerebral blood flow (13). Long bone fractures release fat emboli into the venous circulation which can be detected in nearly all patients with such fractures (34). A dietary fat load in a PFO positive individual has also been postulated to cause fat embolization (13).

Thrombus occlusion of a PFO has been shown in a patient with normal heart function and pressures. The autopsy evidence in this case showed an imperforate foramen ovale with the thrombus extending through the foramen ovale into the left atrium (30). The disintegration of such a thrombus, over time, might easily generate tissue ischemia distal to the thrombus. Additionally, thrombi arising in the left atrium for unknown reasons can cause paradoxical embolism without the presence of a PFO (32).

Surgical procedures that require an incision where the venous pressure is below atmospheric pressure can result in venous air embolism (4, 27). Patient positions in which the operative site is elevated above the right atrium also increase the likelihood and severity of venous air embolism (27). However, introduction of air into the venous system, unless there is massive insult, is generally filtered out effectively by the lung (16-18).

Constant rate infusions of nitrogen, oxygen, and carbon dioxide into the venous circulation in sheep have shown a very high clearance for carbon dioxide and a slow clearance for nitrogen (16). Oxygen was cleared at a rate that was between that of the other two gases (16). Clearance of continuous venous air infusion does not appear to jeopardize lung function in pigs and dogs at infusion rates from 0.1-0.2 ml/kg/min for periods of up to one hour (4, 18, 27). The injection of air into the pulmonary artery of dogs has shown that these emboli lodge at the pulmonary arterioles and appear to be eliminated at that site (18). The vascular and hemodynamic response to foreign intravascular emboli, particularly in the arterial circulation, likely potentiates the primary insult in paradoxical embolism (3).

It is clear that emboli can be formed by many different mechanisms and that there are a multitude of types of emboli. Once appearing in the venous system, most foreign emboli, regardless of type, are filtered by the lung. There are clearance limits in the lung which, if exceeded, will allow passage of emboli into the systemic circulation. The tissue bed distal to the embolus is subject to ischemic injury once emboli enter the arterial circulation. Of particular concern are the cerebral and coronary circulations because of brain and heart sensitivity to ischemic damage. Autologous emboli (fat, thrombi, tumors) generally elicit vascular and hemodynamic responses similar to exogenous emboli (air embolism, infusion, and surgical induction) although the physiological mechanisms of clearance can be different.

ALTITUDE DECOMPRESSION SICKNESS

A significant reduction of barometric pressure over an individual can cause gas to evolve from dissolved stores in body tissue fluids. The pathophysiological symptomatology resulting from these evolved gases is commonly referred to as decompression sickness (39) with nearly identical mechanisms associated with both altitude and diving decompression. The etiology, decompression parameters, bubble composition and dynamics, sequelae, prophylaxis, and treatment are definitively addressed in aerospace medicine texts to which the reader is referred (38, 39). Of particular concern here are the intravascular, mainly venous gas emboli that are present in asymptomatic altitude- and dive-decompressed subjects.

Gas bubbles arising from micronuclei as a result of Henry's Law are generally considered to be responsible for the symptoms manifested in altitude decompression sickness (9, 10, 11, 21). Altitude decompression differs from dive decompression mainly in the rate and magnitude of pressure change. There are a few physiological responses unique to each environment with respect to bubble production and the resultant manifestation of decompression sickness. Examples of these differences include the fact that altitude DCS generally presents cerebral neurological symptoms, while dive DCS evokes predominantly spinal nerve symptoms, i.e., paresthesia. Differences in posture, hydrostatic vascular column effects, and physical workload may explain some of these differences in the manifestation of the disease.

Scrutiny of the mechanisms common to both altitude and dive decompression sickness has been useful historically in the prophylaxis, prediction, and treatment of manifestations common to both. In general, risks common to both include: mechanical expansion and compression of gas in the body cavities, carbon dioxide abnormalities, pressure equilibration difficulties, thermal stress, and life support system hazards.

The acute response to altitude DCS in such an individual needs to be more fully evaluated in light of the increasing likelihood of exposure. The physiological residuals from chronic episodes of altitude DCS in individuals harboring PFO are unknown. Recent dive DCS studies have suggested that extensive central nervous system damage may be taking place to an extent far greater than has been shown by classical clinical examination (35). Unexpectedly extensive areas of cerebral hypoperfusion and ocular fundus lesions may represent evidence of damage by gas emboli (33). It has been postulated that PFO may be a risk factor for the development of neurological decompression sickness (6, 8, 9). This may be of significance in assessment of the altitude decompression sickness risk in the modern aerospace environment.

OPERATIONAL CONCERNS IN THE MODERN AEROSPACE ENVIRONMENT

The atrial pressure gradient reversal caused by events common to the aerospace environment may be sufficient to permit right-to-left atrial shunt in PFO positive individuals, even in normal pressure environments. These events can momentarily cause rapid and substantial venous return to the right heart and include the cessation of a Valsalva maneuver, cessation of positive pressure breathing, cessation of the L-1 or M-1 anti-G straining maneuver, coughing, a Müller maneuver, and negative pressure breathing. Also, acute pulmonary hypertension resulting from hypoxic pulmonary vasoconstriction induced by high altitude exposure elicits rapid and sustained elevation of right heart pressure (25, 26).

Accidental decompression in the modern aerospace environment will most likely expose individuals to high altitude without the benefit of denitrogenation. The prevention of acceleration atelectasis and oxygen system endurance and concentration characteristics all weigh against the uninterrupted breathing of 100% oxygen - an effective prophylaxis for altitude decompression sickness. Unprecedented aircraft altitude capability will increase the magnitude of the decompression and enable a larger population to be exposed. Fortunately, most of these altitude decompression situations will permit rapid descent, reducing the physical and physiological stress presented.

Intentional decompression or mission completion at low pressure allows for control of many factors that influence decompression sickness. Denitrogenation by breathing 100% oxygen has traditionally been done at ground level before exposure to altitude. Even so, altitude decompression sickness has still occurred. Fatigue, dehydration, and some concern for oxygen toxicity also determine practical limitations

to the duration of ground level pre-breathing that can be expected of an exposed individual. Routine altitude decompression operations may eventually be afforded some equivalent DCS protection through the use of altitude denitrogenation schedules, but schedules for extreme altitude denitrogenation could probably not be designed with reasonable confidence for the individual.

Emergency contingency planning for immediate and sustained altitude decompression exposure is fraught with uncertainty, which when compared to the system-wide acceptance of risk, becomes insignificant. It is essential, however, to have knowledge of those characteristics which give an individual the best chance for survival. Individuals harboring persistent PFO may incur increased risk in such situations. Variability between individuals and within an individual is substantial with respect to the development of bubble formation induced by altitude decompression. PFO and a high probability of developing altitude DCS may put such an individual at extreme risk in contrast to the non-PFO individual who is highly resistant to bubble formation. Selection pressure in this respect may have favored those populations who currently operate in these environments. The operator population which may be exposed in the future may not be as highly screened. The individual ability of the operator to complete the mission during extreme altitude decompression exposure should be considered in order to best survive the emergency.

Positive pressure breathing systems, delivering high levels of pressure, will enhance protection against acceleration and protect against the hypoxia of extreme altitude. These systems will require significant and recurrent proficiency training in order to realize maximum system potential. In addition, the operator population will be exposed in mass to levels of breathing pressure nominally three times that to which it has experienced previously. Operational altitude decompression, in addition to fleet-wide training exposures to demonstrate the capability of such equipment, will contribute much to the operational exploitation of modern positive pressure breathing systems. Respiratory maneuvers that routinely take place in the modern aerospace environment will assume increasing importance as the environment becomes more complex and severe.

There is some controversy as to whether or not PFO is the primary mechanism that allows paradoxical embolism to occur, but the controversy may be based upon different methods. Mugge et al. (32) conclude that transoesophageal contrast echocardiography demonstrates better reliability in the detection of PFO than the transthoracic approach.

The development of decompression sickness (DCS) is generally attributed to an excessive rate of pressure reduction over an equilibrated subject to the point where a phase change from dissolved gas to evolved gas occurs. The arterial bubble theory, the notion of *in situ* gas formation causing nerve compression, and the theory of venous blockage have all been offered as the primary event (12, 20, 21). Impingement of intravascular and/or extravascular bubbles upon pain fibers has been postulated as the cause of bends (11). In addition to simply reducing the pressure over the

equilibrated subject, pre-treatment with hyperbaric pressure before exposure to subatmospheric pressure has been shown to evoke DCS at as low as five thousand feet (11, 21). The risk factors elucidated in the development of DCS are manifold: decompression rate, absolute pressure reached, time at reduced pressure, physical activity, obesity, and most recently, gender (22). Assuming that the symptoms are caused by nitrogen bubbles, fat emboli, thrombi, vasoactive substances or some combination of these factors, this author suggests that blood-borne substances that are not filtered or metabolized in the lung pose greater physiological risk once they enter the arterial circulation.

Operators, including pilots, divers, and astronauts, have been exposed to DCS-inducing environments for many years. These populations have used many different life supporting breathing systems with admirable success. Recent physiological research combined with new technology have certainly expanded the operating envelope of the human operator. Technology long associated exclusively with acceleration protection is now supporting operators at extreme high altitude. Equipment primarily developed to provide a suitable breathing environment now contributes substantial protection in the high-G environment. The exposure of the human operator to extreme physiological stress is not only likely but probable. Exposure to extreme low pressure without the benefit of denitrogenation or full protective coverage is likely to be capable of producing silent or overt DCS. Exposure to assisted positive pressure breathing (PPB) in excess of 60 mmHg in a population likely harboring a 25% incidence of PFO may produce right-to-left atrial shunting as a consequence.

Advances in ultrasound instrumentation and techniques now permit non-invasive determination of the presence or absence of PFO (28, 29). Cardiac anatomy, blood flow dynamics, leaflet function, wall thicknesses, and cardiac reponse to stress can all be assessed by a trained investigator. Many of the issues surrounding the relevance of PFO in DCS environments will be elucidated by this new technology.

Questions unanswered: Does PFO put the individual at increased risk when working in environments known to cause DCS? Is screening for PFO appropriate for high altitude pilots and astronauts? Can inferences from the diving experience be applied to the altitude environment with respect to PFO? What event or combination of events reliably causes right-to-left shunt in PFO positive individuals? Are silent bubbles truly silent for the long term? Studies are being designed and a few are currently underway in this laboratory in order to answer some of these basic physiological questions.

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MR. WALIGORA: You mentioned the 20 to 30% incidence of PFO; however, there is also an incidence of maybe half that, that is detectable without provocation. There may be 10-15% of individuals who normally pass blood from right to left.

MAJOR GARRETT: Yes, quiet breathing in some individuals is enough to show right to left shunt in people who are asymptomatic. Probably one in four people sitting in this room has such a potential defect. Were we to generate bubbles in those people from diving or flying, and were they to Valsalva at altitude or on descent, there may be right to left crossing of bubbles into the arterial system.

MR. WALIGORA: Would you predict that that could also happen without the Valsalva? In some people you get this flow of blood without any provocation, at certain points in the cycle.

MAJOR GARRETT: Yes, that is correct.

MR. WALIGORA: We have been doing altitude protocols that involve Grade 3 and Grade 4 bubbling in 50% of the subjects. We have done a lot of them, and we might expect that some of these people have had PFO bubbles if they are representative of the population. You would think that in some of these subjects bubbles must be passing into the arterial system.

MAJOR GARRETT: The magnitude of the bubbling would be a factor. The delta P between the right atrium and left atrium is another variable. Timing is also important. This is a dynamic situation and unless these variables come together just right, you may never see bubbles in the arterial system.

DR. BUTLER: There are several other criteria to consider. Even if there is a foramen ovale, even if there is normal communication of fluid between the two, there is also some sort of skimming effect. Is the blood that crosses normally a representative sample of the atrial blood which has the bubbles? There may be eddies, or some sort of skimming effect. What is the representative sample of blood that is actually passing through?

In addition, in order to impact a response, the bubbles have to go to an organ where you're going to see an effect. If your bubbles are indeed oxygen bubbles, or indeed they are early on in the decompression, or they are small enough, or they do not distribute to the heart, the coronary arteries, or to an area in the brain where you are going to get a gross motor disfunction, then you may not see the impact until there is enough gas accumulated. Previous studies have involved injections of massive amounts of air into the carotid artery of animals, some without anatomical configurations that could tolerate it, and there were no symptoms. You may have to

get into the more subtle symptom analysis, such as neuropsychiatric. That may be why previous investigators did not see gross symptoms.

DR. VAN LIEW: I do not expect oxygen bubbles to last very long. I would think that the situation that you would be most worried about would be when bubbles get into the brain, and that organ has a high nitrogen load. Probably in the altitude situation with preoxygenation the brain is no longer at risk because any bubble that gets there will be rapidly absorbed. But in the diving situation, where the brain may not have denitrogenated yet, a bubble could lodge and then grow.

MR. GILBERT: Does anybody know of a current active research project in which PFOs are being evaluated with altitude decompression sickness, using transcranial Doppler or any other means of determining whether you are getting arterialization of gas emboli?

MAJOR GARRETT: Here at Armstrong Laboratory I plan to investigate that exact thing in pigs. Pigs have a very similar PFO incidence to that of humans. In addition, they are echogenic.

MR. GILBERT: Are you using echocardiography?

MAJOR GARRETT: Yes.

DR. BAGIAN: If PFOs are that prevalent and if right to left shunting with bubbles occurs, then why do we not see bodies stacking up like cord wood? I am not saying bubbling does not happen because I think it does. But, if you cannot establish that there are actual clinical sequelae to these bubbles, you might ask that question. Do we sometimes learn more than we can really deal with appropriately?

Also, you mentioned at the very end of your talk that PFO is especially of interest or more critical in looking at EVA applications. I was wondering from what perspective you were saying that?

MAJOR GARRETT: The perspective was that EVA involves operating for a long time at what is envisioned to be fairly low pressures, high workloads, with coughs, sneezes, Valsalvas, and other straining kind of maneuvers that would lend themselves to changing the atrial dynamics and causing crossover.

DR. PILMANIS: It should also be noted that weightlessness results in higher central venous pressure, as in water immersion.

COLONEL WOLF: Do you know if anyone is looking at the aircrew autopsies done at AFIP to confirm the incidence of patent foramen ovale?

MAJOR GARRETT: I think that would be a valid inquiry. I am not aware that it has been done.

DR. FRANCIS: One way of considering your problem of not having a pile of bodies from bubbles and PFO is to use the analogy of the situation with open heart surgery. For many years people were leaving gas behind in the heart, and people were getting measured gas bubble emboli to the brain. There was apparently no problem as a consequence of this. There is a very good reason why: people did not look for it. I do not think that just because we do not see mass morbidity there is not a problem. You need to look for it, and then if you find nothing, fine, there is not a problem.

MR. WALIGORA: At NASA, the last 20 subjects in our altitude chamber studies were screened for PFO. The reason is because in a previous study we had two Type II DCS. We screened those two individuals with an echocardiograph and made the erroneous determination that those two had PFOs. Later with saline injection and very careful analysis we found that only one of those two had PFO. Nevertheless, we had already begun screening all subjects and found that with a very careful saline analysis, we got some pretty high PFO rates. They are probably not representative of the whole population. We got 40% with provocation and 28% without provocation. Thus, with subjects taken off the street, we had almost the same incidence as in the two people who had Grade 2 symptoms. In addition, the subject with the more significant Grade 2 symptom was the one that did not have it and the more questionable one was the one that did.

So now we are about to go back to not screening our subjects. And yet if you know that you have people bubbling and that you have PFO, screening seems like a reasonable thing to do. However, there is a big impact financially and NASA is not planning to continue screening.

LT COMMANDER CLARK: It might be helpful to look at some of the literature. In Haymaker's autopsy study of the fatal cases of altitude DCS , he found that the incidence of PFO was approximately 30-40%. That is probably consistent with the incidence in the general population.

In Moon's series published in Lancet, the 176 controls had 14% patent foramen ovale detected by bubble contrast. He felt that bubble contrast was superior to color flow Doppler. Of that 14%, 5% were spontaneously detected, i.e., without any provocative maneuvers, and 9% were detected with Valsalva. He then studied 31 divers with CNS DCS histories, which he subdivided into serious DCS, and mild DCS. None of the mild cases had a detectable patent foramen ovale, but of the 18 serious cases, over 60% had patent foramen ovale detected with bubble contrast, both with Valsalva and spontaneously.

The U. S. Navy has been looking at this problem now for the last two years. We have completed a study of 24 Type II altitude DCS cases. Of that 24 we have only had 3 that had bubble contrast detected patent foramen ovale. We are now thinking that maybe we are being overly aggressive. The 12% PFO in 24 cases of Type II altitude

DCS is consistent with the background population. Statistically, I do not think we are seeing a higher incidence of patent foramen ovale in serious or Type II altitude DCS.

Moon did some statistical analysis and felt that PFO was a significant risk factor for DCS and the probability of DCS was 1%. This is about a tenfold increase over the standard risk in sport diving of about 1 in 1,000. But based on our series of altitude DCS, we have not found that to be the case.

DR. VANN: Concerning the difference between altitude and diving, at altitude you are generally breathing oxygen, and oxygen is the most important factor in eliminating that bubble in the lung. You have a much greater driving force, so this may be why intravenous bubbles are more tolerable at altitude. When I see Grade 3 or 4 bubbles in a diver, at sea level breathing air, I worry a lot because that is associated statistically with Type II decompression sickness, whereas it is not as important at altitude because breathing oxygen clears those bubbles much more rapidly.

MR. WALIGORA: But ultimately you can get Type II symptoms at altitude and we do.

DR. VANN: Yes, indeed that is correct, and I think it is a problem. It is probably less of a problem at altitude, by virtue of breathing oxygen, and maybe divers should also breathe oxygen when they have high bubble grades.

DEATH FROM ALTITUDE-INDUCED DECOMPRESSION SICKNESS: MAJOR PATHOPHYSIOLOGIC FACTORS

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INTRODUCTION

The procedures presently followed to avoid life-threatening symptoms associated with severe bubble formation in altitude-induced decompression sickness (DCS) are cabin pressures usually greater than 10 psi or a variety of denitrogenation breathing periods suited to the exposure. The latter are variously planned, though are frequently based on trial and error efforts to decrease the nitrogen content of the blood and other similar tissues. These tissues may be considered to be equivalent to the most "at-risk" theoretical tissues having short half-times for nitrogen equilibration.

For flying exposures, the supersaturation risk is magnitudes less than that which occurs in diving, as shown by Masland's reference to nearly a half million exposures to high altitude with few deaths as of 1948 (10). To date, the number of deaths caused by DCS is still relatively small. Consequently, flying is safe. Training in altitude chambers is safe, too, because 30 minutes of denitrogenation, the norm, is usually adequate. Incidence of DCS per 100,000 exposures in chambers is near 100 and deaths are unheard of even following training exposures to FL 430 (13).

The most recent death from altitude-induced DCS during flying (13) primarily reminded us that overweight individuals are at high risk and taught us to reemphasize safe procedural methods in order to avoid exposures that create severe bubbles. In this one case, as well as historically, the pathophysiological lesson has been less instructive, however. The following review provides a summary of the DCS deaths that are documented and emphasizes particular points about severe DCS for which solutions still remain unclear.

PROCEDURES

Case files were reviewed from available and published documentation of deaths caused by actual in-flight exposure to altitude or exposure to decreased ambient pressures during training in a hypobaric (altitude) chamber. In addition to seventeen of eighteen cases reported by Fryer in Gillies' aviation physiology text (5), two other cases were included.* Altogether, a synopsis is presented to delineate

*This author agrees that case number 3 of Robie et al. (18) which Fryer (5) included, and which Davis et al. (1) did not include, is of doubtful validity. DCS bubbles were undoubtedly possible, and may explain some of the postmortem findings. However, in the author's view, hypoxia is the likely explanation for this fatality.

pathophysiologic commonality and similarity of etiology, if any. Case by case variability precluded statistical application.

RESULTS

The cases are listed and cross-referenced in Table 1. All deaths involved males, and eight were due to in-flight exposure to decreased atmospheric pressure (versus exposure in a hypobaric chamber). All of the exposures occurred with little or no denitrogenation according to the standards used today. Only one individual (case 19) received hyperbaric treatment, alas, without benefit. Symptom onset was at altitude in all but one case. Time to symptom onset varied. However, considering that nitrogen off-loading prior to high altitude exposure was minimal, it is not surprising that onset of symptoms was most often reported within less than an hour (see Table 2).

The pressure decrease from 1 ata that caused death was, in most instances, at or less than 200 Torr. Table 2 shows that fifteen of the cases involved exposures to altitudes greater than FL 300, though one case occurred at less than FL 250. In general, time and altitude were inversely correlated, as would be expected.

The individual medical history of each case provided some valuable information. The preexistent health problems summarized in Table 3 point to individuals who demonstrated a proclivity for excessive body weight. Commensurate with obesity is a usual tendency for fat-infiltrated hepatic cells and elevated serum lipids. Eight of the thirteen persons with fatty livers were described as overweight/obese. Only the Neubauer et al. (13) report provided documentation about a medical history showing hyperlipidemia and hypercholesterolemia in a hypertensive, obese individual. Only one individual had previously reacted badly to a high altitude exposure.

Cardiovascular pathology is of interest. A patent foramen ovale in five cases may have had a tendency to complicate the clinical picture by providing a convenient avenue for the passage of bubbles to the greater circulation. Table 3, however, indicates that this pathologic condition is not the primary threat in determining vulnerability to altitude-induced DCS. Fourteen cases involved atrial septa which were closed, whose condition was unknown, or were presumed normal. Alternatively, pulmonary arteriovenous shunts were not addressed by the preceptors, and present an enigmatic and questionable problem (see Discussion).

The results from autopsy (see Table 4) provide ample evidence of the disruption in the circulatory milieu caused by severe DCS. Throughout the vasculature of cerebral, pulmonary and cardiac circulations (also other systems) the response appeared to involve overwhelming loss of plasma as capillary permeability increased. Thus, effusion, congestion, edema, and in some instances, hemorrhage ensued. Control of plasma volume and immune response was sometimes affected, as evidenced by the hemoconcentration and leucocytosis in some cases. Uncontrolled diaphoresis and fever were also seen in other cases.

TABLE I. FATALITIES CAUSED BY ALTITUDE-INDUCED DCS* CASE EXPOSURE REFERENCE CROSS-REFERENCE

1. Chamber (7) Haymaker Case 1, Davis et al. Case 1, AFIP Access. No. 095412

2. Chamber (7) Haymaker Case 5, Davis et al. Case 2, AFIP Access. No. 100893

3. Chamber (7) Haymaker Case 2, Davis et al. Case 3, AFIP Access. No. 103767

4. Chamber (7) Haymaker Case 3, Davis et al. Case 4, AFIP Access. No. 100822

5. Chamber (2) Fryer Case 1, Davis et al. Case 5

6. Chamber (5) Haymaker Case 6, Davis et al. Case 6, AFIP Access. No. 113646, Masland Case 5

7. Chamber (7) Haymaker Case 4, Davis et al. Case 7, AFIP Access. No. 127451 Masland Case 6

8. Chamber (5) Haymaker Case 7, Davis et al. Case 8, AFIP Access. No. 293075

9. Chamber (2) Fryer Case 3, Davis et al. Case 9, Haymaker Case 9

10. Chamber (5) Haymaker Case 10, Davis et al. Case 10

11. Chamber (10) Masland Case 7

12. Flying (2) Fryer Case 2, Davis et al. Case 11, Haymaker Case 8

13. Flying (5) Haymaker Case 11, Davis et al. Case 12, AFIP Access. No. 570889

14. Flying (5) Haymaker Case 12, Davis et al. Case 13, AFIP Access. No. 638482

15. Flying (2) Fryer Case 4, Davis et al. Case 14

16. Flying (2) Fryer Case 5, Davis et al. Case 15

17. Flying (16, 18) Odland Case, Davis et al. Case 16, Robie et al. Case 1

18. Flying (18) Robie et al. Case 2, Davis et al. Case 17

19. Flying (13) Neubauer et al. Case

* All but cases 11 and 19 are also listed in Reference 5.

TABLE 2. ETIOLOGY

	NUMBER OF CASES
Little or no denitrogenation	19
Symptom onset at altitude	18

Time to Onset
(minutes)

≤30	30-59	60-119	≥120
5	7	5	2

Altitude of Exposure
(Flight Level)

≤200	200-249	250-299	300-349	≥350
0	1	3	7	8

Time from Symptoms Onset to Death
(Hours)

5	5-8	8-16	16-24	≥24
1	2	8	3	5

TABLE 3. PRE-EXISTENT HEALTH PROBLEMS

Health, General

5 - Average build, healthy
9 - Overweight, obese
4 - Health unknown
1 - Other

History of:

1 - Renal problems
1 - Hyperlipidemia
1 - Hypercholesterolemia
2 - Hypertension

Myocardial Defects

5 - Patent foramen ovale
14 - Closed foramen ovale or condition unknown

Pulmonary Defects

0 - Arteriovenous shunts ?

Fatty Liver

13

Prior DCS Reaction

1 - Neurologic (paralysis)

TABLE 4. AUTOPSY RESULTS

18 - Pulmonary Effects	11 - Fat Emboli
13 - Pleural effusion	8 - Pulmonary
11 - Congestion	6 - Cerebral
8 - Edema	1 - Cardiac
	4 - Renal

13 - Cardiac Effects	15 - Cerebral Effects
5 - Hemorrhage	3 - Hemorrhage
3 - Congestion	10 - Congestion/engorgement
4 - Edema	6 - Edema
8 - Pericardial effusion	2 - Ischemic foci

Other

6 - Hemoconcentration (Hematocrit 57-70%)
7 - Congested abdominal organs
5 - Renal congestion
3 - Leucocytosis (May be 8-10 times normal count)
2 - G.I. system hemorrhage
2 - Brainstem edema

Fat emboli were present in eleven of the cases. The source of fat emboli is unclear, though Fryer (3) provides strong implication for fatty liver cells. Hepatic cell vacuolation was only described in two of the autopsies. Omental adipose tissue, which was excessive in many of these cases, should not be discounted as a major source of nitrogen for bubble growth and, perhaps, some fat emboli released via the splanchnic circulation. Fat emboli, though, were never implicated as the cause of death.

Neurologic and neurocirculatory symptoms arose early, but not exclusively. Pulmonary symptoms probably accounted for shock seen in many of the cases. With enough time, damming of the bubbles in the pulmonary circulation occurred. Pulmonary effusion, congestion and edema resulted. Microcirculation in other tissues was just as susceptible, provided that the arterial gas bubbles, by chance, lodged there. Blood pressure frequently remained stable for a long time, masking the seriousness of the illness. Invariably, though, there was development of hypotension and peripheral vascular collapse.

DISCUSSION

Since Fryer's report (3) annotating the preliminary symptoms in 15 fatal cases, the evidence remains that the type of symptoms at onset does not predict the eventual severity of a case. DCS entails a spectrum of insults which, given enough time without treatment, invariably overwhelm the system (pulmonary DCS, shock, etc.). Because the right heart's output goes to the lungs, involvement of the pulmonary circulation is no surprise.

In severe DCS cases as seen here, though, eventually the greater (arterial) circulation is brought into the developing drama. Unfortunately, a synopsis of these nineteen cases does not explain all the reasons for the existence of arterial DCS gas bubbles nor the likely sites for the bubbles to settle. Indeed, the bubbles were apt to lodge anywhere.

Only five of the cases involved indisputable evidence of foramen ovale that were patent. Probably not all of these were functionally patent (9), and even if they were, the incidence does not help to explain mortality with the same ease as has been concluded in diving DCS (11, 12). The statistics for patent foramen ovale in these 19 cases closely compare with other population studies (6).

The existence of a patent foramen ovale does not indicate it is functional or that bubbles shunt from right-to-left through it. In ten of these cases, symptoms indicative of pulmonary DCS developed. Were the pulmonary resistance to blood flow great enough and the damming of bubbles sufficient enough to back up the venous bubbles to the level of a right atrial septal defect, then right-to-left shunting would be likely. Otherwise, the pressure difference across the septum would be inappropriate for bubble passage. Still, fourteen cases were without septal defect, and thus, another explanation is necessary to explain the presence of bubbles in the greater circulation.

Given sufficient nitrogen to seed the gas nuclei of the arterial circulation, rapid bubble growth and insult of the tissue as seen in autopsy (Table 4) would be inevitable. Neuman and Bove (14) consider this as one alternative in severe diving DCS combined with trauma-induced arterial gas embolism that is refractory to immediate recompression therapy. The degree of supersaturation, given the obesity, fatty liver, altitude and duration for most of these cases, is probably adequate (Tables 2 & 3). Thus, gas seeding of arterial gas nuclei may be a contributing cause to the tissue pathology seen in these cases.

Another alternative may be sanctioned by the fact that eleven of the cases exhibited fat emboli in tissues that were dissected during autopsy. Fat emboli do not conclusively demonstrate disruption of cells with high fat content (e.g., bone marrow and liver cells). Higher than normal circulating lipids may explain some fat emboli, and even account for their post-mortem finding in lung tissue. Their presence in other tissues, however, indicates that fat emboli passed to the arterial circulation. Four of the cases with patent foramen ovale exhibited fat emboli downstream of the pulmonary

filter. Yet, seven other cases without potential right-to-left atrial shunt also demonstrated fat emboli.

Population statistics of the incidence for arteriovenous (A-V) pulmonary shunts and anastomoses are apparently undocumented. The studies (8, 17, 19, 20, 21, 23,) which have looked at the existence of a circulation which allows for bypassing the small lung vessels or allows collateral filling via interarterial anastomoses suggest that unusual pulmonary flow patterns may not be rare. Pulmonary shunts should receive at least as much consideration in right-to-left passage as atrial septal defects.

Prevalence of pulmonary A-V shunting may be as high as 61% in neonates and involve as many as 1-2 shunt vessels in each lobule (23). Other studies using lungs from adult accident victims and injecting glass spheres to ascertain the degree of A-V passage point to shunts with diameters of 200 microns in transitional/respiratory zones (21). Near the alveoli, shunt vessel diameter may be smaller (20-25 microns), but within the conductive zone is as large as 500 microns (15, 20). If pulmonary artery pressure rises or alveolar oxygen tension diminishes (as one may expect in pulmonary DCS), then the degree of flow and/or recruitment of shunt vessels is increased (15). Conversely, 100% oxygen decreases shunt flow (15).

Gas seeding and/or bubble shunting to arterial circulation, then, may explain the extensive damage that ultimately caused death in less than a day the majority of the time (Table 2). As with most renewable tissue, the blood cells and the endothelial lining of blood vessels can withstand some small level of insult by intravascular bubbles without clinical consequence. However, when the number/size of arterial gas nuclei increases to the embolism stage (during DCS) and pervasively interacts with red cells, coagulation proteins and the proteins responsible for the integrity of the endothelial barrier, then significant impairment of normal circulation can be expected (22). The consequences fit the autopsy results seen in many of these deaths.

CONCLUSION

Fatalities from altitude-induced DCS are exceptionally rare, in contrast to diving-induced DCS. The nineteen cases synopsized here are least instructive of the physical threat involving such parameters of time and altitude. In short, most people can withstand the exposures that killed these men. These cases, on the other hand, delineate situations in which an exceptional nitrogen store is found in obese and overweight individuals which makes routine exposure to low pressures very life threatening to this segment of the population. This is not to say, however, that all other individuals can fly to high altitude with relative impunity. Given the proper circumstances, all people are at risk at high altitude. Five of the deaths occurred in healthy men of average build. Patent foramen ovale may have played a role in some, but not all, of the other cases. Gas seeding of arterial gas nuclei and pulmonary arteriovenous shunts may provide additional information for explaining some of the threat of exposure and resulting pathology.

RECOMMENDATION

A better indication of the prevalence of pulmonary A-V shunting in humans is needed. Additional research would be prudent in order to determine normal pulmonary vascular response throughout the lung during mild, moderate and severe DCS, or insult by continuous venous gas embolization.

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"SILENT BUBBLES": THE ASYMPTOMATIC GAS PHASE

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This section will attempt to provide the reader with a synoptic view of "silent bubbles." To this end, topics such as their formation, detection, and pathophysiology will be reviewed. It will become evident that the distinctions between the "silent bubbles" and the "clinical" ones are often vague; contrasts become blurred on the borders.

Workshops lend themselves to speculation, and, to this end, latter portions of this paper are of that nature; many advances arise from cases of "over-reading" of the data, but it is hoped that this will not usher in error. Much of the data we have today are conjectural; there is often an absence of a theory to unify them. Some attempts are made herein, siding with the aphorism of renowned British astrophysicist Sir Arthur Eddington, "Be suspicious of all data until it has been confirmed by theory." It will also be evident that the author often groups together tissue gas micronuclei and asymptomatic decompression bubbles as dimensional extensions of one another. I believe that the progression of decompression calculations from statistical predictors to deterministic predictors will arise from a better understanding of these interrelationships.

I. BACKGROUND

A. THE *IN-VIVO* GAS PHASE: AN HISTORICAL INTRODUCTION

It was while crossing the Andes in 1590 that the Jesuit, Juan de Acosta, observed the effects on humans of exposure to high elevations. He was later to write, "the element of air in this place is so thin and so delicate that it is not proportioned to human breathing." His vivid descriptions and grim accounts of mountain sickness were of special scientific interest to European naturalists who coupled these with the findings of Torricelli and Pascal and the newly-invented barometer. We exist at the bottom of an "ocean of air," and with the invention of the pneumatic pump by the German engineer Otto von Guericke, they could test its effects upon living creatures.

In 1670, the English scientist Robert Boyle [1] published his findings describing studies of animals in evacuated chambers. Contemporary research by Pascal on the Puy-de-Dome had demonstrated the reduced pressure of air on mountain summits. The concept of distinct species of gases was not yet known to scientists nor was a knowledge of oxygen. Thus they reasoned the problem lay in the inability of this rarefied air to rid the body of its heat (the Aristotelian concept of the function of the lungs).

Boyle commenced his investigations of reduced air pressure by utilizing the newly invented vacuum pump, and, engrossed in these studies, even created his own version. He placed into his "exhausted chamber" all manner of living creatures in an attempt to study their reactions to the newly-described "rarefied atmosphere." In one such study, he decompressed a snake and noted that it writhed in its death throws. Boyle carefully noted "a small gas bubble to be seen within the eye of the viper." He wrote,

"Another suspicion we should have entertained concerning the death of our Animals, namely, That upon the sudden removal of the wonted pressure of the ambient Air, the warm Blood of the those Animals was brought to an Effervescence or Ebullience, or at least so vehemently expanded as to disturb the circulation of the Blood, and so disorder the whole Oeconomy of the Body."

It is apparent that Boyle even laid plans to study men, for in that same communication he was to write:

"And I have also had thoughts of trying whether it be not practicable, to make a Receiver, though not all of glasse, yet with little glasse windows, so placed so that one may freely look into it, capacious enough to hold a Man, who may observe severall things, both touching respiration, and divers other matters; and who, in case of fainting, may, by giving a signe of his weakness, be immediately relieved by having air let in upon him."

This was the first observation of gas bubbles produced by a change of pressure in living creatures. It is of interest to this Workshop to note that these decompression bubbles occurred during studies of what would today be termed "hypobaric or aerospace physiology."

Erasmus Darwin [2], ancestor of Charles Darwin, also noted gas bubbles in 1774 in decompressed blood. He was investigating the proposition that "elastic vapors" existed in blood that could cause "lunar and equinoctial maladies" attributable to variations of atmospheric pressure. He noted, however, that while extracted blood would bubble and froth at reduced pressure, this did not occur if the fluid remained in a ligated and excised vein.

We concern ourselves in this section with an *in-vivo* gas phase that is asymptomatically present in the body. The presence of such an entity was evident to Haldane and coworkers [3] from the correlations of pathology and symptomatology. The term "silent" was first appended to the bubbles by Behnke [4], in recognition that they might exist apart from overt distress. It is, however, a relative term just like "inaudible." And as one might ask, "inaudible" to whom, one could also ask, "silent" to whom?

The focus of the great majority of workers in the field of decompression physiology has been on dissolved gas dynamics with the objective of creating more

efficient or safer decompression tables. To those of us who first learned decompression pathophysiology from autopsies of rats in all stages of post-decompression stress, there never existed "bubble-free decompressions."

B. FORMATION OF THE *IN-VIVO* GAS PHASE: STRESS-ASSISTED NUCLEATION

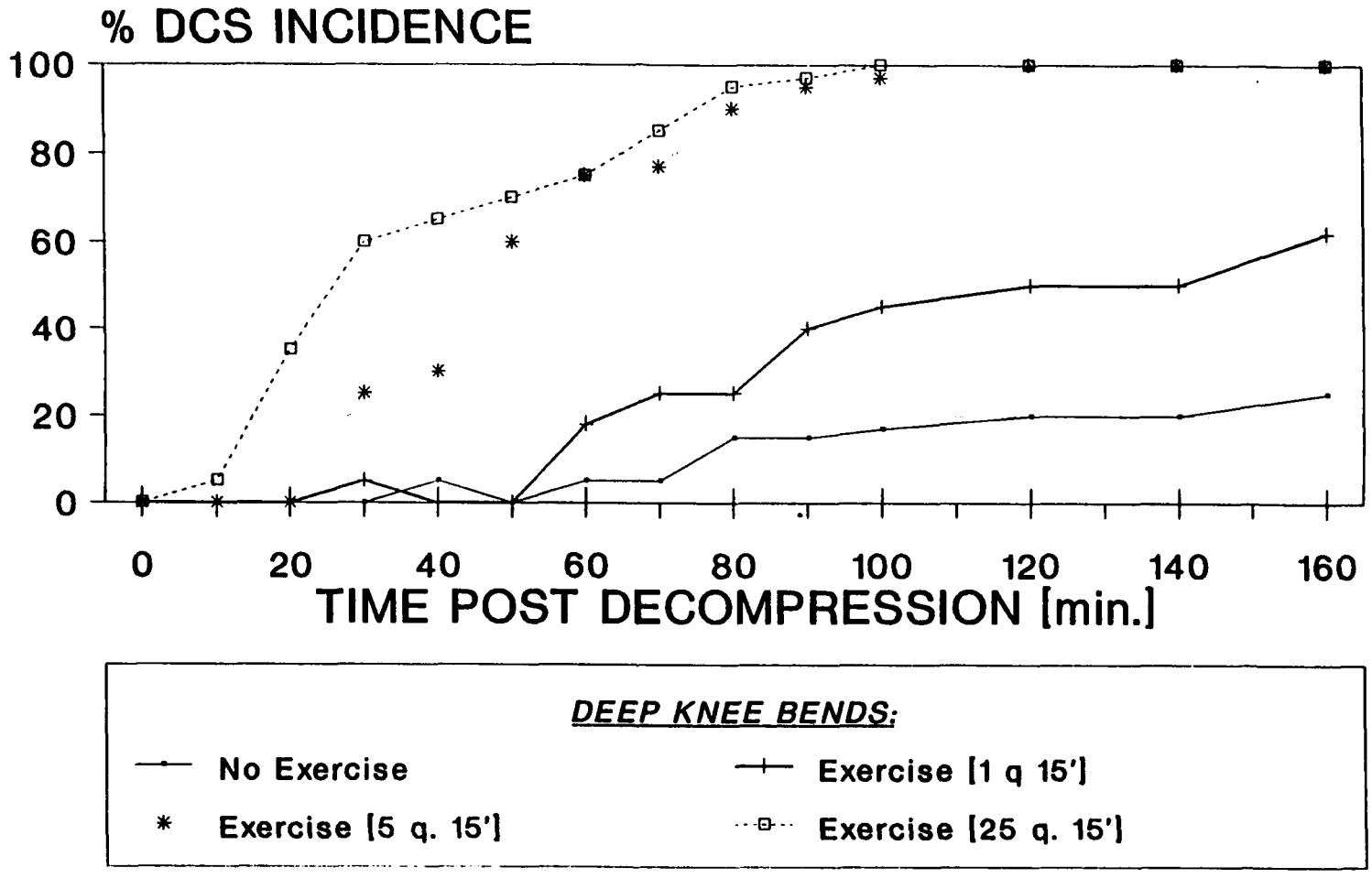
Behnke and Hempleman were the two individuals responsible for the genesis of the concept of the "silent bubble." Behnke first postulated that such an entity could arise in individuals not afflicted with frank decompression sickness (DCS), that is, the disease would be sub-clinical or "silent." Hempleman's studies with repetitive decompressions in goats led him to suspect tissue gas phase formation termed by him the "tissue-bubble complex."

1. *In-vivo* Gas Phase Formation

Earliest work on the question of gas phase formation in living creatures began in earnest during the Second World War. The prevention of this formation was paramount; since exercise should increase tissue gas elimination via increases in perfusion (according to J. S. Haldane and A. V. Hill), one could infer that exercise should be beneficial and reduce the incidence of "aviators' bends" in a population of men. Studies surprisingly indicated that human volunteers displayed an increased tendency to DCS when exercising during altitude decompression. Figure 1, redrawn from Ferris and Engel [5], shows the number of men who developed DCS in their lower limbs while performing step (stair climbing) exercises at simulated altitude. Additionally, the response was proportional to the degree of exercise, and there was an indication that the subjects reached a plateau where further exercise did not produce an increase in the number of responders (or decrease in time to response).

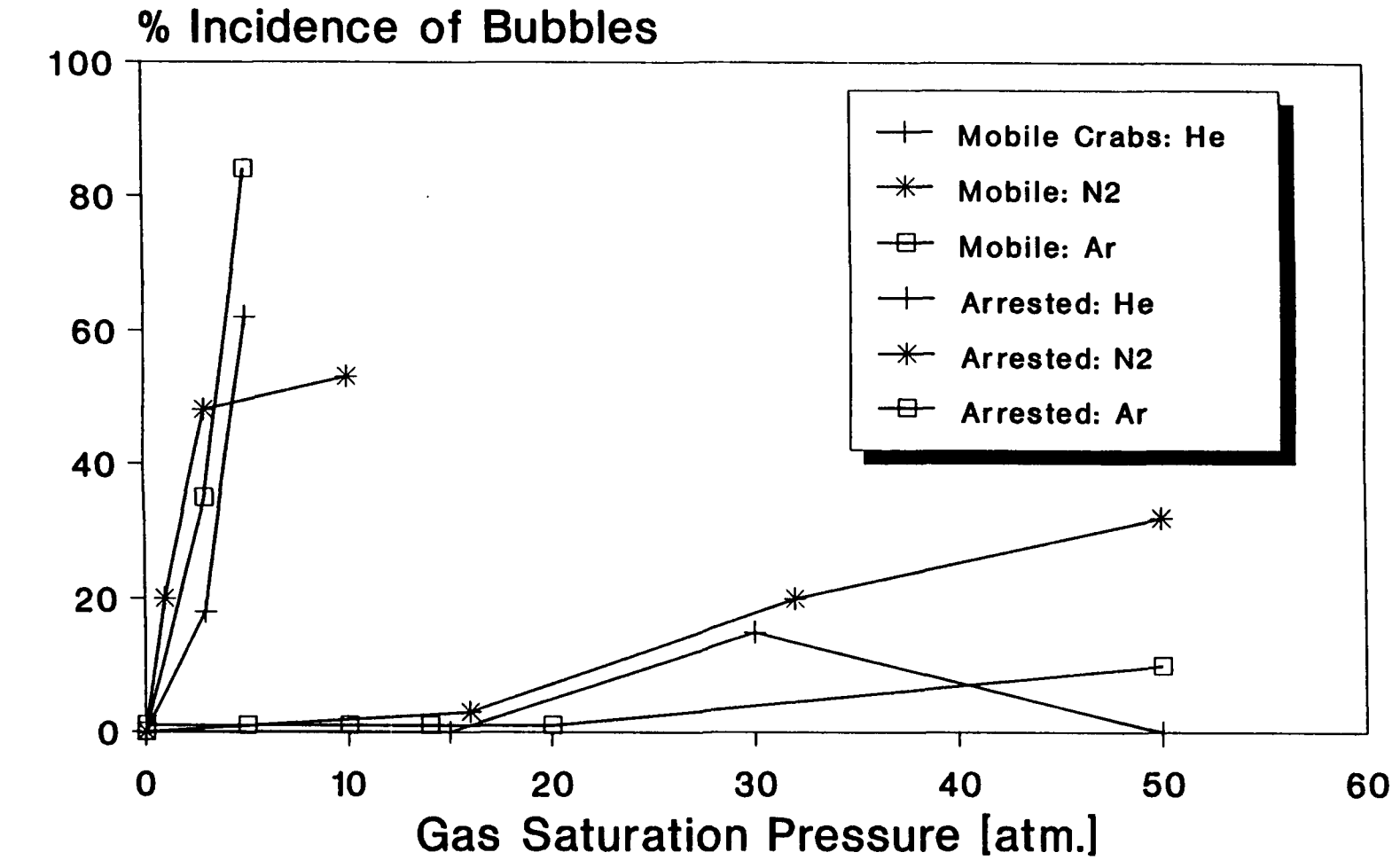
Partly from this work was spawned the question of tissue nucleating sites, and the vast majority of evidence for muscle and joint kinetic activity as a provocative agent for stress-assisted gas phase formation derives from animal experimentation. Since these studies were conducted during World War II, the work was directed towards the genesis of a gas phase in the crews of high altitude bombers. The research of Harvey et al. [6-9] indicated the presence of stable tissue gas micronuclei, possibly sequestered in hydrophobic conical pores; as did the more contemporary studies of Evans and Walder [10], Vann et al. [11], and Daniels et al.[12].

Other work by Blinks et al. [13] indicate that stable tissue gas micronuclei are of lesser importance and are overshadowed by other mechanical mechanisms operating under physiological conditions. These mechanisms include kinetic activity of muscles and joints (to be described below as "viscous adhesion"). Hemmingsen's experiments [14] using crabs as subjects demonstrated a resistance to the formation of a decompression gas phase (seen as bubbles through the carapace) when the feet of the crabs were immobilized with epoxy adhesive. A similar decompression without the kinetic restraint produced numerous visible gas bubbles (Figure 2).



Redrawn from Ferris and Engel (1952)

Figure 1. Altitude decompression sickness during graded exercise vs. rest.



redrawn from Hemmingsen, 1989

Figure 2. Bubble formation in joints of mobile and resting crabs.

The intensity of exercise, the particular muscles involved, and the point in time during the compression/decompression cycle play a role in determining the outcome [15,16].

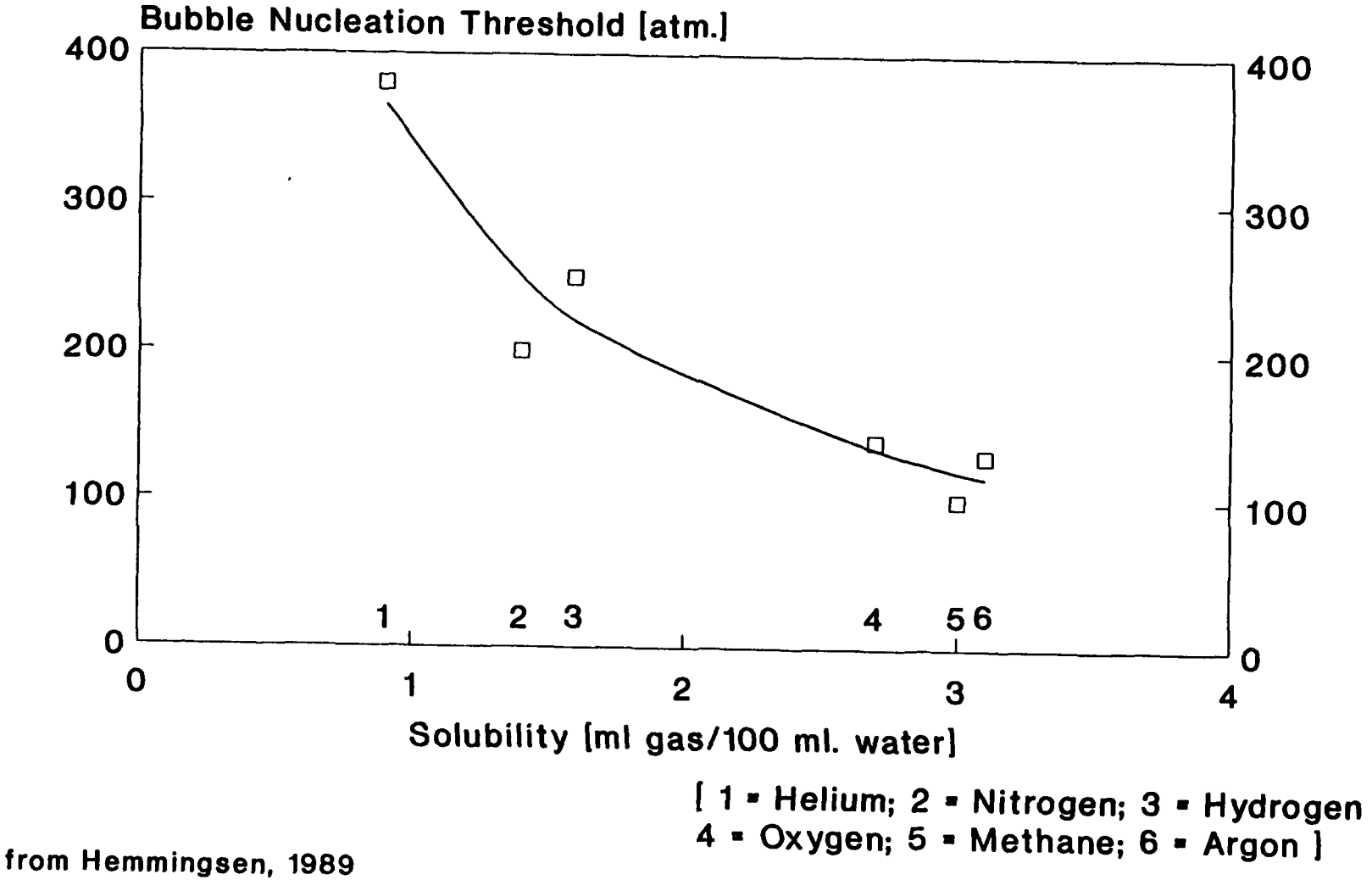
2. Mechanisms of Tissue Gas Phase Generation

The production of a gas bubble in a liquid is a process initially requiring the creation of a void or cavity in the fluid, a process referred to as "cavitation." Water is quite unique in its high cohesive energy, a consequence of its hydrogen-bonded structure; these bonds not only result in a relatively high boiling point for a liquid of its molecular weight, but also in the difficulty in fracturing the fluid and creating the vaporous cavity that, during decompression, quickly can fill with the dissolved inert gas.

To fracture pure water, the theoretical limit requires that the gas supersaturation be on the order of 200 to 4,000 atmospheres [atm.] [17]; the variation is the result of initial assumptions made. A deep-sea diver composed of kinetically undisturbed, nuclei-free water could theoretically ascend directly to the surface without a requirement for decompression after spending a day at a depth of six miles; in actuality, experience has shown it to be more in the range of 25 to 30 feet.

The generation of a separated gas phase from a supersaturated solution involves a large entropy change (that is, the complete separation of the liquid and gaseous components into two distinct phases); for inert gases (i.e., not reactive with water) the change is favorable with regard to enthalpy (and overall free energy) but the reaction proceeds sluggishly. Such a process is referred to as **"suppressed transformation,"** and examples include the superheating and supercooling of water, the formation of fog droplets, and supersaturated solutions of solids in a liquid. Phase transformations proceed more easily when "assisted," which is often accomplished in real physical systems by the presence of either impurities or mechanical forces or stresses.

Some form of "stress assisted" mechanism is postulated to be responsible for the genesis of a decompression gas phase in living tissue. For the last century, the greatest input of effort in decompression physiology has been directed towards the study of (primarily) whole-body gas uptake and elimination. Most likely the result of the legacy of J. S. Haldane, far less thought has been in the direction of the mechanisms by which an *in-vivo* gas phase forms; theoretical treatments have been deficient and recourse has been taken to experiment to obtain the requisite parameters. It is evident that, depending on solubility, a supersaturation of several hundred atmospheres is required [18,19], and it has been known for one and a half centuries that a change of two atmospheres of pressure is the maximum attainable in humans (or other large animals) (Figure 3).



from Hemmingsen, 1989

Figure 3. Homogeneous nucleation thresholds for various gases in water.

Once a spherical bubble has been formed, the conditions for stability are given by the Laplace equation:

$$P_g = P_{ambient} + 2y/r, \quad [1]$$

where P_g is the pressure of the gas within the bubble, *y* is the surface tension of the fluid medium, and r is the radius of the bubble. Because of the effect of surface tension, the internal pressure of a gas bubble of r = 1 *u* at 25°C is 143 atmospheres; smaller, nascent bubbles would have considerably higher internal pressures. Clearly some mechanism must bring the bubble through the formative process to a radius that can be stabilized by inert gas partial pressures of the magnitude actually encountered.

a. Nonspontaneous Nucleation

To solve what is an apparent dilemma, several possibilities have been tendered, all of which find their origin in the knowledge that kinetic activity provokes bubble formation in tissue [14]. **Seed micronuclei** may exist as small volumes of a gas phase stabilized by adsorbed shells or in hydrophobic crevices of surface asperities exposed to the aqueous environment. It is commonly observed *in vitro* (in instances not involving decompression) that sessile gas bubbles do arise from micropores in hydrophobic materials previously exposed to the air. These surfaces lose their ability to promote bubble formation when subjected to hydrostatic pressure since, it is surmised, their nuclei collapse. Within living organisms, there are no instances where internal structures (such as within a blood vessel) have been directly exposed to the gas phase since conception and development. However, it has been shown by calculation that in acutely angled hydrophobic crevices, a gas phase may spontaneously arise by statistical fluctuations in the thermal motion of the dissolved gases [20,21]. The location in cells of such regions of hydrophobicity directly exposed to the aqueous environment is unknown.

Albano [22] refers to pycnometric experiments performed to determine tissue volume decrease at 11 ata. The compressibility of gastrocnemius muscle is given as 39.7×10^{-5} [cc gas/cc tissue]. There is no evidence, however, that these volume decreases can be attributed solely to resolution of a micro gas phase and not to conformational changes in the biomacromolecules. Liebermann [23] studied gas phases during growth and decay following repeated compression/decompression cycles. From observations with the microscope, he noted that a small deposit remained when the gas phase shrank, and it was from this deposit that the bubble could again reappear if there was again another decompression. The volume of the gas in the residue was calculated to be between 10^{-4} and 10^{-5} ml.

There may exist solid surfaces that are hydrophobic and foster the adsorption of inert gas or the quasi-dissolution of gas into intermolecular spaces of the solid structure [14]. A stable gas phase would thus form, but would be destroyed by the application of pressure. Only very weak dipole-induced dipole forces would be involved with typical diving gases, however, and these dispersive forces may not be

strong enough to lead to a consequential adsorption of inert gas from an aqueous solution. It is known, however, that an aqueous solution of proteins dissolves more inert gas than a like volume of pure water.

b. Spontaneous Nucleation

"Spontaneous" refers to the ability for a gas phase to form without the prior condition of the presence of a stable gas micronucleus. It is necessary that sufficient energy be deposited in a small volume within the liquid such that phase separation can result. Under this heading are grouped "homogeneous" and "heterogeneous" nucleation.

Homogeneous Nucleation

This process involves the rupture of the water structure to create a cavity; surfaces containing gas are not involved. As mentioned, cavitation effected solely by supersaturated gas in water does not readily occur [18, 19]. **Negative hydrostatic forces** capable of rupturing the water structure in an environment free of nuclei can be created by mechanical forces. For example, fracturing can occur when liquids pass through a constriction and is termed Reynold's cavitation [24]. The viscoelastic nature of connective tissue means voids could be created in the resting tissue during elongation by opposing muscle groups.

Nucleation resulting from **shock waves** is the product of rupture of the water structure induced by tensile forces of the compression and rarefaction. These shock waves might be produced in the lower appendages when walking.

Tribonucleation [25] can effect the formation of a void and a subsequent stable gas phase when two surfaces are brought into contact with one another and then separated. It is a well-known fact that a solid body, such as a stirring bar, when rubbed against a vessel wall, will elicit the formation of crystals in a supersaturated solution or cavitation bubbles in a superheated one.

In a process described by Campbell [26] and termed "**viscous adhesion,**" negative pressures are generated when adjoining surfaces are separated; the negative forces during withdrawal can be considerable. The theoretical negative tension generated by the separation of two round, smooth surfaces is given by:

$$P = 3\, n\, v\, r^{2/l^3}, \qquad [2]$$

where P is the negative pressure [dynes/cm^2] generated between the surfaces and within the liquid, n is the viscosity [dynes-sec/cm^2], v is the velocity of separation [cm/sec], r is the radius of the surfaces, and l is the separating distance. As an example, when v = 0.1 cm/sec, r = 0.1 cm, and l = 10^{-4} cm, then P = 30 atm (in water).

Heterogeneous Nucleation

Heterogeneous nucleation is produced by the adsorption of gas onto hydrophobic particles. The presence of the surface per se is the important characteristic and not the shape, e.g., a cavity. The surface acts as an accumulator to bring a sufficient number of gas molecules into close proximity to form the nascent nucleus. This process is not considered particularly efficient or of much biophysical importance.

C. ULTRASOUND AND HYPERBARIC PHYSIOLOGY

1. Nature of Ultrasound Waves and Gas Phase Detection

Sound waves, or matter waves, involve the rapid translocation of molecules in the transmitting medium with the detectable frequency in the range of 20 to 20,000 Hz. Above those frequencies lies the region of "ultrasound;" for bubble detection, the frequencies are on the order of 10^6 Hz. A sound wave passing through a medium -- solid, liquid or gas -- is reduced in intensity according to the formula:

$$I = I_o e^{-2\alpha x} \qquad [3]$$

where I is the sound intensity, x the distance traveled, and *a* is the amplitude absorption coefficient. In soft tissue ultrasound will be absorbed to a degree *proportional to the frequency*. It is the absorption that basically limits the depth of penetration; the signal must also traverse the path back.

In decompression gas phase detection, reflection and scattering of ultrasound is a much more pronounced and useful effect. Because of the large differences in acoustic impedance between liquids and gases, almost no transmission takes place at a liquid-gas interface, and thus gas bubbles will effect a strong reflection of the ultrasound beam.

A program to detect a decompression gas phase was initiated at the Canadian Forces Institute of Aviation Medicine in 1962 by Kidd [27,28]. This was a through-transmission system; the presence of a tissue gas phase would reduce the intensity of an ultrasound beam transmitted between two transducing crystals. A gas phase was detected *in-vivo*, but the project was not continued for financial reasons. A combination transmission-reflection system ("back peak") method was reported by Walder, Evans, and Hempleman in 1968 to detect a decompression gas phase in a guinea pig's leg [29]. Mackay and Rubissow [30] in 1971 developed an early imaging device for *in-vivo* gas detection; both devices require that the subjects remain motionless. This caveat obviously limits their utility with both human and animal subjects.

2. The Doppler Effect and Doppler Flowmeter Bubble Detectors

Any time there is a motion of either the transmitter, receiver, or scatterer with respect to each other, a shift of the transmitted frequency will be found. This shift, first derived by the Austrian physicist Christian Doppler to explain variations in the color of stars (his paper was entitled "*On the Colored Light of Double Stars and Certain Other Stars of the Heavens*"), is termed the Doppler Effect and exists for all types of waves. Thus ultrasound waves reflected from moving red blood cells and the gas bubbles contained therein will have a slightly different frequency from the original transmitted signal. The circuitry of ultrasonic Doppler flowmeters will produce a signal output only to reflections that have experienced a Doppler shift (from bubbles in flowing blood, for example); no Doppler shift is produced by reflections from stationary structures (e.g., static tissue gas phases).

In 1968, two groups simultaneously reported the detection of decompression gas bubbles *in-vivo* by means of Doppler ultrasonic flow meters [31,32]. Early work was directed to produce "early warning" devices for decompression sickness [33], though contemporary studies by Powell [34-36] indicated that this probably was not possible based upon the natural history of the development of the decompression gas phases. Additionally, the gas locus of the gas phase responsible for joint pain is most likely **extravascular** and not amenable to Doppler detection; it probably effects pain by nerve ending distortion [37,38].

In-vitro studies by Monjaret, Guillerm and Masurel [39] demonstrated the near proportionality of the amplitude of the Doppler bubble signal to the radius of the bubble, a result expected from the work of Nishi and Livingston [40]. Because of the impedance mismatch, gas bubbles in the blood will scatter ultrasound to a much greater degree than solid particles of the same diameter. In a good signal-to-noise situation, the amplitude of the Doppler signal rises more than 10 dB above the blood flow signal; in its principal frequency band, the reflection can be 20 dB above the blood flow Doppler signal. Gillis [41], using graded glass microbeads injected into the femoral vein of swine, determined the threshold for microbubble detection. Beads of 37 to 43 micra diameter produced individual Doppler "chirps." Solid beads did not produce as large a reflected Doppler signal as the hollow glass microballoons of the same size.

Nishi [42] has provided a careful review of the theoretical acoustic scattering properties of gas bubbles with respect to Doppler detection. His conclusion was that *in-vivo* sizing of bubbles was only crudely possible, and absolute values of size could not be obtained without an internal *in-vivo* reference.

In-vitro measurements by Hills and Grulke [43] indicated that the minimum aurally detectable bubble size in blood was a function of the blood velocity. They found that a bubble of 20 micron radius could be detected if the mean fluid velocity was 55 cm/sec, and the minimum detectable size was 90 microns if the velocity was only 20cm/sec. In the heart and larger arteries, they predicted that the smallest bubble

detectable (on the basis of reflection, velocity, and background noise) would be 20 to 25 micra. This background noise of the heart walls limits precordial detectability as even at resonance, the scattering intensity for a smaller bubble would be an order of magnitude less than that of the 25 micron bubble [44]. It is the definite experience of many investigators that gas bubbles can be easily detected by Doppler ultrasound systems in peripheral veins, while at the same time they cannot be detected precordially.

II. PATHOPHYSIOLOGY OF THE GAS PHASE

A. THE HALDANE PARADIGM

The finding of gas bubbles following decompression is at variance with the Haldanian principle that "clean" decompressions are devoid of a gas phase [3]. In actual practice, gas bubbles are often present and, even when they do not lead to decompression sickness, can change the tissue gas elimination rates as first indicated by Hempleman. This can lead to actual problems in cases of either extended decompressions (e.g., from saturation) or repetitive decompressions [45] since all table calculations assume the entirety of the inert gas to be in the dissolved state.

In the late 1960s and early 1970s, researchers were surprised to find Doppler-detectable bubbles in "bends"-free decompressions; now virtually everyone realizes that some degree of gas phase formation can occur in the body following a decompression. Gas bubbles are detectable with Doppler ultrasonic flowmeters in all representative cases from short bounce dives to slow, saturation-dive decompressions. The equation

Bubbles = "Bends"

is not true under many, if not most, conditions.

Gas phase formation occurs in many tissues of the body and the manifestation of decompression sickness on any given decompression is multifactorial, consisting of at least three facets: (1) the rate of gas uptake and elimination in a given tissue, (2) the tendency for cavitation and gas phase growth to occur in that tissue, and (3) the pathophysiological response to the gas phase that forms [46]. It appears that some tissues can tolerate the presence of a gas phase without damage or evoking pain (muscle, for example), some (e.g., bone) would not be painful but could sustain injury, others produce graded pain (e.g., connective tissue of the diarthrodial joints), and yet others, no pain but severe pathological manifestations (e.g., motor neurons).

No doubt, the best method for detection of a "silent" decompression tissue gas phase would be direct interrogation of the tissue in question with, for example, B-mode ultrasound. Unfortunately, some portions of the body are not easily penetrated by ultrasound (bone) while others (the central nervous system) are very extensive.

B. TISSUE GAS PHASE

One of the most difficult aspects of decompression sickness to study is gas phase formation at the cellular level. At the level of light microscopy, the depth of penetration is on the order of a few millimeters at most. If care is not taken to cover the tissue under investigation (e.g., with a glass slide), dissolved inert gas rapidly escapes and a gas phase is never seen to appear. Whole mount histological specimens of rat and rabbit leg and abdominal muscle tissue indicate that the predominant locus for gas phase growth is in the microvasculature [47]. While the initiating gas micronucleus may be extravascular, growth occurs in the vascular channels in muscle and even predominately intravascularly in adipose tissue. As this microcirculatory gas phase grows, bubbles will eventually appear in veins draining that tissue from which it spawned. There exists no solid evidence, however, that limb-bend decompression sickness results from intracapillary gas phases.

The earliest ultrasound studies of the dynamics of the growth of the tissue gas phase in rats indicated a long time course; this course roughly paralleled that for the development of DCS problems [34]. There were no experimental indications of a lower limit of decompression stress that bounded gas phase formation. Studies by Hempleman [48] had likewise indicated that in goats a residual gas phase remains following asymptomatic decompression that is able to influence the results of a second, normally safe, decompression.

To date, the locus for the pain provocation in joint tissue has not been located, and thus we can only speculate on its *in-vivo* appearance. The tissue distension mechanism of Nims and Inman and Saunders is certainly interesting. While the Ruffini bundle of tendons and ligaments has been proposed as a suitable suspect, no empirical evidence yet implicates it.

In central nervous system tissue, studies conducted over several years have been inconclusive regarding the natural history of lesions. Both autochthonous (in situ) and infarct mechanisms have been proposed. This author favors those mechanisms utilizing stress-assisted nucleation of autochthonous in cord tissue. The kinetic stress derives from the movement and stress upon the cord since it is located within an articulated structure. Numerous "space occupying lesions" have been found in brain and cord in experimental subjects killed just prior to decompression [49, 50].

C. GAS BUBBLES IN THE VENOUS RETURN

1. Doppler Ultrasound Monitoring and DCS Determination

If one understands that "silent" gas bubbles are produced in tissues in proportion to the gas solubility and total volume of that tissue in the body, then it is clearly evident that we are monitoring bubbles not only from "bends-producing" tissue but from many tissues of the body. From autopsies of large rats subjected to increasing time at pressure, it can be easily noted that gas bubbles could be detected

earliest in veins draining abdominal tissue [35, 36]. The femoral vein, for example, is a contributor of bubbles, but it is by no means the only or even a major source.

Gas remaining in solution following decompression will not produce a damaging effect. A gas phase forms in the region of the micro-circulatory systems following a reduction in pressure because this is the region of highest gas tension and low hydrostatic pressure. This gas phase is autochthonous ("thrombic-like," grows in *situ*) at least in muscle tissue; the locus of this growth is intravascular. It is clear from numerous microscopic views [47] that the term "bubble" with its geometrical connotations is incorrect to describe the gas phase. Unquestionably, stagnation anoxia would result from the intravascular gas occlusion.

The preponderance of the gas phase that forms is not in tissues associated with pain-only, limb-bend decompression sickness; muscle and adipose tissue for example are the primary producers because they possess a large portion of the body's soft-tissue weight. When released into the central venous system, the gas phase spawned in tissue accounts for the majority of gas bubbles which are detected by Doppler precordial detectors. When in limited quantity, venous return bubbles appear to produce negligible pathological consequences, although they are, no doubt, associated with some of the reported blood-bubble interactions.

Large quantities of gas will result in an airlock in the right heart with death following [72]. This "decompression death" is the principal effect noted in small rodents following decompression. Its association with "limb-bend" decompression sickness in these subjects must be treated with great reservation. Regrettably, the literature is replete with correlations between various parameters and "limb-bends" in rodents when in fact the authors were studying pulmonary gas embolism.

2. Physiological Effects of "Silent" Doppler Bubbles

Of certain interest are the consequences of the gas phase most amenable to Doppler ultrasonic detection during decompression, viz., gas in the venous return. It has not yet been possible to provide a quantitative determination of gas phase volume reaching the pulmonary vasculature (e.g., with ultrasonic bubble sizing). However, when listening to the often large number of audible bubbles in the pulmonary artery, one is left with the distinct impression they are hearing a large volume of gas. One can even hear a "roaring" sound throughout the cardiac cycle [in animal subjects] following a decompression without signs of distress, and gross manifestations of pulmonary gas embolism are rarely seen in diving.

One semi-quantitative, but invasive, method to determine the volume of the inert gas [present as bubbles] reaching the pulmonary vasculature, is by measurement of the increase in right ventricular systolic pressure (RVSP) [54, 73]. The increases in RVSP following hyperbaric decompression are then compared to similar increases following air injection into the venous circulation at various rates performed while the subject is at the surface. The increases in RVSP are the result of a blockage of

portions of the pulmonary vasculature by the inert gas emboli. It is assumed that the processes, and thus, the RVSP increases, would be similar whether the gas emboli reaching the pulmonary circulation were either internally (from decompressed tissue) or externally (from a catheter) generated.

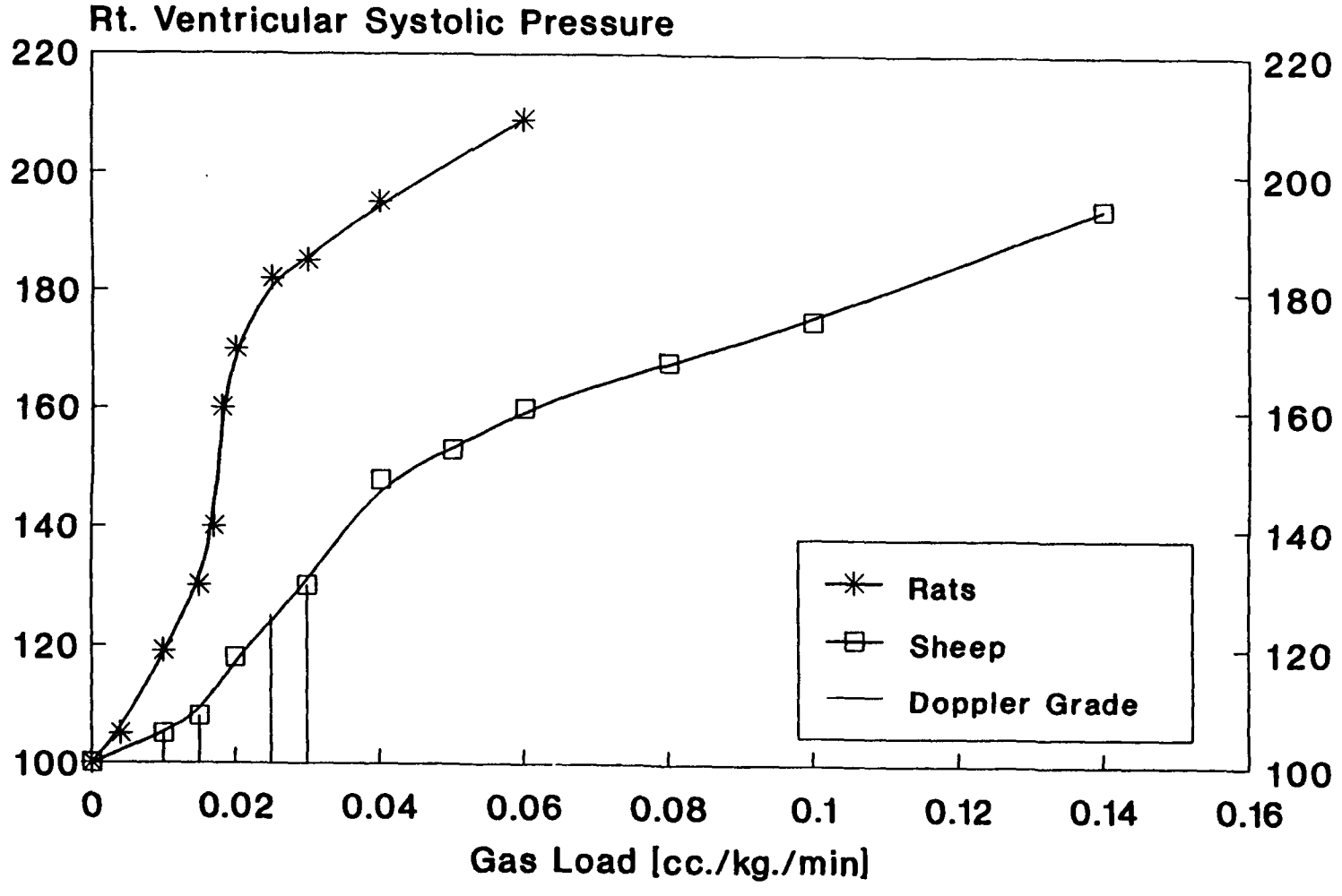
The increase in RVSP with gas load was calibrated by injecting air through a catheter at various rates into five sheep by means of a syringe pump [r = 100 to 300 μ by the rate-of-rise method]. The results of the calibration measurements are shown in Figure 4 where the maximum increase of right ventricular systolic pressure (percent of control) is plotted against the gas load. The data points for the sheep were collected from five different subjects and are combined with data from an earlier study by Spencer and Oyama [74].

RVSP rose with air injection following a brief lag and reached a steady-state in 10 to 15 minutes; it is the maximum RVSP values that are plotted in Figure 4. The return of the RVSP with approximately the same time course as the rise allows the conclusions that the pressure increases are most likely the result of: (1) embolic blockage or (2) reflex closure of the pulmonary vasculature resulting from irritation. This time course is contrasted with blockage by fibrin clots. In the latter case, the time course of the "decrease curve" would be expected to be considerably longer than the "increase curve." The presence of residual gas bubbles in the lungs has been found by Butler et al. [75] using the technique of expansion by nitrous oxide.

In a similar procedure, measurements were made of the increase in RVSP following decompression. Figure 5 shows the result of 22 dives by seven sheep made with compressed air and then decompressed directly to the surface [76]; thus measurements of the post-decompression RVSP were made at 1 ata as were the calibration measurements. The increase in RVSP (following decompression) was also measured in conjunction with the precordial Spencer grade, also on Figure 4. One notes that RVSP increases are small even up to Grade IV. Obvious is the conclusion that Doppler Grade IV+ is not the maximum volume of venous gas; in this range the precordial Doppler is operating in a "saturated" capacity and simple aural discrimination of "high" and "low" Grade IV+ is not possible.

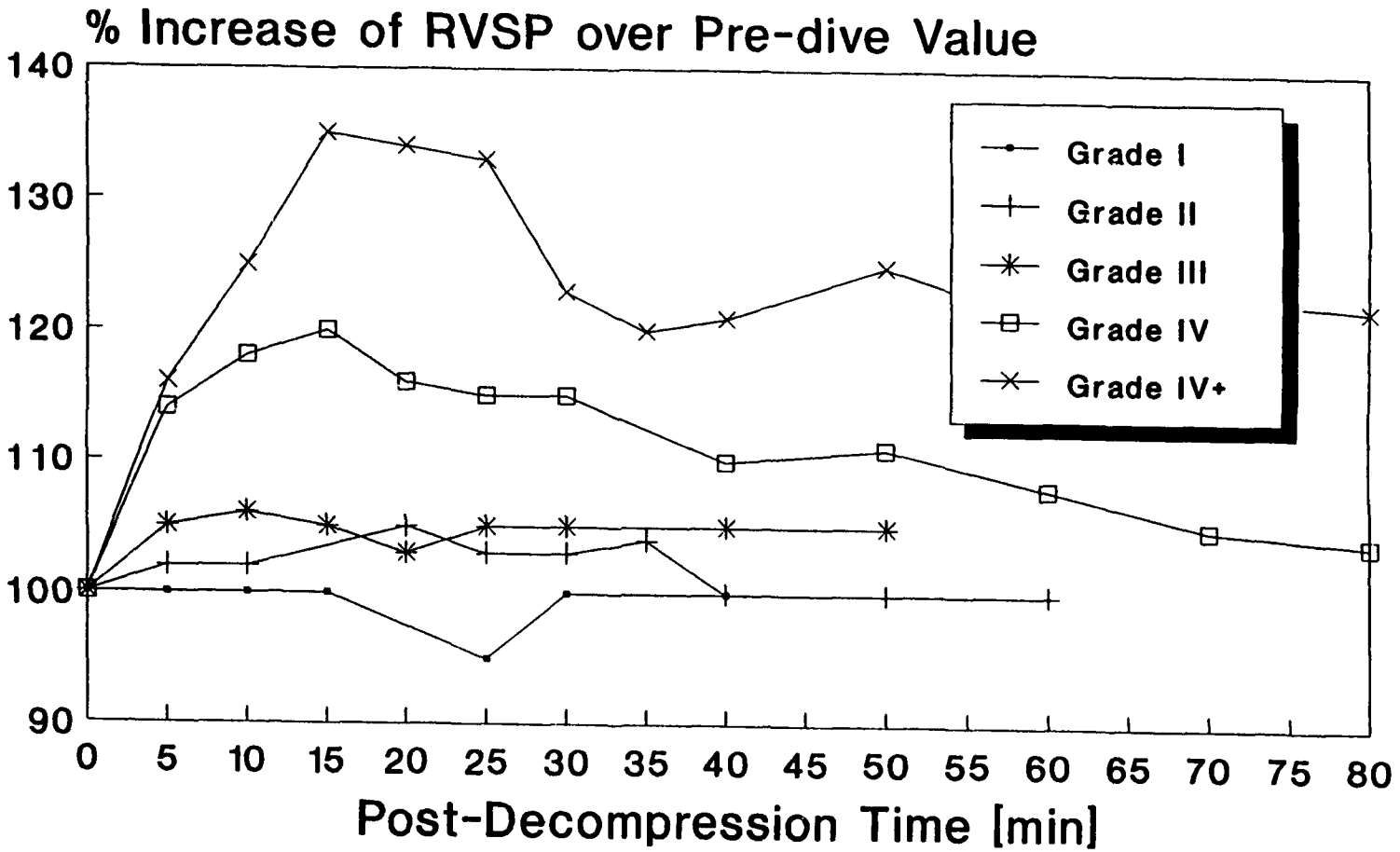
3. Volume of "Silent Bubbles" in the Central Venous System

Decompressions which do not result in limb-bend decompression sickness produce increases of RVSP on the average of not more than 20 percent above the predive value, that is, Grade IV and less. Comparing this to the estimated gas uptake load gives 7 percent of the decompression-released inert gas appearing in the vena cava in the gaseous state [77]. In that these are moderately severe dives, we could extrapolate this finding and state that gas phase separation, in the body as a whole, is small (5-10 percent) in "clean" to "limb-bends-only" producing dives. These measurements of the gas phase in the central venous system do not, of course, indicate the degree of gas phase separation in any given organ which, locally at least, may be large. This is similar to a finding of Hills determined from the elimination kinetics following either gas switching or decompression [78].



from Powell and Spencer (1980)

Figure 4. Right ventricular systolic pressure vs. injected gas loads.



data of Powell and Spencer (1980)

Figure 5. Decompression stress. Elevation of right ventricular systolic pressure (RVSP) with differing gas loads.

Dives which result in evidence of spinal cord involvement may be associated with larger pulmonary gas loads, as Powell and Spencer found that bilateral weakness of hind limbs often occurs in unanesthetized sheep when RVSP increases of 50 percent above pre-dive are noted [76]. This was also found by Bove, Hallenbeck and Elliot in anesthetized dogs [79]. In typical cases, an RVSP of 150 percent of control [predive] is noted up to the point at which sheep are unable to sustain weight on their hind limbs which is equivalent to a gas injection range of 0.04 to 0.06 cc/kg/min. However, it is a rate which the sheep are easily able to tolerate if the gas is injected by catheter. Powell and Spencer also noted that hind-limb weakness has also occurred in sheep in cases of RVSP elevations of no greater than 120 percent [76]. Neuman, et al. [80] have noted a fall in cardiac output and an increase in pulmonary vascular resistance in sheep following a decompression which did not result in an observable stress.

4. Augmented Venous Gas Loads

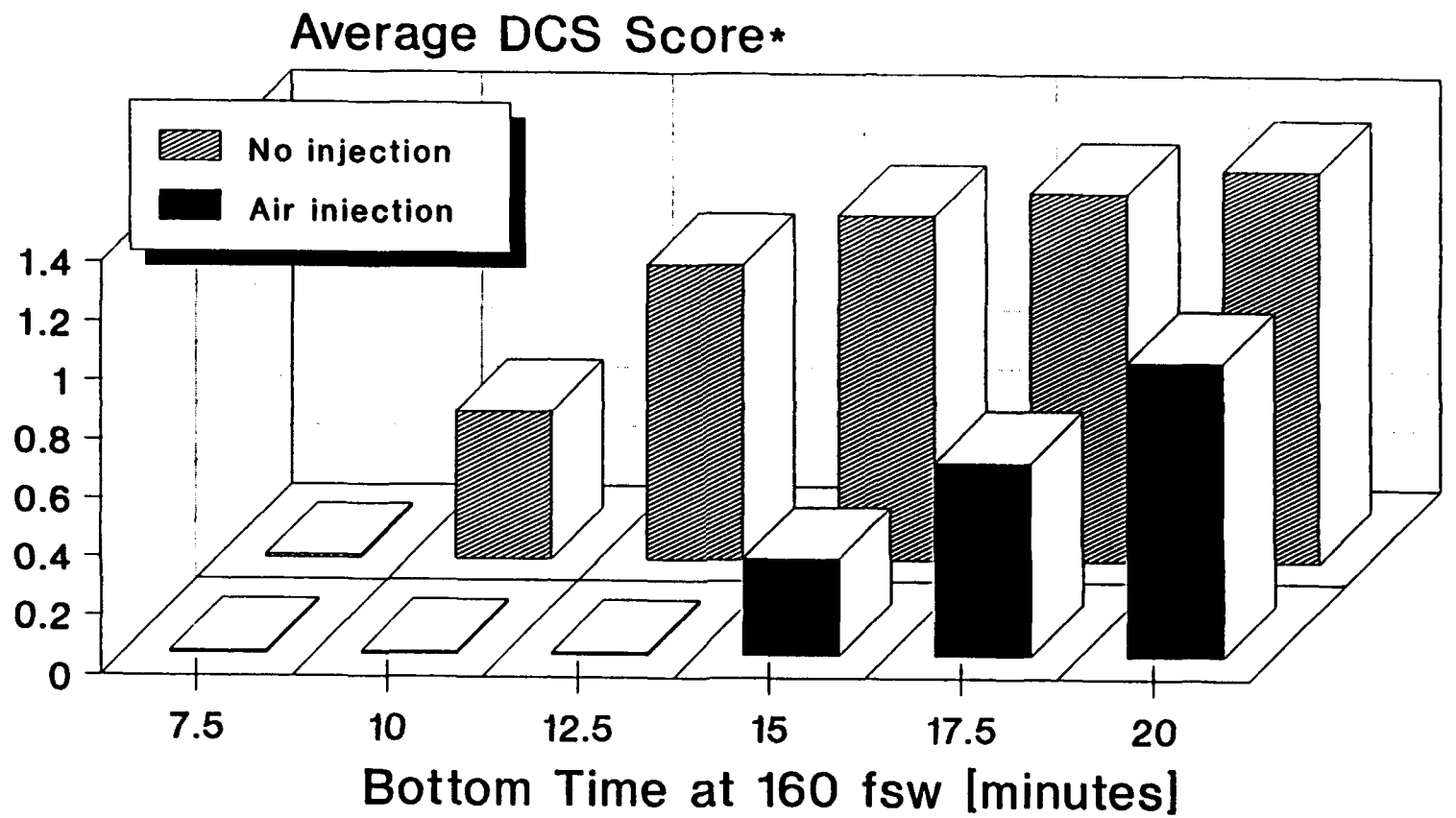
It is not altogether obvious that the venous return gas phase is benign. A study was performed in which air was injected per catheter into sheep following titrated decompressions [those whose bottom times were of different durations]; the augmented gas volumes were constant, of a 1-hour duration, and equivalent to that present with Spencer Grade IV. The augmented gas loads were <u>not</u> found to <u>exacerbate</u> decompression sickness [81], (Figure 6). An increase in severity could be expected if:

(a) ventilation/perfusion abnormalities were created thus hindering the elimination of the inert gas in the pulmonary capillaries and increasing the inert gas tension in the arterial blood,

(b) hindrance of drainage of the epidural venous plexus secondary to an elevation of the RVSP, or

(c) release of products from blood-bubble interactions.

This finding illustrates that the Doppler-detectable gas phase present in the venous return is indeed "silent," and it simply evinces the spawning of a gas phase(s) in upstream tissues. In other words, these Doppler-bubbles do not directly influence the outcome of a given decompression to render it increasingly severe; indeed, if anything, the dives with augmented venous gas were less severe.



data of Powell, Domanie,& Spencer (1987)

* Score: 0 = no problem; 1 = mild;
2 = considerable, reluctant to stand

Figure 6. Titrated decompression stress. Air injection (0.03 cc/kg/min) vs. no injection in sheep after decompression.

D. ARTERIAL BUBBLES

1. Natural History

During air injection experiments (per catheter into the jugular vein of sheep) when large (r=100-300 micra) bubbles serve as the pulmonary embolizing agent, gas bubbles are not detected by perivascular Doppler cuffs on the carotid artery when RVSP is less than 150 percent of pre-injection control [73]. In studies to achieve these steady-state pressure elevations, air injections were conducted for 10 to 20 minutes. In cases where small microbubbles were infused ($r = 10$ to 90), but for short injection periods ($t \leq 2$ minutes), Doppler-detectable gas was again not found on the systemic arterial side [82]. One could conjecture that a combination of a small radius in conjunction with elevation of pulmonary artery pressure act in concert to effect arterialization. Both conditions are necessary and neither alone is found to be sufficient. The very smallest of bubbles ($r < 5$ micra) might be expected to dissolve during transpulmonary passage [83]. Small gas bubbles can pass the pulmonary vasculature of oxygen-poisoned lungs, however [84].

The possible presence of "silent" gas bubbles in the systemic arterial circulation is less clear. Almost assuredly a gas volume of several milliliters is not "silently" present; as little as 0.5 ml has proven fatal and "surgical air" is a serious problem [85]. When large volumes of air are injected into the venous return by means of a catheter, it is found that RVSP will rise considerably over control before arterialization occurs [74, 76]. When, however, the venous gas phase is spawned in (generated by) the tissue capillaries, the bubble radii are smaller and arterialization is perhaps more probable.

A singular contribution of Doppler ultrasonic bubble detectors to our field has been the demonstration that bubbles can appear copiously in the central venous return, but only rarely in the arterial system. Studies on gas separation in two highly perfused organs, kidney and brain, have indicated that these tissues do not readily produce a gas phase following decompression -- even when rather heroic efforts are undertaken to induce one [76].

A highly perfused tissue, such as the brain, seems to be resistant to gas phase formation in all but the severest cases of decompression [86]. Neurologic decompression sickness in the brain could have an origin in arterial gas embolism. The question of transpulmonic passage of the gas phase was first investigated in rats by Emerson, Hempleman and Lentle [87]; their work indicated that a gas phase could not readily pass the pulmonary barrier under normal physiological conditions. Similar studies with rats indicated that arterial bubbles could be found in those subjects which expired, although not all rats with arterial bubbles would necessarily die [34]. Furthermore, the majority of these animal subjects showed no evidence of systemic arterial bubbles following decompressions on profiles known to result only in limb-bend decompression sickness.

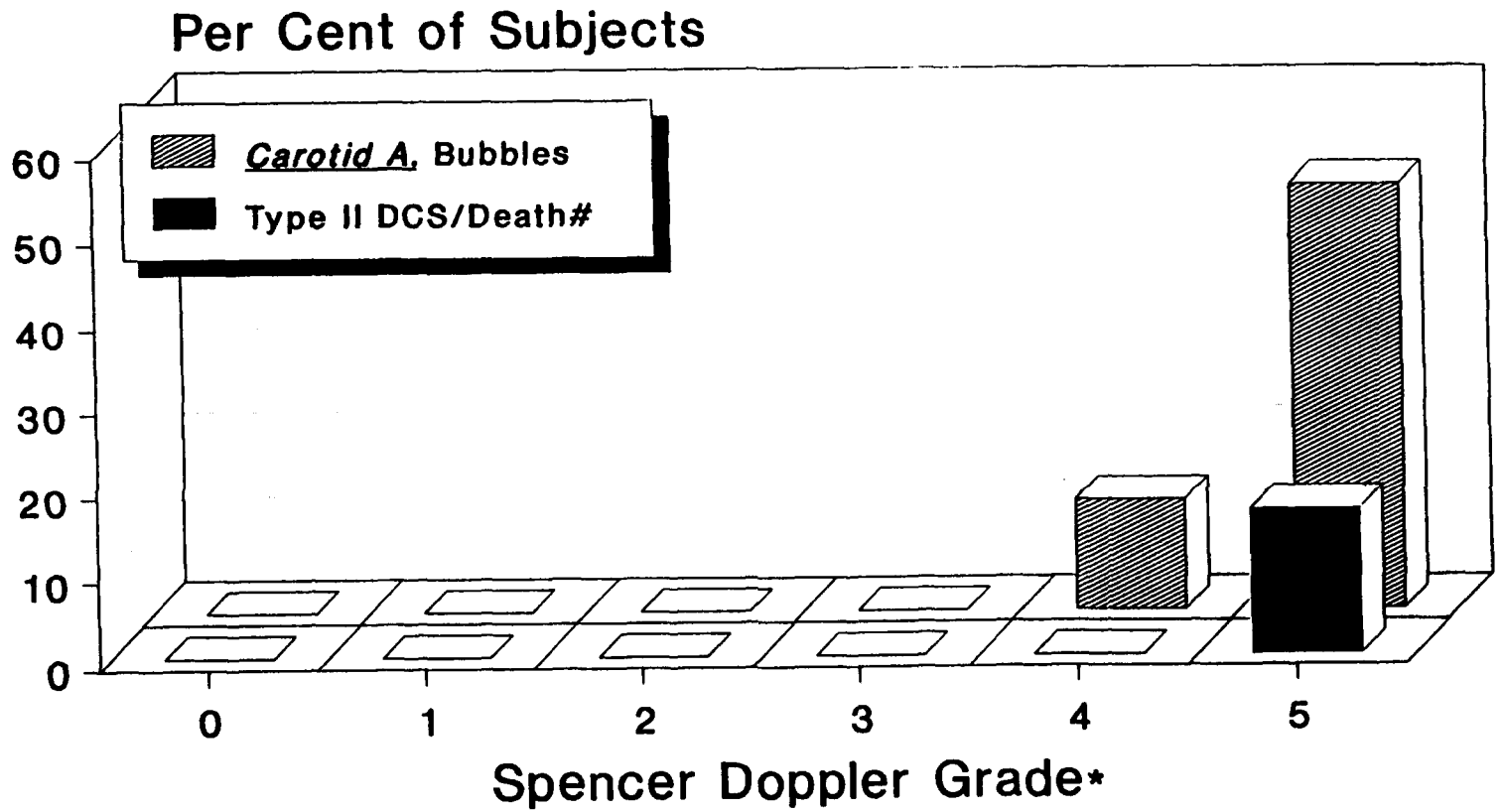
It is logical to assume that since venous bubbles appear first, the source of arterial bubbles is the pulmonary vasculature. It should be stressed here that, in situations where the vena cava is monitored, this vessel will contain a number of gas bubbles before any are noted in the arterial system. In some cases (e.g., viewing a small field through a microscope), researchers have seen bubbles moving *prodromically* in an arterial branch when the conjugate venule is bubble-free. The arterial phase did arise from pulmonary arterialization, and there does not exist an "arterial paradox." Arterial gas tensions are thought to closely follow inspired pressures, not be supersaturated, and not produce bubbles. This lack of nucleation was found to be true in sheep decompressed at the rate of 10 fsw/sec [88]. When Doppler probes were placed around the femoral artery, no gas bubbles were detected even though the supersaturations (from the transit time of blood from lung to leg) were estimated to be 5 ata at the surface.

In Doppler-monitored sheep, during 86 decompressions in which the subjects displayed Spencer Grade III or higher, bubbles were detected in the carotid artery in 7 percent of the subjects displaying Grade IV precordial bubbles, and in 50 percent of those with Grade IV+ [76] (Figure 7). As Grade IV effects the greatest increase in RVSP, one suspects venous bubbles were arterialized by forced passage through A-V shunts [89]. While Grade IV is not commonly encountered in human divers, it is not as rare an event as one might imagine, especially in hypobaric decompressions or in caisson workers. From the Powell and Spencer studies, it appears that the appearance of Doppler-detectable gas bubbles in the systemic arterial circulation is a rare, but not totally improbable, event; it even occurs in the absence of massive pulmonary vasculature overload [76].

The mechanism of arterialization would appear to be straightforward; a rise in RVSP would drive the gas phase through the pulmonary vasculature as seen in a rat study (Figure 8, cf. Powell and Spencer [76]). However, when measurements were made with rats as subjects, the results are inconclusive, as depicted in Figures 8 through 12, where every combination of RVSP and time of appearance of arterial bubbles can be seen. Similar results have also been found in pigs [91].

2. CNS Consequences

Studies by Gorman et al. [102, 103] have shown that gas bubbles in the cerebral arterial circulation could be expected to traverse these capillaries under certain conditions. Cerebral gas distribution is dependent upon the perfusion pressure, and this is an interaction of the arterial blood pressure (BP), cerebrovascular resistance (CVR), and intracranial pressure. The relation is complex since the CVR is a function of the blood pressure as the system maintains a relatively constant flow over a range of blood pressures (cerebral autoregulation).



* A high Grade IV was indicated here by a Grade V

No neurologic residuals/only death

Figure 7. Decompression stress. Gas bubbles detected in the carotid artery of sheep.

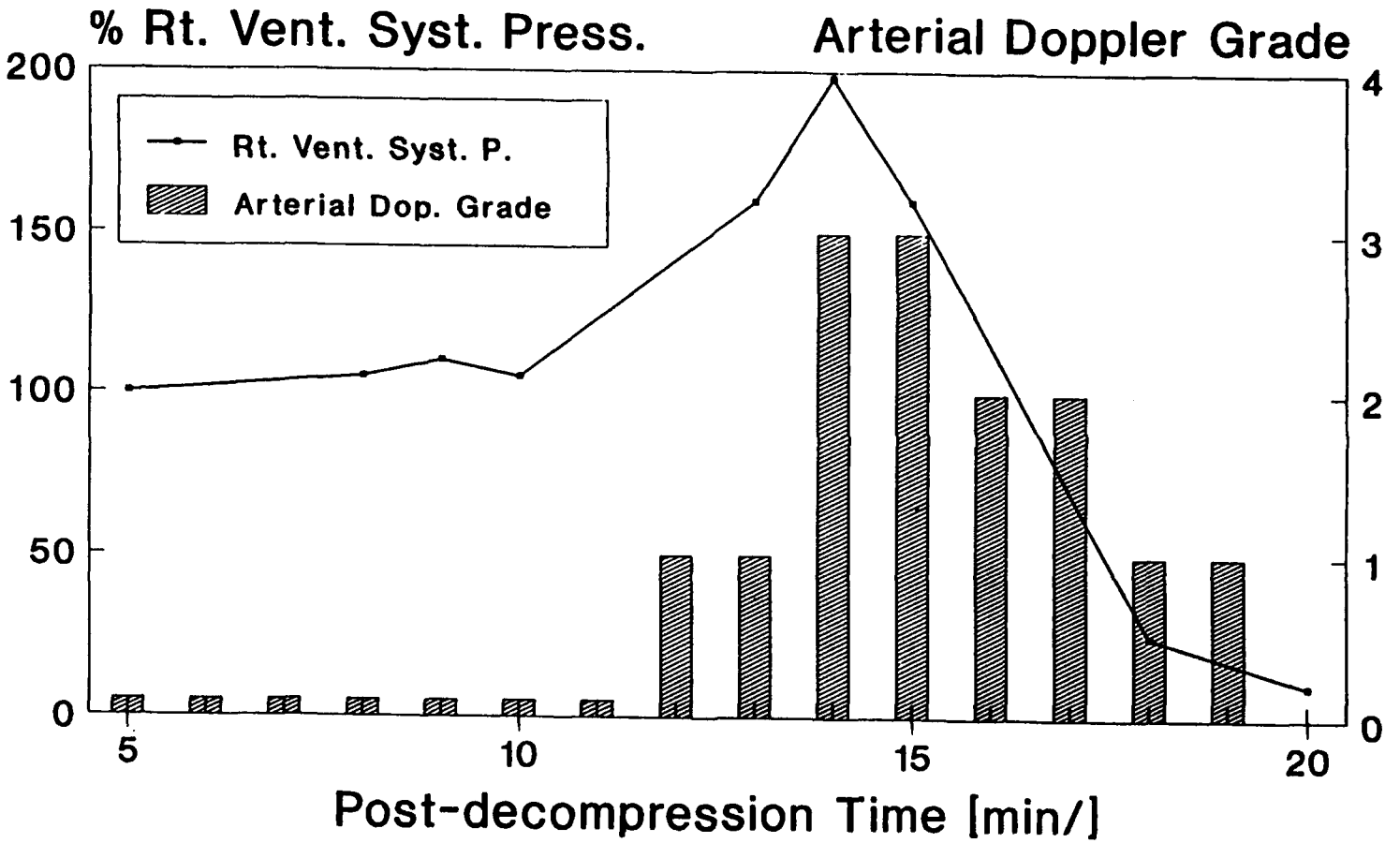


Figure 8. Arterialized gas bubbles. Bubbles detected in the aorta of a rat.

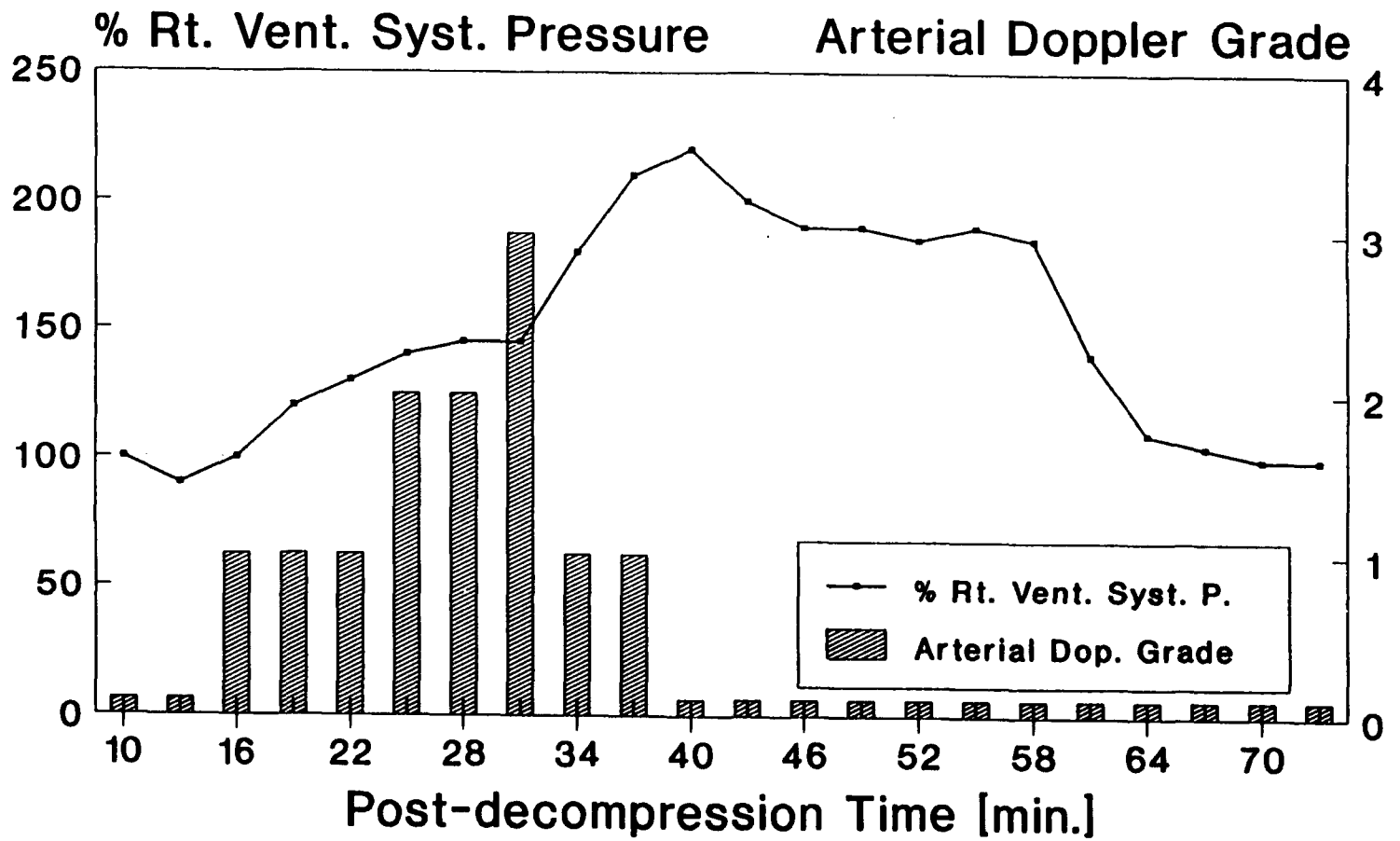


Figure 9. Arterialized gas bubbles. Gas bubbles detected in the aorta of a rat.

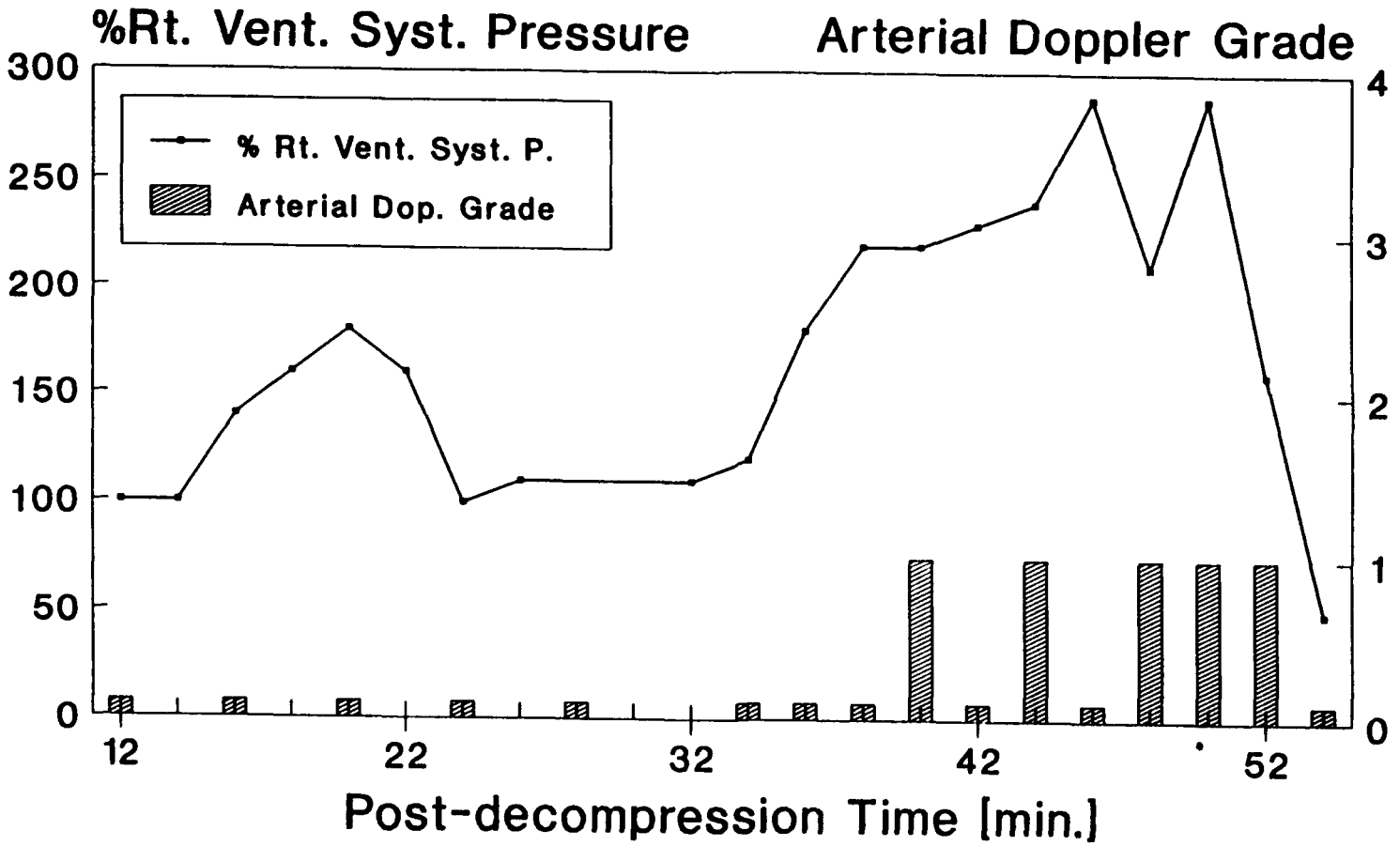


Figure 10. Arterialized gas bubbles. Gas bubbles detected in the aorta of a rat.

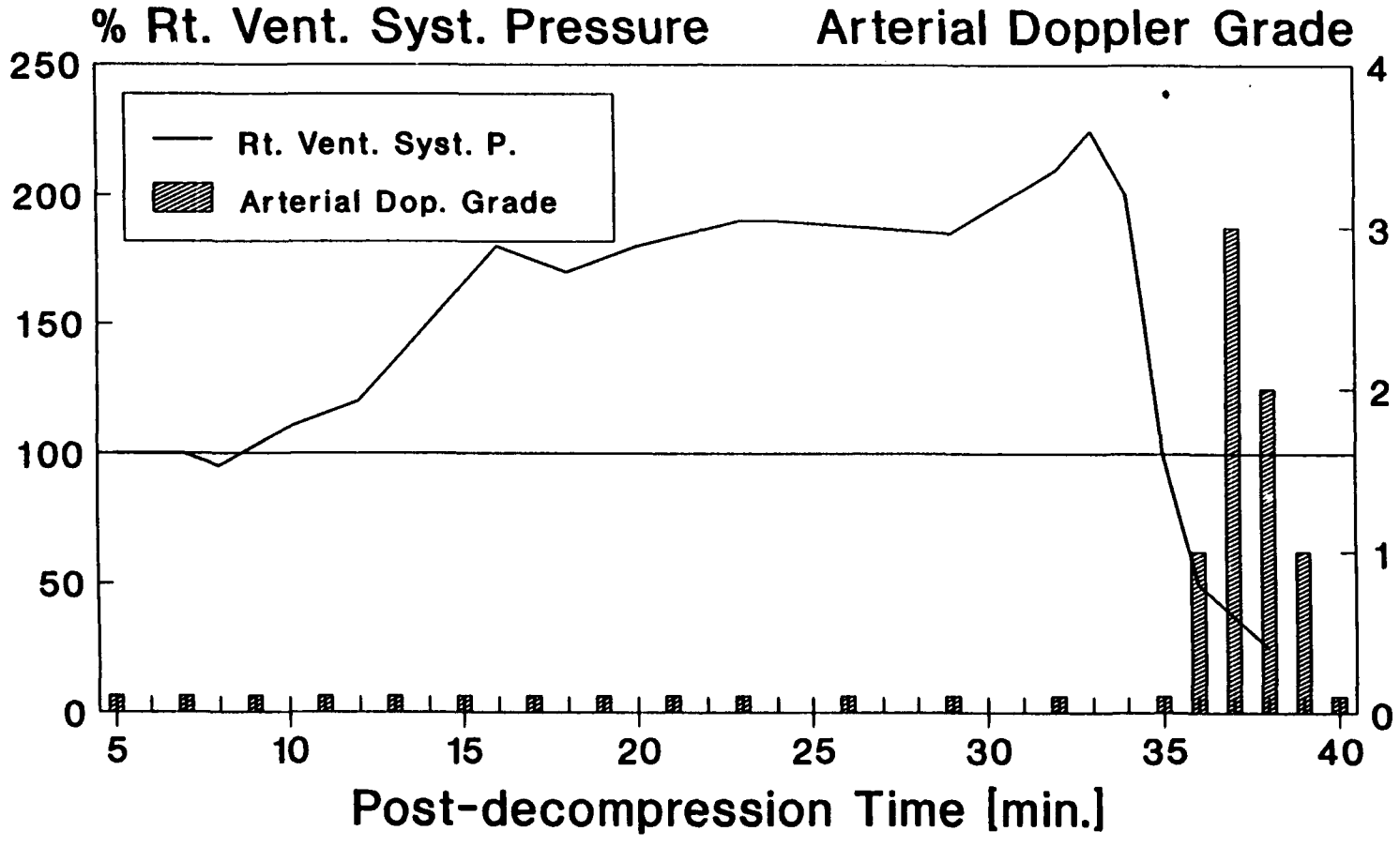


Figure 11. Arterialized gas bubbles. Gas bubbles detected in the aorta of a rat.

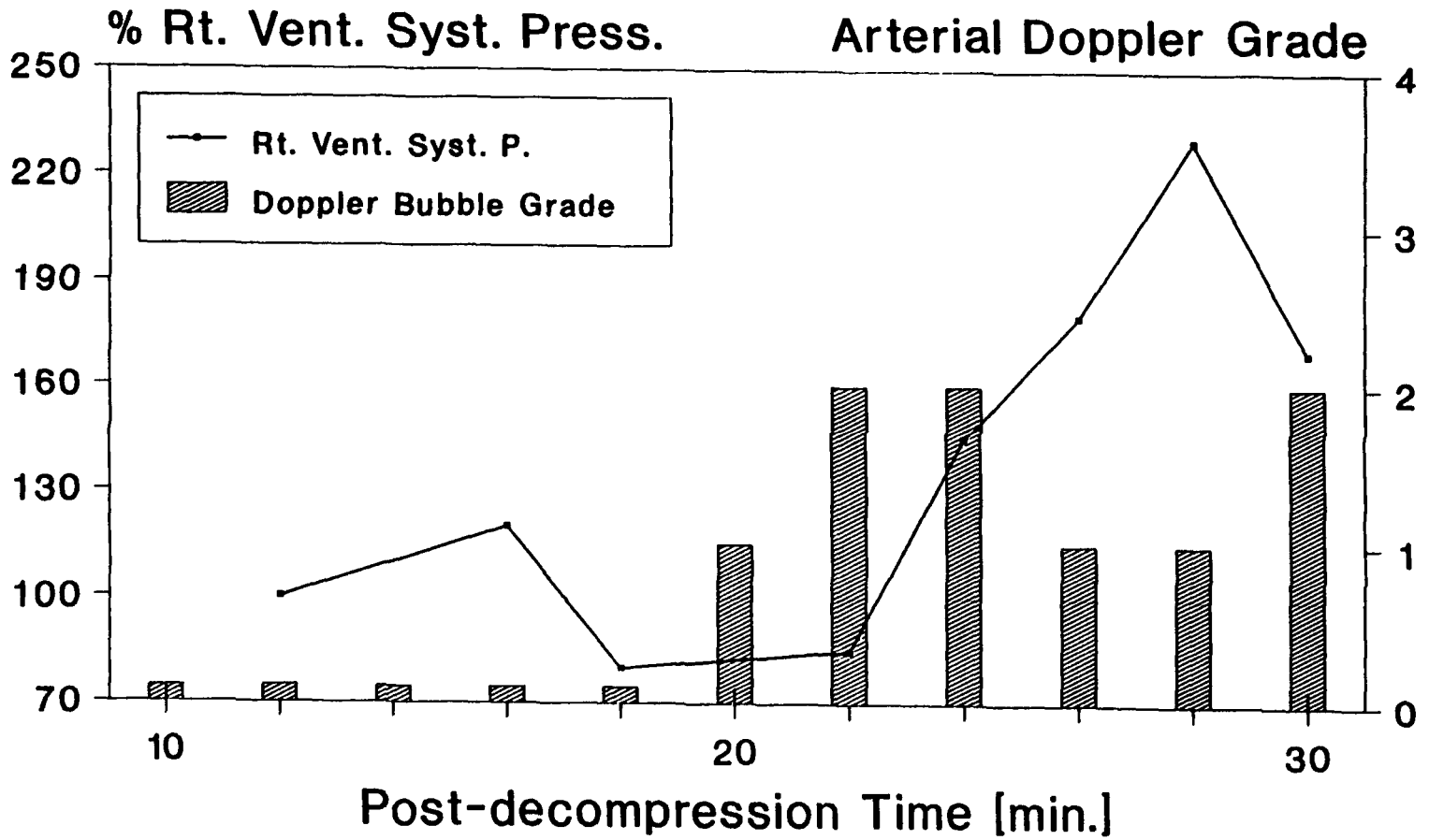


Figure 12. Arterialized gas bubbles. Bubbles detected in the aorta of a rat.

In cerebral gas infusion studies, discrete micro bubbles were not seen after the gas entered the arteriolar circulation, rather coalescence or fusion occurred and cylinders of gas formed. Entrapment occurred in vessels when the diameter was reduced to 50-200 micra. Transarterial passage was dependent on systemic blood pressure and embolus length, l. If the embolus length in this vessel was greater than 5,000 micra, blockade was inevitable, if l < 500, blockage never occurred. Intermediate gas bolus lengths (500 < l < 5000 micra) were found to pass within 3 minutes. These values accord with those of Masurel et al. [104] who calculated that a bolus would become trapped when its length exceeds 10 times its radius (Vik et al., 1989.)

It is possible to make some estimates from these results. If the volume V of a cylindrical capillary of length l and radius r is given by $V = \pi r^2 l$, calculation shows the following:

$$V_{500u} = 7.7 \times 10^6 \, [u^3]$$

$$V_{5,000u} = 77 \times 10^6 \, [u^3]$$

With the lower boundary of detectable gas bubble radii as r = 50 micra, the volume V of such a bubble can be calculated from $V = (4/3)\pi r^3$ as 3×10^5 cubic microns. We could determine the number of such gas bubbles needed to fill the 5,000 micron-long capillary and determine that approximately 260 are needed. Employing Spencer's observation [105] that one human subject who sustained a post-surgical stroke demonstrated gas emboli in the MCA for 14 seconds, this would arithmetically equate to 18 bubbles/second which is an easily detected amount and is more than was found in severe decompressions in animal (sheep) studies [76].

III. CONCLUSIONS AND RECOMMENDATIONS: SOME POSSIBLE FUTURE IMPLICATIONS FOR HYPOBARIC PHYSIOLOGY

A. DECOMPRESSION FROM SATURATION: PERFUSION AND PHYSICAL FITNESS

During decompression, the dissolved tissue inert gas will be eliminated by perfusion mechanisms. At the same time, the presence of [suspected] gas micro nuclei will contribute to the growth of physiologically sizable gas bubbles. The volume of this tissue gas phase will depend on:

(1) the length of time the tissue remains in a supersaturated condition, and

(2) the number of "silent" tissue gas micronuclei.

Exercise is contended to influence these factors in differing manners. Mild exercise performed during nonsaturation, hyperbaric decompression is beneficial since it reduces the time to reach the surface, but strenuous exercise is known to increase the time to reach the surface [15]. Most likely in both cases, perfusion is increased by the exercise, but, in the later case, nucleation is increased by stress-assisted mechanisms.

In hypobaric situations, the individual is almost always in the fully saturated condition in all tissues. Here, the primary variables affecting tissue gas phase formation will be: (1) the local rate of dissolved inert gas washout, and (2) the number of gas micronuclei. Exercise during hypobaric decompression increases the severity [111].

Just as tissue motion tends to increase the probability of stress-assisted nucleation, increased perfusion effects the rapid elimination of local dissolved tissue gas in the presence of a gradient, e.g., reduced ambient pressure. Daily exercise and overall physical fitness is known to increase tissue perfusion through changes in the cardiovascular system [112-114]. These mechanisms include:

1. changes in cardiac output,

2. decreased vascular resistance in the exercising muscle, and

3. increased capillary density.

Weightlessness may also function to increase capillary density. Musacchia et al. (1988) (115) found an increase of 50% in the capillary density [capillaries/mm^2] in the rat soleus muscle.

A test recently completed by Dr. K. V. Kumar at NASA/JSC gives some indication that physically-fit individuals would possess a greater capability of eliminating dissolved tissue gas during decompression and would therefore demonstrate a reduced incidence of both Doppler detectable gas bubbles and limb-pain decompression sickness when compared to the group with the sedentary, nonactive life style. The results from 40 individuals, mostly in paired exposures, demonstrate a marginally significant difference at the level of a classical "bends/no bends" analysis for unconditioned or "untrained" and conditioned or "trained" individuals (sedentary vs. active lifestyle).

DCS and Sedentary Life Style	DCS and an Active Life Style
13	2
(N = 51)	(N = 24)

In a *chi*-square test (with Yates' correction), $X_c^2 = 2.03$ and $p = 0.15$.

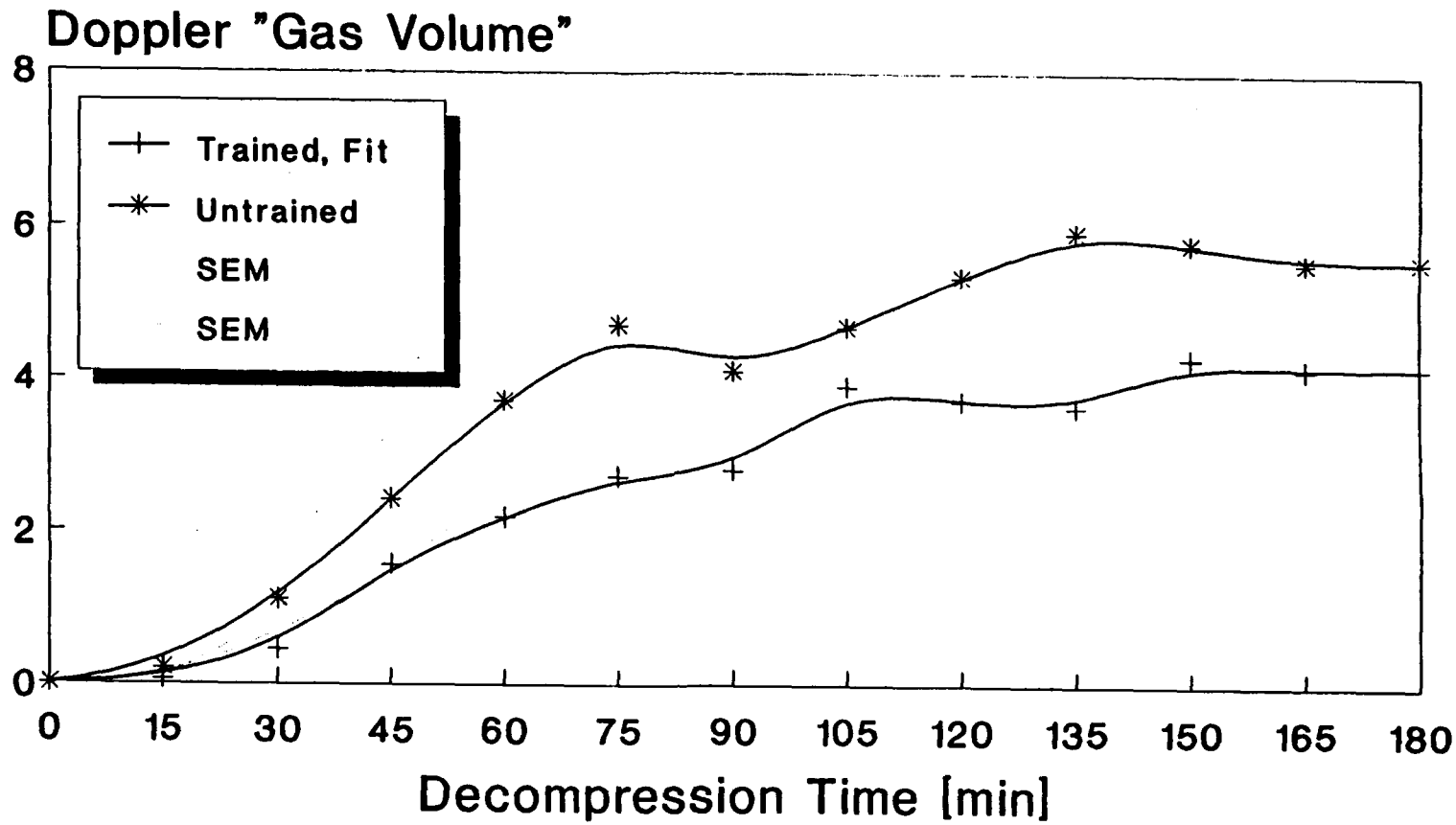
When the data were examined by the method of time-intensity, we find that they indicate a possible difference as shown in Figure 13. It is tempting to regard this difference as real since it would indicate a difference in gas phase formation in physically-fit individuals versus unconditioned subjects. It would be difficult to construct and envision a process whereby exercise results in a decrease in the rate of formation of tissue gas micro nuclei. Rather, the explanation is most likely to be found in the increased perfusion engendered by cardiovascular conditioning that results in an elevated rate of dissolved tissue inert gas during the decompression phase.

Since the pre-exercise or no pre-exercise/decompression protocol was conducted in a pair-wise fashion, it is of value to examine the data in a like manner. When this is done, taking the Doppler "gas volume" for each subject at any give time for the "pre-exercise" and "no pre-exercise" condition, the differences in Doppler scores are more apparent. Figure 14 depicts the sum of the actual differences as well as the average differences. It is still possible that a small increase in the formation rate of stress-assisted gas nuclei could result in the upward deflection in the initial portion of the curve in Figure 14.

It is possible that individual differences in habitus (e.g., greater adiposity) could be an explanation. The lack of differences in the group means does not support this explanation, however.

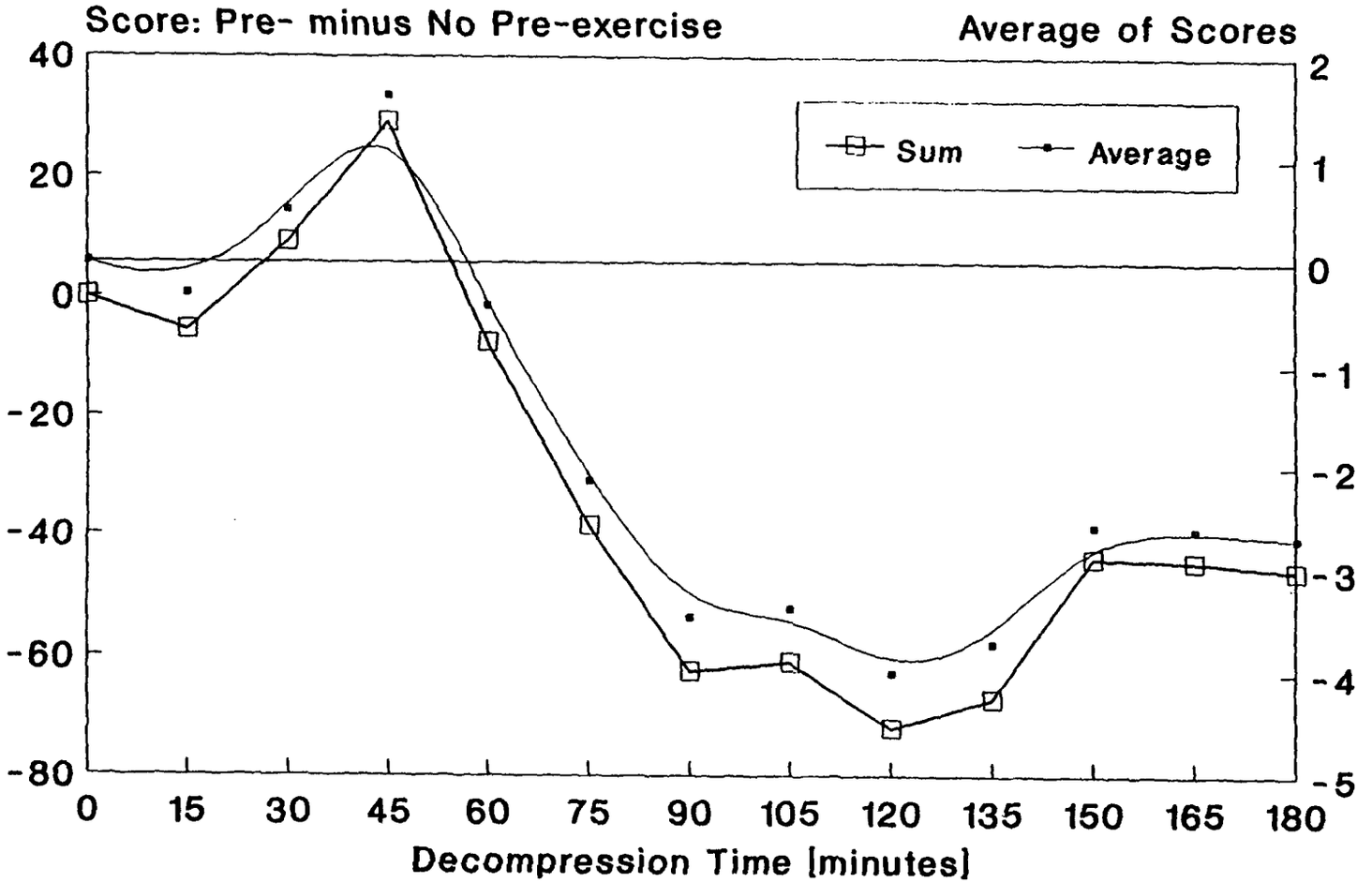
Characteristics of All Males		Males with Active Life Style
Height:	171.5 ± 18.3 cm	175.2 ± 4.9 cm
Weight:	78.6 ± 18.7 kg	76.4 ± 6.6 kg

[Women were not analyzed here as N = 2 in the subset.]



Data of K.V. Kumar, MD
NASA/Johnson Space Center

Figure 13. Decompression stress. Doppler "gas volume" in trained and untrained individuals.



From data of K. V. Kumar, M.D.
NASA/JSC, 1990

Figure 14. Decompression stress. *Pre-exercise (+) vs. no pre-exercise (-).*

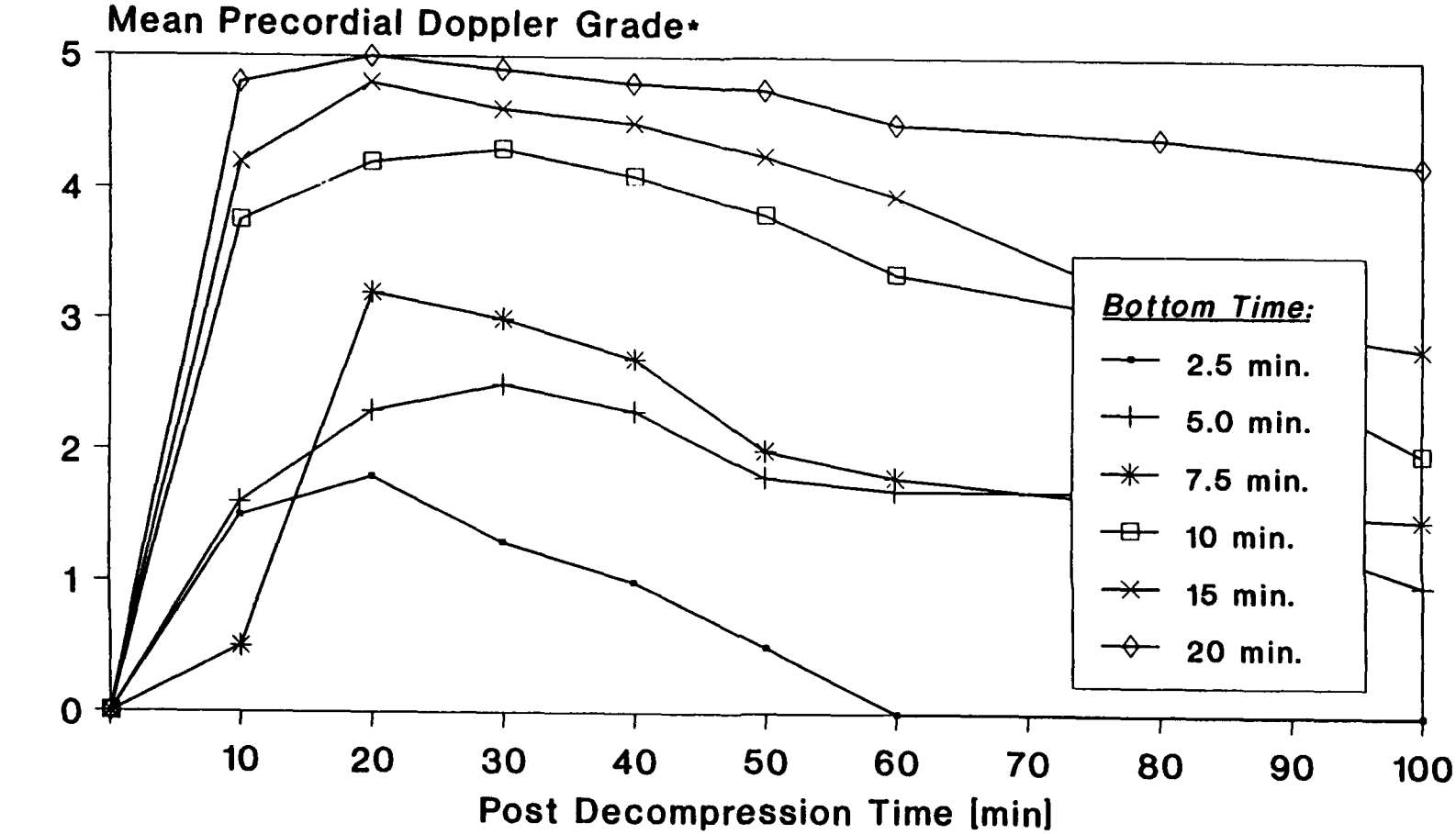
B. ISOBARIC COUNTER TRANSPORT MECHANISMS

1. Secular Patterns

If one closely observes the patterns ("temporal fine structure") of the appearance of Doppler-detectable gas bubbles in **hypobaric** situations (where the subject is usually breathing oxygen), one notes a departure from the usually observed time course of venous gas bubbles in **hyperbaric** situations, i.e., an unusual time delay in the former for the appearance from the instigation of decompression. Gas phase formation (and Doppler-bubble release) usually occur early following the reduction in ambient pressure as can be seen, for example, in Figure 15 for **hyperbaric** cases. This can be contrasted with the formation-and-release curves of Figures 16 and 17 for **hypobaric** situations. These delays seem to be inordinately long especially when one considers that the subjects are breathing against a medium **totally devoid** of nitrogen.

In 1971, it was found that breathing one gas mixture and then suddenly switching to another could under some conditions produce tissue gas phase formation in the absence of decompression [116]. A similar situation of lesions arose when subjects were breathing one mixture while surrounded by another [117]. The effect was shown in both cases to be the result of certain combinations of solubilities and diffusivities such that the sum of the tissue partial pressures exceeded the ambient pressure. This would, in time, effect the growth of those micronuclei already existing in tissue. Similar scenarios have been developed for gas bubble dynamics in treatment situations where nitrogen bubbles are resolved by counter-transport of heliox [118].

In Figure 18, we see the Doppler-detectable bubbles [counts/min] determined by D'Aoust [119] in sheep when isobaric gas switches (three cases where nitrogen was switched to helium and one case of nitrogen to neon) were performed. It is clear from these examples that the rapid rise in the number of bubbles shortly after decompression is absent but rather the pattern is one of a rather long onset (one to three hours) and then a rise and fall of bubble count. This is more congruent with the hypobaric case where the gas phase generated by decompression can be augmented by the influx of oxygen (Figures 16 and 17). It is clear that the rapid initial rise seen in decompressions of nitrogen to nitrogen (air to air) are absent (as seen e.g., in Figure 15). The effect cannot be attributed solely to the fact that "slow tissues" are involved since a similar pattern is seen in a case where a nitrogen saturation decompression is effected without breathing against pure oxygen (Figure 19, after Eckenhoff, et al. [120]). In this latter case, the rise in bubble number is:



*Modified system of Smith and Powell;
cf. Powell and Spencer, (1980)

Subjects: Sheep

Figure 15. Growth and decay of precordially-monitored Doppler bubbles.

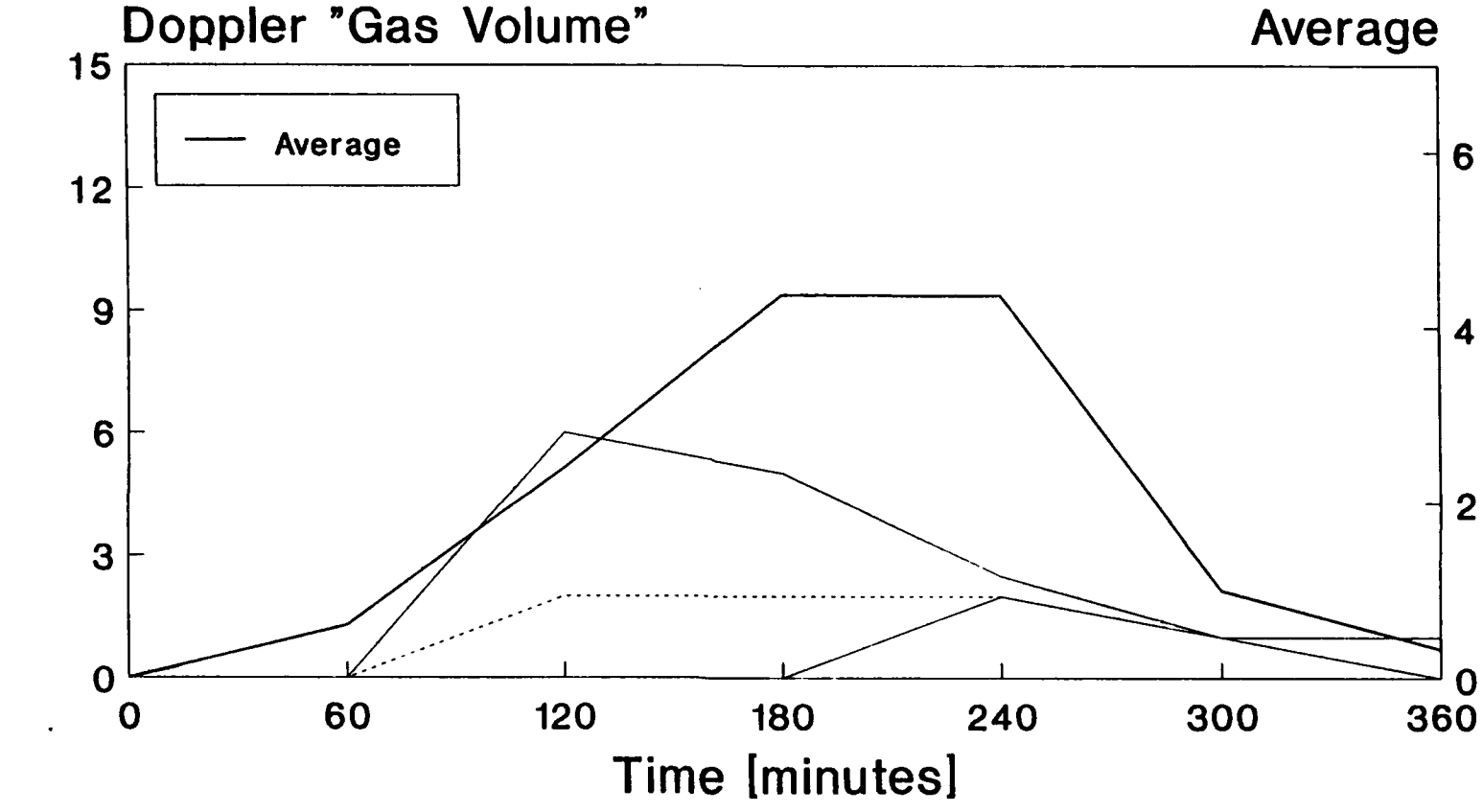


Figure 16. Decompression stress. Doppler-detectable gas bubbles during hypobaric decompression.

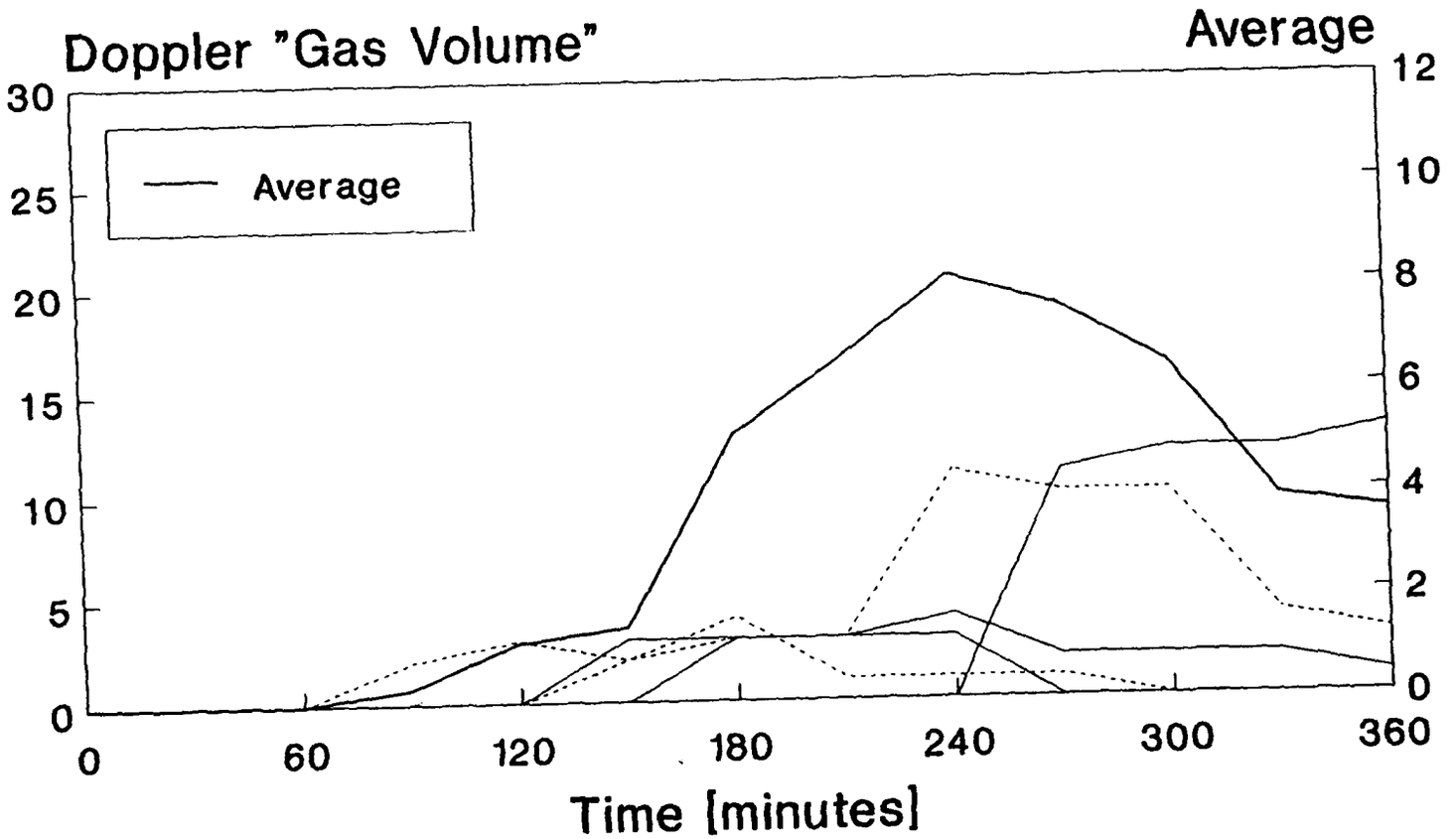
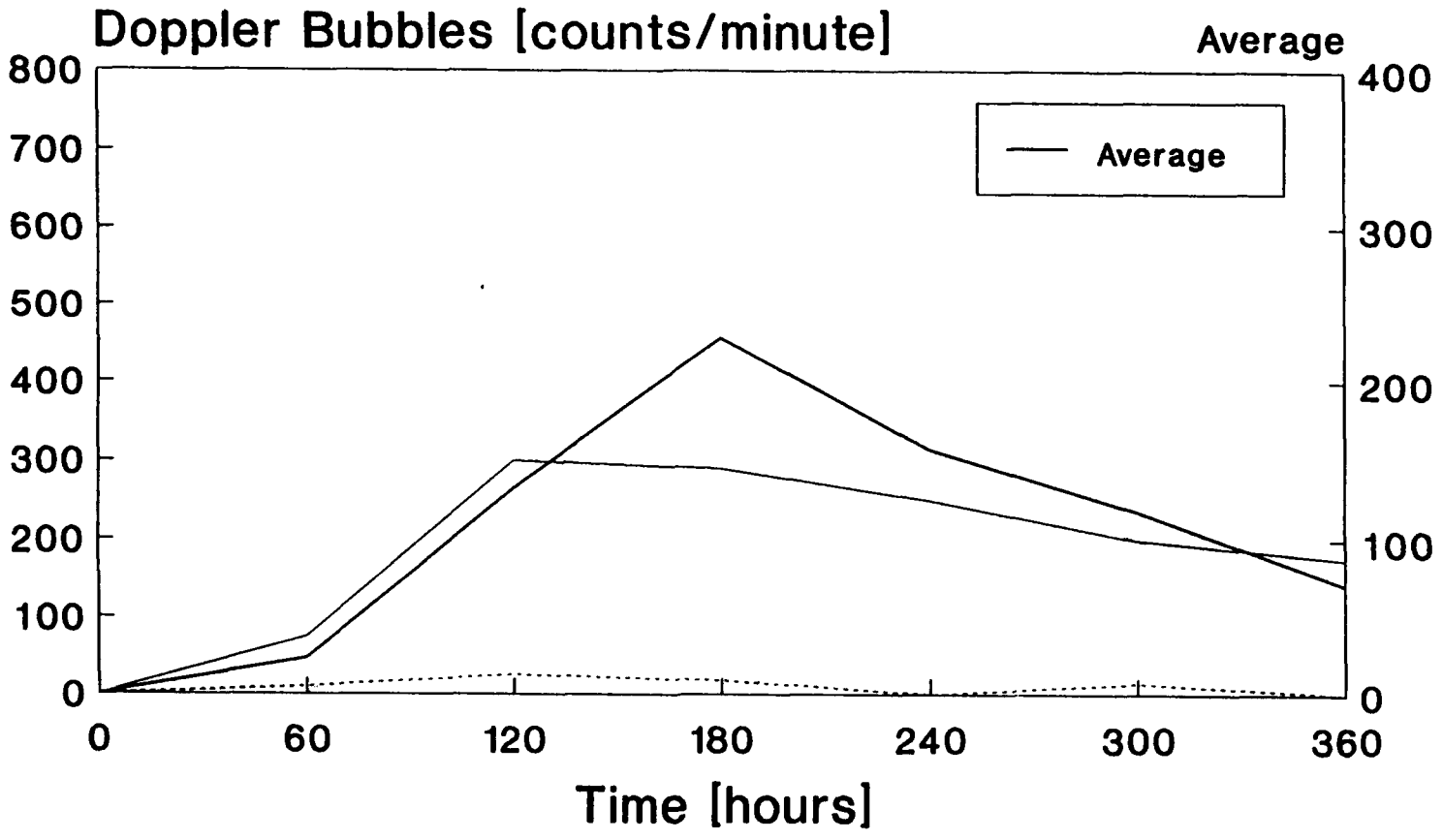
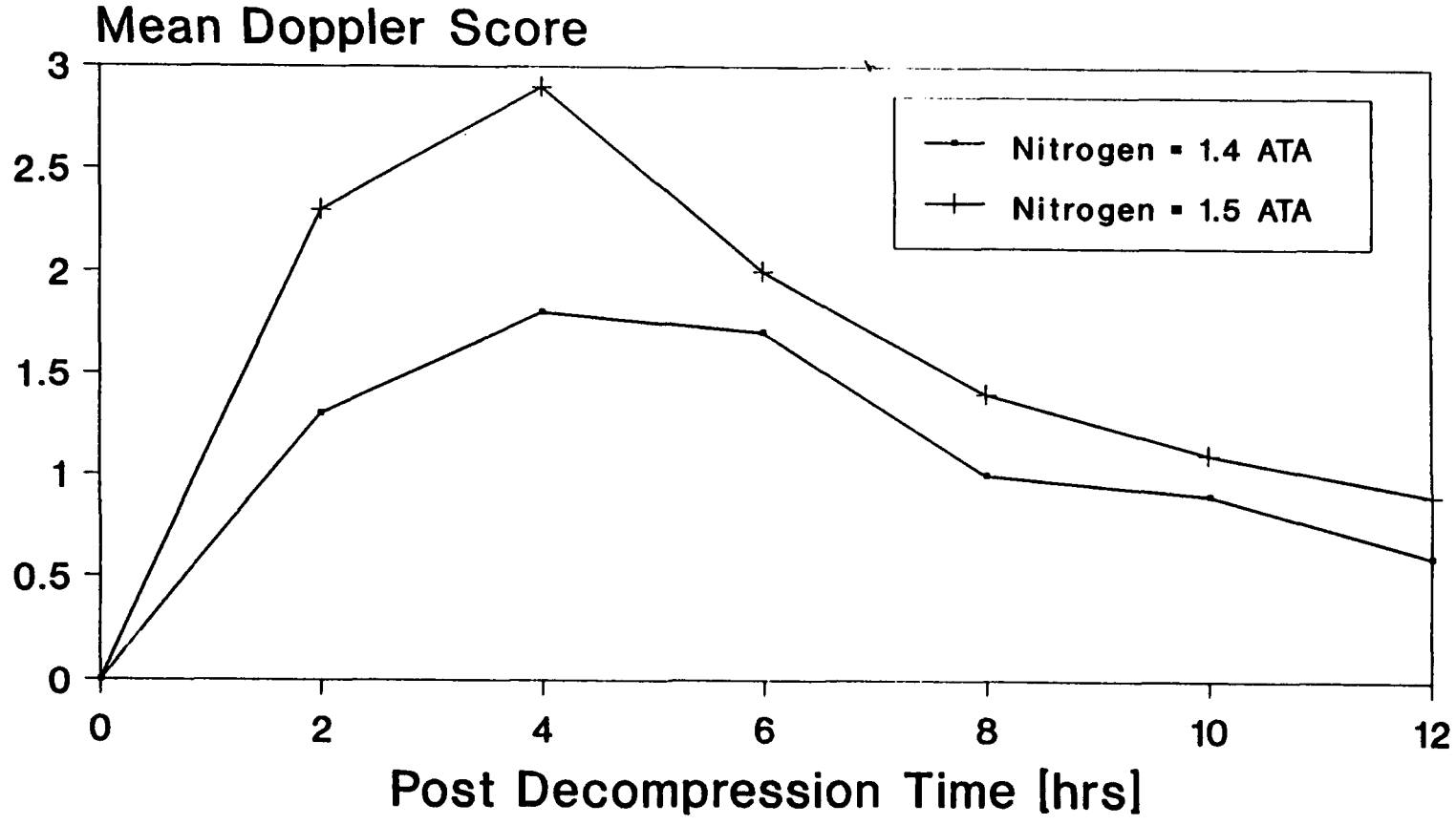


Figure 17. Decompression stress. Doppler-detectable gas bubbles during hypobaric decompression.



from D'Aoust and Lambertsen
In: *Physiology & Medicine of Diving*
(Eds. Bennett & Elliott), 1982

Figure 18. Decompression stress. Isobaric counter transport and Doppler-detectable gas bubbles.



From Eckenhoff et al.,
Undersea Biomed. Res., 1986.

Figure 19. Decompression stress. Doppler-detectable gas bubbles following saturation decompression.

(a) immediately after decompression and,

(b) the Spencer Doppler grades are low (Grade III maximum on any subject) in contrast to the numerous and persistent cases of Grade IV often seen in hypobaric situations.

Some of the most interesting data on this subject are those of Hyldegaard and Madsen [118] describing the effects of gas switches (e.g., helium to nitrogen).

2. Mathematical Treatment

In the mathematical treatment of the flux of gas into and out of gas pockets ("bubbles"), it was shown by Van Liew and Hlastala [139] that the flux of gas out of (or into) a bubble of radius r could be given by:

$$dr/dt = (b_i Q_{amb})(1-P_{ai}/P_{gi})(1+r/O_1), \quad [4]$$

where:

$$h_i = [(B_i Q)/(B_{ti} a_i)], \quad [5]$$

where V is the bubble volume at STP, B_i and a_i, are the coefficients of solubility and the diffusion coefficient of the i^{th} gas, respectively, A is the surface area of the [spherical] bubble, P_i is the pressure of i^{th} the gas dissolved in the tissue at a radial distance of r where Q is the "effective" tissue perfusion (it contains a multiplier factor for end-capillary perfusion), P_{ai} and P_{gi} are the pressures of the i^{th} gas in the arterial blood and tissue respectively, and B_{bi} and B_{ti} are the solubilities of the i^{th} species in blood and tissue, respectively. The three terms contained in Equation 4 are, respectively, from left to right:

(a) constants specific to the i gas species,

(b) partial pressure of the i gas in the bubble and blood and tissue, and,

(c) the reciprocal of the bubble radius and constants specific to the i gas species.

Hills [121] presented a similar situation in which different gas species with solubilities and diffusivities as above were separated by a tissue diffusion barrier of area A and thickness x. In this model, the gas phase could form in the blood vessels (if the sum of the partial pressures exceeded ambient). The model would allow the slow growth of a tissue gas phase (either formed by decompression or growing from micronuclei) tissue gas phase.

In this model, the flux for gas 1 is

$$F_1 = Aa_1b_{t1}(P-p_1)/x = Qb_{b1}p_1 \quad [6]$$

and for gas 2:

$$F_2=Aa_2b_{t2}p_2/x = Qb_b2(P-p_2) \quad [7]$$

substituting:

$$f = A/xQ$$

$$n_1 = a_1(bt1/b_{b1}), \text{ and}$$

$$n_2 = a_2(b_{t2}/b_{b2}).$$

They thus obtain:

$$p_1 + p_2 - P = fP(n_1-n_2)/(1+fn_1)(1+fn_2) \quad [8]$$

Hills noted that there would be supersaturation of venous blood, and the gas phase would grow, if

$$n_1 \geq n_2 \quad [9]$$

One notes that in both presentations of counter transport, steady-state growth of **"silent," preformed** (i.e., **preformed by <u>decompression</u>**) gas phase will occur if the characteristics of the **incoming** gas species with respect to the **exiting** gas species are:

$$b_{in}\, W_{in}^{-1/2} \geq b_{ex}\, W_{ex}^{-1/2} \quad [10]$$

Here the square root of the molecular weight W is used to correct for the differences in the diffusion constants. In the hypobaric situation, tissue nitrogen is gradually being eliminated (either by perfusion or by gas bubble formation and dispersion by the venous blood) so the tissue gas phase is not stable for long periods. In Table I are listed the solubilities and diffusivities of various gas used in hyper- and hypobaric environments [122].

It is evident from this compilation that the solubility-diffusivity ratio for oxygen to nitrogen is 1.7 which would indicate that, in some situations, it might be possible for oxygen to enter dysbarogenic tissue gas bubbles at a rate faster than nitrogen would

exit. Oxygen would be continuously removed via cell metabolism, and any bubbles generated would naturally be small.

TABLE I. DIFFUSIVITIES AND SOLUBILITIES OF GASES

GAS	SOLUBILITY x DIFFUSIVITY($\times 10^8$)*
O_2	1.70
CO	1.30
A	1.87
N_2	1.00
H_2	1.94
He	1.44

*cm^2 torr-min.

C. REPEATED DECOMPRESSIONS TO ALTITUDE

Repeated decompressions to altitude are also found to produce bubbles and decompression sickness in spite of the fact that all tissues are being reduced in gas loadings during each repetitive exposure [123-126]. Residual gas bubbles, chaotically generated, appear to be a viable explanation rather than the physiological or biochemical changes proposed.

NASA data on repetitive decompressions [69] give an indication that repetitive decompressions made within 24 hours produce more Doppler-detectable intravascular gas than might be expected from a single exposure. Figure 20 shows the results of Doppler monitoring studies performed at NASA/JSA over the past several years; here the "Doppler Gas Volume" is plotted against the tissue ratio [tissue nitrogen tension/ambient pressure]. We see that repeated decompressions (within 24 hours) appear to produce a greater than expected "gas volume."

The exposure of astronauts to reduced pressures in excess of once per two or three days may not allow time for the system to "reset" and result in a greater than expected hazard.

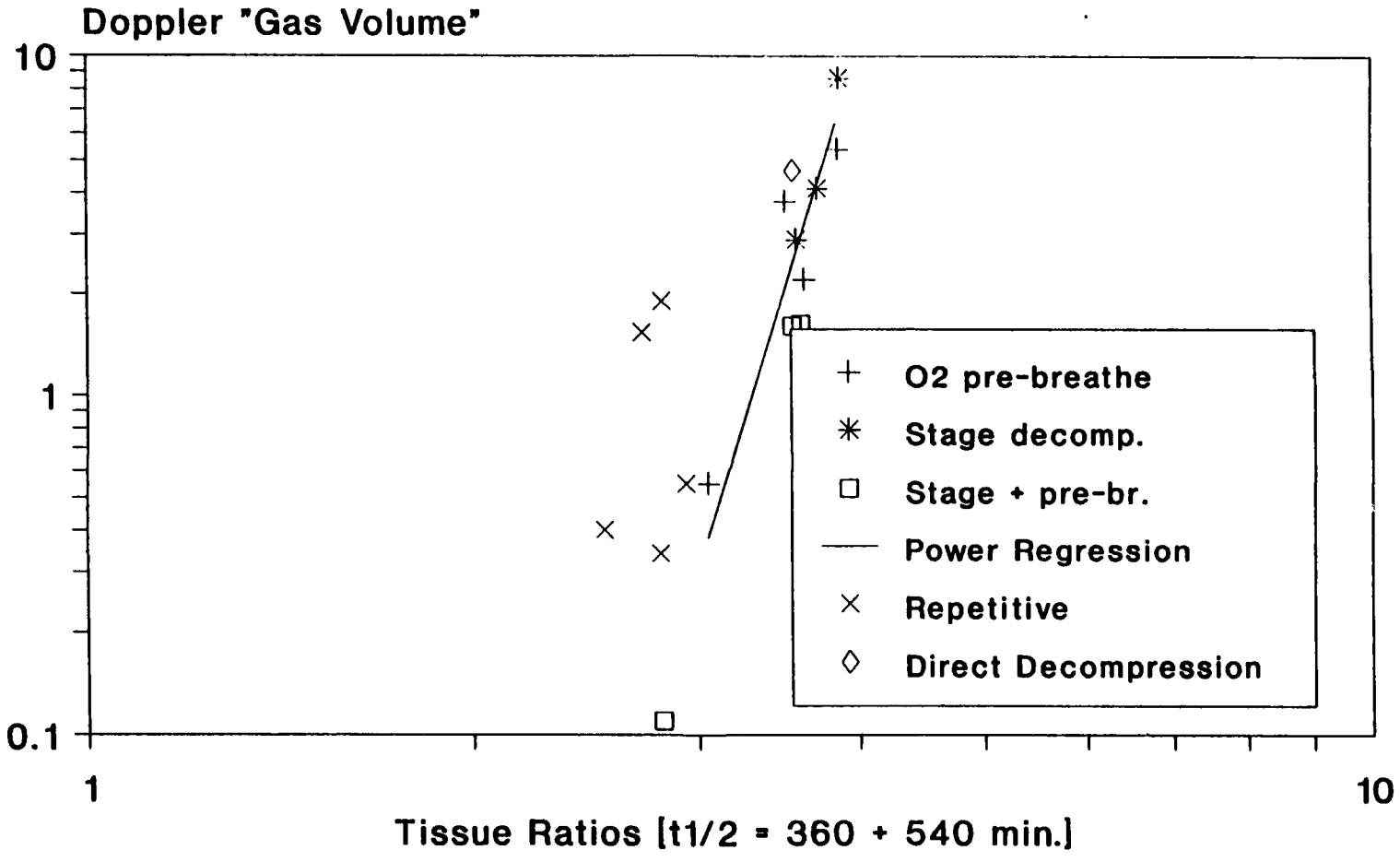


Figure 20. Decompression stress. Doppler "gas volume" vs. tissue ratio: staged and pre-breathe decompressions.

D. THE DECOMPRESSION RISK ANOMALY IN MICROGRAVITY

1. Reduced DCS Risk

Careful ground-based laboratory studies [69-71] conducted with human volunteers over a wide range of tissue pressures ratios, effected either:

(a) by selection of the final pressure and a direct decompression, or

(b) by washout of tissue inert gas by oxygen breathing followed by decompression to a final pressure,

have delineated the incidence of DCS to be expected.

To date, astronauts have performed 26 Shuttle excursions for extravehicular activity (EVA), and no incidences of DCS have arisen. Several explanations have been posited:

(a) unawareness of any problem of joint soreness while intensely preoccupied in EVA; a reluctance to report minor and/or questionable occurrences to the flight surgeon as it might affect their future flight status or denial to themselves that such an event could have occurred,

(b) hormonal, catecholamine or cardiovascular changes (e.g., central fluid shifts) resulting in an increased rate of inert gas washout,

(c) a statistically allowed "cluster phenomenon" of safe, DCS-free decompressions, and/or

(d) a lack of nucleating agents and/or mechanism(s) for the growth of a tissue gas phase.

Reviewing these:

(a) It is possible that unnoted joint soreness could have occurred. Laboratory studies at unit gravity indicate a 25% expected incidence of mild soreness in the lower extremities, a degree of discomfort that could readily be unnoted in an EVA situation. There was a 5% incidence, however, of substantial joint pain in the lower extremities by earth-based subjects, readily noticed by them, that required immediate recompression treatment.

A reluctance to report findings is denied in discussions with the astronauts. They, furthermore, argue against any change in the flight rules to reduce risk by increasing the prebreathe time (to increase nitrogen gas washout), an argument based upon their own experiences during EVA in space.

(b) Hormonal, hematological or other endogenous biochemical agents related to stress (e.g., catecholamine release) could influence the expected incidence of DCS. Altered physiology engendered by the stress/anticipation of EVA might increase the tissue perfusion rate or modify other blood/bubble interactions postulated to play a role in the pathogenesis of DCS (Hallenbeck and Andersen, 1982; Philp, 1990). The magnitude of perturbation(s) resulting from these effects is not currently known; many changes observed in laboratory trials, however, might be epiphenomena unassociated per se with the genesis of DCS.

Cardiovascular alterations in microgravity such as fluid shifts have been known to produce acute changes in tissue gas washout [127, 128] which appear to be engendered by increases in perfusion secondary to elevation of cardiac output (Figure 1). With readjustment in blood volume within a period of approximately 24 hours, less of a gas washout increase is expected. With water immersion, for example, most values return to the air-control conditions within two hours [129].

An increase in **whole-body** gas washout does not necessarily imply an increased gas elimination in "bends"-producing tissue. Subjects put immediately into a -15° head-down tilt position at the initiation of decompression and oxygen breathing, and for the remaining 3 to 6 hours, did not evince either a reduction in DCS or precordial Doppler-detectable gas bubbles (Iseyev, et al., 1988). The incidence of decompression sickness, even in acute head-down subjects, was not reduced when comparing seated vs. head-down supine individuals [16]. Fluid shifts are also produced in deep sea divers as a consequence of immersion; such an immersed condition is generally associated with an **increased risk** of DCS.

Modification of ventilation-perfusion ratios in microgravity would most likely not modify gas washout from long half-time compartments.

(c) The possibility of chance or "good luck" ("cluster phenomenon") is statistically possible and cannot be eliminated at this time. The number n of successful, i.e., "bends free" (probability = p), decompressions in n EVAs is given by the binomial distribution:

$$f(x) = {}^{n}C_{x}\, p^{x}\, (1-p)^{n-x} \quad \text{for } x = 1,2,\ldots,n. \qquad [11]$$

From this it can be found that there is currently an approximately 28% probability of <u>not</u> having encountered a severe DCS problem during the 26 EVAs. In

actual practice, the incidence could be lower since the population in question is not truly random. Since all EVA candidates have experienced hypobaric chamber decompressions, particularly sensitive individuals would have been initially eliminated from further pressure reduction work; thirteen possibly "resistant" individuals performed the 26 EVAs. Volunteers for earth-based hypobaric schedule testing are not "screened" in a similar fashion.

(d) This section focuses upon the last possibility, the reduction in the effects of stress-assisted nucleation and/or the number of tissue gas micronuclei. This would result from the reduction in activity in space of the lower limbs (hypokinesia) and the lack of weight-bearing loads (adynamia) on the legs.

2. Stable Nuclei

Meisel, Nir, Talmon and Kerem [131, 132] performed an analysis of the internal pressures of a spherical gas phase and developed the equation:

$$P_B = p_a + Dp + (3/4)\pi KR_B + 2g/R_B \qquad [12]$$

where p_a is the arterial inert gas partial pressure, Dp is the inherent unsaturation [133], K is the tissue elastic modulus, and g is the surface tension. This equation has been reduced into a dimensionless form (Meisel, Talmon, and Kerem, 1981) [132] and solved for the change of bubble radius R_B during decompression. The equation results in the ultimate shrinkage of the bubble because of the $2g/R_B$ term. The surface tension, it is usually contended, will reduce the bubble to $R_B = 0$.

If instead of the $2g/R_B$ term dominating in cases of small R, the lower radius is considered to be stable (at least for short periods) in accordance with the concepts outlined by Vann and Clark [134], using the treatment of Gent of r polymers [135-138]. Gent assumed that preexisting gas nuclei resided within the polymer with a minimum radius $R_{Bo} = 0.1$ microns; the origin of these nuclei was not elucidated although they were stable. Rather than the $2p/R_B$ term, serving as the compression term from surface tension, Gent introduced,

$$P_B = \Gamma [2.5\text{-}2(R_{Bo}/R_B) - 0.5(R_{Bo}/R_B)4] + 2g/R_B \qquad [13]$$

where R_{Bo} is the radius of the cavity at the time of formation, R_B is the radius at any subsequent time, and p is the shear modulus of the tissue. The actual biophysical system might be expected to be more complicated and anisotropic, but we are seeking a general example. The characteristic of the equation is that it retains small nuclei in a stable state. At $R/R_{Bo} < 1$, large increases in pressure are not accompanied by concomitant decreases in radius. At R the radius becomes unbounded and expands at a rate related to the rate of diffusion of gas into or out of the bubble. The tissue

bubble is in a steady state when the collapsing forces of negative supersaturation are balanced by the elastic resistance of the tissue-bubble complex.

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DR. VANN: We had a group of subjects who were aerobically trained for either distance running or triathlons, and these people had a much lower incidence of bubbles, and no bends when compared to another group who lifted weights, or played basketball or did not work out at all. One hypothesis to help explain this was that the metabolism stays elevated for a period of time, after a period of heavy aerobic exercise. These are people who run five to six miles a day. By virtue of increased resting tissue perfusion, more nitrogen may be washed out during the preoxygenation period, thereby having a lower gas load when they first go to altitude. It seems that there is a categorical difference between the aerobic exercise and other exercise, such as heavy weight lifting.

MR. WALIGORA: How did you identify people who were aerobically active? We have done it by scoring, asking them to indicate how often they exercise and then putting them in grades. We also have done stress tests on everyone to measure maximum oxygen consumption. But we found a rather poor correlation between the two. Some people equate high maximum oxygen consumption to fitness, but I do not think that is always clear-cut.

DR. VANN: I think you are right. We differentiated by training regimens.

MR. WALIGORA: Based on their routine activity.

DR. VANN: That is right. They had to be running or swimming or biking daily, as opposed to two or three times a week for a few miles each time.

LT COLONEL DIXON: Please define viscous adhesion. Is it the same thing as tribonucleation?

DR. VANN: Tribonucleation is a result of viscous adhesion. Viscous adhesion occurs when surfaces slide or when they are pulled apart. One way you can view it is that when you try to pull apart two very loosely separated surfaces, the fluid tries to move in from the outside. The more viscous the fluid, and the closer the surfaces are, the greater the forces needed to separate them. The viscosity keeps the fluid from flowing in, and thereby you generate negative pressure. That is exactly what keeps scotch tape on the paper, it is viscous adhesion. When you try to pull it apart it is very viscous. The fluid does not want to flow in. It is exactly the opposite of lubrication. Lubrication keeps surfaces apart because of the viscosity of the fluid between them. You cannot squeeze the fluid out, and thereby it stays apart. So tribonucleation is the effect, viscous adhesion is the cause.

DR. VAN LIEW: It is interesting to wonder about the lifetime of nuclei that are formed by these processes. If the nucleus only lasts for five minutes after a viscous adhesion event, then you would not expect bubble crushing to work.

DR. VANN: We do not know how long they last.

DR. VAN LIEW: It is not important to know exactly in minutes how long they last, but it is important in relation to the length of time that the pressure exposure occurs and the length of time the decompression occurs.

DR. VANN: There are data that speak to that. Daniels hydrostatically compressed shrimp and would wait different periods of time before he decompressed them. I think he found that after a day the incidence of bubbles had gradually returned to its control level. These were bubbles that were formed as a result of an altitude decompression. He used a pressure spike to eliminate them first. Then he observed how long it took for them to return to their control level of bubble formation. It is a gradual decrease. In 24 hours they were back to normal.

COLONEL SHEFFIELD: I want to clarify one of your statements about exercise before altitude exposure. You are not suggesting that our physiologists encourage their students to do a five mile run before an altitude exposure?

DR. VANN: No. But, that is a difficult question to answer. Exactly what is the critical level of exercise? Our experience has been that heavy weight lifting, i.e., bench pressing 300 pounds, seems to predispose one to DCS. However, I think that aerobic activity does not seem to make that much difference. Exactly where the transition is I do not know. We are going to do a controlled study using heavy weight lifting to see if that does indeed predispose. If we find it does, then we will start cutting back to find the crossover.

MR. WALIGORA: We just completed a study of heavy aerobic exercise 18 hours before the exposure. The reason we used 18 hours was because it was an operational consideration. Eighteen hours is probably the earliest before an EVA that we would have that kind of exercise. There was no effect whatsoever.

NEUROLOGICAL COMPLICATIONS OF DECOMPRESSION ILLNESS - MECHANISMS AND PATHOLOGY

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Introduction

Since the pioneering experiments of Boyle (1) in the mid-seventeenth century, it has been recognised that inappropriate decompression can result in illness and, ultimately, death. We now recognise that much of the morbidity and mortality which Boyle generated in the numerous specimens which he decompressed in his "exhausted receiver" was a consequence of hypoxia. However, Boyle also noted that decompression resulted in the liberation of bubbles of gas in the blood and tissues of the animals he decompressed. Such bubbles he postulated:

> "May by their vast number, and their conspiring distension, variously streighten in some places, and stretch in others, the vessels, especially the smaller ones, that convey the Bloud and Nourishment; and so by choaking up some passages, and vitiating the figure of others, disturb or hinder the due circulation of the Bloud? Not to mention the pains that such distensions may cause in some nerves, and membranous parts, which by irritating some of them in convulsions may hasten the death of the animals, and destroy them sooner by occasion of that irritation, than they would be destroyed by the bare absence or loss of what air is necessary to supply them with."

In the three hundred or so years which have followed Boyle's remarkable work, the dysbaric disorders have been described in man and much work has been undertaken both to try to prevent these conditions and to improve their treatment. It is fair to say that this has been achieved by an approach which has been largely empirical rather than as a result of a profound understanding of the mechanisms involved in the generation of bubble-induced disease. This presentation will review our current understanding of hypobaric neurological decompression illness. A somewhat distressing conclusion is that, as far as our understanding of the mechanisms of the disease is concerned, it is still difficult to improve upon Boyle's inspired conjecture.

Neurological Decompression Illness

By way of further introduction, I will briefly describe the neurological dysbaric conditions. Classically, a distinction has been made between arterial gas embolism and decompression sickness (DCS). In the first instance, gas, which normally resides

in the air spaces of the lung, escapes into the pulmonary vasculature in the form of bubbles and from there, they then enter the arterial blood stream and act as emboli. The condition which provokes this rupture of the pulmonary air spaces is pulmonary barotrauma, which is also known as the pulmonary overinflation syndrome. When not of iatrogenic origin, this most commonly occurs in the hyperbaric environment and happens when a diver, or someone escaping from a submarine, ascends through the water and fails to exhale sufficiently. If intrapulmonary pressure exceeds about 80 mm Hg, this overpressure is sufficient to rupture the delicate alveolar membranes (2, 3, 4). Such a simple explanation of the disease is almost certainly incomplete and other factors are likely to be involved. Being a delicate structure, the alveolus is also vulnerable to shearing forces. In a situation of rapidly changing barometric pressure, such forces could be generated at the interface between volumes of lung with differing compliance (5) or around areas of lung scarring. It is also recognised that the presence of pulmonary cysts or bullae, caused for example by emphysema, predisposes to the condition. In the aviation setting, however, such rapid changes of barometric pressure are unusual and therefore, for practical purposes, this mechanism for generating arterial gas bubbles will not be discussed.

In decompression sickness, it is believed that gas, which is dissolved in body tissues, reaches a level of supersaturation which is sufficient to exceed the bubble nucleation threshold and, as a consequence, leave solution. The physics of how bubbles are formed *in vivo* are still incompletely understood (6). However, given that bubbles do form and are responsible for decompression illness, there are a number of different locations for such bubbles to result in disease, which can be summarised as:

Intravascular -	Arterial
	-Capillary
	-Venous
Extravascular -	Intracellular
	-Extracellular

Finally, it is possible for a gas phase to be liberated within natural body cavities, e.g., the peritoneum and joint spaces. These are probably of little relevance to the symptoms of decompression illness since the collection of small amounts of gas in these spaces is symptomless and can therefore be ignored in this discussion.

Fundamental to our understanding of neurological decompression illness is an appreciation for the role of bubbles in these various possible locations in provoking neurological dysfunction. This review will discuss such possibilities and attempt to relate them to what is known about the pathology of hypobaric decompression illness.

Intravascular Bubble Injury

Sources of intravascular bubbles

Hills (7) considered that, at normal rates of decompression, there is only a limited possibility for arterial blood to become supersaturated to a significant extent because, having recently passed through the lungs, the tension of inert gas will be similar to ambient. Furthermore, even under conditions of rapid decompression, blood pressure is a substantial force which would oppose bubble formation. He concluded that at the rates of decompression from hyperbaric environments generally undertaken by man, the *de novo* evolution of bubbles of inert gas in the arterial blood stream is most unlikely. It should be appreciated, however, that this mechanism may apply to humans *in extremis* and some animal models of the disease, particularly those which involve the explosive decompression of small rodents. Thus, when studying references to the pathology of decompression illness, the severity of the decompression insult should be borne in mind. In the hypobaric environment, except for all but the most dire of emergencies, it is unlikely that the rate of decompression would be sufficient to result in the liberation of gas bubbles, *de novo,* in arterial blood. Thus, for the purpose of this discussion, this potential mechanism for decompression illness will be largely discounted.

In the hypobaric environment, there is, therefore, one primary source of intravascular gas bubbles - the gas dissolved in body tissues. However, it is unclear exactly where these bubbles are formed. The two possibilities are that they nucleate within the tissues and then migrate into the blood stream at the capillary or venular level. Alternatively, gas may escape from tissues and enter the vasculature in solution at any level. From there, it may result in the supersaturation of blood to a sufficient extent for bubbles to form. It is quite likely that both mechanisms operate and the one which predominates varies from tissue to tissue. For instance, there is ample evidence that bubbles of gas can form within adipocytes (8-10) and haemorrhages within adipose tissue occur in DCS (8, 11). It is tempting to speculate that this may be caused by bubbles of gas which form within the fat cells then expand and rupture into the vascular space. Unfortunately, although experiments to demonstrate this possibility have been attempted, the evidence is, as yet, unconvincing (12). Whether this mechanism applies to hypobaric decompression remains far from clear. The work quoted above involved hyperbaric decompression. Catchpole and Gersh tried to demonstrate the evolution of extravascular gas bubbles in rabbits decompressed to altitude without success (13, 14). In other tissues, such as muscle and kidney, where gas bubbles have not yet been found following decompression even from hyperbaric environments, it may be that the gas enters the vascular compartment in solution.

The neurological effects of intravascular bubbles

The effects of intravascular bubbles can be broadly divided into physical and biochemical categories, although there is no clear dividing line between them. The physical effects of bubbles are largely a consequence of their embolic qualities and

therefore depend on their location. Within the central nervous system, the sequence of events which follows bubble embolism of the brain is becoming more clear and the somewhat primitive idea that cerebral dysfunction is due solely to the ischaemic consequences of gas bubbles which physically obstruct blood vessels is being revised. It has been shown in a rabbit model of gas embolism that many bubbles which enter the cerebral circulation pass through without causing obstruction (15). Nonetheless it would appear that a vascular reaction to the passage of these bubbles occurs. Autoregulation of arterial diameter is impaired (16) and initially, this results in vasodilatation and hyperaemia (17-19) which are followed by a progressive reduction in cerebral blood flow and neurological function (15, 18-20). Hypertension, which quite commonly accompanies cerebral embolism (21, 22) may have a synergistic effect on these events, since it has been shown to result in reduced blood flow and cerebral function when associated with gas embolism (23). The reduction of blood flow is far from uniform and autoradiographic studies have shown that focal areas of very low blood flow appear to be adjacent to areas of apparent hyperaemia (24, 25). Ischaemia may be particularly pronounced at the grey/white matter interface (26, 27). The mechanisms involved in these alterations to cerebral perfusion are not understood.

There are additional consequences of the arrival of bubbles in the brain. First is their interaction with endothelium which results in an increased permeability of the blood-brain barrier (28-33). There is also an accumulation of leucocytes at the site of embolism (34) and there is evidence that this may be important in the development both of ischaemia and dysfunction (35, 36). Thus, in cerebral decompression illness, embolic bubbles probably serve as a physical trigger to a highly complex sequence of events which remains incompletely understood.

Similar events presumably occur in the spinal cord; however, embolism of the spinal cord, unlike the brain, appears to be a rare cause of neurological symptoms (37). In the case of decompression illness, there may be a number of reasons for this. First, it is a small organ and has a low blood flow compared with the brain (38); consequently, it has a relatively low probability of receiving emboli. Second, it is likely that the buoyancy of bubbles influences their distribution in arterial blood (39). Thus, if bubbles are distributed to the CNS of upright humans, the buoyancy effect is likely to result in the spinal cord being spared to a certain extent. Finally, compared with the brain, the spinal cord appears to be relatively resistant to ischaemia (40-43). Consequently, even if the cord is embolised, symptoms may not result. In view of these considerations, bubble embolism of the CNS is much more likely to result in dysfunction with a cerebral rather than a spinal cord distribution.

Nonetheless, experimentally, it is possible to induce spinal cord dysfunction as a result of gas embolism. If the cord is examined histologically shortly after the loss of function, there is little evidence of pathology (44). Four hours after the loss of function, it is primarily grey matter pathology which is found (45), as is the case in other causes of spinal cord ischaemia (46, 47). This is at variance with what is generally found in DCS, as will be discussed later.

Another possible way in which intravascular bubbles may interfere with central nervous system function was proposed by Hallenbeck *et al.*(48). Having recognised that the interaction between bubbles and blood is highly complex, and can result in the accelerated coagulation of blood (49) and many other effects (50), they postulated and subsequently demonstrated that following decompression from a hyperbaric exposure, bubbles of gas accumulate in the epidural vertebral venous system (EVVS). This accumulation of bubbles was associated with an apparent cessation of blood flow in these vessels and a loss of spinal cord function (51). However, although further experiments have repeated the observation that bubbles of gas accumulate in the EVVS (52), it is unclear whether this is associated with venous obstruction. There is some doubt as to whether the pathology of venous infarction of the spinal cord is comparable to that seen in DCS (53).

Extravascular Bubble Injury

Evidence for extravascular bubbles in decompression illness

It is possible to interpret the conclusions of Boyle (1) and Bert (54) as proposing a role for *in situ* or autochthonous bubbles in the pathogenesis of decompresssion illness. Keyser (55) was probably the first formally to propose such a mechanism. The problem has been that for many years there was little evidence to support such a hypothesis. The much-quoted observations of Boycott *et al.* on the presence of bubbles of gas in the spinal cord of a goat that had died at depth was disregarded even by the authors (56). This was because, since the animal was deprived of the opportunity to off-gas during decompression, the presence of extravascular gas at *post mortem* examination would be expected. Similar observations have subsequently been made in divers who have died at depth (57) or who perished following explosive decompression (58). There have been occasional mentions of the finding of extravascular bubbles in the spinal cord of experimental animals (59, 60) and in a case of rapidly fatal human DCS (61), but these have been few and far between. More recently, however, evidence has been presented that in hyperbaric DCS, bubbles of gas can be found routinely in the spinal cords of animal models of the disease (11, 43, 44, 62). Furthermore, in one model of the condition, there appeared to be sufficient bubbles to account for the loss of spinal cord function (63).

The neurological effects of extravascular bubbles

As with intravascular bubbles, extravascular bubbles may have both physical and biochemical effects. However, very little is known about the role of these bubbles in generating neurological dysfunction. It is known that as a result of the formation of these bubbles in the spinal cord, white matter axons are displaced and compressed (43, 63). It is possible that the force exerted on spinal axons is sufficient to result in a neurapraxia (64-66). The attraction of this hypothesis is that the onset of dysfunction is rapid and resolves spontaneously provided that the duration of compression is not prolonged. This corresponds with some of the clinical features of spinal cord DCS.

An alternative manner in which spinal cord dysfunction could result from the presence of autochthonous bubbles is if there were a destructive interaction between spinal nerves and gas at the tissue-bubble interface. It is not difficult to postulate that at the high energy interface between bubbles and myelin there is distortion or denaturation of membrane proteins which could result in the impairment of membrane transport and, eventually, nerve conduction may be compromised. After all, there is ample evidence for a similar blood-bubble interface reaction (67).

Hills and James have proposed that the presence of gas bubbles in the spinal cord may interfere with tissue perfusion (68). In their model, the volume of gas which needed to be liberated into the spinal cord in order to exceed arteriolar pressure was about 14-31% of cord volume. In one well documented canine model of spinal cord DCS, in which these bubbles have been found, their volume is at least an order of magnitude less than this (63). Consequently, a global spinal cord ischaemia as envisioned by Hills and James would appear to be unlikely. Nonetheless, considering the distortion of tissues occasioned by the evolution of autochthonous bubbles, it is possible that discrete areas of hypoperfusion are a consequence of disruption of the microcirculation.

It has long been recognised that a feature of the pathology of spinal cord DCS is the appearance of punctate, white matter haemorrhages. It has recently been shown that these may be a sequel to the formation of autochthonous bubbles in white matter (11). It is possible that such haemorrhages eventually result in spinal cord dysfunction and demyelination. If free iron ions are released into the white matter as a result of the breakdown of erythrocytes, they would serve as catalysts for the peroxidation of myelin (69).

Extravascular bubbles as mediators of hypobaric decompression sickness.

Much of the foregoing discussion relates to the events which follow hyperbaric decompression. The reason for this is that much more is known about this process than the consequences of inappropriate ascent to altitude. Hyperbaric DCS in man was described as long ago as 1854 (70) and has been studied more or less continuously since then, whereas hypobaric DCS is a relatively new phenomenon. Furthermore, the clinical manifestations and physiology of hypobaric DCS are often complicated by the effects of hypoxia, unlike in the hyperbaric condition. This has made the study of the condition considerably more difficult. There have been relatively few human fatalities from hypobaric DCS; consequently, there has been little human material to study. In the majority of cases which have occurred, the clinical and pathological picture has been complicated by shock. Of critical importance, hypobaric DCS is not usually rapidly fatal, and thus the hyperacute pathology has not been studied. Perhaps the greatest difficulty of all in the study of human hypobaric DCS, and it is a problem which has afflicted many animal studies, is that following the hypobaric insult, the victim is usually returned to normobaric pressure prior to investigation. Because of this, it is likely that many important clues as to what went wrong are lost, in particular, the bubbles which may have provoked the illness are

given the opportunity to avoid detection. It is for these reasons, and the absence of a good animal model of the condition, that any assessment of the role of extravascular bubbles in the pathogenesis of hypobaric DCS has to be speculative.

A curiosity is the apparent rarity of spinal cord involvement in hypobaric DCS compared with the illnesses which follow hyperbaric exposures. One possible explanation is that alluded to above, namely that the primary mechanism is that of gas embolism and in seated aircrew, the buoyancy of arterial bubbles and the relative blood flow of the organs involved mitigate in favour of a cerebral rather than a spinal cord distribution of the emboli. This is not entirely satisfactory. Although many divers are in a horizontal attitude while in the water, only a small minority of CNS DCS has an onset in the water (71). The great majority occur after surfacing when divers are in a seated or standing position. Compressed-air workers similarly, tend to be upright at the onset of symptoms. There is an alternative explanation for the rarity of spinal cord involvement in hypobaric DCS which is based on an extravascular bubble mechanism.

Although some doubt has been raised as to the validity of perfusion-limited kinetics to describe tissue gas exchange (72), an estimate of tissue time constant can be derived using a standard exponential function. For spinal cord white matter this is of the order of 15 minutes (73). What this means is that within about 15 minutes of a step reduction in atmospheric pressure, the inert gas concentration in spinal cord white matter will be approximately halved. Another background piece of information is that there appears to be a threshold gas tension for the formation of bubbles in spinal cord white matter. A recent series of experiments has estimated this to be about 3.6 atmospheres absolute (ata) for a hyperbaric decompression. An equivalent decompression to altitude would be to approximately 31,000 feet.

Piccard made an important observation with respect to the rate at which gas bubbles are released from water which is exposed to comparable hyperbaric and hypobaric decompressions (74). He found that although the volume of gas released from a litre of water saturated with air following decompression from 5 ata to 1 ata was the same as following a decompression from 1 ata to 0.2 ata, the rate at which the gas was released was different in the two experiments. He states " In the first case the bulk of dissolved air had escaped after 5 seconds, while in case 2, several minutes elapsed before the gas production even approximately ceased." Piccard's explanation for this observation is that for a bubble to be stable, it has to reach a critical radius. The number of molecules of air required to form this stable bubble differed in the two experimental conditions: 29×10^6 molecules in the hyperbaric decompression compared with 690×10^6 in the hypobaric decompression. Clearly, it is more likely for a smaller rather than a larger number of molecules to congregate at a point in the water. Consequently, bubble formation is more rapid following the hyperbaric decompression. With reference to the spinal cord, it may be envisaged that given the fairly short time constant of white matter (and the far shorter time constant of grey matter), bubble formation may occur in the hyperbaric decompression before significant washout of inert gas had occurred. On the other hand, following hypobaric decompression,

there may be sufficient time for tissue gas washout to reduce the inert gas concentration to below the bubble nucleation threshold before significant bubbling occurs.

From this discussion, it may be inferred that autochthonous bubble injury to the CNS is less likely in hypobaric decompression compared with comparable hyperbaric decompressions. This is supported by the observation that extravascular bubbles have not been found in the brains of animals which have been exposed to explosive decompression to altitude (75-77). However, the white matter injury found in the spinal cords of a few experimental animals may possibly have been a consequence of autochthonous bubble injury (76, 77).

Non-bubble mechanisms of decompression sickness.

It is worth mentioning briefly that events which are only indirectly related to the formation of bubbles in blood and tissues may have a bearing on the development of DCS. Mention has already been made of the complex events which occur at the blood-bubble interface (38) and the consequences these have on the rheology and structure of blood (50). End has proposed that these events alone can explain DCS (75); however, this view has not met with widespread acceptance. Other events which may have a bearing on the condition include: increased haematocrit and a loss of plasma volume, which have been found in both animals and man (75-84); the aggregation of platelets (84-89), leucocytes (87) and rouleaux formation (82) and the finding of endothelial cell (87, 90) fat and bone marrow emboli (61, 90-96). While it is not difficult to accept that such pathology may result in general malaise, it is unclear what the role of such pathology may be in inducing primarily neurological symptoms or signs.

Another phenomenon which has received attention recently is the role of complement in the generation of DCS. Ward *et al.* have shown that bubbles activate complement via the extrinsic pathway and that in both rabbits and man, individuals vary in the extent to which this happens. They have shown in man that DCS appears to be associated with those whose complement is readily activated and that decomplementing rabbits appears to protect them from DCS following a standard dive profile (97-99). Again, it is unclear how the activation of complement by bubbles, which occur extensively in the vascular tree following decompression, can result in neurological DCS. Nonetheless, this may be an important mechanism behind the accumulation of leucocytes and platelets and the disruption of the blood-brain barrier which follows the introduction of bubbles into the cerebral circulation.

Pathology of Hypobaric CNS Decompression Sickness

As Fryer and Roxburgh observed: "On the whole, post-mortem findings have been of little value in determining the nature of the condition.... Probably the greatest difficulty lies in the interpretation of the findings in the central nervous system" (100). The reasons for this were outlined previously. As a consequence, despite the title of this presentation, I intend to spend little time describing the pathology of the condition.

Cerebral DCS

Cerebral DCS is not a particularly common condition. Furthermore, it is not usually fatal. A case recently reported in the literature (101) was the first since the case reported by Odland in 1959 (102). Consequently, this review of the pathology of hypobaric CNS DCS is necessarily based on very few cases. As pointed out by Haymaker (103) there is a need to distinguish changes due to DCS from those due to ischaemia associated with hypoxia, chokes and shock which invariably accompany fatal cases (104, 105).

Haymaker and Johnston in their review of altitude decompression sickness (106), noted that generally, the grey matter is spared except at the base of sulci and in Somner's sector of the hippocampus, which they considered could be a consequence of hypoxia. Where changes to grey matter are found, they are limited to sponginess of subpial parenchyma and focal ischemic changes to nerve cells which display pyknotic and hyperchromatic nuclei (103). The primary pathological feature is involvement of white matter which is particularly marked when there has been survival for more than a few hours. Within the white matter there is demyelination - particularly perivascularly and perivascular oedema. The changes to myelin range from fenestration to extensive areas of spongy change. The changes were considered too extensive to be due to hypoxia alone, but since it is recognised that oedema fluid has a destructive effect on myelin, they concluded that the demyelination and perivascular oedema are linked and may be a consequence of bubble embolisation (106). Other findings in the brain are of wedge-shaped infarcts which occur particularly at the interface between grey and white matter (107, 108).

Involvement of the spinal cord in altitude DCS is less frequent than cerebral involvement and it is extremely rare for such cases to reach autopsy. Consequently, yet again, this review is not based on extensive numbers of cases. As in hyperbaric DCS, a case has been reported (91) in which there were scattered, punctate, largely white matter lesions. They tended to be located near blood vessels and showed parenchymal necrosis. There were non-haemhorragic foci of plasma transudation with associated degeneration of axis cylinders and fenestration of myelin sheaths. Unlike in hyperbaric DCS, there was involvement of grey matter. This involved foci of disrupted tissue architecture, again frequently in perivascular locations. Within these foci there were ballooned axons and occasional erythrocytes. Similar changes were seen in two other cases from this paper, but only a very limited amount of cord was available. Essentially, the same findings were reported in the papers by Casey and Dunn (76, 77). It is important to note that the pathology of hypobaric DCS differs somewhat from conventional hyperbaric DCS, in which the white matter is involved almost exclusively (109). However, based upon such a paucity of evidence, this is about the only difference which can be defined between the two conditions and this may indicate that there is a greater role for gas bubble emboli in the hypobaric condition.

Is there residual injury from DCS?

Since the introduction of oxygen treatment tables, it has been generally assumed that a more or less complete recovery can be expected from neurological decompression illness. However, although few would dispute that a clinical recovery is generally achieved, there is growing concern that more subtle, permanent injury may persist, but which avoids detection by conventional examination.

As long ago as 1959, Roszahegyi (110) raised the possibility that psychological disorders may be a sequel of neurological decompression illness. He examined 100 compressed air workers 2.5 to 5 years after neurological DCS and concluded that more than half of them showed evidence of some form of psychological disorder. Of further interest, he detected neurological abnormalities in three quarters of these cases. Unfortunately, Roszahegyi's testing techniques were not objective and he did not study a comparable control group and so, although his findings are interesting, they do not represent evidence that the abnormalities he detected were a consequence of neurological decompression illness *per se*. This work, however, did spawn further studies of the possible psychological consequences of hyperbaric exposures and these were well reviewed by Edmonds and Hayward (111). They asked two questions: Is there evidence of psychological abnormalities following DCS? And, is there any evidence of a cumulative deficit in any aspect of psychomotor function in those who are repeatedly exposed to a decompression stress? They concluded that because of the technical limitations of studies which have been performed to date, it is not possible to be certain in either instance. There remain, however, studies which are at least suggestive that the answer to both questions may be yes (111-117).

Although hard evidence of functional abnormalities in the CNS of divers remains to be gathered, there is evidence that pathological changes may occur in the spinal cord as a consequence of diving rather than as a sequel to decompression sickness (118). There may be evidence of cerebral injury as well, although this is less well defined (119). The possibility of silent cerebral injury in cases of decompression sickness was provided by Adkisson et al. (120, 121). Using a novel technique for measuring cerebral blood flow, they demonstrated cerebral perfusion deficits in cases of neurological DCS in which the brain was not necessarily involved. Furthermore, in a subsequent study, they showed that such perfusion deficits appeared to be persistent (122). Unfortunately, there was no control group in these studies. Current evidence from non-diving controls indicates that these perfusion deficits may be found in individuals who have sustained no obvious injury (D. J. Smith, personal communication). Nonetheless, it would appear that the number and density of the perfusion deficits seen in cases of neurological decompression illness does appear to be greater than in controls and further work is under way to clarify this important issue.

There are further areas of concern. One which was highlighted by a dramatic case report in 1981 (123) is the question: Does clinical recovery equate to the restoration of anatomical normality? In this remarkable case, a diver had made an almost complete recovery from spinal cord DCS and had been carefully examined only 12

days before meeting an untimely end. When his spinal cord was examined, the extent of residual gliosis, demyelination and Wallerian degeneration substantially exceeded the expectation of those who had been responsible for his care. The obvious conclusion is that clinical recovery can occur in the presence of substantial residual injury, presumably as a consequence of the recruitment of alternative pathways.

Finally, in a study of retinal angiography of divers, Polkinghorne et al. (124) have shown that divers had significantly more abnormalities of the retinal pigment epithelium than non-diving controls and evidence of reduced retinal capillary density. Since the retina is a "window" of the brain, this study raises further questions as to the long-term effects of repeated decompression on the CNS.

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THE IMPORTANCE OF HYPERBARIC OXYGEN THERAPY IN THE MANAGEMENT OF ALTITUDE DECOMPRESSION SICKNESS

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The little Bubbles generated upon the absence of the Air in the Bloud, juyces, and soft parts of the Body, may be their Vast number, and their conspiring distention, variously streighten in some places, and stretch in others, the Vessels, especially the smaller ones, that convey the Bloud and Nourishment; and so by choaking up some passages, and vitiating the figure of others, disturbe or hinder the due circulation of the Bloud!
Sir Robert Boyle, 1670 (11)

INTRODUCTION

Over 300 years ago Sir Robert Boyle (1670) postulated the occurrence of an environmental phenomenon that is now known as decompression sickness (DCS). Early information about this disorder came from the caisson and diving communities. Searching for the cause of decompression illness among divers and caisson workers, Paul Bert conducted extensive studies on sudden decompression from pressures greater than one atmosphere absolute (1 ata). In 1878, he published the bubble theory, on which he commented "...I have shown that all the symptoms from the slightest to those which bring on sudden death, are the consequences of the liberation of bubbles of nitrogen in the blood and even in the tissue when the compression has lasted long enough" (8). However, Bert did not relate the disorder to decompression at altitude. Even though he conducted experiments on himself at altitudes of 29,000 feet, he made no mention of symptoms that could be attributed to decompression illness (8). Von Schrotter and colleagues (1900) are credited with coining the phrase "compressed air illness" or "caisson disease" to describe the disorder (22,36).

DISCUSSION

Early Reports of Altitude Decompression Sickness

From their early work on the practical prevention of "caisson disease," Boycott and Haldane (1908) cautioned that this disorder might be possible at very low pressures (10,21). However, Professor Yandell Henderson (1917) is credited with providing a detailed theory that it was possible to experience decompression sickness from exposure to altitude. He wrote, "In order for bubbles to be formed, it is essential, however, that the pressure with which tissues are in equilibrium should be lowered more than half its absolute amount in a few minutes. In the present state of the art of flying, it is scarcely possible for an aviator to rise to a height of 20,000 feet, where the

barometer would be about half that of sea level, in a period sufficiently brief to allow bubbles of nitrogen to form in this way. The disorders from which aviators suffer therefore, are of a different class from those to which workers in compressed air are exposed" (24). The limited operational ceiling of 1917-18 vintage aircraft validated this prediction.

Jungbloed (1929) is credited with providing one of the earliest experimental accounts of altitude-induced DCS in his doctoral thesis. He and his colleagues repeatedly experienced the occurrence of joint pains after exposure to altitudes greater than 42,000 feet. He asserted, "These phenomena are analgous with those found in men after rapid decompression from a high atmospheric pressure, i.e., light cases of the caisson sickness" (21,27). Barcroft, Douglas, Kendal, and Margaria (1931) are credited with experiments that determined the maximum altitude at which subjects could effectively work while breathing 100% oxygen. During prolonged exposure to 30,000 feet, Margaria experienced acute bilateral knee pain after approximately 25 minutes of climbing exercises (ascending a box 13 inches high once every four seconds). The pain did not resolve for several days (2,22). Seven years later, Boothby and Lovelace (1938) reported that a colleague, Dr. J.W. Heim, experienced paralysis from the waist down while exposed to an altitude of 35,000 feet. Dr. Heim was asymptomatic upon reaching ground level (9,22).

Altitude decompression sickness became an operational problem in World War II due to relatively high, unpressurized flight, especially for bomber and reconnaissance aircrew. The risk increased when Germany developed cabin pressurization systems that allowed their aircraft to cruise at altitudes as high as 42,000 feet. In one instance, two of three aviators piloting British Spitfires experienced incapacitating bends while successfully shooting down three German aircraft at very high altitude over Africa. Data from 8th Air Force B-17 combat crewmembers revealed that 24 of 97 of those interviewed had experienced bends pain during flight, many of which occurred at altitudes below 30,000 feet. Bomber crewmembers had an 8% chance of experiencing incapacitating bends pain during flight (39). The German authors, Benzinger and Hornberger (1941), coined the term "Druckfallkrankheit," for which the English translation is "Decompression Sickness" (26).

Early Efforts to Prevent Altitude Decompression Sickness

Technological advances in aircraft design expanded the performance envelope. One important advance was refinement of the aviator's breathing mask by Boothby, Lovelace, and Eulbulian in the late 1930s (9). In 1939, Boeing Aircraft Company test pilots, who had previously canceled "high-altitude" flights due to decompression sickness, were required to breathe oxygen before flight (39). But, it was not until 1943 that the true value of denitrogenation as prophylaxis to altitude decompression sickness was clearly validated by Wright Field Aeromedical Laboratory (37).

During the mid-1930s, Army Air Corps flying officers were tested in low-pressure chambers at Wright Field, OH and Patterson Field, OH. Flying officers were given a physical altitude rating that reflected their ability to tolerate hypoxia. In 1941, the U.S. Army recruited thousands of aviators, which prompted the organization of the Army Air Corps Altitude Training Program in 1942. The program was established to educate flyers about the physiological dangers of flight and teach them how to use oxygen equipment and other protective equipment. Altitude chambers were used to screen out bends-prone individuals. In 1943, the Wright Field Aero Medical Laboratory discovered that prebreathing 100% oxygen before going to altitude would practically eliminate evolved gas problems. This finding resulted in changing the use of the low-pressure chambers from classification of flyers to that of a training tool. Developments of higher flying and faster aircraft near the end of the war brought new requirements for research and training in support of the "man in the system" (37).

In 1941, A. Behnke described "compressed-air illness" as primarily a problem for the US Navy and industrial operations, but added military aviation to the list of concerns in 1945 (3,6). Even though Behnke's work focused primarily on diving decompression sickness, he made significant contributions to the recognition and treatment of altitude decompression sickness (4,5).

The potential negative impact on operational bombing missions was highlighted in 1945 during a series of bombing test flights attempting to reach and maintain a peak altitude of 40,000 feet for a duration of one hour. Over a 20-day period, only one flight reached 37,000 feet. Of the nine unsuccessful flights, five were canceled solely due to physiological reactions, and four were canceled due to a combination of physiological and equipment difficulties. All but one physiological reaction was incapacitating bends; one pilot requiring hospitalization following descent (39).

Because of the associated risks to mission completion, much work was done during the early 1940s to identify aviators who were prone to developing DCS. Brink (1943) reported that only two major symptoms were used to evaluate one's tolerance: chokes and joint pain. Even though he found much individual variability, he reported that 20-30% of men exposed to altitudes as high as 38,000 feet for as long as four hours would develop symptoms that required descent (12). Air cadets were given a series of three-hour exposures to 38,000 feet and were classified into two groups: those prone to bends and those not. When the temperature was lowered, the incidence of joint pain remained about the same, but chokes was markedly reduced. When moderate exercise was added as a test condition, the incidence of incapacitating bends significantly increased. Since exercise caused a subject to experience bends much quicker, this factor was used to make a more efficient selection of immune subjects (12).

In 1943, R.L. Masland published a review of 55 cases of collapse occurring in altitude chambers, and cautioned aviation physiologists to develop a keen eye to

determine which students should be hospitalized following an altitude exposure. He reported that a small number experienced delayed reactions which could prove fatal if left untreated (32).

Contributions to Treatment by Diving Decompression Sickness

Pioneers in the 1940s and 1950s such as Behnke (6), Downey (19) and Armstrong (1) each attested to the similarities between diving and altitude DCS. Perhaps one reason for the slow development of a definitive treatment for altitude decompression sickness was the failure of the aeromedical community to accept the similarity.

Before turning to an in-depth discussion of the current treatment of altitude decompression sickness, let us digress to the use of compression therapy in the treatment of diving decompression sickness. Even though the air pump was developed by von Guericke in 1650, it was not until nearly 180 years later that it was used widely to support manned caisson work that could lead to "compressed-air illness" or "caisson disease." In the mid 1800s when hyperbaric chambers appeared in Europe, the technology was complete for the treatment of DCS. In 1854, Pol and Watelle reported what is thought to be the first account of the effectiveness of recompression when they noted the pain of a caisson worker disappeared when he returned to the pressure in the caisson (35). In 1878, Bert specified the treatment for decompression sickness as a combination of oxygen and recompression. "If auscultation indicates some gaseous gurgling in the region of the heart, immediately make the patient inhale oxygen as pure as possible, which should always be at hand in a rubber balloon, or better, compressed in quantity in some steel reservior. Then when the gases have disappeared from the heart, and death no longer seems imminent, subject the patient immediately to a pressure greater than that from which he came, then make the decompression very slowly....When the decompression shows its effects by paraplegia, recompression must be carried on at once, without losing time in inhaling oxygen, especially when the symptoms did not appear until some time after the return to the open air, for in this case we have to do, not with a general obstruction of the pulmonary circulation, but with some bubbles of gas lodged in the vessels of the medulla, whose volume must be reduced at once so that the blood may drive it out" (8). Snell (1896) advocated the use of a recompression chamber at the Blackwall Tunnel construction site (38). In 1912, Keays reported the first large series of caisson disease cases that established the benefit of recompression to treat DCS (28,29). In 3,278 cases of pain, 90% obtained complete relief with recompression. The treatment profile was to recompress the worker to the depth of the caisson, remain for a brief period then rapidly decompress to 22 to 34 feet of sea water [FSW] depth equivalent followed by a slow ascent to surface. Twelve years later, the US Navy (1924) published its first standardized compressed air treatment schedules for decompression sickness.

Though not widely accepted until many years later, investigators such as Bert (1878), Zuntz (1897), and Heller, Mager and von Schrotter (1900) proposed the use of

supplemental oxygen in addition to increased pressure for the treatment of decompression sickness (4). It was not until Behnke and colleagues (1935) began a series of animal studies that the use of oxygen inhalation was seriously pursued (5). These studies resulted in the modification of the US Navy compressed air treatment tables by adding oxygen breathing at equivalent depths of 60, 50, and 40 FSW after an initial exposure to 165 FSW (45). Because of the high recurrence rate of these tables, Van der Aue, et al. (1945) developed US Navy Treatment Tables 1-4 which remained the principal treatment approach until 1967 (4,40). Figure 1 compares the US Navy Treatment Tables (17).

Early Treatment of Altitude Decompression Sickness

In an attempt to confirm earlier altitude chamber studies on this disorder, the Subcommittee on Decompression Sickness attempted a series of test flights in San Diego in late 1943 by the Consolidated Vultee Aircraft Company. One of the objectives of these flights was to test the efficacy of the "altitude pressure bag." This inflatable rubber bag was designed so that an aviator with the bends could be inserted, zipped up and pressurized until the internal pressure altitude equaled 20,000 feet. This altitude equivalent was chosen because altitude chamber studies of the time indicated that most subjects' bends pain went away on descent to that altitude. The bag was used nine times, seven times successfully, and twice unsuccessfully due to mechanical failure. The "altitude pressure bag" was discontinued due to difficulty in its use, extreme cold for the patient, and technical problems (39).

Until the classic Donnell and Norton (1959) compression treatment of a severe case of altitude decompression sickness, the recognized treatment was descent to ground level and treat for profound shock if required (18). Henry (1946) even mentioned the application of local pressure over the symptom sites, but found it to be impractical as a form of definitive treatment (25). He also noted that inactivity at altitude, denitrogenation, and preselection were the only practical means of prophylaxis. Although the use of supplemental 100% oxygen breathing and plasma administration were reported as early as 1951, their efficacy was not clear (20).

It had long been recognized in altitude decompression sickness that treatment was initiated by descent to ground level and was totally successful in many instances. Those cases that did not resolve upon return to ground level became candidates for compression therapy, hereafter referred to as hyperbaric oxygen (HBO) therapy. The classic case of Donnell and Norton (1959) initiated the use of HBO for treatment of altitude DCS (18). A 39-year-old pilot developed decompression sickness after an altitude chamber flight to 43,000 feet. Upon return to ground level he was noted to have decreased use of his left arm. During questioning, the pilot reported his inflight symptoms and was placed on 100% oxygen. Flight surgeon's examination revealed onset of a severe headache and blurred vision. The pilot was hospitalized for observation, but during admission, his condition rapidly worsened. He became completely disoriented, pallid and cold to the touch, exhibited skin mottling and increased blurred vision. Even with intravenous fluids and 100% oxygen, the pilot's

condition continued to deteriorate. When the pilot became moribund, recompression was recommended, and he was flown by helicopter to a US Navy hyperbaric chamber at Little Creek Amphibious Base, VA. He was successfully treated on US Navy Treatment Table 4, exiting 38 hours later with only a mild deficit in "his ability to deal with symbolic abstractions" (18).

The Donnell and Norton case generated great interest in the aeromedical community. However, some physicians maintained that the treatment of choice was shock management especially if hyperbaric chambers were not locally available. Lack of readily available treatment facilities perpetuated the problem of prompt treatment. Berry (1961) and Malette (1962) acknowledged the use of hyperbaric therapy, but did not recommend it until more experimental data were available (7,31). The classic animal work by Leverett, Bitter and McIver (1963) provided much of these data. In 5 of 26 dogs decompressed to altitude, bubbles existed in the blood upon return to ground level. They recommended further compression to attempt bubble resolution (30). One animal with pulmonary hypertension and tachypnea after return to ground level was pressurized to 3 ata pressure (66 FSW) with subsequent return to normal (33).

Other cases of successful use of HBO in the treatment of altitude decompression sickness began to appear in the literature. Cogburn, et al. (1962) reported the successful treatment of a 47-year-old pilot who experienced loss of motor function in the left leg after exposure in an altitude chamber to 37,000 feet for one hour (14). A third case was reported from Edwards AFB CA (1961) after a photographer experienced an in-flight exposure to a cabin altitude of 40,000 feet. His shoulder pain, numbness, and chokes were completely relieved after treatment on a combination of US Navy Treatment Tables 3 and 4 (33). Additional early cases were reported by Cannon and Gould (13) and Goodman (23).

To consolidate all available information on the incidence of altitude DCS and treatment outcomes (particularly in the Air Force), the United States Air Force School of Aerospace Medicine (USAFSAM) established the USAFSAM Decompression Sickness Management Team in 1963. This milestone, in effect, marked the beginning of the USAF Hyperbaric Medicine Program. During 1965 to 1967 the U.S. Air Force (USAF) installed eight double-lock, hyperbaric chambers at physiological training units where they could complement existing US Navy and civilian chambers and insure expedient treatment availability to our aviators. Today, these eight chambers continue to provide definitive treatment for altitude DCS; they are supplemented by three large clinical hyperbaric facilities located at Brooks AFB, TX, Wright-Patterson AFB, OH, and Travis AFB, CA, which also provide definitive treatment for this disorder. The USAFSAM Hyperbaric Medicine Division is now in its 27th year as the central repository for data collection of all USAF decompression sickness incidents and serves as the Office of Primary Responsibility for the overall USAF Hyperbaric Medicine Program (16). Consultation regarding various aspects of hyperbaric therapy is available 24 hours daily by calling DSN 240-3281 or (512)536-3281 during normal duty hours (0715-1600) or DSN 240-3278 or (512)536-3278 after normal duty hours (1600-0715).

During the time the USAF was developing its treatment capabilities, work was continuing in the US Navy by Workman and Goodman (1968) to develop minimal pressure oxygen breathing treatment tables for diving decompression sickness. Their work evaluated the usefulness of administering 100% oxygen at 60 FSW for a prescribed period with follow-on oxygen breathing during ascent to surface (44). These new oxygen breathing tables (Treatment Tables 5, 5A, 6 and 6A), reported to have a 94% success rate, were formally adopted by the US Navy on 22 August 1967. The initial use of Tables 5 and 6 for the treatment of altitude DCS were equally promising. Five of seven cases (72%) resolved completely after a single treatment, one substantially improved, and one had complete resolution several days following treatment (44). These tables became the standard of care for aviators experiencing altitude decompression sickness and remain so today.

Effectiveness of Current Hyperbaric Oxygen Treatment Tables for Altitude Decompression Sickness

Generally speaking, the physiological rationale for the use of HBO therapy in the treatment of altitude decompression sickness is as follows:

(1) Mechanical compression to reduce the volume of gas (bubble size reduction).

(2) Accelerate the rate of inert gas elimination through enhanced diffusion gradients at the tissue and lung level (bubble resolution).

(3) Increase the amount of oxygen available to hypoxic tissues (tissue oxygenation).

Prior to 1959 when hyperbaric therapy became the recognized treatment for altitude decompression sickness, over 17,000 cases, including at least 17 fatalities, were presented in numerous reports. In the classic review of 145 cases of altitude decompression sickness treated prior to 1 January 1977, Davis et al. (1977) reported on the significant positive effect of hyperbaric therapy (15). Before 1965, nine cases (9/145) were treated with compressed air tables (US Navy Treatment Tables 1,2,3, or 4) (Table 1) with an 88% success rate. After 1965, the remaining 136 (136/145) obtained a 99.3% complete resolution rate when treated on intermittent oxygen/air breathing tables such as Treatment Table 5 (Table 2) and Treatment Table 6 (Table 3) as modified by the USAF (15). The USAF modifications for Treatment 5 include the addition of a 5-minute air break after the second oxygen breathing period at 60 FSW prior to ascent to 30 FSW replacing the second air break at 30 FSW prior to ascent to surface. Treatment Table 6 modifications include delivering oxygen/air on an intermittent schedule of 20/5 for a total of 120 oxygen minutes as opposed to the USN schedule of 60/15 at the 30 FSW level. On both Treatment Tables 5 and 6, the inside medical attendant breathes 100% oxygen from 30 FSW to surface.

TABLE 1. Treatment of Decompression Sickness with USN Tables 1, 1A, 2, 2A, 3 or 4*

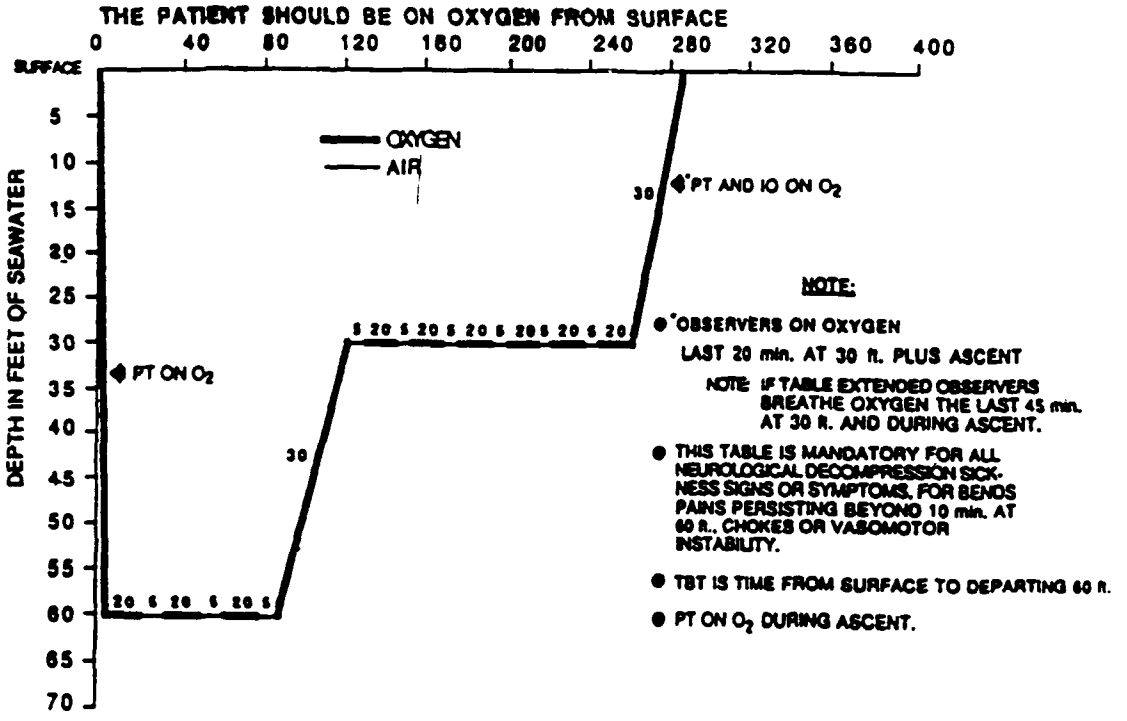
STOPS	BENDS--PAIN ONLY		SERIOUS SYMPTOMS	
RATE OF DESCENT: 25 FEET PER MIN. RATE OF ASCENT: 1 MINUTE BETWEEN STOPS.	PAIN RELIEVED AT DEPTHS LESS THAN 66 FEET. USE TABLE 1A IF OXYGEN CANNOT BE USED.	PAIN RELIEVED AT DEPTH GREATER THAN 66 FEET. USE TABLE 2A IF OXYGEN CANNOT BE USED. IF PAIN DOES NOT IMPROVE WITHIN 30 MINUTES AT 165 FEET, THE CASE IS PROBABLY NOT BENDS. DECOMPRESS ON TABLE 2 OR 2A.	SERIOUS SYMPTOMS INCLUDE ANY ONE OF THE FOLLOWING: 1. UNCONSCIOUSNESS. 2. CONVULSIONS. 3. WEAKNESS OR INABILITY TO USE ARMS OR LEGS. 4. AIR EMBOLISM. 5. ANY VISUAL DISTURBANCES. 6. DIZZINESS. 7. LOSS OF SPEECH OR HEARING. 8. SEVERE SHORTNESS OF BREATH OR CHOKES. 9. BENDS OCCURRING WHILE STILL UNDER PRESSURE.	
			SYMPTOMS RELIEVED WITHIN 30 MINUTES AT 165 FEET. USE TABLE 3.	SYMPTOMS NOT RELIEVED WITHIN 30 MINUTES AT 165 FEET.

PSIG	FEET	TABLE 1	TABLE 1A	TABLE 2	TABLE 2A	TABLE 3	TABLE 4
73.4	165	———	———	30 (AIR)	30 (AIR)	30 (AIR)	30 TO 120 (AIR)
62.3	140	———	———	12 (AIR)	12 (AIR)	12 (AIR)	30 (AIR)
53.4	120	———	———	12 (AIR)	12 (AIR)	12 (AIR)	30 (AIR)
44.5	100	30 (AIR)	30 (AIR)	12 (AIR)	12 (AIR)	12 (AIR)	30 (AIR)
35.6	80	12 (AIR)	12 (AIR)	12 (AIR)	12 (AIR)	12 (AIR)	30 (AIR)
26.7	60	30 (OXYGEN)	30 (AIR)	30 (OXYGEN)	30 (AIR	30 (OXYGEN OR AIR)	6 HR (AIR)
22.3	50	30 (OXYGEN)	30 (AIR)	30 (OXYGEN)	30 (AIR)	30 (OXYGEN OR AIR)	6 HR (AIR)
17.8	40	30 (OXYGEN)	30 (AIR)	30 (OXYGEN)	30 (AIR)	30 (OXYGEN OR AIR)	6 HR (AIR)
13.4	30	↓	60 (AIR)	60 (OXYGEN) ↓	2 HR (AIR)	12 HR (AIR)	FIRST 11 HR (AIR) THEN 1 HR (OXYGEN) OR (AIR)
8.9	20	5 MINUTES ASCENT TO SURFACE ON OXYGEN	60 (AIR)	5 MINUTES ASCENT TO SURFACE ON OXYGEN	2 HR (AIR)	2 HR (AIR)	FIRST 1 HR (AIR) THEN 1 HR (OXYGEN) OR (AIR)
4.5	10	↓	2 HR (AIR)	↓	4 HR (AIR)	2 HR (AIR)	FIRST 1 HR (AIR) THEN 1 HR (AIR) OR (OXYGEN)
0	SURFACE	———	———	———	———	———	———

NOTES: (1) TIME AT ALL STOPS IN MINUTES UNLESS OTHERWISE INDICATED.
(2) TIME OF ASCENT FROM 10 FEET TO SURFACE IS 1 MINUTE ON AIR UNLESS OTHERWISE INDICATED. 1 MINUTE ASCENT FROM 10 FEET TO SURFACE IS MADE ON OXYGEN FOR TABLE 4.

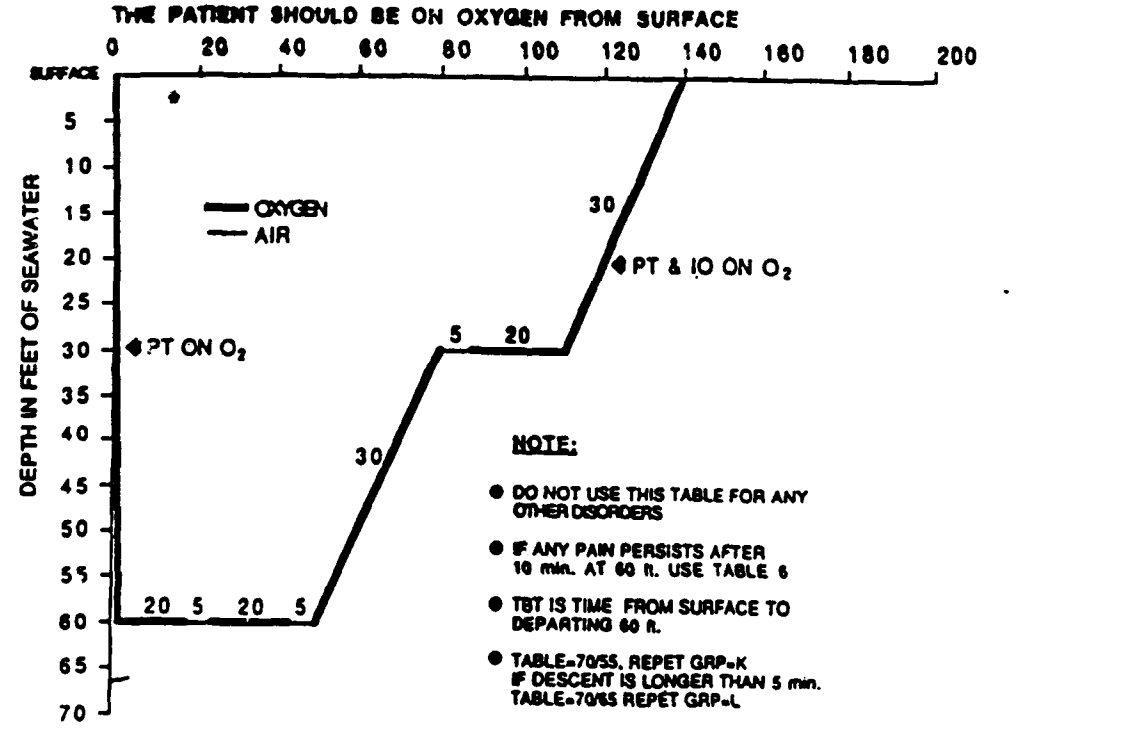
*from AFP 161-27 (5 July 1983)

Table 2. U.S. Navy Treatment Table 5



1988
XXHM-JB-3

Table 3. U.S. Navy Treatment Table 6



1988
XXHM-JB-1

Wirjosemito et al. (1989) reported the outcome of hyperbaric therapy in Type II altitude DCS cases occurring before 1 January 1987 (43). This review determined that 94.7% (126/133) of Type II altitude DCS cases that were treated with HBO therapy had resulted from altitude chamber exposures, and that HBO therapy produced fully successful outcomes in 97.7% (130/133) of the cases. Residual deficits were noted in 2.3% (3/133) of the cases, but there were no fatalities. Treatment Table 6 (as modified) was used in over 80% of the cases. The 1977 Davis report (15) was complemented by Weien and Baumgartner (1990) in a review of 528 cases of altitude decompression sickness occurring between 1 January 1977 and 31 December 1986 (42). Their findings further validated the efficacy of HBO therapy in the treatment of altitude DCS. Complete resolution of symptoms/signs occurred in 98.48% (519/527) of cases. For the period, 1 January 1987 to 30 September 1990, the USAFSAM data repository contains 121 additional cases of altitude DCS successfully treated in USAF facilities. When these unpublished cases are added, the repository will contain 649 total altitude DCS cases reported since 1 January 1977. Since HBO therapy was implemented in 1959, there has been only one fatality. A detailed account of the in-flight case of altitude DCS was reported by Neubauer et al. (34). Table 4 summarizes the success of hyperbaric therapy in the treatment of altitude decompression sickness.

TABLE 4. ALTITUDE DECOMPRESSION SICKNESS SUMMARY OF HYPERBARIC THERAPY RESULTS

REPORTER	TOTAL # OF CASES REPORTED	OVERALL OUTCOME	TREATMENT FATALITIES
DAVIS, et al. (1977)	9/145 136/145	88.0% (TT* 1-4) 99.3% (TT* 5/6)	0
WIRJOSEMITO, et al. (1989)	130/133 (Type II Only) in text	97.7% (TT* 6)	0
WEIEN, BAUMGARTNER (1990)	519/527	98.5% (TT* 5/6)	0

*TT = Treatment Table

CONCLUSION

Clearly, altitude DCS continues to occur in unpressurized operational flight or during exposure to reduced pressures in altitude chambers. Even though preventive measures such as denitrogenation are used, aviators will continue to experience this disorder. Since 1959, there has been a growing recognition and understanding of the

pathophysiology of the disorder, refinement of appropriate treatment regimens, enhanced educational efforts on behalf of the aviator and clinician, and increased availability of hyperbaric chambers to adequately treat this disorder. Further, the success rate for HBO therapy is better than 98%, particularly if the patient presents to the hyperbaric facility within 2 hours of onset of symptoms. All of these factors, have contributed to firmly establishing the use of HBO therapy as the standard of care for altitude decompression sickness.

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COLONEL SHEFFIELD: During the break we will show a very brief film which was used to encourage AF pilots to report their cases of decompression sickness. When the hyperbaric chambers came on-line in the 1960s, the facilities were there, but no one came. As a result this film was made to try to encourage, through education, the pilots to come in and report their symptoms of decompression sickness. Early in the hyperbaric program there was a restriction such that if you had a case of bends, and you reported it, you would probably be grounded. As a result there was a resistance of the crew members, as well as our own technicians, to report decompression sickness. And those of us who are older and gray-haired can verify the absence of reporting in our own cases. In the mid 1970s, we had an Air Force Academy outbreak of decompression sickness, and at that time the Commandant found it unacceptable to ground his cadets. As a result, a review of the policy occurred and Type II decompression sickness then was allowed as a waiverable event. Since about 1978, Type II decompression sickness, or central nervous system decompression sickness, has been waiverable. Currently in the USAF, bends (Type I) is treated effectively with hyperbarics. The local flight doc will return the person to duty 72 hrs later. If it's a central nervous system hit, a waiver is required from the Surgeon General's Office. The Air Force's Surgeon General has been quite liberal in return of Type II decompression sickness to flying duties.

LT CMDR CLARK: Would you treat someone who developed DCS symptoms at altitude but resolved upon reaching ground level with hyperbaric recompression?

COLONEL SHEFFIELD: If there's a chamber on site, the policy is they treat the person in the hyperbaric facility. If there's not a treatment chamber on site, then the policy is to put them on oxygen for a two-hour observation period and arrange for aircraft flight to the facility enroute. If the problem clears up in the meantime, then the transfer is cancelled.

LTC WORKMAN: If the pain recurs after resolution during that two-hour period, then transportation and treatment are continued.

COLONEL SHEFFIELD: That treatment also occurs at the site of the chamber. If it's a recurrent case, and the symptoms clear, or resolve during the transportation to that chamber, the patient is treated upon arrival at the facility.

DR. PILMANIS: Colonel Workman, can you relate the unpublished evaluation of Type II cases done at USN (EDU) a few years ago?

LTC WORKMAN: I do not recall the exact numbers. There were about 3,000 cases of Type II diving DCS that were evaluated for recurrence. The results showed that there was no statistically significant increase for recurrent Type II DCS among the Navy diving population.

LT CMDR CLARK: As I understand it, the only restriction to returning a diver to diving duties after being bent is the presence of residual symptoms. In serious cases, they're usually taken off diving status for five months. They are given that time period to resolve and are then re-evaluated. That brings to light whether we should consider a similar scenario for altitude DCS in our aeromedical disposition.

DR. VANN: I have a question concerning the USAF category of peripheral nervous system (PNS) symptoms, as opposed to CNS. Could a physician here give us a little background on that and a little more information?

MAJOR WEIEN: PNS grew out of the USAF's Type II DCS grounding policy. The general feeling was that PNS symptoms were less severe than CNS symptoms. An administrative category of Type I peripheral nervous system was created in order to avoid the waiver problem for the aviators that might get a little numbness or paresthesia in a limb. I think most of us would consider that a Type II, but administratively the USAF calls that a Type I-PNS.

DR. LAMBERTSEN: There doesn't seem to be any good medical reason for this category.

DR. VANN: Because it affects one's career, is it a reasonable category for getting people to report? Bends has to be decriminalized. Is PNS a way to do that?

DR. FRANCIS: Did those that you sent back to flying with the PNS classification recur?

MAJOR WEIEN: Not in my group of 528.

DR. FRANCIS: Fine, well why not call them Type II and send them back to work?

MAJOR WEIEN: Because the USAF at one time said, "Type II DCS. You're grounded."

DR. VANN: There's the problem.

MAJOR BISSON: I was working as a line flight surgeon during some of that time period. Occasionally we would have someone that had pain in an arm and then complained of weakness. Someone called it " neurologic," and really the weakness was due more to the physiologic effects of the pain than anything neurologic. In the USAF, the reasoning was that we can't destroy this guy's career just because he had a

little bit of pain and weakness in an extremity. Thus it was an administrative solution; it was not practical to lose a valuable USAF member because of some minor findings.

DR. FRANCIS: I think there's one other observation that may be pertinent and that is that classifying decompression illness as Type I and Type II may not be entirely helpful.

DR. BAGIAN: If I were an aviator and reported Type II and I resolved, would I be glad I did it or not?

COLONEL SHEFFIELD: I would say that you've got a better than 50% chance you'll return to status. I can't give you the numbers today.

DR. BAGIAN: Then it's not just like a rubber stamp?

COLONEL SHEFFIELD: No. As a matter of fact, two weeks ago an individual was grounded because of a repeated series of decompression sickness cases. This person was effectively treated, but yet had 3 cases in 12 exposures. As a result the Surgeon General chose not to return that person to flight status. The extremes will not be returned to flight status.

DR. BAGIAN: How about your one-time, U-2 or TR-1 pilot that comes in, has an incidence of bends, or Type I?

COLONEL SHEFFIELD: There have been waivers granted to TR-1 pilots with Type I. Later, the local flight doc came back and said these pilots also had mottling of the skin. The ruling was changed to ground or return to another weapon system. It was a Type I, but it was felt that the mottling of the skin was a very serious thing for these pilots who are going to be exposed deliberately to 29,000 ft cabin altitude.

1990 Hypobaric Decompression Sickness Workshop

Session Two: PREDICTION AND PREVENTION

R.W. Hamilton, PhD, Chairman

THE BASICS OF PREPARING DECOMPRESSION PROCEDURES

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Abstract

Most practical decompression computation methods to date use derivatives of the method originally developed by J.S. Haldane. This method considers the body to be divided into a number of hypothetical compartments which take up and release inert gas in an "exponential" manner; each compartment is defined by its rate of gas transport, its half time. A decompression table computation determines the gas loading in each compartment at the beginning of decompression in terms of partial pressure of the inert gases present, and this depends on the time, depth, and gas exposure of the dive. On ascent the inert gas loadings in the compartments are compared with empirically determined limits, and decompression is periodically stopped or slowed until enough gas is carried away to reduce the theoretical loading to the predetermined limit. Gas partial pressure ratios or differentials are used as limits, the latter being called M-values. Accepted limits are actually determined by a tolerable bubble load or an acceptable risk of decompression sickness, regardless of the parameters calculated. Many other computational methods exist, but most are based on inert gas, and most use predetermined limits to control ascent; some use bubble size or number. One common trait shared by all methods is that they rely on experience from previous dives to set the computational parameters and ascent limits.

Introduction

This workshop is addressing decompression, an environmental stress long associated with aviation and altitude; much of the basic physiology concerning the mechanism of action of decompression and the resulting decompression sickness (DCS) has come from work with altitude. Paralleling this development has been an equally productive field of study—physiology, medicine, and technology--concerned with diving and compressed air work. From the latter field have come some techniques that have proven helpful in managing human exposure to decompression. This paper describes the most important methods for preparing decompression procedures—decompression "tables." It focuses primarily on exposures to elevated pressure followed by reduction back to atmospheric pressure; the principles are valid for altitude exposure as well, but the sequences are different. The paper does not pretend to teach anyone how to prepare decompression tables, but it attempts to introduce the processes used and some of their strengths and weaknesses.

A few important principles should be mentioned. First, an exposure to increased pressure leads to an uptake by the body of excess inert gas. If pressure reduction—often called "ascent"—is slow enough the extra gas can be eliminated uneventfully by the lungs. The big question is, how slow is slow enough. When asked for help by someone who wanted to go diving in a Tibetan lake at 5000 meters altitude (16,000 feet), after some deliberation on how an existing procedure might be extrapolated I finally had a quite serious discussion with him based on my request, "Why don't you just ascend very slowly?" He did, and it went well.

Second, after more than a century of looking, researchers still have not been able to pinpoint either the mechanism of DCS "bends" or the true physiology and biophysics of decompression. We do know a lot, and we are learning more every day. We have some biological/mathematical models based on physics and physiology, but even with the best of them we still have to make some educated guesses (SWAG's: this might be a Scientific Way At Guessing). What we do know for sure is that the problems due to decompression are the result of gas phase separation, or stated another way, the formation and growth of bubbles. The task of a decompression table is to prevent bubble formation and growth, or, since total prevention may not be practical, at least to keep the number and size of bubbles at a tolerable level. What is tolerable, of course, may be one of the guesses; this has to be determined by experience.

As support for our informed guesses, we draw on previous experience. At present the most important principle of all methods of decompression table development is that it is an empirical process. The computation serves to apply yesterday's experience to tomorrow's dive. As models get better we may one day be able to rely on them to account for all the many variables, but for the present and near future the decompression table developer must rely on experience. What works, works.

One more principle, then a couple of warnings. As mentioned, the task is to control the release of inert gas. The process of defining the necessary "slow ascent" is generally based on the inert gas component of the gas being breathed. The nature of the inert gas has some definite effects on decompression. The shape of the ascent profile (within limits) has some relatively minor effects, as does accounting for the carbon dioxide and water vapor in the lungs. But the overwhelmingly most important aspect of creative decompression is the judicious use of oxygen. At high levels and within temporal limits oxygen probably does act somewhat as an inert gas (Berghage and McCracken, 1979; Thalmann, 1986), but within the exposure ranges easily tolerated it can be ignored and the decompression based on the inert gas alone. Justification for this position has been described well by Vann (1989), and verified in careful experiments by Weathersby, Hart, and colleagues (1986). Dr. Lambertsen has been trying to tell us this for nearly 50 years.

Thus a secondary aspect of preparing a decompression table is to manage the exposure to the resulting hypoxia.

The first warning applies to anyone seriously involved in decompression research at the computational level. Do not take the numbers too seriously. Brian Hills called this "computer narcosis." When one is able to manipulate profiles and account for many of the operative variables it is easy to get lulled into believing that the numbers are real. One might modify a table by "reducing the inert gas in the 5th compartment" or some such, and the modification may indeed improve the table, but it is misleading to believe too steadfastly that this is what is really happening.

We seem to be bouncing between, "What do we care as long as it works," and, "If you don't know what you are doing then it is potentially dangerous...."

The second warning concerns the nature of DCS. No matter how conservative the profile, if a person has been exposed to anything other than a trivial decompression, there is a chance of getting DCS. Both those doing the computing and those doing the decompressing should plan for it.

The Haldane Model and Its Descendants

The most famous early report of bubbles at reduced pressure was by Robert Boyle, but the grandfather of aviation physiology, Paul Bert, did not believe that bubbles formed on ascent to altitude. It was the respected respiratory physiologist and our patron saint, John Scott Haldane, who derived the first practical method of calculating a decompression table, early in the 20th century. His method has been the basis for almost all existing decompression tables. It is worth reviewing the development of this method, because it shows the principles, and because with the right relation to experience and some good SWAG's, its derivatives can be made to work. We are concerned here more about an understandable description of the method than about historical accuracy or computational precision. The Haldane method consists of accounting for gas uptake in the body, and controlling outgassing during ascent based on empirically determined limits.

An early Haldane contribution was on how to use the goat as a decompression subject. Using goats, he learned that after a "long" exposure an absolute pressure reduction to half the previous level could be tolerated, for example from 8 to 4 atm absolute (abs), or from 4 to 2 or from 2 to 1 atm abs. This was a pressure ratio of 2:1; he used total pressure, but others point out this is a reduction of 1.58:1 when only the inert gas is considered. Haldane actually used more finely tuned ratios for his tables.

Haldane knew that blood picked up gas in the lungs and delivered it to the tissues, and vice versa. This process is "perfusion." Diffusion also has to take place; physiologists have long argued the question of whether perfusion or diffusion is the limiting factor in gas transport. The current consensus leans toward perfusion, but this has little bearing on the validity of models using either diffusion or perfusion mathematics.

Once blood delivers gas to the tissue, the level in the tissue is raised. This "level" is best described as the partial pressure of the gas, but this interacts with solubility, gas properties, temperature, diffusivity, and numerous other parameters. The partial pressure of a gas is the fraction of the total pressure made up of that gas, calculated as PP = F * TP. Fraction is (% / 100).

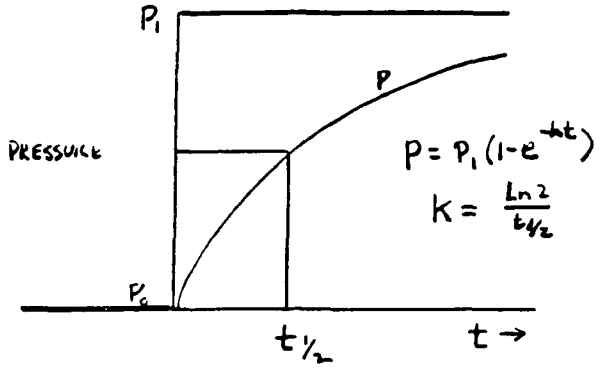

Figure 1. Exponential change in a gas partial pressure. Partial pressure P in a compartment changing in response to a change in ambient pressure from P_0 to P_1.

The Haldane approach considers that gas loading in the tissue builds up on exposure to a new ambient pressure in proportion to the differential pressure. Another way of saying this is that the process is exponential. This is common in physical systems, a good example being radioactive decay. As the process proceeds and the pressure in the tissue approaches that of the new pressure the rate of increase slows down. The shape of the uptake curve is described by its half time, the time it takes to change halfway to the new pressure. This relation is shown in Figure 1 and described by the equation shown there. The curve is gas partial pressure in the tissue as it builds up with time following a sudden exposure to a new pressure, P_1. The half time is shown. The e is a mathematical constant and the half time is included in the k. The equation gets more complex as other factors are considered, but the basic idea is here.

It was clear to Haldane that the entire body does not handle gas with the same kinetics. He considered the body to be made up of 5 tissue compartments, each described by its half time. These are still called "tissues" by some, but it is better to call them compartments to reduce ever so slightly the tendency to believe that this is what is really happening at the physiological level. A compartment with a given half time is whatever part of the body that handles gas in that manner. Haldane determined the half times of his compartments on the basis of estimated physiological blood flows, using 5, 10, 20, 40, and 75 minutes as the half times. For the record these proved to be inadequate to describe all but rather short dives. Current workers use as many as 17 compartments with half times to as much as 1280 min.

When the ambient pressure is reduced, the gas is presumed to leave the compartments according to the same equations, but there are reasons to doubt that this concept of symmetry holds up. Once the pressure is reduced to the point that the inert gas partial pressure in the tissue exceeds the ambient pressure the tissue is said to be supersaturated. The lifetime of supersaturation in living tissue is probably short in most situations; even very low supersaturations (a few cm of excess pressure) can lead to bubble formation after long times (hours).

The final step of the Haldane process consists of doing bookkeeping on the buildup of gas in the body as a result of an exposure to pressure (which in the real world involves many factors in addition to just pressure), then to set limits on the gas loadings allowed as ambient pressure is reduced. Haldane used ratios as ascent limits, using the 2:1 as a guide but refining each of them according to experience and some good SWAGs. The limiting ratios are depth specific, such that a complete set of limits involves all compartments and all depths at which stops are likely. During ascent a diver is required to stop at each depth until the calculated gas ratios in all the compartments are below the predetermined limits. Haldane's tables were conservative for short dives and inadequate for long ones, with a major difficulty being the short half times which he had determined by blood flow rather than diving experience. A good review of Haldane's work is given by Hempleman (1982).

U.S. Navy Experience and Workman's M-values

The U.S. Navy picked up on these methods, preparing tables soon after Haldane and doing a great deal of work in the 1930's. Many of those workers are more than familiar names, but also colleagues some of us have known well, such as Chuck Shilling and Al Behnke. USN increased the compartment halftimes to include 120 min, used partial pressures of inert gases, and added the benefit of more experience. The U.S. Navy air tables done in 1956 are still reliable for short dives, but have never been really adequate for the deeper and longer dives >40 meters of sea water (msw)/130 feet of sea water (fsw) and >30 min, or with decompression times >60 min).

The next USN procedural change was introduced by Workman (1965). He noted the difficulty with deeper dives imposed by the ratio method (although with the right ratios the results could be the same) and defined a method based on limits described by differential pressures. That is, the gas transport equations are the same and the loadings are expressed in terms of absolute partial pressure, but the limits are a set of empirically predetermined gas partial pressures, a separate one for each compartment and each depth during ascent. These partial pressure limits are called M-values, the "M" standing for "maximum." A set of these limits is sometimes called a "matrix." On ascent the current gas loading in each compartment is compared, at each selected stop depth, with the M-value. If the loading in all compartments is less than the appropriate M-value then ascent is made to the next stop depth and the comparison is now made with those M-values. Stop depth intervals are usually 3 msw or 10 fsw. When the 3 msw stop is clear in all compartments the diver ascends to the surface.

A matrix of M-values can be determined empirically for each cell, or if its shape is smooth it can be generated by a set of linear equations, one for each compartment, again generating a separate M-value for each cell.

Workman's method is widely used, has been used for heliox as well as air dives, and is still taught to USN diving medical officers (Flynn, et al., 1981). It has even been programmed to work on a spreadsheet program (Short and Flahan, 1989).

Schreiner's Multiple Gas Modification

Heinz Schreiner (Schreiner and Kelley, 1971) added another trick. Workman's method could work with different gases (which require different M-values) but could not easily work with more than one at a time. Schreiner considered that all gases in a given compartment add up to a total sum of inert gases, and his method compares this sum to the M-value. The different inert gases might have different half times in each compartment, but all gases in each compartment contribute to the sum. This enables one to use as many inert gases as one cares to bother with.

Schreiner's paper describes a set of multigas half times based on measured blood flows. Like Haldane's, they have been modified as experience in its use developed. The half times are not realistically linked to physiological blood flows.

There are some imposing physiological problems with this method and others that use multiple gases; so far they are limited by lack of sufficient data on dives with multiple gases to describe the relationships in any detail, and the data available are difficult to interpret.

Another contribution of Schreiner's is the handling of CO_2 and water vapor. His model (identified as "Tonawanda II," or the Haldane-Workman-Schreiner method) uses CO_2 and H_2O values calculated from the alveolar ventilation equation, and subtracts these from the total pressure before computing the inert gas partial pressures. These values are of small significance in diving, but play a much larger role in altitude decompression.

The Haldane-Workman-Schreiner model has performed well in a number of applications, despite its deficiencies.

Thalmann's Exponential-Linear model

It has long been known that when the M-values (or ratios) are adjusted to make the longer, deeper dives reliable it often messes up the shorter profiles that are working fine. Ed Thalmann, in an extensive program performed at the USN Experimental Diving Unit, developed a method of reducing the rate of outgassing to better match the experimental data (1983; 1985; 1986). He assumes that bubbles form as soon as supersaturation develops and the exponential kinetics no longer apply. An empirically determined linear outgassing pattern (based on physiological

principles) slows down ascent in a way compatible with dive experience. One important aspect of this model is that the experimental program was dedicated to validation of the model, not a specific set of tables. Although the E-L model is effective, the Navy's interests have turned to other data-related, statistical approaches to decompression computation.

The overwhelming proportion of recreational dive computers has been programmed with variations on the Haldane model. The variations include the definition of the compartment half times, the ascent limiting M-values, and various methods of defining the gas. Since dive computers tend to make every dive "worst case" in terms of the exposure, they necessarily have more conservative algorithms than comparable tables. Dive computers achieve their efficiency by enabling a diver to work at several levels during the same dive, without having to calculate it as a "square" dive as would be required by tables. A mechanical (nonelectronic) "wheel" designed by Rogers (Rogers and Powell, 1988) uses a clever presentation of Haldane methods with new M-values, but is not really a new approach; its stratagem is to base repetitive dives in the recreational dive range (no-stop, <40 msw) on the 60 min compartment rather than on the 120 min compartment which is used for the USN repetitive tables.

Other Haldanian or Neo-Haldanian Applications

A workshop organized by Berghage (1980) examined a number of tenets of the Haldane method. Some of these were moot, such as the question of how much supersaturation can be tolerated without bubble formation. Probably very little; most current workers realize that it is a tolerable bubble load, not initial bubble formation, that is being titrated in decompression development. Another point made at that workshop was that if there are enough variables that can be adjusted, then just about any model will do.

Thalmann's efforts were to allow longer decompressions where needed but not to restrict tables that were working. Another approach was taken by Swedish Navy researchers, who selectively used empirically determined conservatism factors for the deeper dives (Hamilton, et al., 1988). Still another approach is taken by Peterson and Hamilton (1987), whose "t-delta-P" algorithm considers that the longer a decompression lasts the more likely DCS is to occur; he uses a factor based on the integral of differential pressure over time to further slow down a long decompression.

Another Haldane-related analytical method is the ratio used by NASA to assess Extravehicular Activity decompressions. This is a ratio of the absolute partial pressure of inert gas calculated to be in the 360 min compartment to ambient pressure (Waligora et al., 1984). It is hardly necessary to mention that it is used empirically.

The Kidd-Stubbs Model

A fresh approach to decompression gas management is taken by the Kidd-Stubbs model used at the Canadian DCIEM and for their tables (Nishi and Kuehn, 1973). It began as a pneumatic analog having 4 small chambers connected in series with tiny orifices (drilled sapphires). Successive chambers filled from and emptied to their neighbors; only the first one was exposed to ambient pressure and gas. The device was originally patterned after the USN tables. It worked, but quality control problems limited its use. The pneumatic device did not prepare a "table" as such, but controlled the diver's ascent. An electronic analog of this model developed at DCIEM and refined by Nishi allows the parameters to be controlled and tables to be calculated. The Kidd-Stubbs model uses numerical techniques to solve the complex orifice flow equations for the gas loadings, but like Haldane it uses partial pressure limits to control ascent; only the first two compartments have any effect, although all four are needed to maintain the gas behavior. This model has been used in producing a set of air tables that has proven to be quite successful, and heliox tables are in the works (Nishi and Lauckner, 1984). The approach used in formulating the air tables was to adjust the parameters so as to ascend more conservatively where the standard USN tables had been observed to be inadequate.

Bubble and Other Models

A number of decompression models use bubble formation and/or growth to limit decompression; there are too many complexities to this approach to include here (for reviews see Vann, 1982 or Wienke, 1989). Many of these use gas loadings calculated the Haldanian way to determine the environment for bubble formation, but let bubble parameters set the limits (Yount and Hoffman, 1986). Wienke's new nucleation model (prepared for a dive computer) addresses some situations not treatable by Haldane methods, such as the observation that "deep before shallow" appears to have a higher risk than a similar set of dives done in the reverse order (Wienke, 1990); the output of these computations are implemented as multipliers of M-values.

One important aspect of bubbles is the observation that microscopic nuclei appear to exist in the body. Bubbles need some sort of nucleus to form at the pressure differentials involved in human decompression. The behavior of bubble nuclei has a big effect on bubble formation and decompression success.

There are yet quite a number of other computational models using slab diffusion, the square root of time, critical volume, thermodynamics, and many other approaches. Most of these models share the common practice of making gas calculations and limiting ascent when certain parameters are reached or exceeded. A few of the non-Haldanian ideas have become validated, useable tables, but most of them remain as proposals.

Decompressions from saturation dives may occasionally be calculated by the methods discussed, but because the ascents are often linear, a straightforward and highly effective method—provided there is a good experience base—involves graph paper and a bit of knowledge about oxygen levels and the nature of the gases in use.

Some Complications

Although some models may approach physiological reality in a single cell or tissue or test tube, the real world involves many complications. Some of these complications are the variation between individuals, exercise levels, and temperature, and other factors that regulate blood flow in body tissues such as the oxygen level. There are also operational complexities such as repetitive dives and surface decompression. A repetitive dive is one performed soon enough after an earlier one to be affected by it. Surface decompression ("Sur-d") involves a diver ascending to the surface before decompression is complete and finishing the decompression in a deck decompression chamber, usually breathing oxygen. The algorithms for repetitive diving and sur-d are not nearly as well developed as those for plain dives. They too rely heavily on empirical SWAGs.

Conclusion

Decompression table computation remains an empirical art. It abounds in scientific inputs and techniques, but ends up largely as the judicial use of available experience. With a suitable base of prior experience, many types of models can be used effectively.

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DR. FRANCIS: You are aware of the work which appears to throw doubt on the validity of exponential tissue gas wash in and wash out. How does that alter the way in which you would compose a table?

DR. HAMILTON: I have not tried to use that information in my work. There are different ways to look at the problem. Early experiments showed that gas behaved exponentially. Some of the curves described earlier today showed similar behavior. There are several other methods used. Bubble formulas are used and compared with the classical approach.

DR. WENZEL: You mentioned that there are several physiological models with which you can describe the outcome of a decompression experiment quite adequately. This does not necessarily mean that the model describes the physiology.

DR. HAMILTON: That is right. However, it may be a usable technique and could be adjusted to make it fit the experience.

DR. WENZEL: These Haldane-Shreiner-Workman models you used. They have about 20 to 50 adjustable parameters. This should really be enough. In fact, with modern computers you can use 1,000 or 10,000 parameters as you like. If you want to increase the number of tissues, you are free to do so, but it does not necessarily improve your description.

DR. HAMILTON: As you add more compartments, you reach a point of diminishing return. But people still do it.

DR. LAMBERTSEN: The very important work that Haldane did, which opened the window to understanding decompression, dealt primarily with the uptake of gas in various compartments, or the elimination of it, but not with anything that had to do with formation of bubbles. Taking gas up does not make bubbles. Making it come out does not make bubbles. Something else makes bubbles. Thus, all of the computer programs start off with something that has really nothing to do with the primary problem, the problem of creating bubbles in certain places in the body. Next, individuals have found that gases did behave exponentially, as Haldane said they should, and you could separate out theoretical compartments and do it by actual measurement. That still had nothing to do with the formation of bubbles. That is why people started talking about other factors involved in the generation of bubbles. The blending of these processes is what makes decompression complicated. The part that deals with the formation of bubbles, we do not really know, we cannot calculate where it is happening. All we can do is run the gas in and run it out and do that over and over and over again without regard for the fundamental process of what is making the bubble happen in the unknown regions.

MR. GILBERT: We have a tendency to confuse the physiology with a mathematical model. A mathematical model describes what we have seen in experimentation. It does not, or should not, attempt to describe the physiology behind what the results show. If it happens to coincide with certain theories on the physiological changes occurring, then there are ways of proving that. In that case, the mathematical model does describe the physiology response.

DR. VAN LIEW: There are two kinds of models. One type starts with physiology, or physics or basic premises, and tries to grow towards the other type, described by Mr Gilbert.

PREBREATHING THEORY AND HISTORY

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Introduction

The physiologic effects of pressure changes in the flying and diving environments are often compared and contrasted. For example, one assumption frequently made is that an altitude decompression cannot be as significant as a diving decompression since the magnitude of change in the altitude environment is much smaller, i.e., a 1/2 atmosphere (ata) total change as opposed to a 3 ata total change. This assumption has led to the opinion that altitude exposures are less significant and less harmful than diving exposures. However, Fryer (6) pointed out that the physiologic stress of decompression was not proportional to the pressure drop, but was related to the ratio of the initial and final pressures. He demonstrated that a pressure reduction from 1 ata to 1/4 ata would have the same physiologic effect as one from 8 ata to 2 ata (not 7 1/4 ata)(6). While the ratio may not be the only important factor, it is undoubtedly true that altitude decompression is physiologically important. Altitude decompression led to significant decompression injury and death before precautions were routinely observed. Prebreathing is currently the most effective preventive measure available to decrease decompression sickness (DCS) morbidity and mortality due to altitude exposures.

Only eighteen deaths due to altitude DCS have ever been reported (5,12): the number of unrecognized DCS deaths are unknown. All but one of the reported deaths occurred before prebreathing was a standard procedure for high altitude flights. Prebreathing as a means of reducing the incidence of DCS appears to have had a significant effect on mortality. Efforts to quantify the amount of nitrogen offgassing while prebreathing have led to a better understanding of DCS and have helped identify appropriate prebreathe schedules. However, even appropriate schedules do not totally eliminate the hazards of altitude DCS. This paper reviews the work leading to acceptance of prebreathing and the theory behind its use.

Classification of Symptoms

There are no standard criteria for quantifying DCS symptoms. Rather, criteria used to stop a particular flight are specific only for that study (6). Grading scales for mild as well as moderate DCS symptoms varied according to the author. For example, in 1945, Clark et al. (4) used the following scale for their decompression subjects:

1+ (mild) - signs and symptoms are noted, and there is a definite degree of discomfort.

2+ (severe) - definite discomfort which had progressed sufficiently to cause interference with performance of normal activities or duties. At this point, subjects were usually removed from the chamber.

2+ (chokes) - characterized by a cough on deep inspiration. Flights were terminated when this symptom was present.

Balke (1), on the other hand, used a simple three-point scale for his subjects: mild, tolerable, and almost intolerable pain. He did not address the occurrence of chokes. His flights were terminated for almost intolerable pain.

Other authors have likewise set their own scales, often without specifying criteria. Such fundamental differences between studies must be kept in mind when data are evaluated. Often, mild symptoms were not reported since only termination symptoms were viewed as significant. This methodology leads to confusion and makes comparisons between databases almost impossible.

Modern researchers still grapple with the problem of classification. While most flights are stopped early in the disease process, some protocols still allow development of symptoms to more severe states. This data is misleading if the reader is not aware of the specific criteria being used for termination.

Altitude Decompression Sickness Deaths

In 1917, Yandell and Henderson predicted the possibility of decompression sickness in flyers (quoted by 10). DeHart reviewed 743 serious cases of decompression sickness prior to 1959; at least 17 aviators were known to have died due to altitude induced illness (10). In 1988, an 18th case of fatal altitude DCS was reported in the open literature (12).

These 18 deaths are detailed in Table 1. With the exception of the final case in 1988, all patients died before it was recognized that the severity of DCS could be decreased by oxygen prebreathing. As can be seen, ages ranged from 22-51; symptoms included chokes, visual changes, neurological symptoms, abdominal pains, and "bends" pains; several patients had multiple symptoms. The minimum altitude of exposure was 22,000 feet for 30 minutes.

Nitrogen Washout

Oxygen prebreathing was suggested by Behnke (3) on the assumption that oxygen decreased the total body nitrogen stores and would, therefore, decrease the severity of DCS. Nitrogen washout depends on the existence of a partial pressure difference between the tissues and blood. This pressure differential drives nitrogen from the tissues and into the blood where it is delivered to the lungs and exhaled (3, 6). Consequently, there is always a drive to release tissue nitrogen stores while breathing 100% oxygen.

Table 1. Altitude Decompression Sickness Deaths.
Adapted from Fryer (5) and Neubauer et al. (12).

Number	Age	Peak Alt. (ft.)/ time (min)	Interval btwn Sym. onset & LOC (hrs)	Symptoms At Altitude					Time from first sym to Death (hrs)
				Bends	Chokes	CNS	Abd Pain	Collapse	
1	25	38,000/72	1-1 1/2		yes			Impend.	8 1/2
2	26	38,000/35	brief	yes		?	yes	Impend.	71
3	22	38,000/85	Approx 1-6			yes		Impend.	16 3/4
4	38	30,000/60 & 38,000/sec	1	yes	yes	yes		Impend.	17 1/4
5	28	37,000/120	1/4					Impend.	13-14
6	22	30,000/19	Partial 1-2		yes	yes		yes	38 1/2
7	23	30,000/23	Brief		yes			yes	55 1/2
8	33	40,000/8	Nil	yes		yes		Impend.	45
9	37	25,000/25 & 40,000/1-2	1/2		yes			yes	5 1/4
10	28	35,000/120	2 1/4	yes	?				17
11	29	30,000/55	? Brief				yes	Impend.	10 3/4
12	50	35,500/60	Nil				yes	yes	11 3/4
13	34	26,000/80 & 29,000/2 1/2	Nil			yes		yes	5 1/2
14	38	35,000/8 & 30,000/4-7	Nil		yes			yes	15 1/4
15	34	30,000/40	Nil					yes	11 1/2
16	36	22,000/22	Nil		yes		yes	yes	12 3/4
17	32	33,000/120	? 3-4	yes			yes	Impend.	9
18	51	28,000/30	7		yes			yes	7

In 1953, Lundin (11) demonstrated that nitrogen elimination could be quantified by measuring the end-tidal air with a nitrogen meter during oxygen breathing. Various individuals have predicted nitrogen desaturation curves from these measurements (2,3,9,11). These curves show that 95% +/- 2% of the total body stores of nitrogen are eliminated in the first 4 hours of prebreathe, and that by 6 hours 98% +/- 2% of total body stores are removed. Therefore, Behnke (3) predicted 5 hours prebreathe with 100% oxygen would completely desaturate tissues and give perfect protection against DCS.

As nitrogen is eliminated from the body, the pressure differential between tissues and the blood is decreased, and offgassing slows down (6). In fact, nitrogen elimination from the lungs follows an exponential curve (Figure 1). As the time of prebreathing increases, the amount of gas released from the tissues gets progressively smaller. After approximately 100 minutes, the slope of the release curve approaches asymptotic levels.

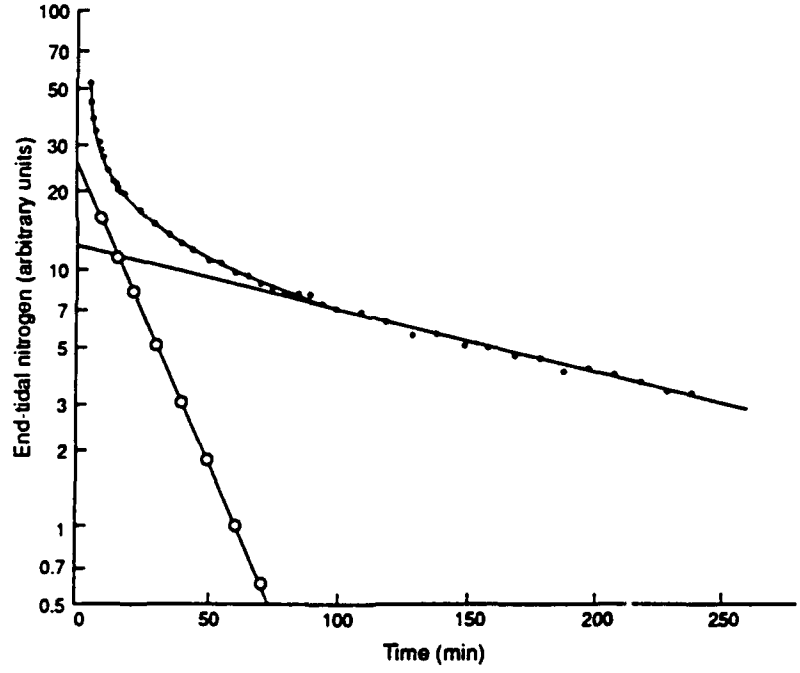

Figure 1. Tissue nitrogen washout over time while breathing 100% oxygen (11). Open circles represent fast tissue desaturation, solid line represents slow tissue desaturation, and closed circles represent total body washout times.

A second factor influencing nitrogen elimination is the perfusion rate of the tissues. As more nitrogen-poor blood passes through tissues, more nitrogen should be removed (6), but the rate of elimination varies for each tissue type. For simplicity's sake, three phases of elimination have been identified based on tissue vascularity.

The rapid phase which has an elimination half time of about 1.5 minutes and corresponds to the blood, the liver, and other highly vascular tissues. The intermediate phase has an elimination half time of approximately 12-13 minutes and represents the muscle. Finally, the slow phase has an elimination half time of 110-200 minutes and represents fat (11).

Prebreathe Requirements

Prebreathing has significantly reduced the occurrence of altitude DCS and is now standard procedure prior to high altitude exposures. Results of previous prebreathing experiments are difficult to interpret, however, due to varying definitions of "significant" symptoms.

Even without oxygen prebreathing, a certain percentage of subjects will remain at altitude for long periods and not require descent. In a study by Fryer (6), 90% of males aged 20-30 remained at 35,000 feet for 1 hour. Fifty-five percent were at this altitude for 4 hours. While not requiring descent, almost all of these subjects had symptoms (considered insignificant at the time) that would now terminate the exposure.

When subjects remained at altitude with mild to moderate pain, the natural progression of the disease was seen. Some pain resolved while at altitude. In other subjects, pain never increased to the point requiring descent. However, at the end of two hours, greater than 20% of runs were aborted. The information in Figure 2 indicates that the number of "failures" and reported symptoms increased with time.

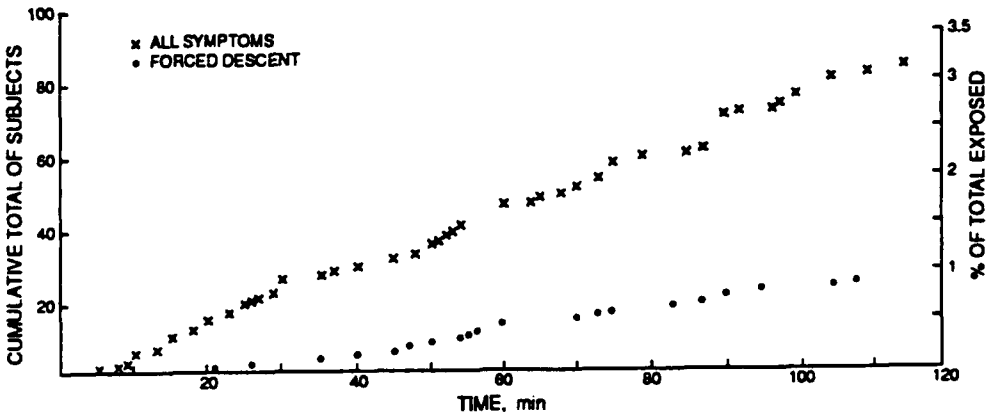

Figure 2. Development of altitude DCS symptoms over time (6).

Predictions of the degree of protection against DCS are confusing. Oxygen prebreathing does delay the onset of severe symptoms. However, the onset and occurrence of mild symptoms may not be altered by oxygen prebreathing. Thus, Gray (8) exposed subjects to 38,000 feet for 2 hours with and without a 45-minute ground level prebreathe. For the prebreathe group, the occurrence of severe symptoms was decreased by 88% for bends and 100% for chokes. However, the occurrence of mild

pain-only bends symptoms was not changed. Hence, Gray concluded that breathing 100% oxygen for 45 minutes before a two-hour simulated flight at 38,000 feet only reduced the incidence of severe symptoms (8).

Conversely, Waligora et al. (13) found that prebreathing eliminated both severe and mild symptoms when subjects were taken to 4.3 psia (30,000 feet) to simulate space flight. They determined that an 8-hour prebreathe would completely eliminate DCS symptoms and venous bubbles (as measured by precordial Doppler). This work is summarized in Table 2 and Figure 3.

Table 2. Results of Exposure to a Decompression to 4.3 psia (30,000 Feet) Simulating Extravehicular Activity (from Waligora et al. (13)).

Hours of Oxygen Prebreathe	n	% Symptoms	% Venous Bubbles
3.5	23	30	65
4.0	28	21	46
6.0	38	10	29
8.0	8	0	0

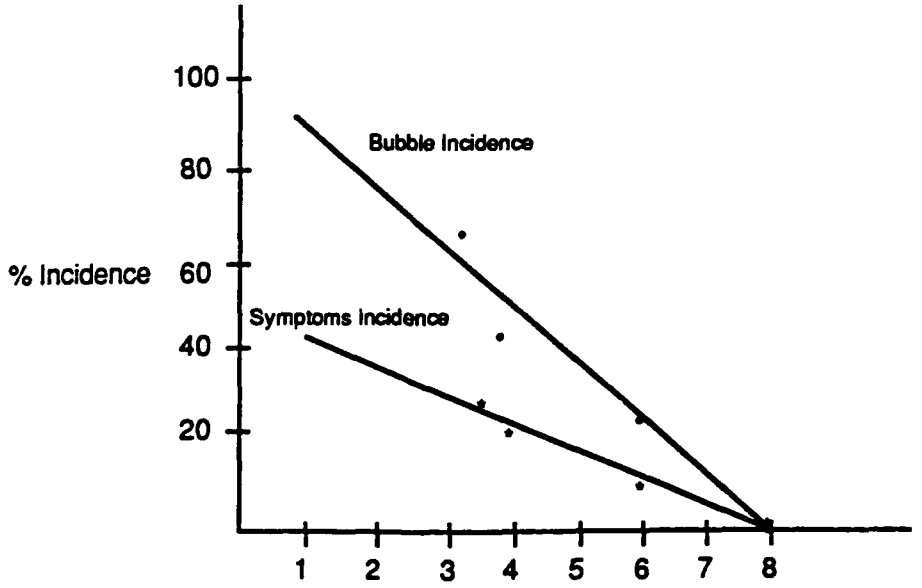

Figure 3. The incidence of venous gas bubbles and symptoms of decompression sickness during simulated EVA at 4.3 psi (from Waligora et al. (13)).

Other studies show mixed results about the ability of prebreathing to decrease mild symptoms. For example, a two-hour prebreathe followed by ascent to 38,000 feet resulted in 2 of 7 symptoms-free flights and 5 of 7 subjects being able to double their time at altitude (1) despite performing deep knee bends at altitude to provoke DCS symptoms . Forced descents occurred on an average of 24 minutes into the flight. The onset and frequency of mild symptoms were not reported, however.

Clark et al. collected data from 500 flights to 38,000 feet (4). Again, flights were not terminated unless the subject experienced severe pain. By varying the length of the preexposure oxygenation period and rate of ascent, the authors were able to demonstrate a significant improvement in ability to tolerate exercise at 38,000 feet. Despite these added measures, protection was not universal and there was great deal of intrasubject variability. Onset of symptoms was delayed as prebreathe times increased. These data are summarized in Table 3.

Table 3. Protection Against Decompression Sickness: Data from Clark et al. (from Bateman (2)).

Hours O2	No. Subjects	No. Exper.	Age (ave)	Conditions of Test			Onset of Symptoms (min)	
				Altitude (feet)	Rate of Ascent (ft./min)	Time at Altitude (hrs)	Grade 1	Grade 2
0	6	18	28	38,000	5000	1.5	33	40
1	6	18	28	38,000	5000	1.5	71	76
2	6	18	28	38,000	5000	1.5	100	100

Altitude Thresholds

Bateman (2) developed the concept of threshold decompression altitudes by calculating tissue half times for both slow and rapid phases (Tables 4 and 5). He predicted the nitrogen load remaining in both slow and fast tissues after oxygen prebreathe; he then determined the maximum altitude a person could be exposed to and expect DCS protection based on both tissues' nitrogen loads. This method predicted that a 2-h prebreathe of 100% oxygen would provide complete protection up to an altitude of 36,419 feet. Only after exceeding this level would the nitrogen differential between the body and the ambient environment be high enough to cause bubble formation.

Table 4. Threshold Decompression Altitudes for Bubble Formation in Slowly Desaturating Tissue Following Breathing of Pure Oxygen (from Bateman (2)).

Time Spent Breathing Oxygen at Sea Level (Hours)	Fraction of Initial Nitrogen Remaining	pN2 (mmHg)	Equivalent Total Pressure for Person Breathing Air (mmHg)	Threshold Decompression Pressure (mmHg)	Threshold Decompression Altitude (feet)
0	1.000	573	760	266	26,263
0.5	0.908	520	697	240	28,630
1.0	0.820	470	635	208	31,765
2.0	0.672	385	528	167	36,419
2.5	0.608	348	482	150	38,800

Table 5. Threshold Decompression Altitudes for Bubble Formation in Rapidly Desaturating Tissues Following Breathing of Pure Oxygen (from Bateman (2)).

Time Spent Breathing Oxygen at Sea Level (Hours)	Fraction of Initial Nitrogen Remaining	pN2 (mmHg)	Equivalent Total Pressure for Person Breathing Air (mmHg)	Threshold Decompression Pressure (mmHg)	Threshold Decompression Altitude (feet)
0	1.000	573	760	182	34,616
0.2	0.817	468	632	139	40,261

Interrupted Prebreathe Periods

In an effort to make the prebreathe period more convenient and still protect flyers from DCS, attempts have been made to interrupt the prebreathe period. Gibson and Manning (7) believed that oxygen breathing during sleep would provide protection even if the aviator went about daily activities on ambient air. Furthermore, no additional prebreathe would be needed prior to flight. Some investigators have had subjects breathe oxygen for 8 hours, followed by as much as 5 hours of air breathing prior to altitude exposure. Gibson exposed 13 subjects to 35,000 feet for 3 hours on this prebreathe schedule. After a total of 65 exposures, he found he could extend the time at altitude for a group of "bends susceptible" people from an average of 126.7 minutes to an average of 173.4 minutes. The subjects descended once symptoms became severe. After the flight, he noted significant side effects which he attributed to oxygen breathing, including dry cough, fatigue, "oxygen bends" (sudden pains which came on when oxygen was breathed at ground level day after day), chest pain, irritation of the throat and trachea, and nosebleeds. In addition, he noted that all

subjects had "residual pains" after exposure ranging from transient-mild to persistent-uncomfortable. It is unclear if these residual pains were considered to be due to oxygen or to altitude exposure.

Another study found that a 3-hour oxygen prebreathe followed by 90 minutes of air breathing was equivalent to 1 hour of uninterrupted denitrogenation (1). Finally, Bateman (2) listed the following as being equivalent to a continuous 2 1/2-h prebreathe:

4-h of oxygen followed by 1.7-h of air breathing
6-h of oxygen followed by 2.9-h of air breathing
8-h of oxygen followed by 3.6-h of air breathing
10-h of oxygen followed by 4.0-h of air breathing.

A summary of Clark's work in interrupted prebreathing appears in Table 6.

Table 6. Protection Against Decompression Sickness: Effect of Inhalation of Oxygen Interrupted by Breathing of Air Before Ascent. (Data of Clark as Presented by Bateman (2)).

Hours O2	Hours air	No. Subjects	No. Exper.	Age (ave)	Conditions of Test			Onset of Sym (min)	
					Altitude	Rate of Ascent (ft./min)	Max. Time at Altitude (hrs)	Grade 1	Grade 2
9	4	6	6	28	38,000	1267	1.5	83	87
0	0	9	18	27	38,000	1267	1.5	57	62
1	1.5	9	18	27	38,000	1267	1.5	63	70
2	1.5	9	18	27	38,000	1267	1.5	90	97
3	1.5	9	18	27	38,000	1267	1.5	110	112

Staged Decompression

Staged decompression is used during diving decompression and the same concept can be applied to altitude. The space shuttle provides the most recent example of such staged "altitude" decompression. Prior to spacewalks, astronauts prebreathe 100% oxygen for 2 hours, then the cabin pressure of the shuttle is lowered from its normal sea level pressure of 14.7 psia to 10.2 psia (about 9,750 feet). A 28%/72% oxygen/nitrogen mix is used for 12 hours at this pressure. For 40 minutes just prior to the extravehicular activity, the astronauts return to breathing 100% oxygen. Finally, they don their pressure suits and depressurize to 4.3 psia (30,000 feet) for exit from the orbiter. By using this schedule, a long prebreathe with 100% oxygen while in the spacesuit is avoided. To date, there have been no reported cases of DCS in shuttle crews.

Staged decompression works because "silent" bubbles are not considered to be significant until altitudes above 20,000 feet are reached (2). Prebreathing at altitudes below this level will denitrogenate tissues without causing symptoms. If oxygen breathing is added at these altitudes, greater protection is achieved. Preoxygenation at altitudes above 20,000 feet seems to be less effective than below 20,000 feet (2): Fryer showed that denitrogenation at 8,000 feet provided better protection than it did at 20,000 feet. (5) The results of the Fryer and Canadian work are shown in Figure 4 and Table 7.

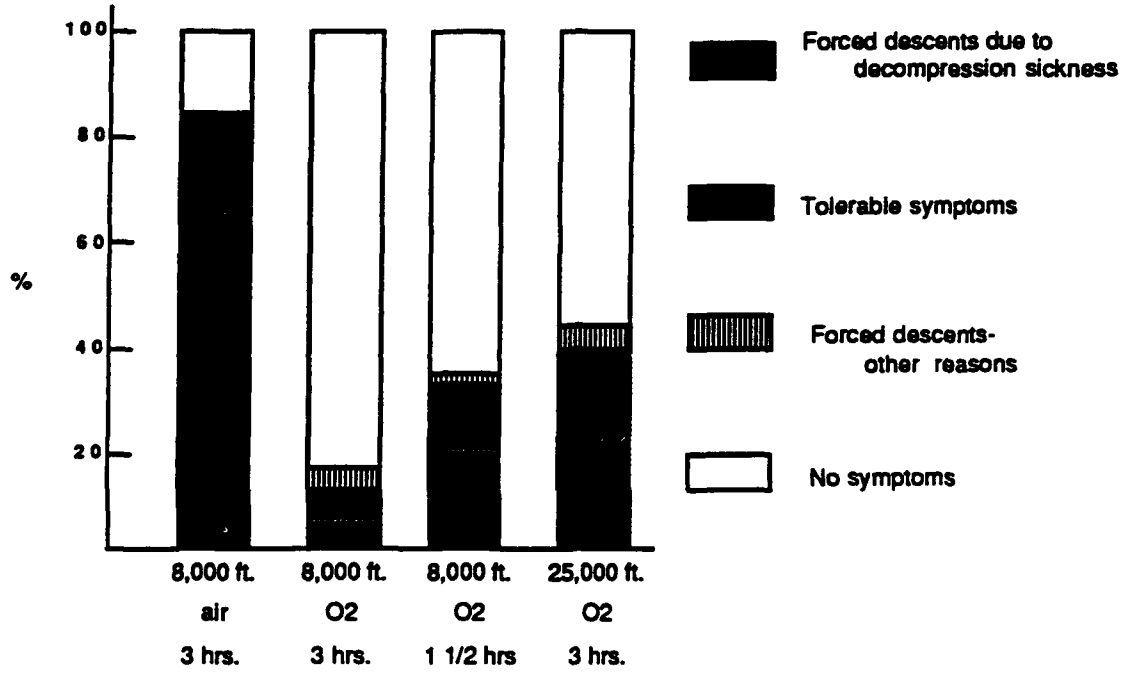

Figure 4. Comparison of the effectiveness of the four forms of denitrogenation (5).

Table 7. Effect of nitrogen elimination at various altitudes upon the incidence of decompression sickness (from Bateman (2).

Altitude of Stage Decompression (feet)	Time (Hours)	Final Altitude (feet)	Effect
20,000	2	30,000	Symptoms not prevented
30,000	4	35,000	Symptoms not prevented
--	--	35,000	45% moderate and severe symptoms
10,000	1	35,000	12% moderate and severe symptoms
20,000	1	35,000	21% moderate and severe symptoms
--	--	35,000	40% moderate and severe symptoms
27,500	1	35,000	28% moderate and severe symptoms

Conclusion

The incidence of severe DCS symptoms has decreased dramatically with the use of oxygen prebreathing. But the problem has not been eliminated. As reported elsewhere in these proceedings, significant numbers of DCS cases during training chamber runs are still seen in the U.S. Air Force and U.S. Navy, and the reports of altitude DCS during operational activities are less numerous, but may be under-reported due to career concerns. Various prebreathing schedules have been shown to be effective in decreasing the incidence of severe symptoms, but the ability of the prebreathe to alter the occurrence of mild symptoms is still in question. Continuous prebreathe periods, interrupted prebreathe periods, and staged decompressions all provide protection against DCS; however, routine protocols for their use have not yet been established. Furthermore, inconsistencies in the reporting of DCS symptoms have made it impossible to understand the impact of these methods. Further research to clarify these problems is clearly indicated and is currently underway in many centers.

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CAPT GARDETTO: Currently, in high altitude parachuting above 18,000 feet, the regulations require prebreathing be done at all altitudes below 10,000 feet. That allows for a lot of leeway for the aircraft commander. Should we prebreathe on the ground before taking off? Should we prebreathe at altitude on the way?

DR. STEGMANN: We are currently completing a study for USAF SAC on "in-flight" denitrogenation. The preliminary results indicate that there is no significant difference in bends/bubbles incidence between prebreathing at ground level and prebreathing at altitudes up to 16,000 ft.

DR. BALLDIN: Your conclusion that an interruption in prebreathing doesn't substantially affect the incidence of decompression sickness seems to contradict the view of the space medicine community which has reported a tremendous DCS impact.

DR. STEGMANN: My understanding of that work is that the nitrogen on-gassing increased significantly after prebreathe was broken with even one breath. But I don't believe that the incidence of DCS was looked at. It's difficult to review since the work was not published. According to the papers I reviewed, denitrogenation can be interrupted, up to 5 to 10 minutes, without increasing the incidence of DCS. It just does not appear to significantly increase risk.

COLONEL SHAFFSTALL: Isn't there a time factor involved? Are you talking about long denitrogenation times?

DR. STEGMANN: The longest denitrogenation time Cooke's study had was 3 hours. A 10-minute interruption in a 3-hour protocol did not change their DCS incidence.

COMMANDER BASON: But we don't know what a 10-minute interruption would do for a 30-minute prebreathe situation. That is what is currently used in our altitude chambers.

DR. STEGMANN: True. However, we do know that a 5- to 10-minute interruption in a one-hour prebreathe does not significantly change the incidence of DCS.

DR. BAGIAN: You stated that it didn't significantly change the incidence. However, you also said that the only difference that did occur was in the people that had breaks.

DR. STEGMANN: The study had one or two subjects who bent, and the only time they bent was when they interrupted their prebreathe.

DR. BAGIAN: How big was the study? The fact that they didn't have significance doesn't mean it's not so.

DR. STEGMANN: They had 10 to 15 subjects. The sample was not big enough to be statistically significant.

DENITROGENATION

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Abstract

A faster elimination of nitrogen from the body through the lungs may decrease the risk of hypobaric decompression sickness. Various methods have been found to increase the nitrogen elimination rate. Physical exercise, mild hypoxia and carbon dioxide admixture to the breathing gas in a hypobaric environment seem, however, to have other disadvantages. Head-out immersion, supine body position, raised ambient temperature, negative pressure breathing and vasodilator drugs, on the other hand, have been shown to be of value to decrease the risk of decompression sickness. The enhanced nitrogen elimination through the lungs by these methods has also been shown to decrease the amount of intracardial gas bubbles in hypobaric provocation tests used in humans and in animals. The increased nitrogen elimination in these environments or by these methods seems to be accompanied by an increased cardiac output and increased peripheral tissue perfusion, as indicated by increased Xe^{133}-elimination from muscles and subcutaneous adipose tissues.

An accelerated nitrogen elimination to reduce the duration of the necessary preoxygenation prior to extra vehicular space activity with reduced space suit pressure may be accomplished by application of physical as well as physiological fundamentals (Lambertsen 1967). A physiological approach is to increase respiratory inert gas elimination by improving tissue perfusion and thus tissue gas elimination by different methods, such as changes in the ambient environment, physical activity, body position, respiration or by the use of drugs.

Physical exercise may increase the inert gas elimination through the lungs during oxygen breathing by an increased skeletal muscle perfusion (Behnke and Willmon 1941; Jones 1951; and Dick, Vann, Mebane and Feezor 1984) and be beneficial during decompression from diving (Vann 1984). On the other hand, only small amounts of nitrogen are solved in the muscles, and the circulatory adjustments to exercise may reduce the inert gas elimination in other more nitrogen solving tissues as for instance subcutaneous fat tissue. It is also reported that exercise might actually increase the incidence and intensify the symptoms of altitude decompression sickness (Cook 1951 and Roth 1967).

Mild hypoxia has a stimulating effect on blood circulation as well as on ventilation and might thereby increase the inert gas elimination. It has also been reported that artificially induced hypoxia slightly reduces the frequency of symptoms of

altitude decompression sickness (Smith 1951). To achieve a hypoxia during 1 ata conditions to increase the nitrogen elimination involves, however, the admixture of another inert gas than nitrogen. That means that complex conditions will appear, where nitrogen is eliminated, while another inert gas is solved in the tissues. The problems with counter diffusion must, therefore, also be taken into consideration. Hyperoxia, on the other hand, has both been shown to decrease muscle blood flow, $Xenon^{133}$ elimination from tissues (Balldin, Lundgren, Lundvall and Mellander 1971) and cardiac output, as well as to cause an unchanged muscle, fat and visceral blood flow (Plewes and Fahri 1983). A digital computer model indicated a minimal change in the pattern of nitrogen gas elimination by hyperoxia by Plewes and Fahri (1983). To achieve hyperbaric hyperoxygenation to increase nitrogen elimination seems, however, to impose big practical problems, if a hyperbaric chamber or hyperbaric space suit is to be installed in the space craft.

Carbon dioxide admixture to the breathing oxygen may also improve nitrogen elimination probably by an increased peripheral blood perfusion, at least in some tissues, as well as by an increased lung ventilation (Margaria and Sendroy 1950). The influence on the incidence of decompression sickness seems, however, to be questionable when using carbon dioxide admixture to the breathing gas with both unchanged (Gray 1944) and increased risk (Hodes and Larrabee 1946). Balldin, Lundgren and Westling (1971) also pointed out that carbon dioxide accumulation by an increased respiratory dead space might increase the severity of decompression sickness once it occurs.

The inert gas elimination through the lungs and thereby the total body nitrogen elimination has been shown to be substantially increased by head-out immersion. When sitting subjects were immersed in thermo-neutral water of 35°C, a mean of 40% increase in nitrogen volume was registered in a spirometer system over a 30-min period and an increase of about 30% over a 2-hour period compared to a similar oxygen breathing period sitting dry in thermo-neutral temperature of 28°C (Balldin and Lundgren 1972) (see Figure 1). Head-out immersion is in many aspects similar to the conditions during weightlessness. However, the effects of head-out immersion were studied during acute exposures, not lasting more than 2 hr, or occasionally 4 hr. The hemodynamic effects of weightlessness during long-lasting space flights, therefore, seem to be different from the findings during short, acute exposure to head-out immersion.

In the explanation of the findings of head-out immersion it was shown that immersion increased the cardiac output by about 30% using the dye dilution technique. This was concomitant with a central blood volume increase of about 0.7 l, a decrease of about 30% in systemic vascular resistance (Arborelius, Balldin, Lilja and Lundgren 1972a). The increased cardiac output during immersion is accompanied by an increase in peripheral circulation. Thus, a more than 100% increase in $Xenon^{133}$ elimination and hence in blood flow in leg muscular tissue was shown during immersion (Balldin, Lundgren, Lundvall and Melander 1971). A similar increase in both blood flow and $Xenon^{133}$ elimination was also seen in subcutaneous adipose

tissue (Balldin 1978), which may be a more important tissue due to the greater inert gas solving capacity in the fat tissue. The xenon eliminated from the tissues is finally eliminated from the body mainly through the lungs.

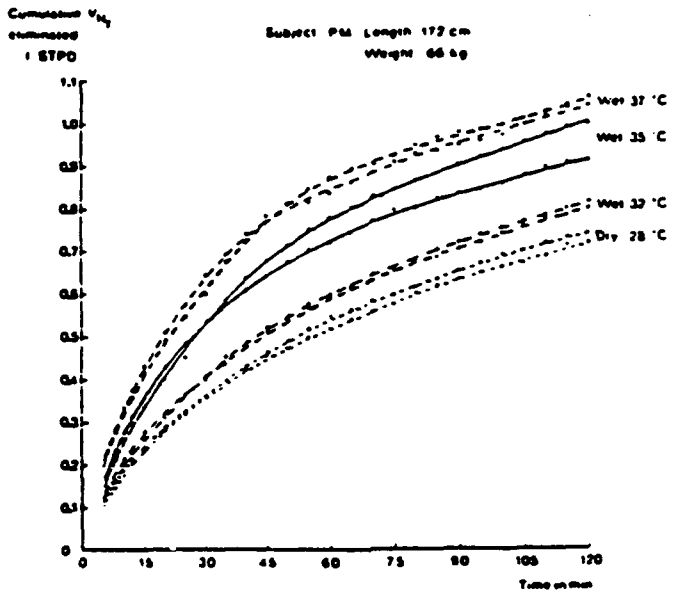

Figure 1. Nitrogen elimination through the lungs during oxygen breathing, sitting dry at 28°C and immersed at 32°C, 35°C and 37°C (from Balldin and Lundgren 1972).

The regional lung function was also shown to be improved during immersion (Arborelius, Balldin, Lilja and Lundgren 1972b). Thus, a more even ventilation-perfusion ratio between the bases and apices of the lungs could be shown with Xenon^{133} radiospirometry. This improvement was, however, considered insignificant for the enhanced nitrogen elimination during immersion as this elimination is largely blood flow dependent (Balldin 1973a). Only major deterioration in lung function during immersion might influence the exchange of nitrogen and other gases with low solubility in the blood and then in a negative way. Such deteriorations in lung function may be the lung vital capacity decreases or possibly atelectasis formation described during immersion and oxygen breathing (Balldin, Dahlbäck and Lundgren 1972; Dahlbäck and Balldin 1983; and Baer, Dahlbäck and Balldin 1987) (see Figure 2) similar to findings during increased G-loads (Haswell, Tacker, Balldin, Burton 1986). The increased inert gas elimination (nitrogen as well as xenon) through the lungs during immersion may thus be explained mainly by the increases in both central and peripheral blood circulation and not by the improved ventilation-perfusion ratio of the lungs.

Changes in body position have in some ways similar influences on blood circulation as immersion. Thus, supine compared to erect body position increases the

Xenon^{133} elimination and blood flow in muscular tissue by about 100% (Balldin, Lundgren, Lundvall and Mellander 1971) and in subcutaneous fat by about 35% (Balldin 1976). These increases are accompanied by an increase in nitrogen elimination through the lungs during oxygen breathing by a mean of 24% after 30 min of supine compared to erect body position and by 15% after 2 hours (Balldin 1973b). These findings may, in the same way as during head-out immersion, be ruled out as acute, short exposure effects, with no relevance to long-lasting space flights with weightlessness.

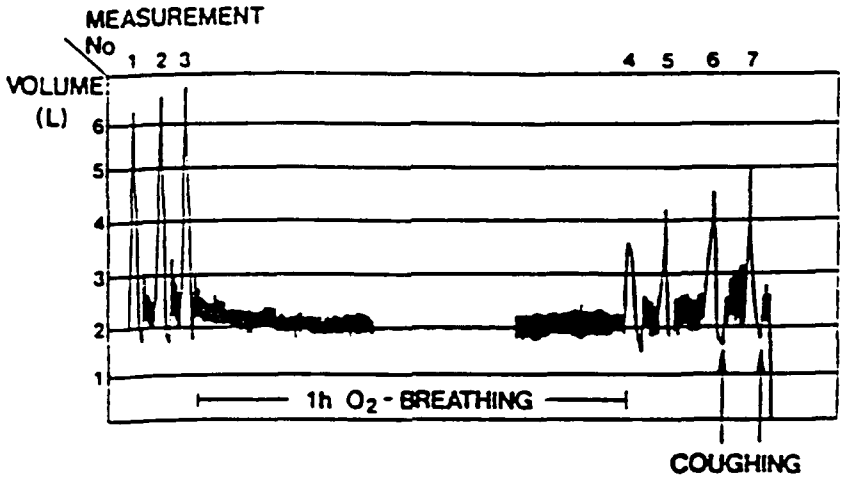

Figure 2. Spirogram from a subject indicating reduced vital capacity after 1 h oxygen breathing during immersion (from Dahlbäck and Balldin 1983).

Temperature also influences the central as well as the peripheral blood circulation and thus the inert gas exchange. The mean subcutaneous adipose tissue blood flow and Xenon^{133} elimination during immersion was thus increased with 37°C (warm water) compared to 35°C (thermo-neutral water) by a mean of about 90% (Balldin 1978). A similar increase in total body nitrogen elimination through the lungs of about 6% was seen when the temperature was increased from 35°C to 37°C during 2 hours immersion (Balldin and Lundgren 1972).

The ambient temperature, and thus the body temperature, in dry conditions, is, however, of more interest in a spacecraft situation, when preoxygenation prior to an EVA is performed. When the temperature is increased in dry conditions from 28°C (thermoneutral) to 37°C in sitting as well as in supine subjects the nitrogen elimination has been found to increase by about 17% after 2 hours (see Fig. 3) (Balldin 1973a). Heat stress has also been shown to increase whole body xenon washout in rabbits (Bove, Hardenbergh, and Miles 1978). The combined effect of immersion and raised ambient temperature increased the nitrogen elimination by 49% after 30 min and 36% after 2 hours, compared to sitting in dry conditions in a thermo-neutral environment

and thus seemed to be most effective (Balldin and Lundgren 1972). Similar increases in Xenon^{133} elimination from adipose tissue in humans were found with immersion in warm water (Balldin 1978). The combined effect of raised temperature in dry conditions and supine compared to sitting body position increased the whole body nitrogen elimination by about 30% after 30 min as well as after 2 hours (Balldin 1973b).

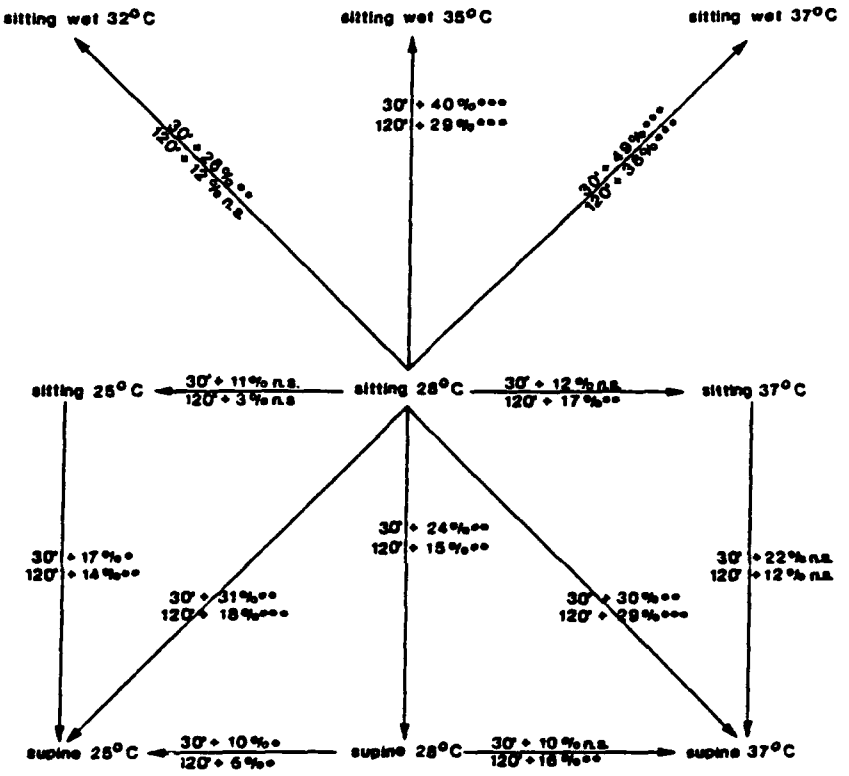

Figure 3. Mean relative differences in tissue nitrogen volumes eliminated through the lungs after 30 and 120 min immersed at 32°C, 35°C, and 37°C compared to sitting dry at 28°C. Sitting and supine body positions in dry conditions are also compared in 25°C, 28°C, and 37°C. * p<0.05, ** p<0.01 and *** p<0.001 (from Balldin 1973).

On the other hand, lowered ambient temperature, and thereby lowered peripheral and later body core temperature, decreases mainly the subcutaneous adipose tissue blood flow through vaso-constriction. Consequently, the total body nitrogen elimination during oxygen breathing could be shown to decrease slightly (about 10%) in the immersed subjects with water temperature lowered to 32°C (Balldin and Lundgren 1972). In the dry conditions the effects of a lowered ambient temperature to 25°C were not so consistent with both unchanged and increased nitrogen elimination observed in humans (Balldin 1973b). In rabbits, cooling caused

unchanged whole body xenon elimination in combination with an increase in muscle xenon elimination probably due to shivering (Bove, Hardenberg, and Miles 1978).

The most effective way of increasing the nitrogen elimination in these studies, namely the combined effect of immersion and raised temperature (see Figure 3), was tested in actual decompressions in humans (Balldin 1973c). Denitrogenation by oxygen breathing for 25 min in water at 37°C or during dry thermo-neutral conditions was followed by provocation of decompression sickness at an ambient pressure of 155 mm Hg (20.7 kPa). The occurrence of decompression sickness (bends) was 20% after warm water denitrogenation and 90% during dry thermoneutral conditions, thus supporting the effectiveness of the increased nitrogen elimination during warm water immersion.

Negative pressure breathing (NPB) has been shown to increase cardiac output as well as adipose tissue blood flow in humans (Balldin 1976). Thus, 20 cm H_2O NPB increased Xenon^{133} elimination in subcutaneous fat tissue by about 70%. As a consequence of these findings it could also be shown that denitrogenation during NPB compared to normal oxygen breathing decreased the amount and delayed the onset of intracardial gas bubbles detected with precordial Doppler ultrasound in decompressions to altitude (Balldin and Borgström 1977). It also delayed the onset of symptoms and reduced the risk of decompression sickness. The question of whether NPB also increases the nitrogen elimination during a weightlessness condition remains to be investigated.

Positive pressure breathing (PPB), in contrast to NPB, decreases the cardiac output (Balldin and Wranne 1980). PPB of 20 cm H_2O has also been shown to decrease Xenon^{133} elimination from subcutaneous adipose tissue by about 40% (Balldin and Linér 1976), which consequently should impair inert gas elimination as well. PPB has, however, also been shown to reduce the atelectasis during oxygen breathing in immersion (Dahlbäck and Balldin 1983) and in diving (Dahlbäck and Balldin 1985) as well as at high G-loads (Tacker, Balldin, Burton, Glaister, Gillingham, and Mercer 1987). Even though atelectasis may develop during oxygen NPB, and especially in combination with immersion, the improved blood circulation during NPB and immersion may be more important to gas elimination.

The therapeutic use of drugs for treatment of decompression sickness and the prophylactic use of drugs in decompressions have been investigated in many studies (c: Catron and Flynn 1982). Perorally administered terbutaline, a symphathomimetic beta-2-receptor stimulator with mainly vasodilator but also with bronchodilator effects, has been shown to increase the Xenon^{133} elimination in subcutaneous adipose tissue by more than 100% in humans (Balldin 1976). This increased inert gas elimination by terbutaline could also be shown to have a preventive effect on the occurrence of hypobaric decompression sickness in rabbits, who were denitrogenated during oxygen breathing with terbutaline given prophylactically (Balldin and Linér 1978).

The factors to increase inert gas elimination seem to be most important as prophylactics, before gas bubbles have been formed in a decompression. Once decompression gas bubbles have been formed, the increase in inert gas elimination through the lungs does not substantially help to eliminate the gas trapped in gas emboli in the circulation or as gas bubbles in situ. The elimination of inert gas from a bubble in the tissue or a gas pocket is slow compared to the elimination of gas solved in the tissue (Van Liew, Bishop, Walder and Rahn 1965). The bubble might disturb the microcirculation by blocking the blood flow and preventing the gas transport from the bubble. Preoxygenation prior to altitude exposure preceded earlier by a dive giving rise to silent bubbles is, thus, not as effective to reduce the risk of decompression sickness as is preoxygenation without a preceding dive (Balldin 1980).

The methods to increase nitrogen elimination (e.g., immersion, raised ambient temperature, supine body position, NPB and vasodilators) might be used in a preoxygenation procedure to reduce the risk of decompression sickness in diving, at altitude or to some extent before an extravehicular space activity. In the last situation, the raised ambient and body temperatures and the use of vasodilators might be of value in a preoxygenation procedure, while NPB is more dubious during weightlessness in space.

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DR. BUTLER: How much of terbutaline effect do you think is due to vasodilation versus increase in cardiac output? There may be two different mechanisms.

DR. BALLDIN: The cardiac output does not influence the elimination of the nitrogen very much. The important thing is that there is vasodilation in the periphery. Therefore more gas can be transported from the tissues.

DR. BUTLER: What about with exercise?

DR. BALLDIN: Exercise mostly affects the blood flow in the muscles. The muscles do not contain very much nitrogen. They are water soluble tissues with very little nitrogen compared to fat tissues. But exercise will, as a secondary effect, increase the body temperature, and in turn increase the tissue blood flow in the fat tissue, and that increases the N_2 elimination.

DR. BUTLER: Dr. Vann, with exercise during preoxygenation, how much of the N_2 elimination do you think is due to temperature or vasodilatation versus cardiac output?

DR. VANN: When we compare thermoneutral immersion (35°C water) with light exercise (25 watts), the exercise results in higher N_2 elimination. Although the 35°C immersion results in higher N_2 elimination than any other variation of body position, the exercise results in the highest elimination. Certainly, warm water immersion is the most effective resting procedure. But the exercise seems to be more effective.

DR. WENZEL: You are measuring cumulative N_2 elimination?

DR. BALLDIN: Yes, that is right.

DR. WENZEL: Is there really a correlation between DCS and the cumulative N_2 elimination? In other words, do you really look for an increased elimination at the sites where DCS is occurring?

DR. BALLDIN: I have not looked at the correlation. However, I tested this method in the subjects exposed to warm water immersion preoxygenation and dry preoxygenation. There was a distinct difference in susceptibility to decompression sickness. Thus, even if I have not correlated it, I have tested the model, and it seems to be effective in decreasing the risk of decompression sickness. I also did this with negative pressure breathing. The finding was the same. If you increase the nitrogen

elimination by warm water immersion, or negative pressure breathing, you will have decreased the amount of gas bubbles, and decreased the amount of decompression sickness symptoms.

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The most important development in decompression theory since Haldane's pioneering work of 1908 (1) is the statistical methods published in 1984 by Weathersby of the U.S. Navy (2).

Statistical Decompression Models

We will view an individual as having a binary probability of developing decompression sickness (DCS) -- one or zero -- who either gets DCS or does not. A large population of individuals, on the other hand, has a nearly continuous probability which can assume almost any value between one and zero.

Statistical decompression models attempt to rationalize this dichotomy within the bounds of two extremes -- biometric and physiological. The biometric extreme estimates a continuous probability from the binary responses of a population. This is a common statistical tool in which binary data are correlated with measured variables such as pressure and time using empirical relationships. No underlying mechanisms are assumed.

The physiological extreme is the desired but unachievable goal. It predicts the exact occurrence of decompression sickness for each individual in a population. Mechanisms are exact and parameters such as blood flow, bubble volume, nitrogen solubility in tissue, etc., may differ for individual exposures.

Statistical decompression models developed to date are clustered towards the biometric extreme. Kumar et al. (3) used a classic biometric model based on linear logistic regression to estimate the altitude threshold for decompression sickness.

Conkin et al. (4) stepped away from the purely biometric approach by incorporating a Haldane tissue and supersaturation ratio. With this model, they analyzed the results of NASA and U.S. Air Force (USAF) altitude trials and estimated the risks of untested procedures. Dr. Gilbert describes this model elsewhere in this workshop.

Weathersby and his coworkers in the U.S. Navy, of course, have the longest record of using statistical decompression models (2, 5-12). They place particular emphasis on data quality, insisting on verifiable accuracy of both pressure profile and symptomatology. Their most frequently used model (5) postulates that the probability of DCS increases with sustained supersaturation. Supersaturation is integrated over time using one or more exponentials to describe nitrogen kinetics. This model has been applied only to hyperbaric data.

All these are excellent approaches, superior to the nonstatistical models of the past. Further improvement should be possible, however, by incorporating hypotheses concerning the mechanisms of decompression. Models of this nature might be called "quasi-physiological" in recognition of the impossibility of achieving complete knowledge. Let us consider such a model in which pain-only decompression sickness is precipitated by stationary extravascular bubbles.

A Bubble Model Based Upon a Population with Varying DCS Susceptibility

The traditional approach to a bubble model assumes that decompression sickness will occur for every individual in a population when the bubble volume exceeds a critical value (13). In a statistical application of this model, every individual in the population can have a different critical volume. These volumes are distributed across the population by a density function such as that shown in Figure 1. A small fraction of the population is highly susceptible and develops DCS at low volumes. Most of the population is at risk at intermediate volumes. Another fraction of the population can tolerate large bubbles and is highly resistant to bends. The critical volume an individual can tolerate may change from one exposure to the next. Thus, the population of exposures represented in Figure 1 may be for the same individual or for a group of different individuals.

Integrating the population density (Figure 1) over the volume results in a dose-response function (Figure 2) which describes the relationship between bubble volume and the total fraction of the population which develops DCS. Thus, if the bubble volume is known, the risk to the population can be determined. In contrast, the dose-response function for a nonstatistical model (13) is a unit step from zero to one when the critical volume of the population is exceeded.

Now, let's consider the dynamics of bubble growth as illustrated by Figure 3 (14). Bubbles grow in response to gaseous supersaturation and are absorbed as a consequence of the oxygen window. Bubble growth and resolution occur by diffusion, simulated in Figure 3 by a barrier around the bubble. Inert gas exchange between blood and tissue is assumed to be perfusion-limited. The equations of bubble dynamics observe conservation of mass for dissolved and gaseous nitrogen. Bubble dynamics, therefore, are controlled by both perfusion and diffusion.

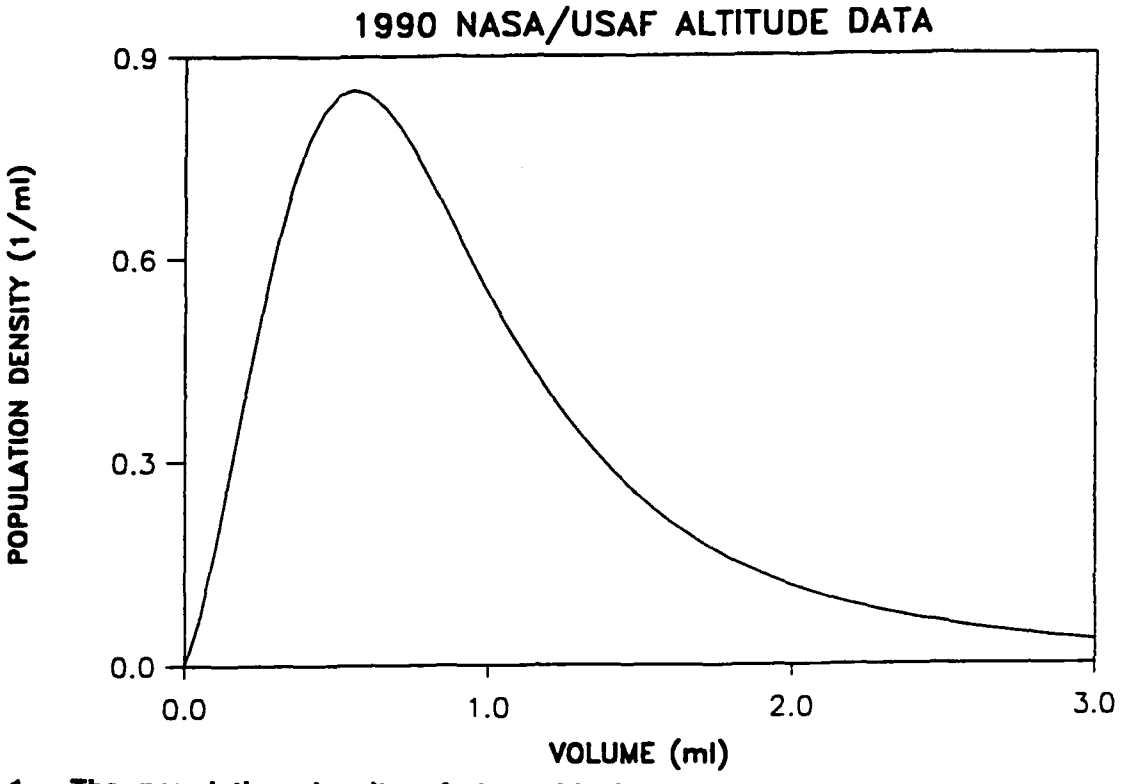

Figure 1. The population density of the critical bubble volume as distributed over bubble volume for the 1990 NASA/USAF altitude data.

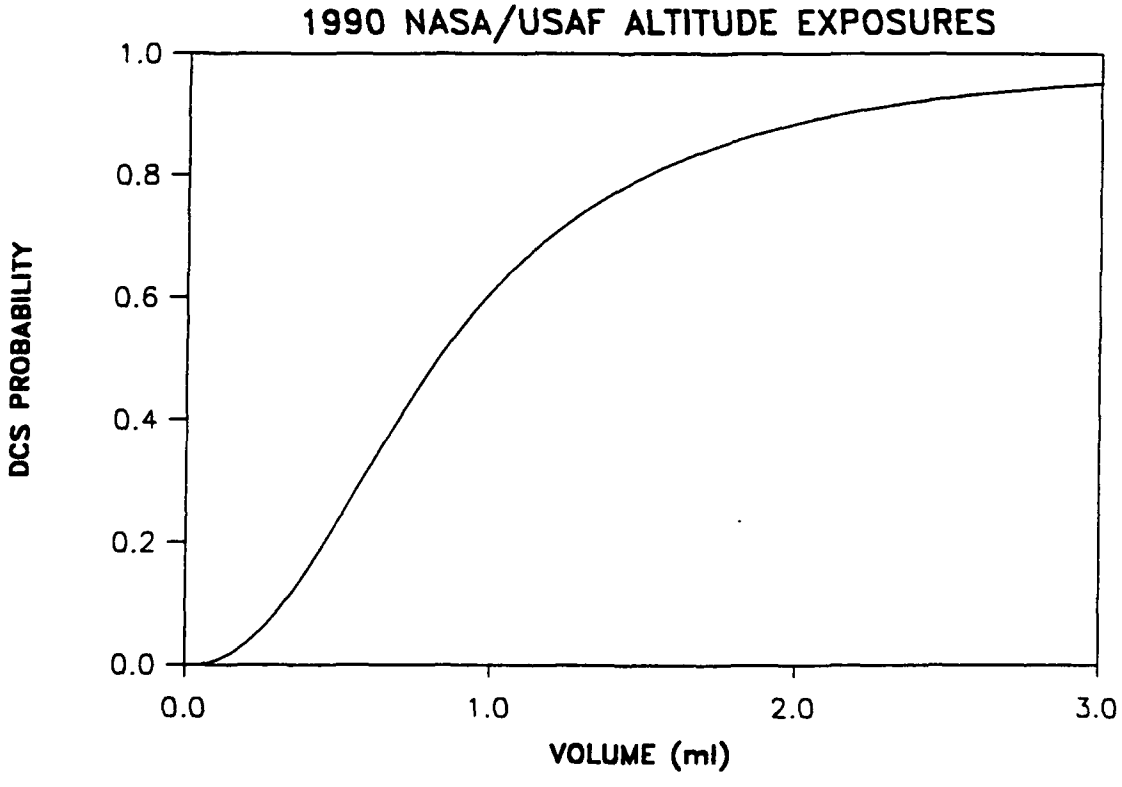

Figure 2. The dose-response function of the 1990 NASA/USAF altitude data where the dose is the bubble volume and the response is the DCS probability. The dose-response function is the integral of the population density of Figure 1.

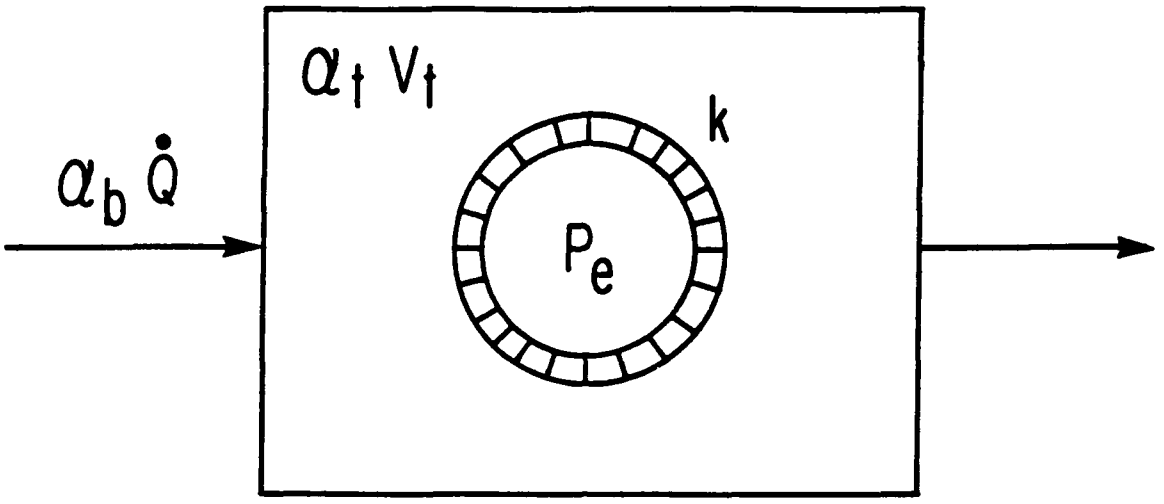

Figure 3. A perfusion-limited tissue with a bubble surrounded by a diffusion barrier (14). Alpha refers to inert gas solubility in blood (b) or tissue (t). Q is blood flow, Vt is tissue volume, k is the diffusion barrier inert gas permeability, and Pe is the tissue pressure.

Diffusion results in gradual bubble growth consistent with the delayed onset of DCS symptoms observed at altitude. Gradual bubble growth is shown in Figure 4 during an exposure at 30,000 feet after prebreathing oxygen for 3.5 hrs at sea level. The volume of the bubble determines the fraction of the population which develops decompression sickness (Figure 2). In Figure 4, the bubble reaches a peak volume of 0.4 ml after 30 min at 30,000 feet, corresponding to a maximum DCS incidence of 22.5%.

There are alternatives to the peak bubble volume hypothesis upon which Figure 4 is based. Bubble surface area or a linear measure of size such as radius might be the parameter which provokes symptoms. Tissue forces might render the bubbles aspherical. The probability of symptoms might accumulate during a bubble's lifetime. A great strength of statistical modeling is that any hypothesis which can be formulated mathematically also can be tested quantitatively and objectively compared to other hypotheses. This powerful tool facilitates hypothesis testing.

The rate at which a bubble grows and resolves is governed by tissue parameters that relate to blood flow, tissue volume, tissue pressure, diffusion barrier permeability, and blood and tissue nitrogen solubilities (Figure 3). In the ideal physiological model, there would be a different set of parameter values for each exposure in the population. In a quasi-physiological model, however, we must accept the single set of parameters which best describes the overall behavior of the population. The many sources of individual variability are lumped into the population

density function (Figure 1) and the dose-response function (Figure 2). These are "fudge-factors" which account not only for physiological and environmental uncertainty, but also for theoretical error.

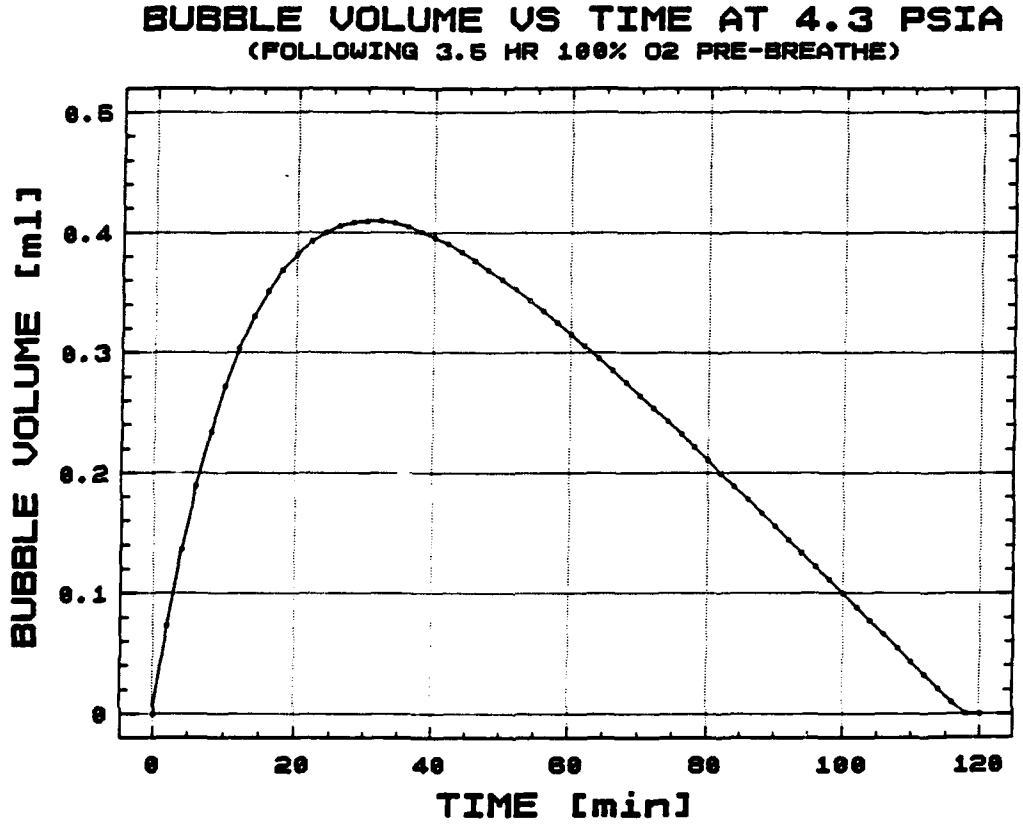

Figure 4. Bubble growth at 30,000 feet following 3.5 hours of oxygen pre-breathing at sea level.

Maximum Likelihood and Parameter Optimization

The next step is to find the set of parameter values which determines the best correlation between the dose-response function and the binary results of the individual exposures. This is analogous to linear regression in which the slope and intercept of a straight line are found to determine the best correlation between a line and experimental data.

In linear regression, the least-squares error is minimized to find the optimal parameters. With binary data, however, the appropriate measure of agreement between theory and experiment is the likelihood which is maximized to find the optimal parameters. Indeed, there is a characteristic maximum likelihood for every model and population of data. In comparing two models, the model with the greater maximum likelihood may be a better description of the data.

Likelihood is not a new concept, but Weathersby deserves the credit for introducing it to decompression (2). Likelihood is the theoretical probability of an experimental outcome. The theoretical probability, P(DCS), is between zero and one, while the probability of no DCS is 1 minus P(DCS). The experimental outcome, X, is 1 if DCS occurs and 0 if DCS does not occur.

Expressed quantitatively for a single exposure,

$$\text{Likelihood} = P(\text{DCS})^{X} * P(\text{no-DCS})^{1-X}$$

Likelihood can be extended to multiple exposures by multiplying the likelihoods of the individual exposures. This approach is analogous to finding the probability of a series of coin tosses by multiplying the probabilities of the individual tosses. Pooling data in this manner significantly improves confidence limits over the binomial confidence limits in which individual pressure profiles must be treated separately. Since multiplying likelihoods results in a small number, the natural logarithm of the likelihood is commonly reported and appears as a negative number.

The likelihood is maximized by a nonlinear optimization procedure which adjusts the parameter values until the maximum likelihood is achieved. We use an optimization procedure developed by Bailey and Homer (15) of the Naval Medical Research Institute and based on the Marquardt algorithm (16). This procedure provides confidence intervals for parameters values and prediction intervals for DCS risk estimates. These measures of uncertainty are essential to the design of trials for validating decompression procedures.

Data Analysis and Risk Estimation

The statistical bubble model was fit to the results of tests conducted by NASA and the USAF during trials of 49 pressure profiles. These trials involved 923 individual exposures in which there were 201 cases of mild to moderate pain-only DCS for a 22% DCS incidence. Included were ascents to pressures of 7.8-10 psia without oxygen prebreathing, ascents to the Shuttle space suit pressure of 4.3 psia (30,000 feet) following 3.5-8 hours of oxygen prebreathing, and stage decompression to 10.2 psia followed by ascent to 4.3 psia.

After the parameters of the bubble model were optimized to these data, decompression risk could be estimated for any pressure profile. Figure 5, for example, shows the effect of oxygen prebreathe duration on the DCS risk of an exposure at 30,000 feet. With a 3.5-hr prebreathe, the estimated risk of mild to moderate pain is 22.5%.

Also shown in Figure 5 are the 95% prediction intervals around the estimated risks. The prediction interval varies with the risk and is as small as +/-7% for a 60-min prebreathe and as large as +/-22% for a prebreathe of 280 min. This variation reflects the nature of the underlying database. A large prediction interval on an estimated risk indicates there are little data in the vicinity of that exposure.

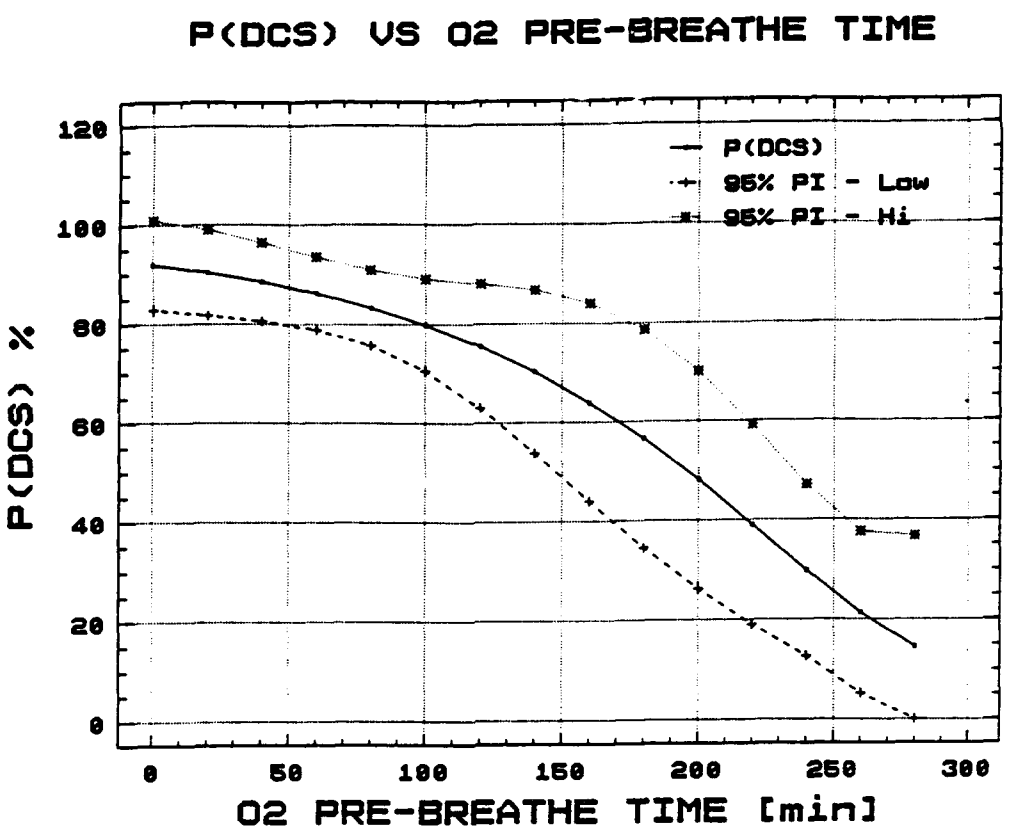

Figure 5. The effect of oxygen prebreathe duration on the estimated DCS probability at 30,000 feet. The 95% prediction intervals (PI) on the probability estimates are indicated.

Acceptable DCS Risk

With the capability for estimating risk, comes the difficult task of deciding what risk is acceptable. Figure 6 shows the results of a survey of 26 opinions concerning acceptable risk (17). Acceptable risk is defined as the maximum permissible DCS incidence or the incidence at which a decompression procedure must be modified to be made acceptable.

Opinions fell into high and low groupings. The highest acceptable risk, 6% moderate or Grade 3 pain, was recommended for Shuttle operations where pain-only symptoms have been the rule in ground-based studies (18). Other opinions in the high grouping were 3-4% for the U.S. Navy (19) where medical coverage and recompression facilities are readily available, and 2% for compressed air work (20). For Station operations where flight crew will be less accessible than in the Shuttle, acceptable risk was reduced to 1% moderate Grade 3 pain (18).

Those with opinions in the low grouping felt that acceptable risk should not exceed 0.5% although many believed that more than 0.2% was unacceptable, and a substantial number thought that no decompression sickness at all was tolerable. The primary factors influencing opinion were the seriousness of the symptoms and the availability of treatment and medical support.

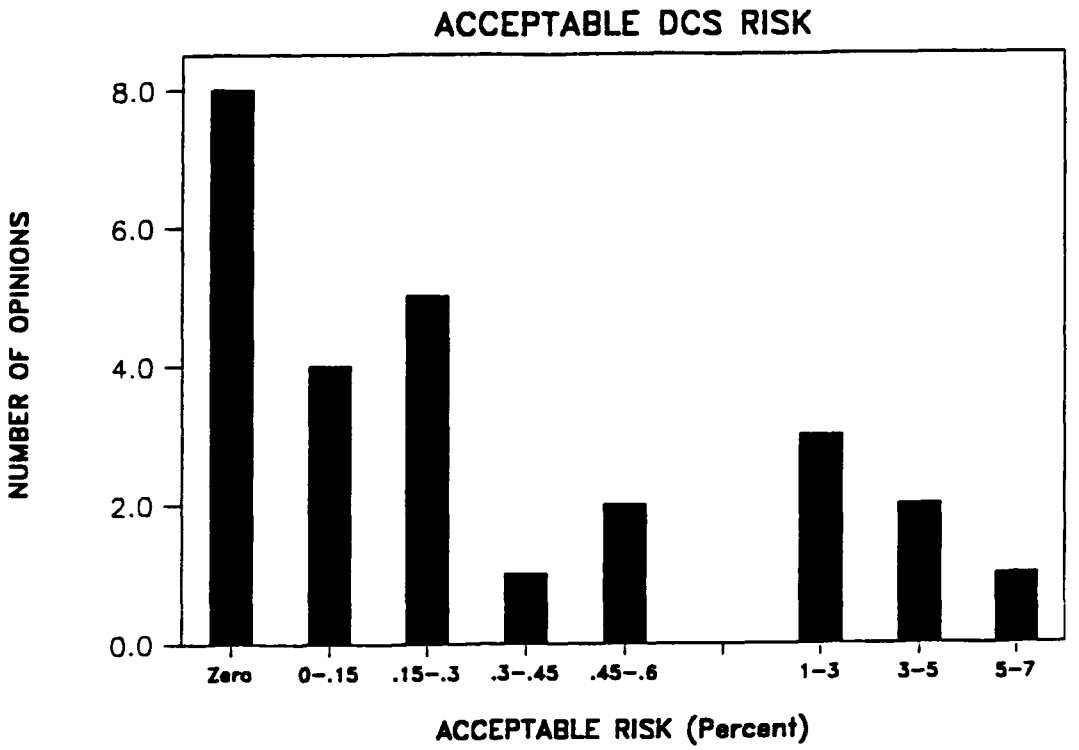

Figure 6. A survey of 26 opinions concerning acceptable DCS risk.

Most of the conservative opinions were from the diving community where serious symptoms such as spinal DCS are a particular problem. Serious symptoms are much less common in altitude exposure, and this was the rationale for selecting 6% as an acceptable risk for the Shuttle after tests of a 3.5-hr prebreathe resulted in only mild to moderate pain (18). Current trials at Brooks AFB, however, indicate that neurological symptoms can occur at 30,000 feet after prebreathe durations of one and two hours (21). Unfortunately, the incidence of these symptoms is uncertain because reporting accuracy appears to be affected by the extraneous factors addressed by Dr. Pilmanis elsewhere in this workshop.

The occurrence of neurological symptoms with 1-2 hr prebreathes but not with 3.5 hr prebreathes suggests that nitrogen is eliminated more rapidly from the tissues responsible for neurological symptoms than from those responsible for pain-only symptoms. A similar conclusion follows from dives where neurological symptoms are rare during slow saturation decompression (22) but are common after bounce dives with fast decompression (23). Thus, while there seem to be pressure profiles which predispose to neurological symptoms, existing data are inadequate to accurately define their circumstances. Risk estimates based on an analysis of pain-only symptoms are inappropriate for estimating the risks of neurological symptoms. Accurate reports of serious symptoms are essential if data are to be accumulated from which to make the needed risk estimates.

The Validation Cycle

Indeed, decompression modeling and the validation of decompression procedures is a cyclic process which begins and ends with data as indicated in Figure 7. Data should specify pressure profile, breathing gas, temperature, workload, symptoms, and treatment. Models for data analysis can be empirical or quasi-physiological (Figure 3). Parameter optimization by maximum likelihood allows models founded on different hypotheses to be compared. An acceptable risk must be declared based upon value judgment or risk-benefit analysis before decompression procedures can be calculated for trials (Figure 6). Procedures selected for trials are those whose risk estimates have the greatest uncertainty as indicated by their prediction intervals (Figure 5). As trials are conducted, their results are entered into the database (Figure 7). Validation is complete when multiple circuits of Figure 7 reduce the uncertainty to an acceptable level. Combining data from compatible sources can decrease the number of new trials that are needed to develop risk estimates having the desired confidence.

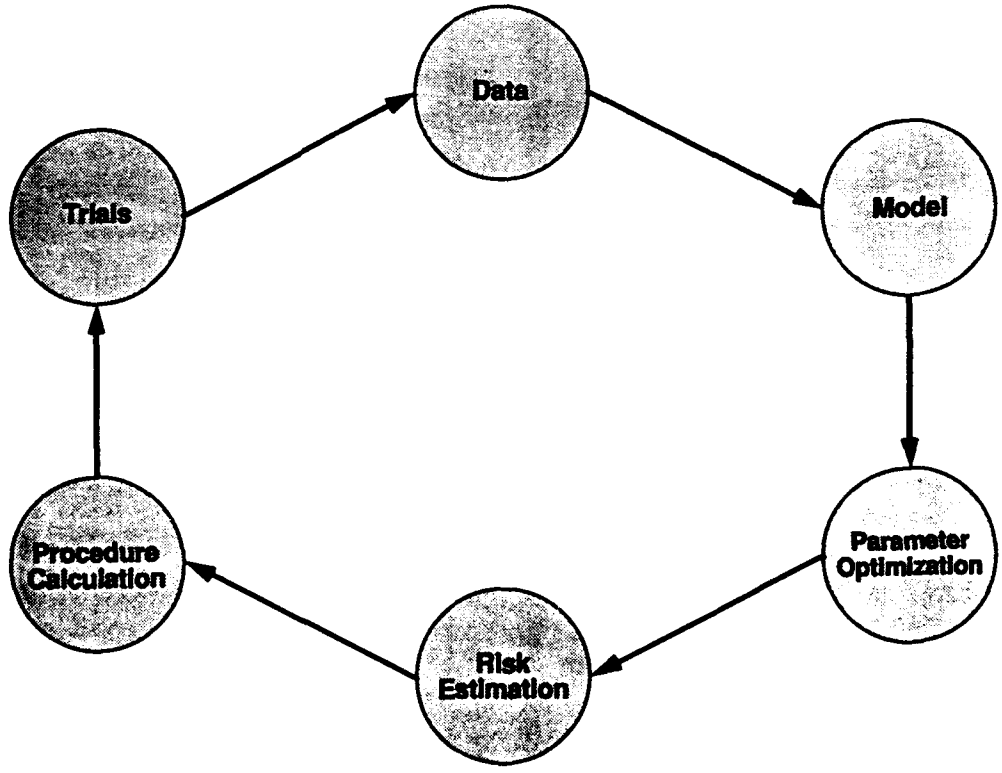

Figure 7. The cyclic process by which risk estimation is used to develop decompression procedures.

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DR. VAN LIEW: Can you subject to the maximum likelihood procedures models that consist of only two tissue compartments or one tissue compartment?

DR. VANN: It does not make any difference. You can be totally empirical and approach it from strictly a statistical point of view. You do not need to introduce any physiology at all.

DR. VAN LIEW: But it seems to me that a model that does not have a lot of different tissue halftimes is not a realistic model at all.

DR. VANN: There is more involved than just the number of tissues. You can find some single-tissue models that will be more successful than some multi-tissue models. The proof of the pudding is really in the statistics, how well does it fit the underlying data.

I agree that as one introduces more valid physiology you are going to have a better model, but the only way to make that judgment is statistically, how well the model fits the data, not on the basis of your assumptions themselves.

MR. GILBERT: We are looking at our data, with a multivariable logistical equation, and we have been able to squeeze the error down slightly by including another tissue. Right now the tissue halftime of the tissue type we are using is 360 minutes. By inclusion of a 480 minute tissue halftime variable within this, and also with the inclusion of a variable for gender, we have been able to squeeze the data so that we can get a smaller error around the estimate. Thus, we are approaching what Dr. Vann was talking about; it is the physiological model as opposed to the strictly mathematical and empirical model.

DR. HAMILTON: The maximum likelihood method enables you to compare a bunch of complete profiles, not just single points. In order to compare profiles and mix them all up and get the equivalent of a least squares agreement or middle ground there, you have to look at those profiles against some model. When Weathersby makes these comparisons, he tries several different relatively simple models. The kind of thing I was describing has so many variables in it that it does not work very well, whereas a simpler model that does not describe the profile quite so precisely, does work.

DR. VAN LIEW: My hope for the maximum likelihood method was that it would examine the whole realm of decompression, and be able to tell us which model was better. But in order to examine over the whole realm you have to take account of all the different tissue types.

DR. VANN: The hypothesis is that there is a different mechanism, and therefore model, for each kind of symptom; for example, chokes, data for chokes could not be modeled with data for pain. The spinal cord neurological symptoms appear to have a different set of kinetics than the pain-only symptoms. You need to model those symptoms separately and actually fit a different model to a different population of data. Cerebral decompression sickness might take another model. So you have to look at the specific symptom that occurs. Getting back to the problem of Type I and Type II DCS, you cannot drop symptoms into those boxes if you are going to use this sort of approach. You must have the condition well described by the clinicians. Then, someone else can make the decision how they want to handle the data. That is far superior to the Type I and Type II approach.

DR. VAN LIEW: In other words, if you choose an end point, like chokes, then you are also choosing the compartment which is going to give you chokes, so one compartment is appropriate, just as NASA says that one compartment is probably appropriate after people are denitrogenated and going to altitude.

DR. LAMBERTSEN: I think it is a mistake to use the term model for what is tested by the maximum likelihood method. It can be anything you feel like putting in there for your test, whether it has any rationality or not. One can do statistics with irrational lines and see which gives you the best fit among these irrational lines.

DR. HAMILTON: There is still a little bit of art involved here.

MR. GILBERT: A model is simply something which is trying to describe the data in as accurate a way as possible so that one can use the model to predict data points that do not occur in the actual data set. The fact that we are using physiological, or quasi-physiological parameters as variables indicates that we are trying to associate them with certain aspects of the physiology, but does not mean that they follow in all respects the physiological parameters in a wide field.

When we are looking at a 360-minute tissue halftime, we are saying, the body has a large number of compartments and from our testing of all these compartments, they seem to average out to a 360-minute tissue halftime. This seems to match the data we have been receiving, the data that we will be working on.

ALTITUDE DECOMPRESSION COMPUTER DEVELOPMENT: A Progress Report

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Introduction

The ongoing development of an altitude decompression model for the USAF is expected to take several years. This report describes the progress to date and defines the future direction of the project. It is not a research paper but rather a descriptive overview for this workshop.

Many decompression models have been created to delineate the boundaries of decompression sickness (DCS). These models have been used to produce decompression procedures (decompression tables) for diving. While the diving models provide a basis for developing an altitude model, important issues not addressed in decompression procedures for diving must be taken into consideration for altitude.

One important difference between the requirements for decompression procedures at altitude versus diving is the nature of the mission. A diver usually begins a mission by descending to some water depth. During a diver's descent and bottom time there is no risk of DCS. The risk is during the return to sea level and after surfacing. At that time the mission is over and decompression time can be extended at will. A diver can plan a dive to eliminate the risk of DCS. Thus, most diving decompression procedures are based on a binary yes/no ouput. It is generally accepted that the form of DCS associated with altitude exposure is milder than that associated with diving. In many flight missions an aircrew must deal with other risks in addition to DCS. Because of these differences and depending on the nature of the other risks, an aircrew may accept relatively high levels of DCS risk. Thus, an altitude decompression model must output a 0% to 100% scale of risk rather than a binary response.

In contrast to diving, an aircrew is most likely to develop DCS at the beginning of a flight during the ascent from ground level or during the early part of the time on station. As tissues denitrogenate at altitude, the risk of DCS diminishes. The recompression of returning to ground level is analogous to a diver's recompression for treatment of DCS.

Another important difference between diving and altitude is the difference in the composition of gases in a bubble. The tension of O_2, H_2O, and CO_2 remains relatively constant at depth and at altitude. However, the partial pressure of nitrogen decreases with reduction of pressure. For example, at 33 feet of sea water (fsw) depth, nitrogen composes 92% of the volume of the gas in a bubble. Similarly, at an altitude of 30,000 feet, nitrogen would compose 44% of the volume of the bubble. An altitude model must include this factor; a diving model does not.

The risk of altitude DCS could be completely eliminated by prebreathing pure O_2 to flush inert gases from body tissues. Unfortunately, the prebreathe time required to completely eliminate nitrogen from body tissues is so long as to be unacceptable for most operational situations. It is important that the model determine the length of time an aircrew must prebreathe to reduce risk to some acceptable level for a mission.

It is known that different tissues give up nitrogen at different rates due to the degree of tissue perfusion. When a diver makes a dive, tissues which are well perfused take up nitrogen first. These tissues quickly become supersaturated with respect to sea level. A diver's "slow" tissues become supersaturated much more slowly and for short dives are not as important in predicting risk during the diver's ascent. In altitude exposures, an aircrew is moving from ground level, where all tissues are equally saturated with nitrogen, to altitude where all tissues, including slow tissues, are supersaturated. Thus, in altitude excursions, in contrast to diving, slow tissues are extremely important.

The overall objective of all decompression procedures is the prevention or reduction of DCS injury. In the diving field, since Haldane's classic work was published in Boycott et al. (1908), there have been decompression guidelines (tables). For over 30 years, the Standard US Navy Air Decompression Tables have been the world standard for DCS prediction and prevention in the diving world. The diver can either look up in a table what needs to be done in decompression, or consult a dive decompression wrist computer which will provide decompression information. A dive computer contains a chip that has a decompression model programmed into it. A sensitive pressure transducer and clock provide the real-time input to the model. It is estimated that over 50% of recreational divers now wear dive computers. Similarly, the USAF altitude decompression computer development is aimed at hardware and software to guide exposure to the low pressure environment. A complete review of the dive computer development can be found in the proceedings of a recent workshop (10).

In contrast to the diving field, the aviation field has never had an organized, standard approach for predicting decompression sickness risk. Currently, in response to an inquiry concerning the DCS risk for a new or unusual altitude exposure, one of three approaches is used. A literature search can be initiated to find a matching study in a "scattergram" of over 50 years of DCS research. This is time-consuming and, often, unsuccessful. If the question is of high priority, a study specific to that issue can be initiated. This is also expensive and time-consuming, but will most likely provide the answer. Most often, however, the "best guess" approach is based on an

individual's memory and experience is used. A more rational approach would be to take this wealth of information from the last 50 years of DCS research and use it as the basis for the development of an altitude decompression model to serve as the operational "standard" for the altitude field. The current development of such an altitude decompression computer has the following objectives:

1. Provide "cockpit or pressure suit" real time readout of DCS risk under constantly changing conditions;

2. Provide capability for evaluating various options in high altitude mission planning;

3. Provide a tool for improved altitude chamber crew safety and manning control;

4. Provide desktop DCS risk analysis capability.

Figure 1 is a conceptual illustration of an altitude decompression computer.

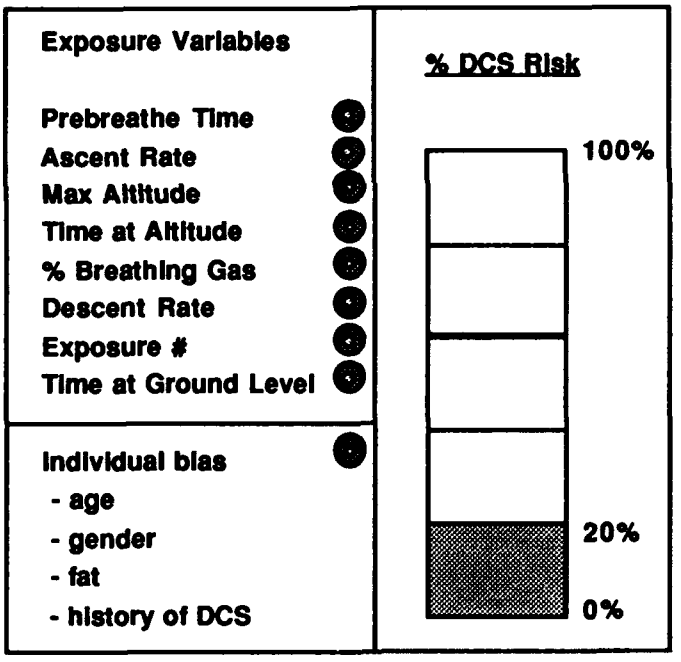

Figure 1. Proposed altitude decompression computer readout.

Approach

In 1989, an altitude decompression model development program was initiated at the USAF School of Aerospace Medicine*. The immediate goal of this effort is to define the architecture (or framework) and the algorithms (or software) for the decompression model. The final product will be a software package that will serve as the altitude decompression "standard" and will lead to hardware development to

*Now Armstrong Laboratory

provide the USAF with DCS risk assessment capability for a variety of operational settings. Figure 2 outlines the development process.

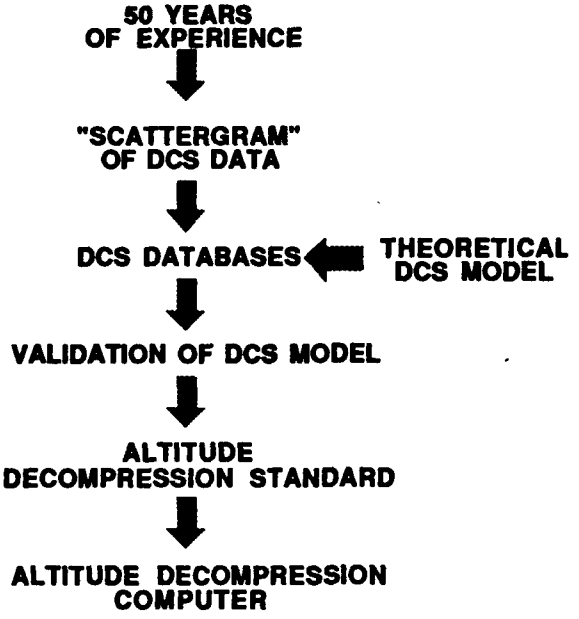

Figure 2. Progression of the development process for an altitude decompression computer.

A set of guidelines was defined that established the philosophy behind this model development.

1. It will permit DCS risk assessment rather than a binary yes/no product;

2. It will be a "compartmental" model incorporating several algorithms;

3. It will allow for biasing with predisposing factors and individual variability;

4. It will be tested against realistic altitude scenarios using existing databases; and

5. It will be validated on human subjects in altitude chambers using echo imaging and Doppler.

The initial stages of this process have been directed toward defining the theoretical algorithms. Existing decompression modeling approaches have been reviewed. Table I lists the "models" considered.

Table I. List of Decompression Modeling Approaches

1. Haldane (2)
2. US Navy Standard (4)
3. US Navy Exponential/Linear (14)
4. RNPL (7)
5. Hills Thermodynamic (9)
6. Hennessy and Hempleman (8)
7. Bateman and Lang (1)
8. Van Liew (15)
9. Yount (17)
10. Gernhardt (6)
11. NASA (3)
12. Maximum Likelihood (16)
13. Pneumatic and Electrical Analogs (12, 13)
14. Eger's Anesthesiology Model (5, 11)

Altitude decompression model framework

The first generation of the model is compartmental; it was used to test various approaches from several of the existing algorithms. It is a framework from which will evolve the working model. Figure 3 shows the 5 major components of this framework. Each of these tasks can be accomplished in a number of different ways. This approach will explore a few of the more promising methods in each compartment.

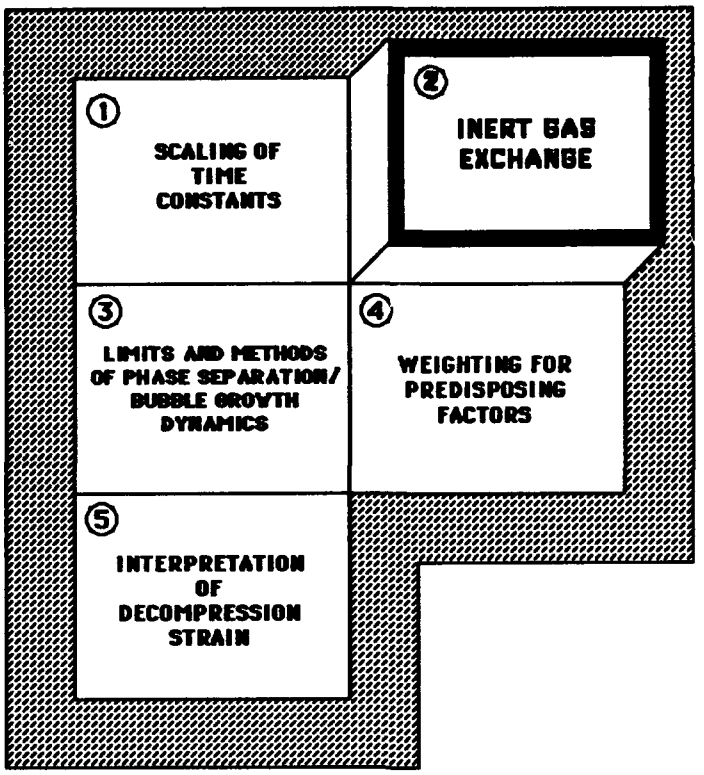

Figure 3. Five major components of the framework for the working model.

1. **Scaling of time constants.**

The three methods to be explored in the development of the model will be:

- arbitrary

- scaled

- statistically chosen

Each of these methods has its own advantages and limitations. Commonly, the tissue compartments are given by a range of representative time constants that are expected to adequately cover the spectrum of possible saturation half-times in the body. Arbitrary constants have been used successfully in diving for many years. However, for altitude, it is possible that since the quantities of gas are much smaller overall, the calculation of inert gas in the tissues may be more sensitive to the time constants used. It has been suggested that these time constants should not be chosen at random, but should be fitted to specifics of the type of exposure, exercise levels, bubble gas composition, etc. Some models choose their time constant(s) to optimize their fit between predicted incidence of DCS, and a specific database. The major

disadvantage of this method is that a statistical fit depends on the available data, which is highly variable for altitude DCS. A universal fit would be difficult to obtain. Still, this method would minimize the number of compartments used and, thus, the run time of the predictive model.

2. Inert Gas Exchange

The second block would determine the inert gas content of the body. The methods being explored are:

- perfusion-limited

  -Haldane method

  - anesthesiology method

- diffusion-limited

- perfusion-diffusion

3. Limits and methods of phase separation

Simple extrapolation of a gas exchange model successfully used in diving to one for altitude does not work. An altitude model requires the integration of bubble theory. Concepts on bubble formation, bubble growth and bubble nuclei will be reviewed and included in the model as appropriate.

4. Weighting for predisposing factors

There are several factors which have been shown to have a significant effect on the outcome of DCS, but which would be difficult to include as part of the overall algorithm. Age, previous injuries, physical fitness, diet, complement activation, etc., have been implicated as affecting susceptibility to decompression sickness. A statistical means of weighting the model to deal with these variables would be desirable.

5. Interpretation of decompression strain

The end result of this model should be a type of decompression strain or decompression sickness risk. Since the incidence of DCS is accepted to be a function of inert gas content of the tissues as it relates to bubble growth, and since bubble growth in turn affects the inert gas content of the tissues, it seems reasonable that the size of the gas phase should be the value to represent decompression strain. This value could, in turn, be fed into a dose-response equation, such as is used by NASA, or it could be evaluated using some other statistical method.

While these portions of the model are not always intrinsically separable, they can be manipulated so that they may be analyzed individually. Since a model can be formed using all or part of these components, it makes sense to tackle these considerations in order of importance, rather than order of execution in the working model. The most important consideration is the determination of the inert gas content of the tissues. Obviously one must know how much gas the body contains before it is decided either how likely the formation of bubbles is, or how large will be the gas phase if bubbles do form -- this calculation forms nearly the entire model for many approaches, including the U. S. Navy model, and the RNPL model. Next would be the calculation of the size of the bubble phase to provide an index of "decompression strain" from which to predict the likelihood of DCS. While it could be argued that supersaturation ratios also provide an index of decompression strain, and with half the amount of calculations involved, bubble formation would delay the denitrogenation process, and a model acknowledging this consideration would be more likely to present an accurate portrait of the risk factor vs time. Going along with this portion of the model would be the determination of the degree of supersaturation that could be tolerated before a gas phase would be initiated -- this step would incorporate a time lag between the event of supersaturation and the initiation of bubble growth, as well as altering the initial conditions of bubble growth. With these calculations completed, the next most important consideration would be to correlate the resulting "decompression strain" in an average individual to a factor expressing risk of decompression sickness.

Validation

As a test bed for this model development process, a set of 15 altitude exposure scenarios were compiled. These scenarios represent a spectrum of operational exposures that will be used as computer simulations to guide the model development and to keep it in touch with reality. These scenarios are currently in use and, in general, the DCS risk is known. The altitude scenarios for model testing include:

1. Type I altitude chamber training flight
2. Type II altitude chamber training flight
3. Type III altitude chamber training flight
4. Type V altitude chamber training flight
5. U.S. Army HALO training flight
6. U.S.A.F. HALO training flight
7. DCS research flight #1
8. DCS research flight #2
9. DCS research flight #3
10. Space Shuttle EVA
11. T-37 cross country flight
12. High altitude reconnaissance flight
13. Hi-Lo-Hi routine flight
14. Hi-Lo-Hi emergency flight #1
15. Hi-Lo-Hi emergency flight #2

Finally, the model will be validated by exposing human volunteers to selected altitude profiles in altitude chambers. The subjects will be monitored for DCS symptoms and venous gas emboli with echo imaging systems.

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DR. BUTLER: Do the current definitions of decompression sickness, Type I, Type II lend themselves to a computer like this?

DR. PILMANIS: No. It is very difficult to compare many of the databases. We are relying on the database that we have here at USAFSAM from the chamber research. We have controlled these experiments as much as possible, or at least we know what we did.

CAPT GARDETTO: How much importance do you place on individual hydration level as one of your biases?

DR. PILMANIS: Our approach at this time to these factors, such as hydration, is to obtain a rough number out of the literature for biasing the model. Hydration is a factor. How much of a factor is yet to be determined.

COLONEL SHEFFIELD: In your model, you will need to have some way of including some of these variables to account for the unusual circumstance; the explosive decompression to altitude, the extreme cold, or the terrible temperature control system in some of our weapons systems that result in limbs being very cold, while the chest is very hot, etc. You have a tough challenge to determine how to account for these very widespread variables in your model.

DR. PILMANIS: Nevertheless, we have reached a point with altitude DCS that requires the development of an organized way of assessing decompression risk. The USAF currently does not have one. Calls from operational units requesting information on DCS risk for specific altitude exposures are often very difficult to answer with any accuracy. The best we can do is search the literature for comparable studies and than make an educated guess. Or, if there is little or no data available, a new study may be initiated. An acceptable altitude DCS risk model would greatly reduce the necessity for case-by-case studies of unique flight scenarios.

DR. NORFLEET: I recommend that all studies have O_2 monitoring in the masks because I am concerned about poorly fitted oral nasal masks.

DR. PILMANIS: Periodically, we do check the O_2 in the masks and the results are always in the 99% range.

COLONEL SHEFFIELD: The measurements that have been done in our chambers have shown very effective USAF aviator oxygen masks. Occasionally, the very small faced person may have a fit problem. Through repeated fittings by our life support shops when they come through the chambers for training, the problems are solved, and they are allowed to use their own equipment.

MAJOR GARRETT: One method to ensure that inside observers are denitrogenated is to put them all on "safety pressure." If there are leaks, they are outboard leaks.

DR. VANN: You really have two kinds of data, primary and secondary. Your primary data, the data that you calibrate your model with, is laboratory data where everything is controlled very carefully. Your secondary data come from field studies in which you cannot control such things as wave height or fit of your oxygen mask or other things. You need to get feedback from the field as to how well the procedures that you design in the lab are working operationally, because people may not be applying them correctly. There may be equipment failures that are easily controlled in the laboratory. It needs to be recognized that it is not as simple as just having good laboratory data. The application is frequently a problem in itself.

DR. PILMANIS: That is a very broad spectrum of databases.

DR. VANN: Yes, but there is no getting around it.

MR. GILBERT: NASA has adopted what we refer to as a "French" mask, a full head mask that uses neck seals to maintain integrity. We have had very, very good luck with this mask as far as reducing the leaking for nitrogen washout, and any leak that does occur is an outboard leak. We sometimes have to use little clips to tighten up the neck band but it is a very effective means of preventing any inboard leaks. We are quite assured we are getting 100% oxygen.

DR. PILMANIS: Here at USAFSAM we also use the "French" masks in all our research flights. However, they cannot be used in the field.

DR. BAGIAN: I agree with Dr. Vann's comment about the need for feedback from the field. However, sometimes feedback is not enough. There is such a wide spectrum of operational environments. The feedback you get may not be as accurate as you might think. Often there is another method. Go out and work with the operators and see what they really do. The way people wear their gear in the chamber when everybody is watching is one way, and the way they fly is another way. The only way to really see that difference is to observe them at the flight lines.

"COMMENTS ON APPLICATION AND SUCCESS OF DECOMPRESSION THEORY"

by R. W. Hamilton, PhD

DR. HAMILTON: I offer two anecdotes to point out some difficulties in the application of decompression theory.

It was mentioned that divers often use some "fudge factors" to make the tables work right. One of the unwritten rules that the old chiefs know, that is not in the book, is called the two plus two rule. If you are within two minutes or two feet of the end of the table, for example if you are at 28 minutes on a 30 minute table, you go to the next table. In other words, there are inaccuracies in measurement, there are inadequacies in the tables, you have got leaking oxygen masks, you have got all these things that give you reason to put in a little bit of "J" factor.

1. A few years ago, the U.S. Navy Safety Center did an analysis of some 6,000 dives. They examined these dives with respect to the two plus two rule. They found that there was no statistical difference between dives that were less than the two plus two limit, i.e., 28 minutes or less for the table to go with a 30-minute bottom time, and dives 28 and 30 minutes for the same table. And there was a bends incidence in both alternatives.

Then they looked at a number of instances when a 30-minute table was used, but the dive was a 31-minute or longer dive. In other words, they had actually violated and gone beyond the table. No bends were reported in that group.

Does that mean it is safer to go beyond the limit of the table? Not at all. It means that if you make a mistake, you do not tell anybody. In other words, if there is no decompression sickness and you did not do it right, you can put that down on the form correctly. But if there was DCS and you did not do it right, you make the form look like you followed the table exactly. This is the kind of thing we are facing in regard to the business of getting good data back from the field to plug into these models.

2. There is an ongoing study in the North Sea, directed at documenting data from the dive logs of diving companies. The information on the logs was not extensive. It included the table that was used and the actual time and depth of the dive, and whether or not there was decompression sickness. One part of the study showed an interesting result.

Some 6,000 of these dives were no-stop dives; i.e., they did not have any decompression stops. There was a very low incidence of decompression sickness in those dives. Perhaps a half dozen cases in some 6,000 dives. However, the cases that did occur were neurological decompression sickness. One interpretation is that the mild Type I cases simply were not reported. Another conclusion could be that, no

matter how well you do things, sometimes it just does not go right and when it goes wrong, it goes very wrong.

LT COL DIXON: I was asked earlier if chokes was overwhelmingly seen in all of these cases. To answer that question, I think it is important to point out that if you wait long enough, yes, you are going to see enough damming up of bubbles in the pulmonary circulation and then you are going to have what we all generally consider to be chokes. But that is not always the first symptom that we see. In fact, most often you are going to see neurologic, or neurocirculatory, symptoms before you see chokes. The physiological mechanism of chokes may involve specific vascular beds. Various researchers have stated that offloading nitrogen is faster with exercise. That also means that the offloading of nitrogen in other beds is reduced. I used the splanchnic vascular bed as the example. That particular bed may not be critical to us, but maybe we need to know that before we discount it.

DR. VANN: Are you saying that perhaps it is the large gas-containing reservoirs that are important for chokes or cardiovascular collapse?

LT COL DIXON: It is a possibility. I do not have supporting data. But I do not think we should discount it. Are we really trying to minimize simple bends or are we trying to minimize the overall threat to the individual, whatever the nitrogen source.

COLONEL SHEFFIELD: I think it is important to point out that many of these deaths, especially in the chamber flights, were 38,000 feet/two-hour exposures, exercising with deep knee bends, deliberately trying to screen out those individuals who are benders. If they did not bend on one day, they would bring them back the second day and try it again. They used the chamber in the 1940s as a screening tool.

DR. WEBB: You mentioned that about 9 out of 20 of the individuals were overweight/obese. What is the population of all global aviators that are overweight/obese? Is it not fairly close to that number? Is fat really important if the individuals are within the USAF criteria in terms of desirable weight for their height?

LT COL DIXON: There are a few of us in this group that deal with this problem administratively. In the 1987 case in which the retired lieutenant colonel died from chokes, he had been refused refresher training in one of the USAF chambers, but managed to get himself scheduled at another one. This is an individual who probably had heard the decompression sickness story six times during his career, and still chose to ignore the symptoms at altitude.

DR. BALLDIN: When talking about obesity, I think you have to differentiate between diving decompression sickness and altitude decompression sickness. In bounce diving, obesity might be a positive factor. On the other hand, with saturation diving and altitude exposures it might be more dangerous to have more fat in the tissues. There is an increased risk of decompression sickness in females, compared to males. Females have more fat. On the other hand, I would also say that in many

provocations of decompression sickness, in both humans and animals, there are certain individuals who always get decompression sickness, and others who do not, with the same exposure. It is important to track the individual thresholds. Thresholds will be pretty constant for certain individuals. Sometimes it does not matter whether they are obese or not.

DR. BUTLER: "Chokes" is probably one of the hardest aspects of DCS to define. I can think of seven or eight specific symptoms that are listed under chokes. If we go through the etiology of each of those symptoms we find that they are completely different. For example, one symptom is described as some sort of a coughing reflex. Is this an irritable feeling in the back of the throat, or is it a neurogenic reflex to profuse pulmonary edema, and is there a mediator or a receptor involved. We need to be careful when we talk about chokes until we can redefine what chokes are.

DR. HAMILTON: Are you regarding chokes as the lung vasculature filling up with venous bubbles?

DR. BUTLER: That is only one aspect. Pulmonary vascular bubbles are related to chokes. I think that has been well established both with decompression bubbles detection and with gas injection studies. However, that definition does not give you the mechanism for what the choke is. This catchall phrase of chokes is not adequate. Some will call chokes a neurological type. What does that mean? Does it involve the central nervous system, does it involve the spinal cord?

DR. LAMBERTSEN: It is very important that we have a restatement of what is the comparative circumstance of diving and aerospace. If you do not start with saturation, then there is no really good comparison between the two. The difference is to get time at altitude before you have troubles. That is not the way it is in diving saturation decompression. The objective is to come from one saturation level to another, without having any problems, no matter how long you stay at that next saturation level. The same has been done in aerospace work by going from air breathing to oxygen breathing for days. This regimen will result in no potential for decompression sickness at all. You can stay there forever if you stayed on oxygen long enough. The highest interest is in the middle ground between that long oxygen exposure where you will never get decompression sickness no matter what you do, and the situation where you do not denitrogenate or prebreathe at all, and are going to get DCS fairly soon.

The question then is what is the level of decompression illness you are going to get in this middle ground of going from saturation to an excursion from saturation? That level is going to differ, depending upon things like exercise, and other predisposing factors. However, you cannot just take exercise as though it is a lump of physiology, and say it is going to do this or that. The importance of these factors will depend upon what stage of decompression you are in. It is going to make a difference whether venous gas embolism has occurred. This circumstance is in contrast to exercise simply resulting in change in blood flow to the muscles, causing constriction

in other tissues, and causing pain because with exercise you have caused a vascular change somewhere else. Therefore, you do not remove the gas from that other place while you are dumping it hard into the lungs through the venous side from other tissues. Thus, words like exercise or chokes or pain or peripheral circulation or neurological should not be used. The terminology here is just too loose.

However, the one thing that is definite is that you are going from one saturation state to a transient excursion in most hypobaric work in contrast to what is happening in diving, going from saturation to another saturation level. The principles have to be the same, but the circumstances are different.

MR. WALIGORA: I would like to further discuss procedures that might reduce prebreathe in an operational situation. NASA is interested because we are currently using very long prebreathe times operationally. Are any of the conditions that have been described to improve denitrogenation currently operationally applicable.

DR. BALLDIN: I think immersion is impractical. Also, you already have the immersion situation in weightlessness. But the increased temperature might be a practical method.

In addition, the drug, terbutaline, might potentially be used. It increases the peripheral circulation and decreases the risk of decompression sickness in rabbits. However, human experiments will be needed. That is my suggestion; try increased temperature and the drug. I do not know if the negative pressure breathing could be used during weightlessness

I think that immersion and zero G, are the same thing, and you will not gain very much more with it.

DR. BAGIAN: The temperature could be controlled with the astronauts liquid-cooled garment.

MR. WALIGORA: The low level of exercise would minimize bubble formation and save maybe an hour on a four-hour prebreathe.

DR. VANN: The three and a half hour prebreathe, which contains 2 hours and 15 minutes of exercise, was equivalent to roughly an 8-hour resting O_2 prebreathe in terms of the decompression sickness. It reduced the DCS to essentially zero. Whether or not that is operationally practical, the individual operational managers are going to have to decide.

DR. VANN: You have to realize that a long period of exercise is required in order to eliminate nitrogen from those slow tissues that you are dealing with for pain only symptoms. During that time, they cannot do anything else. It will fatigue them a little bit, but 25 watts is not much more tiring than walking at three or four miles an

hour. This laboratory investigation suggested that the light exercise has the greatest effect of any other procedure we tried in reducing DCS.

We also looked at 50 watts of exercise which is noticeable in terms of fatigue. There was not much of an improvement over the 25 watts. It may be that the 25 watts of exercise is abolishing some of the vasoconstriction that occurs during oxygen breathing, whereas the 50 watts does not.

Operationally you have got a tradeoff. It is inconvenient to get bent. It is also inconvenient to exercise for that period of time. I do not think it would work with short periods of exercise. I think it has to be for a fairly long period of time.

DR. BUTLER: You have to be careful that these vasodilator drugs are tested when used in situations where there are bubbles circulating. If you lose your ability to control blood pressure, for example, and block off enough pulmonary circulation so that your cardiac output is down and your right heart is straining, and you do not have any afterload, you may go into a hypotensive situation from the drug. We have also found in other studies using pulmonary vasodilators that bubbles went into the arterial circuit easier. One should be very careful with this drug usage. There are two approaches. One is using a drug to prevent bubble formation, vis a vis denitrogenation. The other one is what happens if you use the drug and there are bubbles circulating? These need to be carefully evaluated before application.

DR. BAGIAN: It is going to be difficult to evaluate the blood pressure regulation. Orthostatic loads play a large role in blood pressure regulation, or your inability to regulate. However, in zero G that is not a factor.

It would be reasonable to think that if you had something that was really inhibiting your control of blood pressure, and if you could pass a ground (IG) test, then, on orbit in zero G, you probably could pass for sure because the circumstances are more demanding at IG.

DR. NORFLEET: Cardiovascular pharmacology is advancing by leaps and bounds in terms of highly selective compounds that are convenient to use and that have very rapid onset and offset. I would like to see a lot more effort directed towards coming up with application drugs.

DR. LAMBERTSEN: When using drugs, it should be kept in mind that the local circulation is what is important, not cardiac output. The cardiac output is sought only when you are talking about the whole body. You are not dealing with the whole body when you are dealing with decompression sickness.

When you dilate somewhere in the body, there is a compensatory action and massive constriction may happen elsewhere. Pharmacology may be important but it is not going to be simple because the circulation is built with multiple corrective mechanisms and that is why I said whole cardiac output is not critical. Local circulation is.

1990 Hypobaric Decompression Sickness Workshop

Session Three: DECOMPRESSION IN SPACE

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SHUTTLE AND SPACE STATION EVA

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Introduction: Decompression Procedures in Early Programs

This paper will review the decompression procedures used in the U.S. space program with emphasis on the experience up to the present in the Space Transportation System (STS)/Space Shuttle Program and plans for the Space Station Freedom (SSF) Program. It will be useful in understanding these procedures if we review briefly the protocols used in earlier programs to prevent altitude decompression sickness (DCS) prior to extravehicular activity (EVA).

In the Gemini and Apollo programs, the probability of a crewmember experiencing DCS was greatest at the time of launch when the pressure decreased from sea level to 5 psi (a pressure equivalent to 27,000 feet of altitude). Therefore, the crew breathed 100% oxygen for 3 hours prior to launch and depressurization. The Apollo cabin atmosphere was maintained at 100% oxygen until the Apollo fire on the launch pad, after which it was changed to a 60%-40% mixture of oxygen-nitrogen. This mixture came close to 100% oxygen at the 5 psi pressure in orbit. After several days at this pressure the EVA crew depressurized from 5 psi to 3.8 psi for the lunar exploration (1). There were no reports of decompression sickness on these flights. However, one crewmember later described bends-like pain which he experienced on both Gemini and Apollo missions (2). This pain occurred after launch as would be expected based on the decompression schedule. Table 1 lists the cabin and suit atmospheres and the decompression procedures by program.

Table 2 lists the crewmember hours both in 0-g and 1/6-g for past and current flights. An important parameter in relation to the risk of DCS in space is the work levels of astronauts during EVA.

Table 3 summarizes the estimated metabolic rates by program. The average work rates are moderate. However, short-term excursions to significant higher levels have been experienced, especially in the space construction simulation accomplished in Shuttle flight 61-B in November 1985 (3).

Table 1. Cabin Suit Pressures

PROGRAM	CABIN ATMOS.	PREBREATHING HOURS	SUIT ATMOSPHERE (PSI)
Gemini/Apollo	5 psi/100% O_2 changed to 60% O_2, 40% N_2 on launch after Apollo I fire	3	3.8
Skylab	5 psi/70% O_2/30% N_2	3	3.8
Apollo-Soyuz	Soyuz 10 psi/33% O_2 Apollo 5 psi/100% O_2	No EVA	------
Shuttle	14.7 psi/21% O_2	Staged or 4 hours O_2	4.3
SSF	14.7 psi/21% O_2	TBD	4.3

Table 2. US EVA Experience (Crewmember Hours)

	MICROGRAVITY	1/6-G
Gemini	11.9	158.7
Apollo	7.3	
Skylab	81.4	
Shuttle	136.0	
Total	236.6	158.7

Table 3. EVA Metabolic Rates (kcal/hr)

PROGRAM	MEAN RATE FOR ENTIRE PROGRAM	RANGE OF RATES FOR ENTIRE EVAs	RANGE FOR SELECTED TASKS
Apollo (1/6 G)	235	197-302	99-450
0 G	151	117-504	
Skylab	238	145-330	100-500
Shuttle	197	152-275	100-384

EVAs in the Space Shuttle Program

When the Space Shuttle Program began, it was apparent that nitrogen elimination prior to decompression for EVA was going to have a great impact on crew time. The cabin pressure is 14.7 psi with 21% oxygen and 79% nitrogen. The entire prebreathe scenario must be accomplished 1 orbit just prior to the EVA. At first it was planned to eliminate nitrogen by using a straight oxygen prebreathe of 3.5 hours. However, data from the USAF School of Aerospace Medicine* indicated that astronauts interrupting prebreathing while transitioning from oxygen mask to pressure suit could breathe a significant amount of inert gas (4,5). Estimated makeup times were calculated, and it was concluded that the procedure of transitioning from oxygen mask to suit would incur an excessively high risk of renitrogenation. The alternative procedures were either to prebreathe in the space suit or to reduce the cabin pressure in a staged decompression. The preferred technique was the staged decompression since it permitted the crew to carry out their normal duties while breathing a cabin air with reduced partial pressure of nitrogen (PN_2). Table 4 summarizes the planned and actual durations of staged decompression and duration of the EVAs. In the first EVA during STS-6, the straight oxygen prebreathe was used for 3.5 hours in the space suits prior to decompression to 4.3 psia.

The remainder of the EVAs used the staged method which involves a 60-minute period of oxygen breathing by the EVA crew prior to cabin atmosphere depressurization to 10.2 psia with 26.5% oxygen (2.7 psi of oxygen), equivalent to the oxygen partial pressure (PO_2) at 5,000 feet. With the variation in pressure and composition, the lowest oxygen equivalent that would be encountered is 2.3 psi, which is equal to about 7,500 feet. Likewise the highest nitrogen equivalent that would be encountered is 7.7 psi, the highest pressure to which the crew would equilibrate during the prebreathe. The minimum required duration of equilibration was 12 hours at the time of the past Shuttle EVAs. This duration has since been changed to 24 hours to achieve a more conservative decompression ratio. Most of the EVAs had durations of equilibration considerably longer than the 12 hours planned (Table 4).

At the end of this stage of decompression, the EVA crewmembers donned their spacesuits and breathed pure oxygen for a minimum of 40 minutes. Again, the actual times, as shown in Table 4, were frequently longer than the minimum requirement. When two EVAs are being done on a flight, as was the case on five flights, the cabin remained at 10.2 psia between the EVAs and was not returned to 14.7 psia until all EVAs for that mission were completed.

No signs or symptoms of decompression sickness have been reported during the Space Shuttle Program. However, data collected in 1-g during simulated EVA profiles has shown a significant percentage of subjects with both intravascular bubbles and DCS (Fig. 1). These data indicate that the probability of a DCS incident severe enough to abort an EVA is about 5% with a 1.65 ratio of estimated tissue nitrogen to suit pressure. Most of the DCS cases reported in the altitude chambers are mild limb bends which could remain unnoticed in the operational environment with the normal

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Table 4. Shuttle Denitrogenation Protocol: Planned vs. Actual

EVA #	FLIGHT	PREBREATHE (MIN) PLANNED	PREBREATHE (MIN) ACTUAL	TIME @ 10.2 psi PLANNED	TIME @ 10.2 psi ACTUAL	IN SUIT O2 (MIN) PLANNED	IN SUIT O2 (MIN) ACTUAL	EVA DURATION (HRS)	TISSUE PN2 RATIOS*
1	STS-6	≪ 3.5 HR PROTOCOL ≫						4.0	1.80
2	41-B		75		HRS:MIN 24:24		75	5.5	1.54
3	41-B				39:25		50	6.0	1.58
4	41-C		75		21:37		40	3.0	1.67
5	41-C				60:23		40	7.0	1.63
6	41-G		59		24:32		43	3.5	1.65
7	51-A	60 MIN	87	12 HRS	22:47	40 MIN	76	6.0	1.55
8	51-A				36:40		46	6.0	1.59
9	51-D		68		19:55		54	3.0	1.64
10	51-I		67		25:19		41	7.5	1.65
11	51-I				15:12		49	4.5	1.43
12	61-B		100		20:10		40	5.5	1.67
13	61-B				40:02		45	7.0	1.60

* Ratios based on 360 tissue half-time

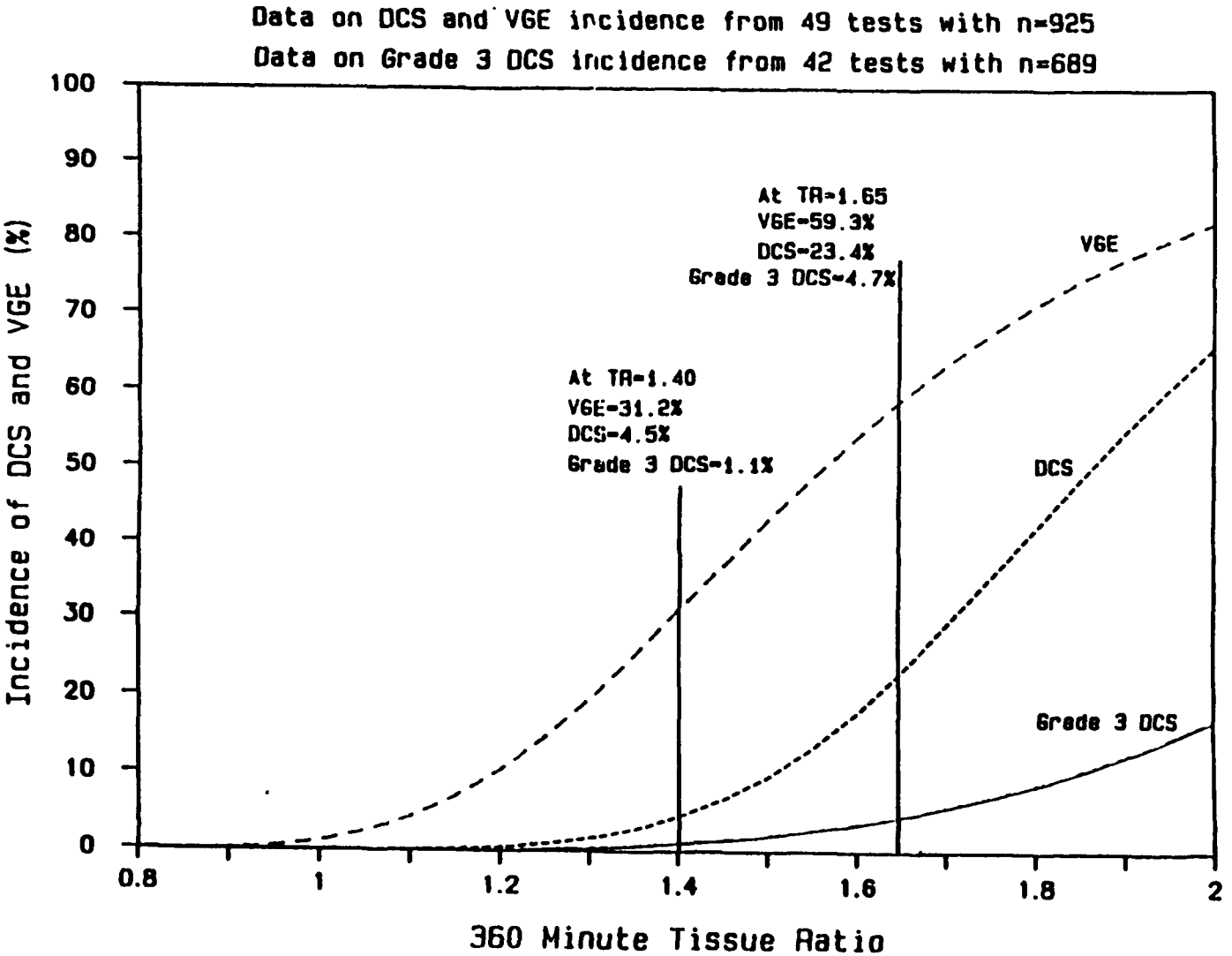

Figure 1. Incidence of VGE and DCS as a function of 300 min tissue ratio.

fatigue of working in a spacesuit. Altitude chamber test and training runs done with test subjects or crewmembers in 1-g seldom have reported cases of DCS, probably for the same reason. The discomfort of working in a spacesuit may mask many of the symptoms detected in unsuited subjects. Proposals have been made for future studies to investigate the possible effects of microgravity on risk of DCS.

Space Station Freedom (SSF)

Based on the data available on risk of DCS (Fig. 1), a more conservative decompression ratio (1.4) was recommended for the Space Station Freedom Program. The proposed decompression ratio is 1.65 for both construction EVA for STS and for the minimal contingency maintenance EVA now anticipated for Space Station. Table 5 lists the estimated crewmember hours of EVA to assemble SSF (6). The projection for assembly alone is greater than the total EVA time of all past programs combined. Although the Space Station program has plans for 12 EVAs per year after assembly, some estimates predict a greater requirement. For example, a recent study by the SSF External Maintenance Task Team estimated the average EVA requirement over a 35-year period to be 5.3 two-crewmember EVAs per week (7). Therefore, it appears that there will be a regular and repetitive requirement for EVA. Moreover, we do not have data to show the physiological effects of repetitive decompressions in microgravity over long durations; nor do we have data to indicate what impact the physiological changes due to microgravity over time have on the crewmembers' ability to perform EVAs. We are recommending that a more conservative R value of 1.40 be used for high frequency EVA missions such as space station construction and on space station EVAs if the EVAs are frequent.

To achieve a more conservative decompression, several methods have been considered, such as higher pressure suits, use of the crew lock for staged decompression, or an extremely long-duration prebreathing procedure. A higher pressure suit is not being further developed at this writing for SSF construction or early SSF operations, and the long prebreathe (5.75 hrs) is not practical. Therefore, it would seem that the use of some type of staged decompression or a reevaluation of the need for a higher pressure (e.g., 8.3 psi) suit is in order.

Beyond SSF

There are now plans for a lunar station and missions to other planets such as Mars within the next quarter century. The use of EVA for construction, repair, maintenance, and exploration is expected. The physiological basis for safe decompressions and other physiological aspects of EVA should be carefully considered in planning cabin and suit environments and mobility systems. Changes in physiology as a function of mission duration will be monitored and used in making decisions related to environmental control and life support systems, as well as planning for appropriate work levels. If a low pressure suit is used, staged decompression, or even EVA modules with a transition atmosphere allowing for 24-48 hours of equilibration before

Table 5. Space Station Freedom Assembly

PROJECTED EVA's	CREWMEMBER HOURS
2 Planned	24
1 Growth	12
1 Contin (SSF)	12
1 Contin (STS)	12
Max EVA Time Per Flight (SSF)	48
Tot. EVA Time (SSF) 29 Flights	1392
Tot. EVA Time (SSF) 34 Flights	1632

going to a suit system, could be developed. If a suit is developed with an atmosphere close to that of the cabin or habitat, it may be possible to simply don the suit system and go outside immediately. Optimization of extravehicular activity requires that all physiological, structural, operational, and safety considerations be considered in an integrated fashion as future cabin, suit, and mobility systems are developed.

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COLONEL SHERMAN: Could you explain the rebreather system? Are you still using it?

MR WALIGORA: No, that rebreather system was eliminated when we went to the 10.2 psia procedure. We currently have two options. The primary option is to use the staged decompression and the 40-minute prebreathe. This is done with an open mask which dumps into the cabin. The other option is to prebreathe for 4 hours. However, that 4-hour period has to be done in a suit. We can't dump gas into the cabin for four hours.

COLONEL SHERMAN: The rebreather system itself did work?

MR WALIGORA: It did work. It gave you warm, damp air to breathe, but it worked. It was never brought into final production.

DR. BUTLER: What are the requirements for repetitive EVAs?

MR WALIGORA: Right now there is a requirement that we do EVAs on alternate days. We don't do them on consecutive days with the same crew. That requirement was not based on decompression sickness. It was based on crew fatigue.

Development of a Predictive Model for Incidence of Decompression Sickness under Experimental Conditions

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Introduction

The capability of predicting the susceptibility to a decompression sickness (DCS) event of an individual who is exposed to hypobaric decompression is of great importance to the U.S. space program. The suits used during Extravehicular Activity (EVA) are pressurized with pure oxygen to 4.3 psia (29.6 kiloPascals/kPa). The standard pre-EVA decompression procedure described in Waligora, et al. (1984) involves decompression of the Shuttle cabin to 10.2 psia (70.3 kPa) 12 - 24 hours prior to onset of EVA. Immediately prior to EVA, astronauts prebreathe 100% oxygen during the 45 minutes they are suiting up. To date, no instances of DCS have been reported to Flight Controllers/Flight Surgeons.

Over the past 10 years, researchers at NASA and the USAF School of Aerospace Medicine* have been engaged in research into the effects of oxygen-prebreathe denitrogenation, of exercise at varied altitudes, and of staged decompression on the incidence of DCS and concomitant circulating microbubbles (CMB), also known as venous gas emboli (VGE) in volunteer human test subjects. To date, over a thousand human exposures have been tabulated with data from the researchers at Duke University and other cooperating facilities. This paper discusses the development of 2 models used to enable prediction of the occurrence of CMB and DCS based on these data.

Procedures and Methods

Two models are described in this paper: (1) a nonlinear regression, using Hill's dose-response model, based on Conkin et al. (1987, 1990) and (2) a logistic regression model, as described in Hallum (1990); both use the 360-minute half-time tissue ratio (TR360) as a covariate. Since the data derived from decompression runs are dichotomous (either 1/yes or 0/no) for each individual exposure, features of the

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logistic regression approach based on binomial rather than normal distribution of data seem appropriate.

While the standard regression model of a response variable y on a covariate x is one that attempts to model the conditional mean, the logistic model looks at the conditional probability of a dichotomous random variable y that takes a value of 0 or 1 modified by the variable x. The logistics model diagnostics rely primarily on Chi-square Goodness-of-Fit as opposed to the squared multiple correlation coefficient (R sup3(2)) used for the Hill dose-response equation (Hallum, 1990). The logistic modeling also takes into account the number of individual exposures, thus allowing for adjustment of the confidence intervals dependent upon sample size. In both calculation methods, the models were modified to reflect zero probability at a TR = 0.78, the tissue saturation at 760 mm Hg, using the expression (TR360 - 0.78).

I. The first model (Conkin, 1987 & 1990) used the Hill's equation in the form of:

$$p = f((TRx - 0.78)sup5(n),[(TRx - 0.78)sup5(n) + (TR50 - 0.78)sup5(n)])$$

where p is the probability of a positive response, TRx is the dose (i.e., 360-minute half-time tissue ratio), and TR50 is the dose where $p = 0.5$.

II. The second model used the logistic form (Hallum, 1990) with one variable (x = [ln TR360]) in the form of:

$$p = f(esup5(a+b\ ln[TR360-.078]),1 + esup5(a+b\ ln[TR360-0.78]))$$

where a = constant; b = regression coefficient for TR360

Additional information regarding the development of the modified Hill's equation model structure or the use of it as a special case of the standard logistic model can be obtained in Conkin, et al. (1987, 1990) and Hallum (1990).

Results

The data used in the development of these models are standardized to a 360-minute Tissue Half Time (TR360), with exposure duration of 6 hours. The rationale for use of the 360-minute tissue half time is discussed in Conkin, et al. (1987; n=607). In support of this initial selection of TR360, we compared the correlation coefficients derived by fitting observed versus predicted incidence rates of both DCS and CMB using Tissue Half Times from 90 through 900 minutes. These calculations showed maximum correlation at 360 minutes. This selection of TR360 as the model tissue half-time has been supported by subsequent survival analysis on NASA/JSC data (n=426), a subset of the larger combined NASA/USAF data set.

The variables in the data sets referred to below are described in Conkin (1987). A brief review is as follows: **CMB**: circulating microbubbles are determined by auditory signals from an ultrasonic bidirectional Doppler system at the precordial location of the pulmonary artery; **DCS**: decompression sickness symptoms are recorded from subject reports of the presence or absence of the generally accepted symptomatology classified as Type I. **DCS Type I - Grade 3** describes a subject's symptoms which interfered with the performance of assigned tasks in the chamber, resulting in early termination of the test. These symptoms were treated with hyperbaric recompression if the symptoms had not resolved upon descent from altitude, or if their onset was delayed during a 48-hour period after hypobaric exposure.

The 1990 NASA/USAF data set (Conkin, 1990) was decreased during the logistic model development, when it was found that data outliers (3 data groups with TR >1.85) degraded the Goodness-of-Fit tests of the logistic model. Hallum (1990) shows that Chi-Square Goodness-of-Fit "p" values increase from 0.00 for DCS and for CMB to 0.358 for DCS and 0.158 for CMB when these three groups were excluded from the model. These groups had been included in the original 1990 data set to extend the Tissue Ratio range, however, subjects in these 3 USAF experiments performed exercises which were significantly different from those in the rest of the data set: chair high knee bends and arm lifts using 5-pound weights. Since the intent was to develop a simple usable model, while providing the most precise estimates of risk by reducing the range of the confidence limits, these groups with TRs above 1.85 were dropped (screened) from the model data. Thus, the total number of individuals included in the data set is 782. Because USAF test termination criteria use a DCS Grade 2 pain level as an endpoint, these TR groups did not present any Grade 3 data, leaving the sample size of this data set unscreened.

The equations modeling each of these respective data sets are presented below.

RESULTS FOR SCREENED CMB (VGE)

(Conkin et al. 1987)

%CMB = 100 * f((TR360 - 0.78)sup5(3.08),(TR360 - 0.78)sup5(3.08) + 0.47)

(Conkin-unpublished, 1990 [6 hour])

%CMB = 100 * f((TR360 - 0.78)sup5(3.56),(TR360 - 0.78)sup5(3.56) + 0.38)

Please see Figure 4 for graphic representation.[1]

[1] Figures 1-6 are located following the Discussion section.

(Hallum, 1990)

p = f(esup5(.9675+3.615 ln[TR360-0.78]),1 + esup5(.9675+3.615 ln[TR360-0.78]))

Please see Figure 1 for graphic representation.

Chi-Square Goodness-of-Fit: C.C.Brown=2.450; df=2; p-Value=0.105

RESULTS FOR SCREENED TYPE 1 DCS

(Conkin, et al. 1987)

%DCS = 100 * f((TR360 - 0.78)sup5(4.24),(TR360 - 0.78)sup5(4.24) + 2.16)

(Conkin-unpublished, 1990 [6 hour])

%DCS = 100 * f((TR360 - 0.78)sup5(6.29),(TR360 - 0.78)sup5(6.29) + 0.93)

Please see Figure 5 for graphic representation.

(Hallum, 1990)

p = f(esup5(.0034+6.323 ln[TR360-0.78]),1 + esup5(.0034+6.323 ln[TR360-0.78]))

Please see Figure 2 for graphic representation.

Chi-Square Goodness-of-Fit: C.C.Brown=2.057; df=2; p-Value=0.358

RESULTS FOR TYPE 1 - GRADE 3 DCS

(Conkin, et al. 1987)

%DCS3 = 100 * f((TR360 - 0.78)sup5(2.50),(TR360 - 0.78)sup5(2.50) + 17.61)

(Conkin-unpublished, 1990 [6 hour])

%DCS3 = 100 * f((TR360 - 0.78)sup5(4.22),(TR360 - 0.78)sup5(4.22) + 11.13)

Please see Figure 6 for graphic representation.

(Hallum, 1990)

p = f(esup5(-2.423+3.657 ln[TR360-0.78]),1 + esup5(-2.423+3.657 ln[TR360-0.78]))

Please see Figure 3 for graphic representation.

Chi-Square Goodness-of-Fit: C.C.Brown=2.450; df=2; p-Value=0.294

Discussion

Figures 1, 2 and 3 detail the CMB, DCS and DCS Grade 3 plots of predicted incidence using the Logistic Model of the Hill equation. Figures 4, 5 and 6 show the original Hill Equation plots of the predicted incidence of CMB, DCS and DCS Grade 3 as modified in the unpublished 1990 revision of the Conkin (1987) paper. The collection of additional data over the succeeding years did not significantly affect the curve values or the 95% confidence interval (CI) shown in Figures 4, 5 and 6, although the exponent, and therefore the slope of each equation did change. This tendency is seen in the fact that the predicted incidence of any event, but most especially CMB, do not change dramatically between the 1987 Technical Memo and the 1990 unpublished revision of that memo. During the period between the preparation of these two models, the number of exposures increased from 607 to 927, with the number of groups increasing from 34 to 49 (see Table 1).

The significant area to note when comparing curves 1-6 is the reduced range of the CI surrounding each of the logistic model curves. At the TR of **1.65**, the maximum level at which astronauts may start their EVAs, the predicted incidence of CMB using the Hill's equation (1990 revision of Conkin,1987) is approximately 60%, with a CI of 39-76% (Fig. 4) versus 61% incidence and a CI of 56-67% with the logistic regression plot (Fig. 1). The Hill's equation plot of the DCS (Fig. 5) shows an incidence of about 23% (CI=5-38%), with the logistic regression (Fig. 2) showing an incidence of 29% (CI=24-35%). Figure 6, a plot of DCS, Type 1, Grade 3, predicts an incidence of 5% (CI=0-9%) using the revised Hill equation. Using the logistic regression of the Hill equation gives a similar predicted incidence of 5%, but again with an reduced CI of 2-8%.

In tabular form, these comparisons are as follows:

Table 1. Model Comparisons

EVENT & EQUATION USED	N of SUBJ/ GROUPS	PRED. PROB.	LOWER 95% LIMIT	UPPER 95% LIMIT	LIMIT DELTA
CMB - HILL(Conkin,87)	607/34	58%	38%	77%	39%
CMB - HILL(Rev.90)	927/49	62%	39%	76%	37%
CMB - LOGISTIC	782/27	61%	56%	67%	11%
DCS - HILL(Conkin,87)	607/34	20%	5%	38%	33%
DCS - HILL(Rev.90)	927/49	31%	5%	38%	33%
DCS - LOGISTIC	782/27	29%	24%	35%	11%
DCS(3)-HILL(Conkin,87)	568/31	4%	0%	9%	9%
DCS(3)-HILL(Rev.90)	698/42	5%	0%	9%	9%
DCS(3)-LOGISTIC	698/24	5%	2%	8%	6%

As can be seen from the above comparisons, in all cases the logistic regression of the Hill equation provides significant reduction in the 95% confidence interval. The incidence of all three events increased when both 1990 models are compared with the 1987 model. One of the factors involved in these changes is that the model based on the Hill equation predicted only the group incidence of an event, not taking into account the size of the group, i.e., 24% of a Group X would develop DCS at a TR of 1.65. Also, confidence intervals were determined by taking an average of **n** in all the groups, rather than using the **n** of each group.

The 6-hour data standardization used in the 1990 revision of the Hill equation also appears to slightly overestimate the CMB and DCS incidence, when compared to the Logistic Model. However, in all calculations using the 1990 models, the numbers are consistently close, tending to support the validity of the use of logistic modeling in the generation of a predictive equation.

Using the logistic regression special case of the Hill dose-response equation, we are now able to make predictions regarding the probability of event incidence in an individual exposed to hypobaric conditions at a specific TR. This is a significant enhancement of the utility of the model-based predictive system.

Additional work by Dr. K.V. Kumar of the NASA Governmental Physiology Laboratory is now in progress to evaluate the association between the time to onset of symptoms and other independent variables. The epidemiologic approach being used is primarily that of analyzing "censored" data (incomplete data due to cessation of a test before the occurrence of an outcome event) by using survival analysis techniques (Kumar, 1990). Preliminary indications are that by using this type of statistical approach to data analysis, the predictive precision of the model will be improved. Of course, some of this improvement will be a result of the continually increasing number

of human hypobaric exposures included in our database. Further comparison of the log likelihoods for the Hill logistics and survival predictive equations is being conducted to determine the most robust method on which to base our predictive CMB/DCS Model.

Acknowledgments

The authors wish to express their sincere appreciation to K. Vasantha Kumar, MD, for his invaluable and patient assistance in the preparation of this paper.

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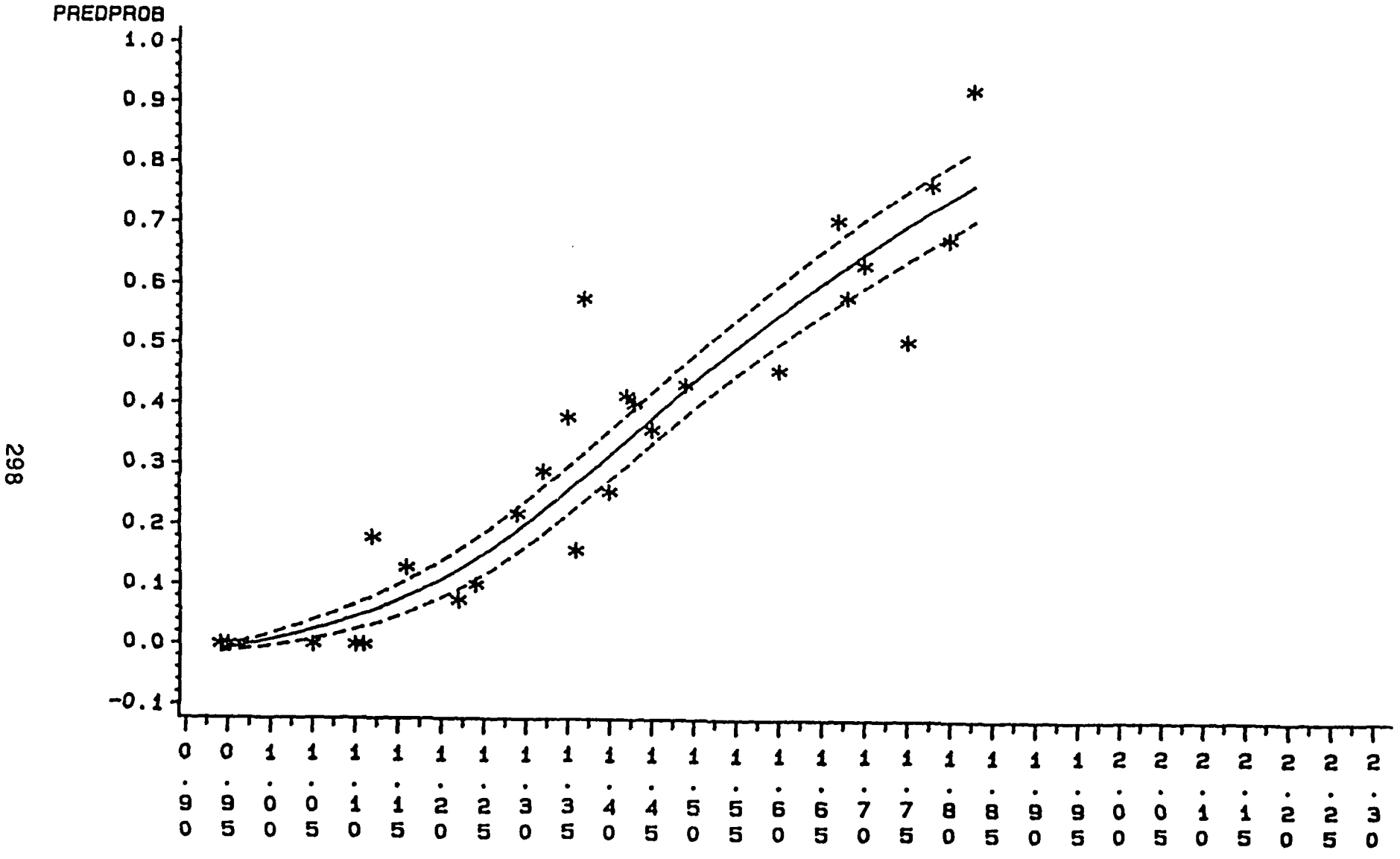


Figure 1. VGE confidence plots.

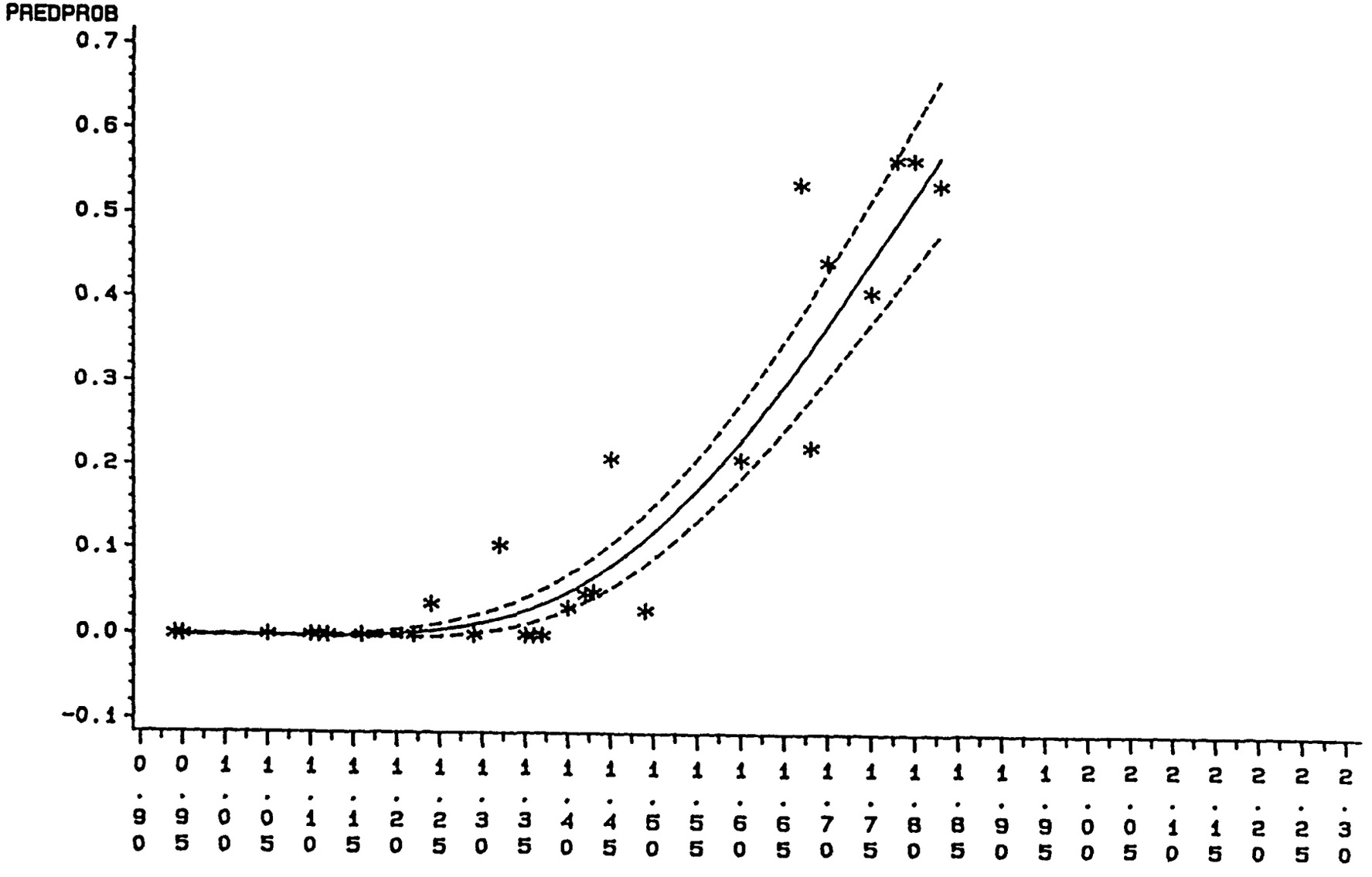
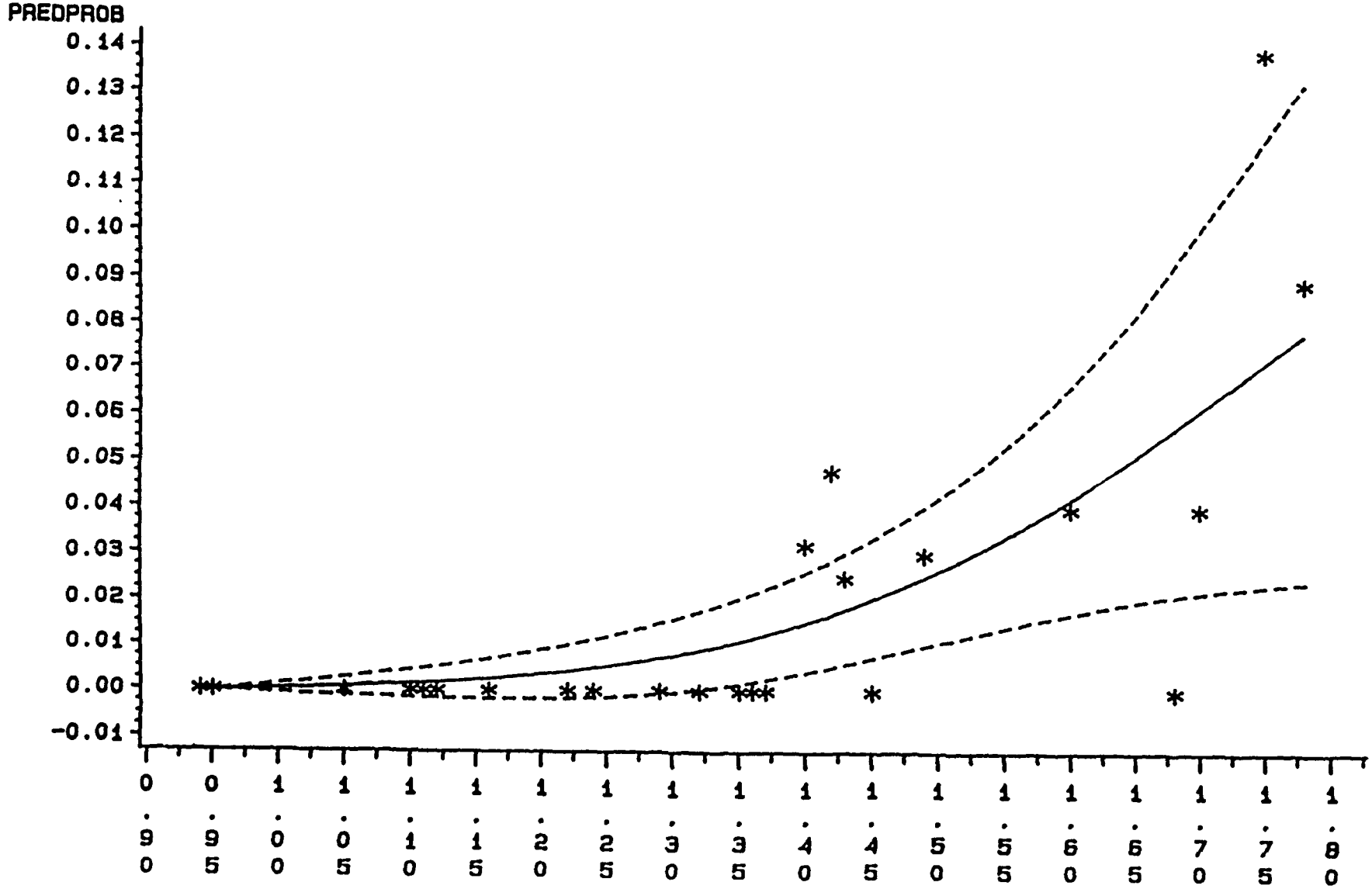


Figure 2. DCS confidence plots.



300

Figure 3. DCS3 confidence plots.

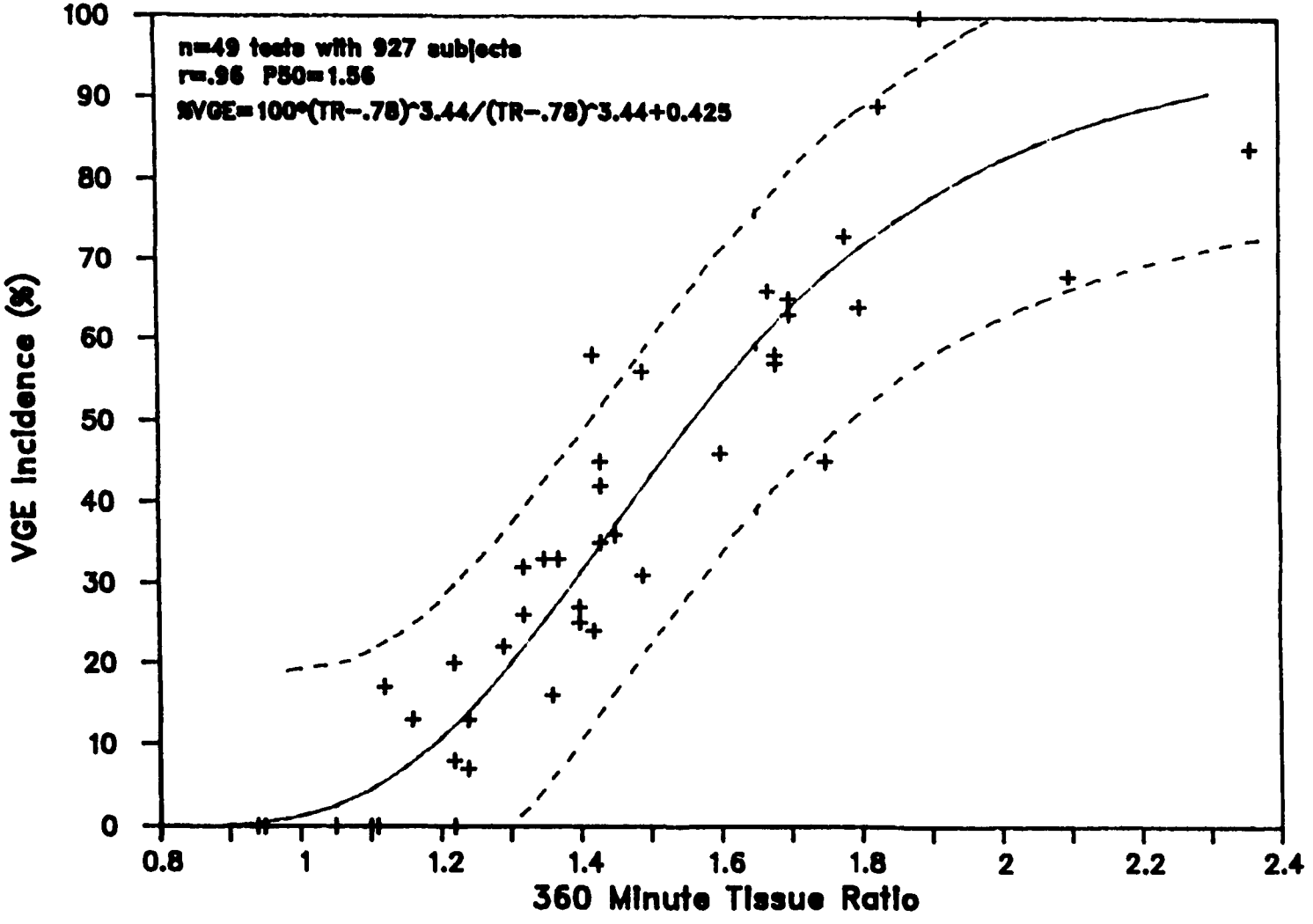


Figure 4. NASA/USAF VGE incidence versus 360-minute tissue ratio with 95% confidence interval.

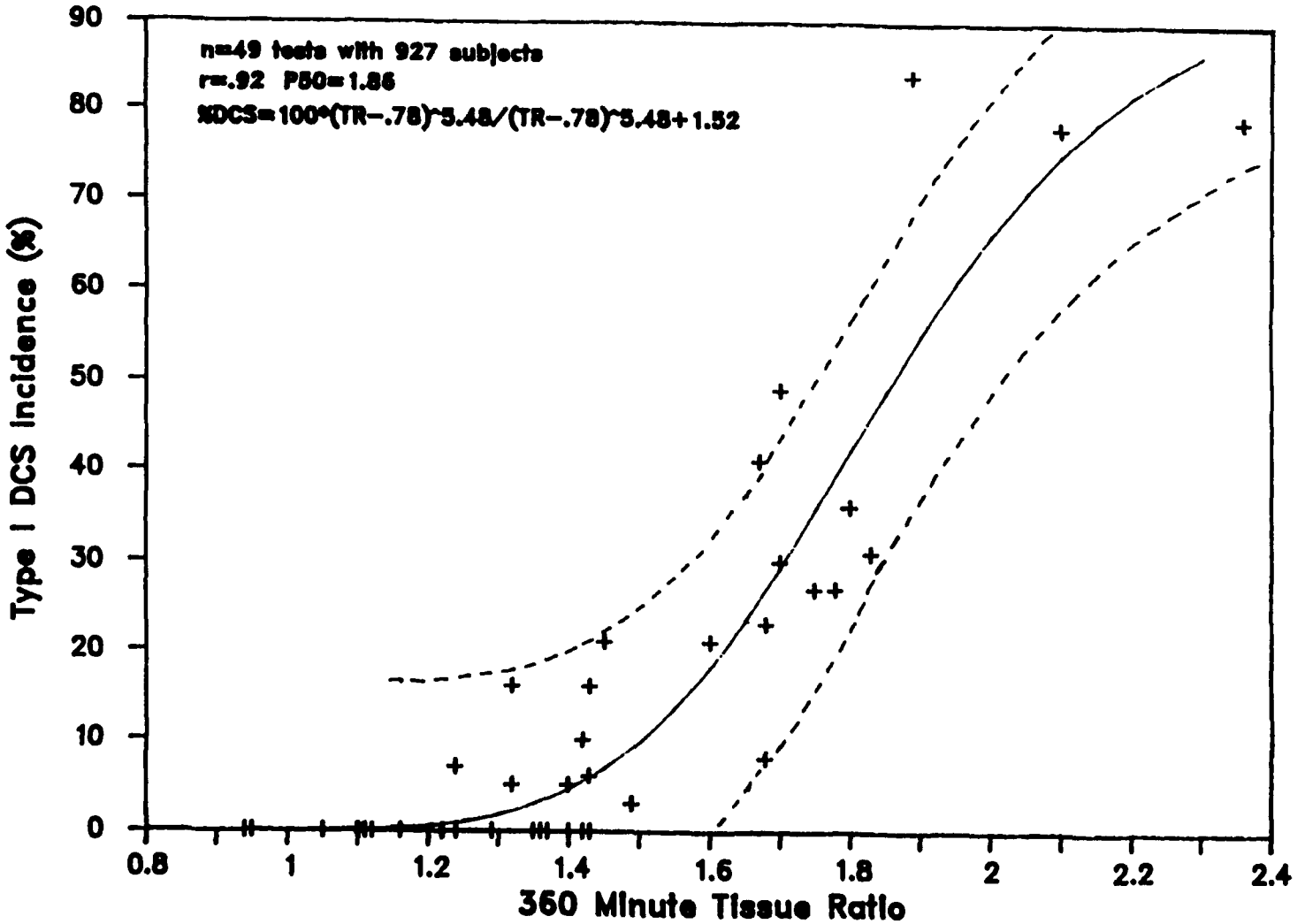


Figure 5. NASA/USAF type 1 DCS incidence versus 360-minute tissue ratio with 95% confidence interval.

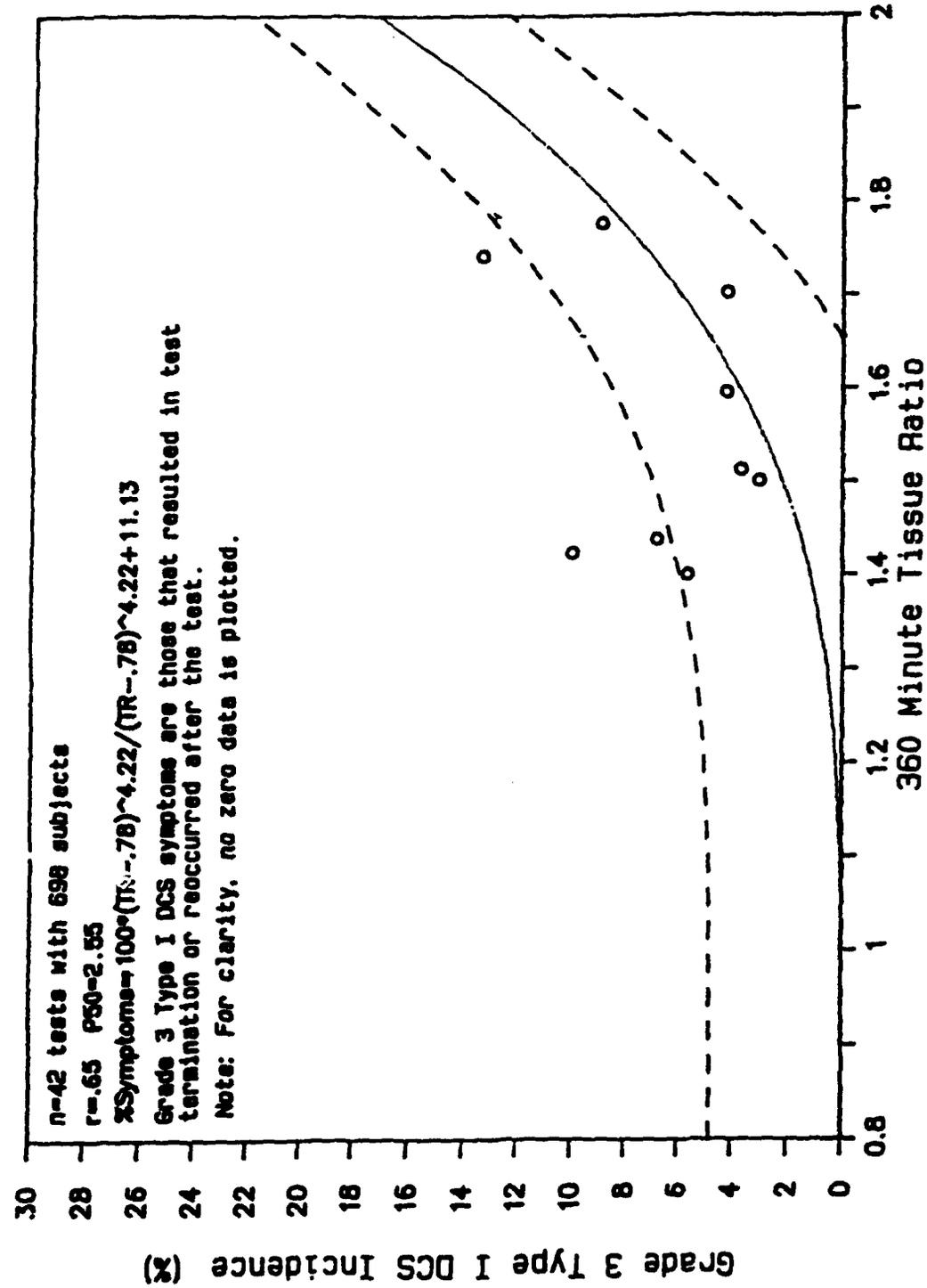


Figure 6. Type 1 grade 3 DCS versus 360-minute tissue ratio with 95% confidence interval.

CURRENT CONSIDERATIONS ON EVA DECOMPRESSION

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Introduction

Extravehicular Activity (EVA) has been widely used in both US and USSR space programs. With the development of European space initiatives, the European Space Agency (ESA) decided to evaluate the options for an original European EVA capability. One severe disadvantage of existing systems is the necessity of prolonged prebreathing prior to decompression into the low pressure suit. After reviewing the existing suit technologies, ESA aimed its development efforts towards a space suit with an operating pressure of about 500 hectoPascals (hPa) (7.3 psia). To estimate the physiological advantages of such a system, a "Study on Decompression Risks for European EVA" was initiated and Deutsche Forschungsanstalt für Luft-und Raumfahrt (DLR) was contracted for this study[1]. The considerations presented here are derived from this study which was finished in August 1989, without representing official ESA opinions.

DCS-Study

Figure 1 gives the outline of the study. In addition to the main body of the work, which was concerned with the risk assessment and quantification of EVA decompression, there are packages dealing with therapy, crew selection, medical monitoring, and ground facilities. The study was prepared by a scientific team from the Underwater Medicine Department at the DLR Institute for Aerospace Medicine, together with a Dornier team, which contributed in technical and administrative matters. T. Hennessy of Brunel University participated as a contractor.

The first part of the study reviewed the current knowledge of decompression and decompression sickness (DCS) started by defining the conditions to be encountered by European EVA. A close similarity between saturation diving and EVA was established, thus suggesting that saturation decompression data could be used for the risk assessment of EVA decompression procedures. In this respect, the use of shallow nitrogen saturation and long exposure compressed air work data appeared to be especially promising (Fig.2). The R-factor derived from these data for a safe one-step decompression from elevated to normal pressure was 1.2 and was very similar to the results of NASA experiments with a Zero-Prebreathe-Suit (where 'safe' means a risk of about 1% or lower).

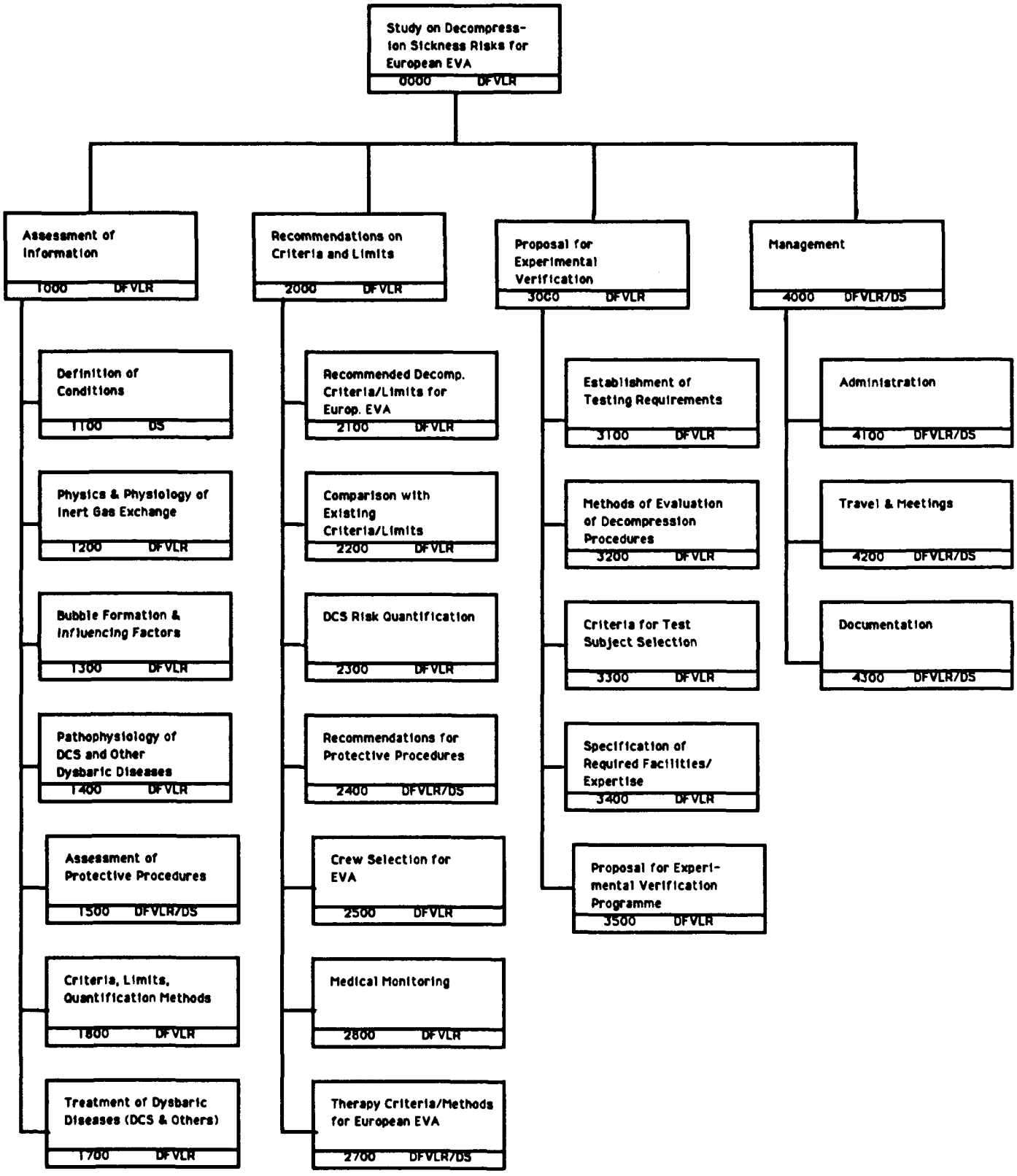

Figure 1. Outline of study on decompression risks for European EVA.

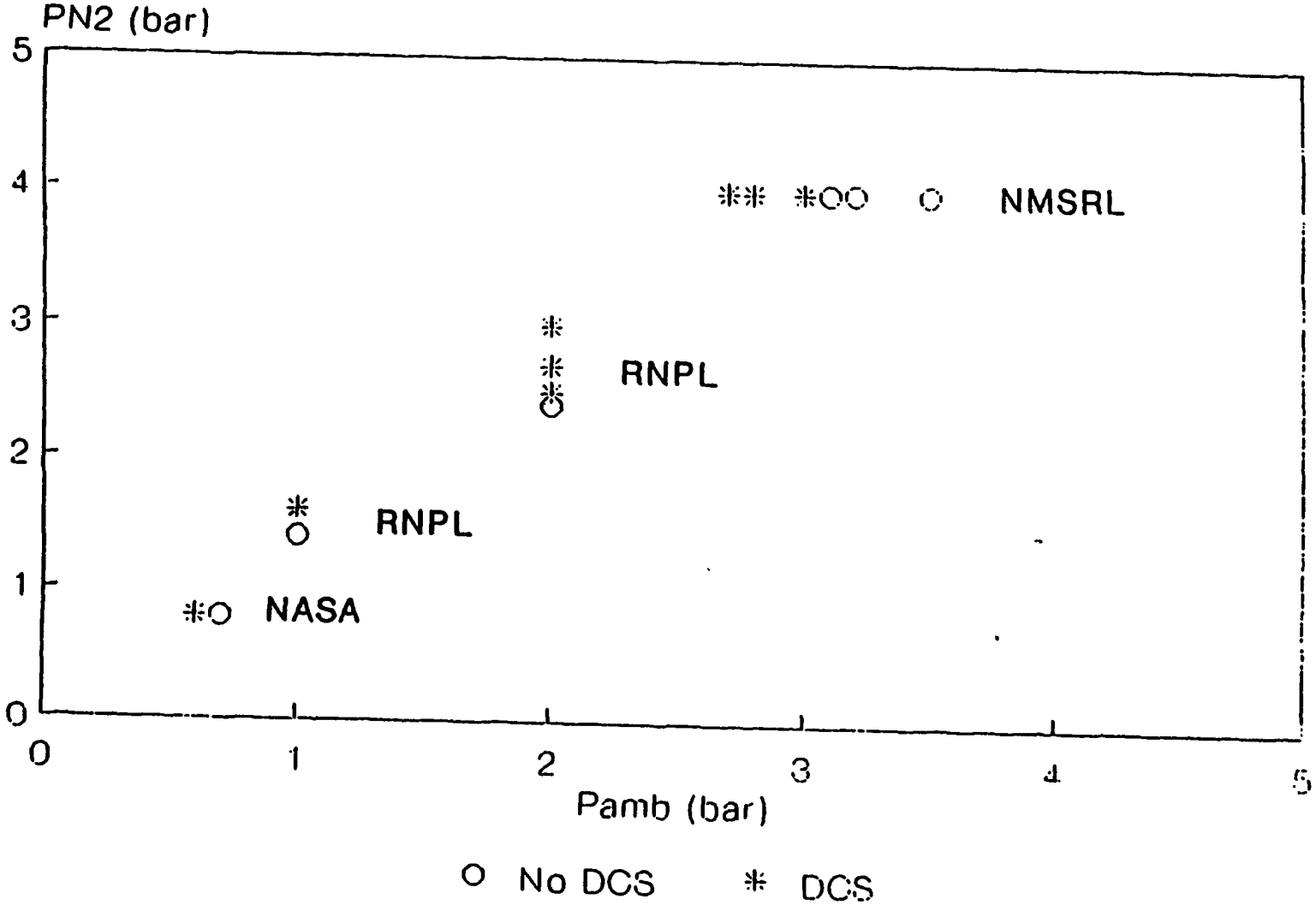

Figure 2. Critical pressure curve - nitrogen.

The second part of the study recommended a 500/430 hPa space suit and was based on the information gathered in part one. The main information source was the NASA/USAF database on hypobaric decompression which consisted of about 600-man decompressions to target pressures below one bar. Unlike the (shallow) hyperbaric decompression data mentioned previously most of the decompressions in this database involved complex decompression protocols instead of a simple one-step decrease to the target pressure. These decompressions usually employ oxygen prebreathing as the general protective measure prior to EVA which points out the necessity of using inert gas exchange kinetics to estimate the inert gas loading of the organism during the pressure reduction. Inert gas exchange is usually expressed in terms of "tissue" half-times, where the organism is considered to be an ensemble of different compartments, each with an independent gas exchange and different time constants. It is clear that for saturation decompression only the slowest tissues' data are relevant; it therefore represents the most meaningful physiological half-time for gas exchange. These half-times were estimated in a NASA paper by Conkin et al.[2] where the tissue loading calculated from a single (long) half-time prior to decompression was correlated with the empirical DCS incidence resulting from the corresponding schedule, assuming a linear relationship. Of course, there was considerable scattering of data points, but by taking the linear regression coefficient corresponding to different half-times as a measure of the quality of description, a maximum could be found around 400 min.

We again tried to compare this result with saturation diving data, this time using data from continuous decompression from deep heliox diving. If we assume that in a long decompression of several weeks even the smallest supersaturation will cause bubble formation (as it is never decreased), then the transition of half-times beyond Tissue Ratio = 1 with the given decompression rate will indicate the point where half-times grow beyond physiological reality. In general, this point was found between 300 and 500 minutes (corrected from helium to nitrogen).

In a second step, tissue supersaturation has to be correlated to the risk of decompression sickness. Although this approach for the identification of relevant tissue half-time using linear regression was used in the NASA report mentioned previously, it is clear that the correlation will follow a more S-shaped curve, as in pharmacological dose-response functions. A widely accepted mathematical description of this kind is the two-parameter Hill function

$$P(D) = \frac{D^n}{D^n + D50^n}$$

where P(D) is the response probability related to the dose D, while the exponent n controls the steepness of the transition from zero to 100% response. The parameter D50 is the dose at which approximately 50% of the population show response.

For decompression problems, it is convenient to define or fit a dose to approximately "zero." This zero figure is not identical to a TR = O, but considers the

fact that with no pressure reduction at all there is surely no risk of decompression sickness. Starting from normal atmosphere, the lowest meaningful TR for a starting point of the dose response function with P(DCS) = 0. It is .79, therefore, taking into account the correlation between tissue saturation and the necessary driving force for bubble formation and growth, a TR of 1.0 would be a reasonable choice. If a TR is selected as the starting point of the response function and denoted as a critical TR or TRc (D = 0), then the decompression dose corresponds to TR - TRc and the Hill equation can then be rewritten for decompression problems as

$$P(DCS) = 1 / (1 + ((TR_{50} - TR_c) / (TR - TR_c)^n)$$

where TR_{50} is the TR with 50% incidence of decompression sickness. In the NASA paper which analyzed hypobaric decompression data using the linear regression method, TRC has been, a priori, set to 0.78 (assuming that only when there is no pressure reduction at all will there be a zero risk of decompression sickness), and with an appropriate iteration algorithm the following parameters for the Hill equation were found (using an operational tissue half-time of 360 minutes; then r is the correlation coefficient, and gives an estimate of the quality of data representation):

P(DCS): $n = 4.24$, $TR_{50} = 1.97$, $r = 0.84$;

Similar to the dose response analysis of NASA, the maximum likelihood evaluation of Vann et al. [3] tried to establish a Hill function for the combined NASA/USAF hypobaric decompression data. Contrary to the NASA analysis, the maximum likelihood approach did not employ an a priori estimation of the TRC. Instead TRC was included as a part of a group of parameters that had to fit the different models tested, so that in the case of the single tissue model the four parameters

TR_{50}, TR_c, n, and tl/2

had to be accessed. The result for tl/2 was 508 min., and the TRC was found at 1.0007, which closely agrees with the assumption that a decompression within the oxygen window does not impose a decompression risk (compared to the NASA a priori adoption of TRC = 0.78). The two remaining parameters of the Hill function are not given in the paper, but the overall result is given as a dose response plot.

The DCS risk with a TR of 1.2 is 1.3%; without a TR of 1.3 is 3.5%

With this additional information it is possible to calculate the two remaining parameters of the Hill function, i.e.,

$TR_{50} = 2.11$ and $n = 2.52$

This may be compared to the NASA values, also calculated with the above parameters:

Procedure #	1	2a	2b
Recommendation	acceptable	preferred	preferred
Cabin pressure level	1013 hPa only	1013 hPa then 700 hPa	1013 hPa then 700 hPa
Prebreathing time for EVA astronauts before reducing cabin pressure	N/A	1h	0
Time of reduced pressure before first EVA	N/A	12 - 24 h	48 - 72 h
Prebreathing time for EVA			
- P = 500 hPa	2.5 h	0	0
- P = 430 hPa	3.5 h	1 h	1 h

Figure 3. Summary of protective procedures for nominal EVA.

TR = 1.2: P(DCS) = 1.16% and

TR = 1.3: P(DCS) = 2.81% .

Thus, both the least squares method and the maximum likelihood approach give very similar results.

As our contract included the delivery of some recommendations concerning decompression with a 500 hPa suit, we had to decide on an acceptable risk level. Taking into account the very limited treatment options on orbit, our decision was limited by the medical considerations and an R-factor of 1.2 was selected corresponding to a risk of about 1% (keeping in mind a considerable error may have been incurred with some of our estimates and due to the limited number of experiments). The recommendations are summarized in Figure 3 (employing a relevant tissue half-time of 360 min).

Conclusion

This very conservative approach results in relatively long prebreathing times for the direct transition from normal atmosphere to suit pressure of 500 hPa. This time is longer than ESA had expected. However, final decisions for decompression procedures will be set when the suit pressure is finalized, and it will be a political responsibility to set the risk level at which astronauts are requested to perform.

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DR. VANN: In decompression modeling, one normally assumes that gas exchange in the lung occurs so rapidly that it can be omitted. Is it justifiable to assume instantaneous gas equilibration?

DR. WENZEL: There is a time constant, between lung alveolus and blood, of the hundredths of a second. We have no reason to assume that this has changed, even in deep diving. This is current physiological knowledge. There is no real diffusion limitation in the lung.

DR. LAMBERTSEN: We all keep talking about ratios as though they were useful, when you are really talking about a gradient.

DR. WENZEL: It is very simple for visualizing, but for the real articulation of decompression schedules, you have to use differentials.

DR. VAN LIEW: In this process of attempting to find low risk schedules, the relevant tissue half-times seem to be getting longer. I am not sure I understand where the estimate comes from. If the estimates are based on oxygen prebreathing, then there should be very little nitrogen left in the body, and you may be getting artifacts. Some of the artifacts I would worry about would be nitrogen leakage around the mouthpiece or hood, and nitrogen that comes through the skin. In which case you could prebreathe forever and you would still have the same amount of nitrogen because there would be a continuous current coming in through the skin.

DR. WENZEL: That is why we tried to compare these prebreathing experiments for hypobaric decompression with high pressure diving experiments, in which we will not keep below any supersaturation to form bubbles. Interestingly, we came out at a similar time constant for the slowest relevant tissues. We were not so confident with just taking one rule as evidence, so we looked around in the hypobaric world and tried to gather some experience which would correlate with this data, and, in fact, we were successful.

DR. BALLDIN: Just a short comment on the leakage or the diffusion of nitrogen through the skin. To my knowledge, it is very, very little and could be negligible. I have another question. What is the policy in NASA and ESA on the acceptable risk of decompression sickness? You talk about it as if it is 5%.

MR WALIGORA: Right now the procedure that we use operationally is a 5% incidence of symptoms that interfere with performance.

DR. BAGIAN: It is not so much a policy, but rather that is what we are using?

MR WALIGORA: We accepted it sometime ago with the caveat that we were going to improve on it. We have done the test that would show how you could improve it. However, in the meantime, there has been no DCS incidence and people are reluctant to change.

DR. WENZEL: I was very pleased when Mr Waligora talked about decreasing the R factor to 1.4, or even to 1.2, to have zero risk, because this was our approach also. At the moment, there is no opinion about this at [illegible]A. They were not very pleased with our recommendation of 1.2 and the very long prebreathing times, because they hoped to have no prebreathing at all for a 500 hectoPascal suit. So they did not really like our support and the current opinion is that we have no opinion. But our recommendation of 2.5 hours of prebreathing for 500 hectoPascal suit has been evaluated for further technical development.

DR. BAGIAN: The fact of the matter is that operationally with your 7.3 psi suit, you get an hour prebreathe normally. So, while you might not treat it as a requirement operationally, you will get it anyway.

DR. VANN: Regarding the diffusion across the skin and the incidence of bends, we looked at 10 flights with subjects immersed in air in a closed bag and 9 flights with them immersed in oxygen. There were 3 bends in either case. There was no distinguishable difference in the whole body nitrogen elimination. So within the limits of these numbers there is really no difference whether you are immersed in one gas or another.

DR. FRANCIS: Back to this business of risk of decompression sickness, if what we have heard this morning is correct, NASA can competently predict 5% incidence. That amounts to policy.

DR. BAGIAN: Instead of management coming down and saying this is the risk we will accept, and then going to the research arm and saying what do we need to do to satisfy it, instead it percolates the other way, and, originally, we went in with a 1.2 request. However, management said, that is no good, let us try again. We are now at 1.4.

DR. WENZEL: Acceptable risk is surely connected to the therapy options we have. You talk about 1.4 or 1.6 and have it in mind to use an airlock, as a sort of hyperbaric recompression facility. We were required for ESA to look at the scenario where no such option is available, and so we have insisted on nearly zero risk. As we are medical people and not technicians, we have to take responsibility for our questionable patients, and not what would be nice to have from an operational point of view.

OPTIONS AND PLANS FOR TREATMENT OF DECOMPRESSION SICKNESS ABOARD SPACE STATION FREEDOM

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KRUG Life Sciences
Houston, TX

Introduction

Decompression sickness (DCS) is a likely consequence of extravehicular activity (EVA) from Space Station Freedom (SSF), given the nature and frequency of EVAs currently projected. Estimates of the probability of DCS in these operations are somewhat uncertain due to a paucity of chamber trials of decompression procedures and difficulties in extrapolation from ground-based studies to microgravity. However, utilization as planned of an extravehicular mobility unit (EMU) containing a 30 kPa (4.3 psi) environment will probably result in symptoms of DCS that require hyperbaric therapy in 1-5% of all person-EVAs (a person-EVA is defined as an exposure of one individual to EVA conditions; a typical EVA involves two crewmembers and, therefore, entails two person-EVAs). Given current demands for approximately 100 person-EVAs per year, the need for hyperbaric therapy capabilities aboard SSF is obvious as a means for providing prompt treatment and precluding costly medical evacuations to Earth.

A brief overview of the facilities and procedures planned for hyperbaric therapy aboard SSF follows. The description of hardware will be preceded by a discussion of the diseases that will be confronted along with a short compilation of some of the most pressing questions that need to be answered before optimum therapy can be provided.

Types of DCS

Decompression sickness and related disorders are known to take the following forms in the aviation and/or diving communities:

1) extremity pain

2) spinal cord dysfunction

3) cerebral dysfunction

4) vestibular dysfunction

5) pulmonary manifestations (the "chokes")

6) dysbaric aseptic osteonecrosis

7) ebullism (exposure to hard vacuum)

8) arterial gas embolism

9) cardiovascular manifestations

Obviously, the above disorders are not necessarily exclusive, and, in many cases, may be manifestations of the same disease process (for example, arterial gas embolism can cause spinal cord dysfunction). However, arranging a list of potential problems in this manner is useful for preparing treatment capabilities.

Undoubtedly, the most common manifestation of DCS aboard SSF will take the form of extremity pain, a.k.a. "limb bends." In a ground-based study of a denitrogenation method which may be used on SSF, Waligora et al. (1) found an incidence of DCS of 23% (26 of 111 subjects). In 23 of these symptomatic subjects, the symptoms were mild, did not interfere with work, and disappeared upon recompression to 101 kPa (14.7 psia). Most clinicians would not treat such symptoms with hyperbaric therapy, and there are no plans to do so in these cases on SSF (except that afflicted crewmembers would be restricted from EVA for a period of several days). However, 62% of the subjects in this study had very heavy venous gas embolism burdens as reflected by grade 3 or 4 Doppler bubble scores (refer to (2) for a discussion of Doppler monitoring). The risk of repeated, weekly exposures to this kind of protocol as planned for SSF operations is not established. Although there are no plans for specific therapy for evanescent "limb bends" symptoms when they happen, will there be a need to confront, at some point delayed, manifestations such as the cumulative result of small neurological "hits" or, perhaps less likely, dysbaric osteonecrosis (3)?

Waligora et al. (1) observed the following symptoms in the study described above: 1) a sudden onset of exhaustion in conjunction with a cold sweat and a red and white pattern of marbling on the chest, 2) a mild pain in the knee had a reoccurrence of pain one hour after pain disappeared upon recompression to site pressure, and 3) slight sensations of numbness moving from one leg to the other.... It appears, therefore, that mild neurologic symptoms and persistent or recurrent limb pain after recompression to 101 kPa (14.7 psi) are possible outcomes of EVA activity from SSF. In a further study by Waligora et al. (personal communication) with a similar denitrogenation procedure, 6 of 426 subjects required hyperbaric therapy, including 4 subjects who exhibited neurologic manifestations. Prudence would dictate that SSF retain a capability for similar treatment given the anticipated high frequency of EVAs.

Clinical experience has demonstrated that DCS in aviators is manifest almost entirely as mild limb bends, and that persistent symptoms respond well to hyperbaric therapy (4, 5). This pattern contrasts somewhat with that of the diver where profound and recalcitrant neurologic problems can be more common. The astronaut is frequently assumed to be more analogous to the aviator than the diver from the point

of view of DCS symptomatology. This assumption is open to question. Certainly, in terms of hemodynamics, the astronaut, with intrathoracic fluid shifts and the lack of dependent blood pooling, is more like the diver than the upright, seated aviator. If central fluid shifts and engorged epidural venous plexuses can predispose to blood stasis that interferes with inert gas elimination from the spinal cord or that causes bubble accumulation, coagulation, thrombosis, and cord infarction as postulated by several investigators (e.g., Bove (6), Hallenbeck (7)), then the Crew Medical Officer (CMO) aboard SSF may be confronted with serious cases of neurologic DCS. Also, given the high bubble scores observed by Waligora et al. (1), pulmonary manifestations may be distressingly common.

Concerning ebullism, the most likely circumstance that would give rise to this problem would be the loss of pressure integrity of an Extravehicular Mobility Unit (EMU) or a pressurized habitable module. The risk of these events during the course of 30 years of Station operations and thousands of EVAs is unknown, and clinical experience with ebullism is confined to a few case reports (8). Given these uncertainties, should special procedures over and above conventional hyperbaric therapy be made available to treat this disorder, and, if so, what would these methods be?

Perhaps arterial gas embolism (AGE) is the most controversial disorder in terms of the risk of occurrence aboard SSF and the optimal treatment of the problem. AGE could result from a sudden, unexpected loss of integrity of a pressurized module or an EMU. The risk of structural failure and the likelihood of AGE occurring in such a catastrophe are unknown. However, a scenario which produces AGE does not have to include equipment failure. Several cases of AGE have occurred during the course of seemingly entirely normal decompressions, especially during submarine free ascent training (9). Through screening of SSF crewmembers for occult pulmonary pathology and intracardiac shunts, the likelihood of these events will be minimized, but some residual risk may remain. Therefore, it appears that a small but real risk of AGE exists during operations aboard SSF.

The optimal treatment for AGE is a matter of debate. A U.S. Navy Table 6A with a maximum pressure of 6 atmospheres absolute pressure (ata) is the standard treatment method, but recent animal and clinical data (e.g., (10), (11)) indicate that 2.8 ata may be adequate. These data have limited applicability to SSF where emboli would arise from hypobaric exposures, and some scenarios would produce predominately oxygen rather than air gas emboli. After consultation with hyperbaric specialists and considerable debate, NASA management has recently selected 2.8 ata as the maximum working pressure of the chamber aboard SSF.

Options for Hyperbaric Treatment Facilities Aboard SSF

1) No hyperbaric capabilities. Given the use of an EMU with a 30 kPa (4.3 psi) environment and the planned frequency of EVAs from SSF, decompression sickness will probably be a common occurrence. Hyperbaric treatment will clearly be a medical

requirement. Return of a victim to Earth will be very costly and will entail a delay in treatment between hours and days, depending upon the transport methods available. For these reasons, some hyperbaric facility is necessary on SSF.

2) Monoplace inflatable hyperbaric chamber. The SSF Program has debated this issue for many years. The 1986 Ad Hoc Committee on Hyperbaric Medicine (12) pointed out the limitations posed by monoplace chambers and recommended against their use. The main difficulty is the inability to provide direct patient care during therapies. Caregivers cannot treat true medical emergencies or assess a patient's neurologic state or such as by a neurologic examination to evaluate the tension pneumothorax, or control seizures, e.g., those encountered with neurologic oxygen toxicity. The care of an individual with neurologic symptoms, hemodynamic instability, or a compromised airway or sensorium in a monoplace chamber is difficult. Because confrontation of such patients aboard SSF is a reasonable possibility, the use of a monoplace inflatable chamber does not seem to be a viable option.

3) Multiplace hyperbaric chamber. This is the option that has been chosen for use on SSF and will be discussed in some detail below.

The Hyperbaric Airlock

The design for SSF includes one multifunctional airlock located on the starboard side of the Station attached to Node 1 (Fig. 1). This airlock will perform the following functions:

I) allow egress and ingress of two crewmembers to and from space;

2) transfer large objects to and from space;

3) service and check out EMUs;

4) provide a single-lock multiplace hyperbaric treatment chamber with a maximum working pressure of 2.8 ata.

For planning purposes, a Hyperbaric Worst Case Scenario has been developed. It is depicted in Figure 2 along with details of the pressure profile and breathing gas schedule for a U.S. Air Force Table 6 protocol. The Worst Case Scenario calls for one maximally-extended Table 6 plus three additional unextended Table 6 treatments. After completion of this scenario, a patient would be assumed to have received maximal benefit of hyperbaric treatment within the resources of SSF.

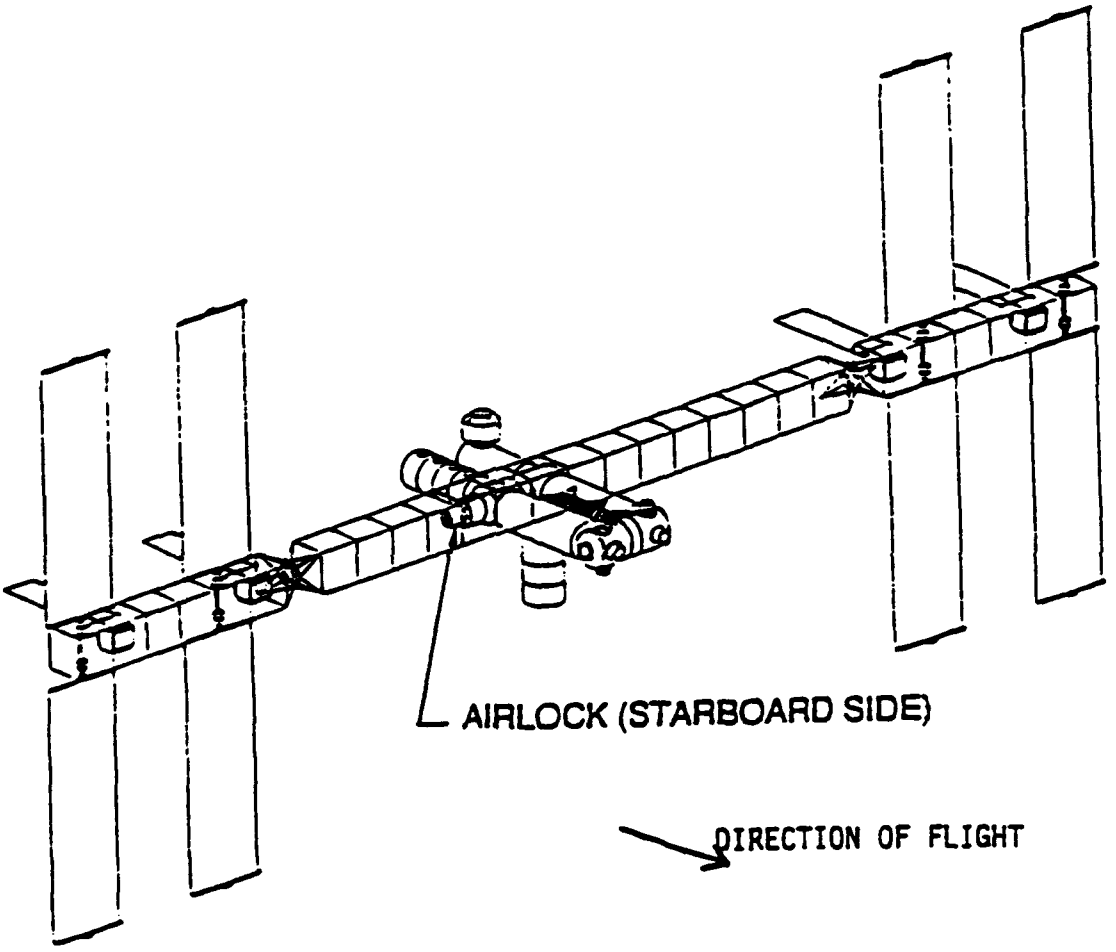
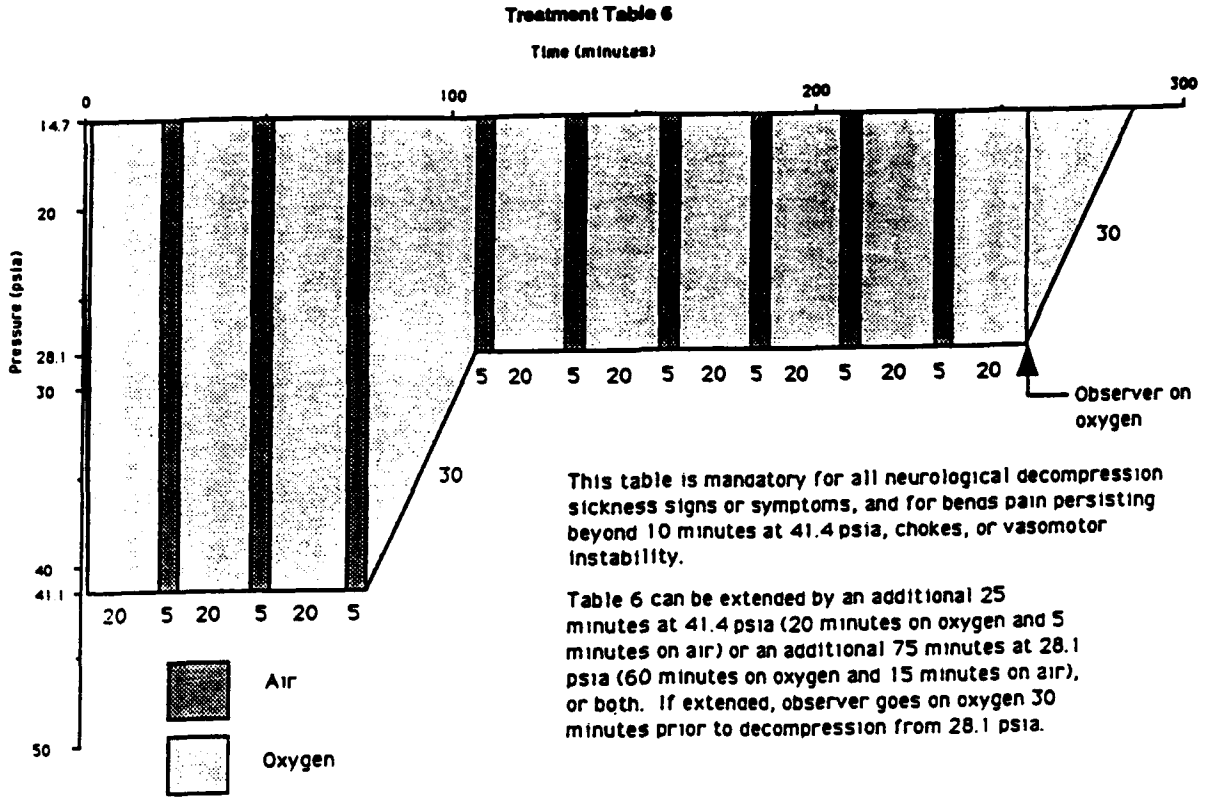
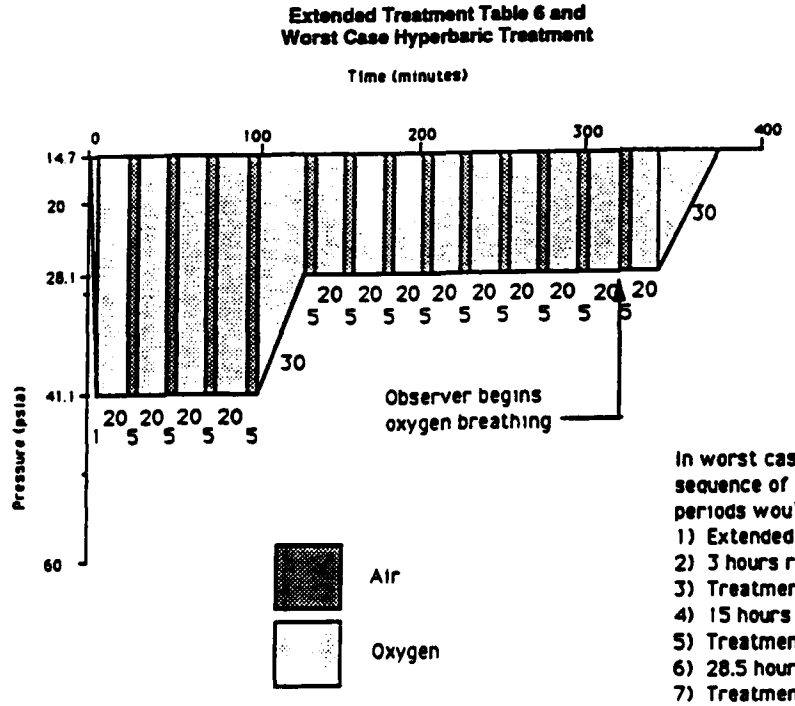


Figure 1. Overall configuration of Space Station Freedom and specific location of the hyperbaric airlock.



This table is mandatory for all neurological decompression sickness signs or symptoms, and for bends pain persisting beyond 10 minutes at 41.4 psia, chokes, or vasomotor instability.

Table 6 can be extended by an additional 25 minutes at 41.4 psia (20 minutes on oxygen and 5 minutes on air) or an additional 75 minutes at 28.1 psia (60 minutes on oxygen and 15 minutes on air), or both. If extended, observer goes on oxygen 30 minutes prior to decompression from 28.1 psia.



In worst case treatment, the following sequence of treatment tables and rest periods would take place:

1) Extended Treatment Table 6
2) 3 hours rest
3) Treatment Table 6
4) 15 hours rest
5) Treatment Table 6
6) 28.5 hours rest
7) Treatment Table 6

Figure 2. Worst case hyperbaric scenario and details of Hyperbaric Treatment Table 6 and Extended Treatment Table 6.

To meet the above requirements the Airlock is divided into two sections, the Equipment Lock (EL) and the Crew Lock (CL). In moving from Node 1 to space, one would first pass into the EL where EMUs would be donned, then move into the CL where decompression of that compartment to vacuum would take place, then egress "out the door" to space (Fig. 3). The CL serves a dual function in that it is also capable of sustaining a positive pressure of 2.8 ata; the CL and the Hyperbaric Airlock (HAL) are one and the same structure. The dimensions of the HAL are, relative to many commercial chambers, fairly generous (Fig. 4), and a patient and tender along with medical equipment can be accommodated. The hatch between the CL and EL is designed to accomplish pressure-assisted sealing for both hypobaric and hyperbaric operations through the use of an oval hatch on an articulated support. The hatch would be rotated, swung through, and seated on either side of the EL/CL bulkhead depending on the pressure anticipated in the CL. This hatch supports a transfer lock with an inner diameter of 41 cm (16 in.) and an inner depth of 51 cm (20 in.). The HAL is a monolock design, i.e., a crewmember cannot transfer into or out of the lock while the HAL is pressurized.

Controls for operating the HAL are located in the EL. A manual pressurization and depressurization system with computerized prompting of operations is planned. Gases are drawn from compressed (probably cryogenic) sources. During decompression, gases are exhausted to vacuum; there is no gas reclamation system.

A conventional Built In Breathing System (BIBS) supplies air or oxygen to oronasal masks within the CL. Exhaust gases are vented to vacuum through an overboard dump system.

Total gas consumption for an entire Worst Case Scenario (Fig. 2), including supplies for both chamber pressurization and BIBS usage, is about 133 kg. This gas would be drawn from specific stores designed to cover a variety of contingencies; hence, the mass of SSF's gas stores probably will not be increased by the requirements of this scenario.

Visual contact between EL and CL personnel can be maintained through two 18 cm (7 in.) diameter ports in the CL/EL hatch. Television viewing through one of these ports for transmission to Earth is also provided. Audio communications are accomplished through conventional noise suppressing headsets and microphones in the CL and EL.

Lighting is provided through sealed fluorescent bulbs. Alternatives, including conventional light pipe methods were considered, but strict power constraints forced the choice of a well sealed and shielded fluorescent system.

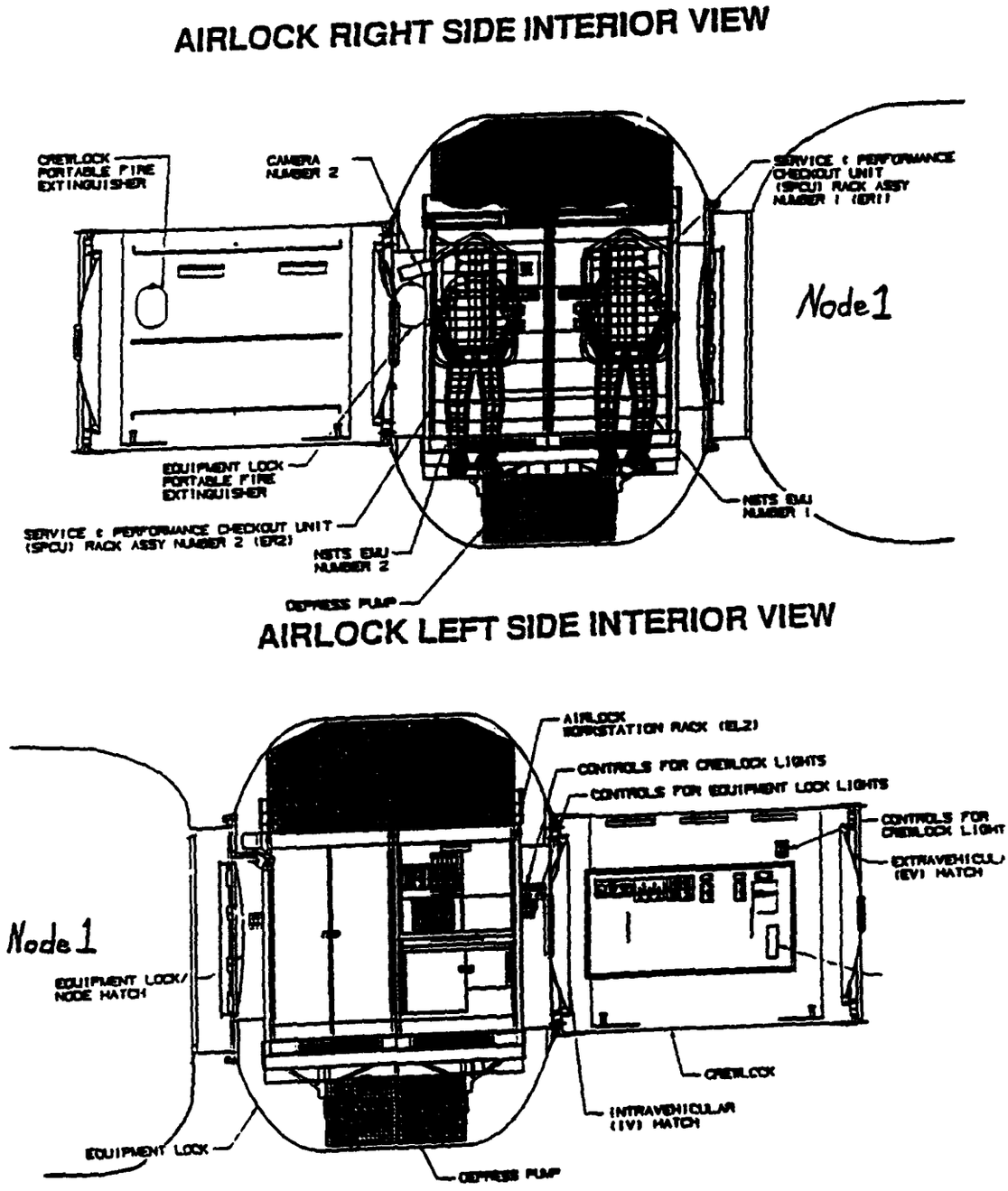


Figure 3. **Details of the Airlock. The Airlock is composed of the Equipment Lock and the Crewlock. The Crewlock can serve as a hyperbaric chamber and is also referred to as the Hyperbaric Airlock.**

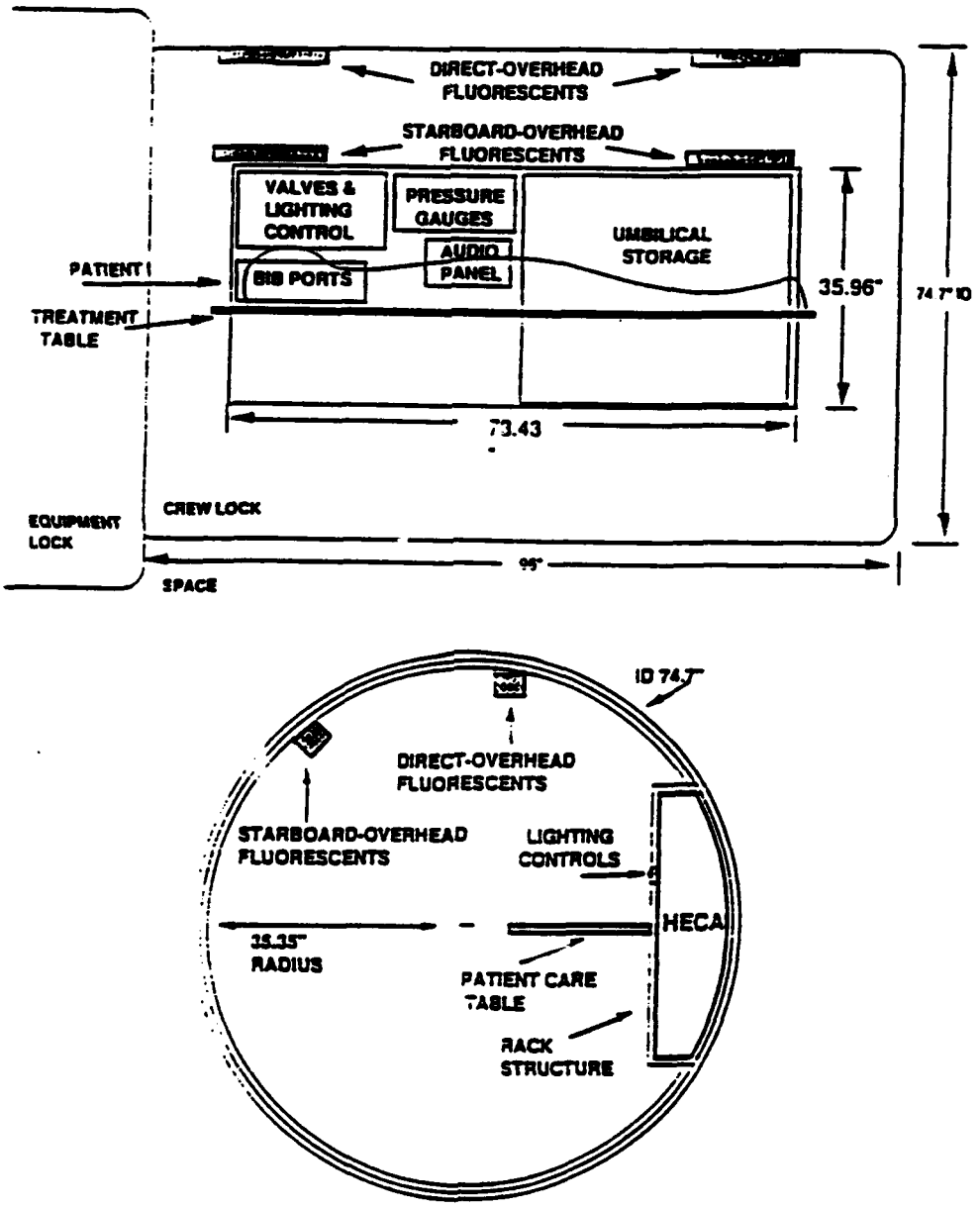


Figure 4. Further details of the Hyperbaric Airlock (a.k.a., Crew Lock).

Control of temperature and environmental gas composition within the HAL is provided by the Hyperbaric Environment Control Assembly (HECA) located in the CL. For a variety of reasons program managers have chosen to place the HECA with its associated electronic components including blower fans, within the hyperbaric environment of the CL rather than forming these elements into an external life support loop as is the practice in terrestrial facilities. Careful design is anticipated to reduce the fire hazard of this configuration to acceptable levels. The HECA uses molecular sieve technology for carbon dioxide and water vapor scrubbing. A heat exchanger controls temperature. Atmospheric composition is maintained at 20% oxygen/80% nitrogen, by HECA.

Some aspects of the basic functional design of the HAL that have not been clearly defined at this time include fire detection and suppression methods. Another difficult problem is the detection and removal of contaminants such as hydrazine and nitrogen tetroxide that might be "tracked into" the CL by a crewmember returning from an EVA. Because no terrestrial chamber facility has had to fight a fire in microgravity or deal with rocket fuels, these problems await unique solutions.

Medical support devices are designed to monitor a critically ill patient during hyperbaric operations and to provide necessary ancillary treatment for decompression sickness such as intravenous fluids and medications. Pumps for four intravenous lines are being developed. Intubation and mechanical ventilation will be supported. Non-invasive and invasive hemodynamic monitoring will be available. Insertion of a chest tube will be possible.

Conclusion

Given current denitrogenation strategies prior to EVA, the need to transition from an environment with air at 101 kPa (14.7 psi) to an EMU pressurized at 30 kPa (4.3 psi), and the anticipated high frequency of EVAs, DCS requiring hyperbaric therapy may be a common occurrence aboard SSF. Through appropriate training of Crew Medical Officers and development of capable hardware, treatment of stricken crewmembers with a level of care comparable to terrestrial facilities is planned.

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DR. PILMANIS: I do not know if the term bothered anybody else, but could you define a small neurological hit?

DR. NORFLEET: You can certainly define things in terms of the degree of symptoms. However, I get your point. I believe Dr. Lambertsen has said it best, if you see a little you have found a lot.

DR. LAMBERTSEN: It is a neurological hit in a small mind, that is what it is.

DR. PILMANIS: Secondly, you are defining the worst case as four Table 6s, with the first one extended.

DR. NORFLEET: That is correct.

DR. PILMANIS: Was the end point oxygen toxicity?

DR. NORFLEET: The end point was basically a compromise between a lot of things including gas stores, and oxygen toxicity.

DR. HAMILTON: Do you store nitrogen onboard the station so that you can make up the atmosphere of the chamber?

DR. NORFLEET: There are, at this time, nine options for gas stores being debated. Some of them involve plumbing pure oxygen and pure nitrogen to the chamber control console and some involve plumbing air and oxygen. So the answer to that is to be determined.

DR. HAMILTON: So we could store some neon?

DR. NORFLEET: There is certainly room for a lot of creativity in this design.

MAJOR BISSON: Essentially, the pressure suit is a monoplace chamber. Why not gain at least a little bit of added advantage of using the pressure suit and whatever type of chamber you have, since most of the time you are going to be treating simple bends?

DR. NORFLEET: If you are in the suit you have the limitations of a monoplace chamber. Are you proposing decreasing the working pressure of the chamber by using the suit in the chamber?

MAJOR BISSON: Yes, you could gain that because you are talking of a differential in pressure. If you do need access to the patient then you are stuck with a multiplace chamber.

DR. BAGIAN: In current shuttle ops we use that approach. However, it does not buy you much. We can go up to 7 or 8 psi, or to a half atmosphere, but that is the end of that suit, that trashes the suit.

DR. STEGMANN: Can that suit function in a 2.8 atmosphere environment?

DR. BAGIAN: As long as you have your umbilical attached to the wall you can function indefinitely because then the shipboard side supplies your O_2 and you have your cooling from the shipboard side.

COLONEL SHEFFIELD: I was reviewing some of my old archives recently and came across an article from the senior physiologist in the field at about the 1965 time period. They had tried to treat decompression sickness using the pressure suits. At that time pressure suit technology was quite good, and we had not yet gotten the hyperbaric facilities on-line. They tried a couple of cases with pressure suit. It did not work, so they came out with a notice to the field to disregard that as a method for treatment.

DR. BAGIAN: Thank you. Yes sir.

LT COLONEL FLANNIGAN: During hyperbaric treatment in the lock, will access to the outside be lost? Do you have any other access to the outside, ingress and egress?

DR. NORFLEET: If the orbiter is present there is a mast that connects to the wall. The orbiter normally does not berth at this point. It berths at another node via another mechanism. However, if the orbiter is present you could get in and out through that mast. If you really had to get out somewhere else you could use the node essentially as a lock. One of the four nodes at the ends of the soda cans. However, that is very wasteful of gas.

LT COLONEL FLANNIGAN: So you essentially shutdown outside operations during a hyperbaric treatment?

DR. NORFLEET: Essentially. During the early phases of space station operation there are only four crew members onboard. When one of them is sick and one of them is taking care of him, you run out of minds, hands, and eyes very quickly.

DECOMPRESSION SICKNESS DRIVEN OPERATIONAL PROBLEMS IN SPACE FLIGHT

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NASA Astronaut
NASA Johnson Space Center

Every US, and for that matter, Soviet, space flight is associated with the risk of decompression sickness (DCS) resulting from planned or unplanned hypobaric exposure. To minimize the chance of a DCS episode and thus maximize the probability of mission success, potential problem areas need to be identified and addressed. Once this has been accomplished, methods of operationally dealing with these problems might be possible.

During Space Shuttle operations, the first opportunity to encounter DCS occurs during the launch phase of flight. At this point each crew member is wearing the Launch/Entry Suit (LES) which is designed to provide both anti-exposure protection, in case of bailout into the water, and altitude protection sufficient to protect the crew against exposure to the ambient pressure at an altitude in excess of 180 nautical miles. The LES provides this protection through its dry suit and partial pressure suit capabilities. While the LES has been successfully human-tested at 100,000 feet, Shuttle procedures do not allow adequate denitrogenation time prior to liftoff for the LES to provide its optimal level of crew protection. Obviously, if the crew were permitted to prebreathe 100% oxygen for an adequate time, the partial pressure suit would furnish excellent hypobaric protection. However, such a prebreathe would result in the crew member exhaling nearly 100% oxygen into the cabin, creating a situation of high Shuttle cabin O_2 partial pressure which would pose an unacceptable flammability hazard. For this reason, the Shuttle program management has decided to equip the crew with a partial pressure suit, but not allow a formal prebreathe to take place prior to liftoff. Such a decision accepts the chance of DCS in the relatively remote case of an unexpected cabin depressurization rather than subjecting the crew to the increased flammability hazard during every launch.

During any manned space flight, the potential for performing a space walk (Extravehicular Activity [EVA]) exists. Upon leaving the confines of the Shuttle to perform an EVA, a crew member will experience a change in ambient pressure from 14.7 psia (80% N_2/20% O_2) to 4.3 psia (100% O_2). Such an exposure would result in DCS if certain denitrogenation strategies were not employed. The two current methods during Shuttle operations are 100% O_2 prebreathe and/or "long" term reduction in the ambient Shuttle cabin pressure. If only a prebreathe is employed, the crew member is required to breathe 100% O_2 for 4 hours while in the suit. This means that the crew member is basically of no operational use during this period and also increases the ultimate suit time for a given EVA by nearly 50% in many cases. To

minimize these shortcomings, a decrease in the Shuttle cabin pressure to 10.2 psia for 12-24 hours may be performed, thus reducing the prebreathe required immediately before EVA by approximately 65-85%. This technique is not without its own disadvantages, which include the requirement to power down all unnecessary electrical equipment on board the Shuttle for the duration of the 10.2 psia cabin to avoid overheating equipment. So, while two apparently physiologically adequate denitrogenation techniques exist, they are both associated with significant operational impacts: reduction in crew member availability, or reduction in available electrical equipment due to thermal constraints.

In the future, Space Station construction will bring its own set of problems. The amount of EVA required for the construction of Space Station will be at least an order of magnitude greater than all our experience to the present. After looking at the incidence of DCS in ground-based EVA prebreathe studies, one cannot help but conclude that episodes of DCS will be seen during construction of the Space Station (assuming our ground based model is appropriate). This is a sobering thought as the operational impact of a DCS case could be disastrous for the Space Station in some situations, not to mention the welfare of the involved crew member. For this reason a variety of depress, prebreathe, and treatment strategies are under consideration, each one of which has definite operational impacts.

Questions exist in some quarters as to why the reported operational incidence of DCS associated with EVA differs from the observed ground based experience. This discrepancy between the lab and field operations is not new and exists in other venues as well. The lack of crew reported DCS may be attributed to absorption and focus on task, misinterpretation of muscle vs. DCS symptoms, career-specific considerations, and 0-g peculiar phenomenon. The greater observed ground-based incidence of DCS may be explained by the heightened awareness and/or anxiety caused by the test subject briefing, the type of activity during the test, the test facility and environment, and 1-g peculiar phenomenon. When all these causes for discrepancy are considered, it seems reasonable to conclude that, in general, the operational incidence is probably, if anything, under-reported and conversely, the laboratory incidence may suffer from over-reporting. (It is important to note, however, that anecdotal information from both operational and laboratory populations exist which supports the premise of under-reporting.) Unless the penalties to the individual for reporting a case of DCS are removed, there is virtually no chance of reconciling the observed discrepancy between lab-based data and data from any operational source.

In such an uncertain environment, how does any program decide on a course of action? Implicit in answering this question is the notion of acceptable risk. The question then becomes "Who decides?" and "How safe is safe?" In the U.S. Space Program, the "Who decides?" boils down to a question of management vs. researchers/clinicians. The roles here are often blurred, but ultimately might be reduced to the following: The researcher's duty is to quantify the risk with respect to the various operational options and supply this information to management, while management's job should be to weigh this information in light of mission requirements,

probability of mission success, cost, schedule, public opinion, etc., and the nebulous area of safety. As for "How safe is safe?" unless a given situation can be said to be 100% safe (i.e., a zero probability of onboard event), the definition of safe is a purely arbitrary one and is a programmatic decision. A cost/benefit analysis must be undertaken to arrive at what is "safe enough," and ultimately deciding what constitutes an acceptable risk is an administrative call by management after carefully weighing all input. The fact that the ultimate operational policy decision is not a technical one is sometimes difficult for the researchers/clinicians to accept, but it is the way the system works. The onus, therefore, is on the research community to clearly communicate to management the available courses of action and their consequences for various operational scenarios so that the best decision can be reached for each situation. Tradeoffs are often necessary and they are, in the final analysis, the province of management. Just as in the Shuttle program where compromises between pre-breathe vs. flammability and cabin depress vs. Shuttle power down have been made, so too will similar decisions be made affecting Space Station. If optimal decisions are to be made, it will be the researcher/clinician's responsibility to provide the appropriate facts and communicate them in an effective manner (i.e., assess the risk), so that management can arrive at policies which will result in an acceptable risk to the program and the ultimate accomplishment of mission objectives.

Additional Comments by Dr. Bagian

The Soviets have the ability to vary the pressure in their suits; in an EVA they will often go out at a higher pressure so they are still kind of denitrogenating and then they will go down (to a lower pressure) later. They will go down lower for brief periods when they have intense periods of activity so they can work more with their hands, and then they will go back up in pressure again. They are a little more flexible. Why do we have a discrepancy between what we have seen operationally and in the lab incidence of DCS? It is not a problem that is peculiar to NASA. I think there are several problems here. We see differences and they can be categorized in two areas. One is operational factors and the other is lab factors.

On the operational side let us start with crew motivation. When the crew you are out in EVA doing a job, it is not like a lab experiment where you are concentrating on "how do I feel?" You are busy doing a job. The suit is not comfortable. The way you fit in the suit, the geometry of the shoulder joint for instance is not natural and it is painful. I usually end up sloughing the skin over my clavicles on both sides after every run. I mean I get to the point where I have a periosteal bruise. So wearing the suit hurts a bunch. It tends to mask. That, combined with your focus of attention on your job, tends to distract you from what could be DCS symptoms that you do not even notice. You are just busy working. You are used to hurting. Third, there is misinterpretation of symptoms.

Then we come to the real problem. The fourth reason *is career consideration.* I do not think any crew member wants to ever own up, if they do not have to, to having DCS. I think this exists regardless of whether there is a written penalty that says, "if you have DCS you are out of here," or whether it is just the whispering campaign, "he had DCS, he is now a flawed crew member." I think most crew members believe that once they are identified as having one of these maladies, they are flawed compared to the person who has not had the problem. So it is not considered a career enhancing move to have an incidence of DCS. You would like to avoid reporting it. If you really did notice DCS, do you want to report it? "I will just watch it a little bit and if it goes away I will just forget about it. "

It is just a matter of career considerations. It is the same in the Air Force. Pilots do not report anything unless it has real, true operational safety impact. The system is not flexible enough to accept that. Even when you go for waiver, you can be off status a year or more while a medical board decides the fate of your career. What motivated individual would want to accept that uncertainty if it could be avoided?

On the lab factor side, by the time the test subjects are briefed, many can be very apprehensive about what is going to happen if they have a DCS episode. So they are going to be more inclined to report events that maybe are not limb bends.

In addition, the type of activity these subjects do does not emulate what we do in EVA.

DR. PILMANIS: I would like to comment on your assessment that operationally DCS incidence is an underestimate, and in the laboratory it is an overestimate. I cannot speak to the operations. However, I would venture the opinion that in the laboratory, at least in our laboratory using echoimaging, DCS is not overreported. I would call it pretty close to what is happening physiologically.

DR. BAGIAN: I think one of the reasons we see a discrepancy is that one set of subjects may tend to go one way because they have vested interests, and the other may tend the other way. I am not sure what the spread is. I think what you say now is probably more true than it was ten years ago because the technology is improving.

DR. PILMANIS: Has a walk-around O_2 bottle system been looked at? You talked about usable crew time. Our Inside Observers use a walk-around bottle for prebreathing routinely. Could you not have a simple walk-around system and remain productive during prebreathing?

DR. BAGIAN: We have talked about that, and I do not know where it currently stands. The crew acceptance of that was terrible, I mean they did not want to hear it.

MR WALIGORA: One of the options that is being talked about for the station is to lower one of the modules to 10.2 psia overnight and then to do whatever additional prebreathe they need on a walk-around system and then put the suit on. Thus, prebreathe would be reduced.

DR. WENZEL: We talked a lot about the discrepancies between ground level DCS and orbit DCS and, apart from these reporting problems we talked about the only serious physiological argument which is left, the difference between one G and zero G. Should we then do ground level experiments under immersion or head-down tilt.

MR WALIGORA: We are going to do that. We have the protocol approved by the human use committee and should be starting in a couple of months.

DR. VANN: Getting back to the question of acceptable risk, making DCS risk prediction for any given pressure profile, the relevant figure is the upper 95% prediction interval on that risk. Thus, it becomes a little worse than the predicted risk would indicate. That is one of the reasons why the statistical methods become important. You try to reduce the number of undetermined parameters to a minimum because that makes your prediction interval smaller.

DR. BAGIAN: We are discussing the role of assessment of risk versus acceptable risk.

DR. VANN: I think that is right. Acceptable risk determination requires value judgments. You have to make a risk/benefit estimation. There is no analytical way you could do that. You can however, assess your risk analytically based on data.

MR WALIGORA: When presenting the risk of DCS and the tradeoffs, I think it is appropriate for the medical people who are involved to make a recommendation. It is also appropriate to state all the options and the impacts.

DR. BAGIAN: I would consider that part of the assessment. Our job is education, to educate the managers to understand what the tradeoff is between a T.R. of 1.4 and 1.2, as an example, and then let them make an informed decision. I think we hurt ourselves in the long run, and we end up with some of the problems that we have on station design because we have not educated management, upper management, enough to what the costs are. We have just given them answers, solutions, and they do not understand where the solutions came from. Then they make far reaching programmatic decisions inappropriately.

DR. PILMANIS: An example where this might be put into practice is the development of a suit for Mars. None of the suits we currently have will work, because they are too heavy for Mars' gravity. That means a total redesign of an EVA suit. We cannot even go back to the moon with the soft suit because so many things have been added, it is too heavy even on the moon. If we are going to redesign the suit, it is a good opportunity to look ahead and redesign the system with DCS consideration included from the start.

1990 Hypobaric Decompression Sickness Workshop

Session Four: DCS INCIDENCE AND REPORTING

James R. Francis, PhD, MSc, MB, BS, Chairman

USAFSAM HYPOBARIC DECOMPRESSION SICKNESS RESEARCH SINCE 1983

James T. Webb, PhD
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Introduction

Six major decompression sickness (DCS) protocols, one of which includes five studies, were initiated or completed during 1983 to 1990. Data from these experiments are stored on the USAFSAM VAX computer. The studies have resulted in numerous publications which are listed as references in the annotated bibliography handed out to participants in this workshop (Webb et al., 1990). Two protocols addressed the incidence of DCS at higher altitudes and another investigated the threshold of bubble formation at lower altitudes. The results of these efforts are pertinent to our theme.

Methods

During all of the hypobaric DCS studies at USAFSAM, volunteer subjects were monitored for venous gas emboli (VGE) with ultrasonic Doppler equipment; intravenous gas bubbles were graded by the method of Spencer (1976). During Doppler monitoring, joint flexion aided in release of bubbles from extremities and increased the reliability of ultrasonic bubble detection.

Spencer Scale for Grading Doppler Bubbles

Grade 0 — No bubbles;

Grade 1 — An occasional bubble signal, with the majority of the cardiac cycles being bubble free;

Grade 2 — Bubbles in many, but less than one-half of the cardiac cycles;

Grade 3 — Bubbles in most of the cardiac cycles; and

Grade 4 — Numerous bubbles that obscure the heart sounds.

Anytime prebreathing was required by the protocol, 100% oxygen was supplied to the mask. An Intertechnique neck-seal mask was used during prebreathing and during the exposures.

Results and Discussion

Protocol:	Bends Screening Index Study
Prebreathe:	60 min
Exposure Duration:	8 h
Exposure Breathing Gas:	100% oxygen

ALTITUDE FEET MSL	# OF MALE SUBJECTS	EXPOSURES TOTAL #	EXPOSURES % W/DCS	MEAN TIME TO DCS (MIN)	% EXPOSURES W/GRADE 3-4 VGE	% EXPOSURES W/GRADE 1-2 VGE
30,000	31	38	82	87	79	3
27,500	33	83	80	142	64	4
25,000	27	28	79	149	69	9
22,500	19	46	52	188	57	2

During the Bends Screening Index Study, male subjects prebreathed 100% oxygen for 1 hour prior to ascent and throughout the exposures which lasted approximately 8 hours. The exposures were terminated at the onset of Type I DCS symptoms (Grade 2; continuous mild to moderate [or greater] joint pain) or Type II DCS. Bends screening exercises (Krutz and Dixon, 1987) consisted of five upward extensions of the arms while holding 5-lb weights and five chair-height knee bends every 15 minutes.

The results indicate generally greater incidence of DCS and reduction in average time for symptoms to occur with increasing altitude. The incidence of Grade 3 and 4 bubbles above 25,000 ft is considerably above 50%.

Protocol:	Decompression Sickness Protection Using an 8 psia Suit Environment (5 studies)
Prebreathe:	None
Exposure Duration:	6 h
Exposure Breathing Gas:	50% oxygen

ALTITUDE FEET MSL	# OF MALE SUBJECTS	EXPOSURES TOTAL #	EXPOSURES % W/DCS	MEAN TIME TO DCS (MIN)	% EXPOSURES W/GRADE 3-4 VGE	% EXPOSURES W/GRADE 1-2 VGE
16,500	32	94	0	N/A	37	16
16,000	25	25	0	N/A	44	4
15,000	20	20	5	261	25	0
14,500	10	10	0	N/A	10	20
13,000	22	23	0	N/A	22	13
11,500	6	6	0	N/A	0	17
10,250	9	9	0	N/A	0	22
9,000	2	2	0	N/A	0	0

The protocol titled "Decompression Sickness Protection Using an 8 psia Suit Environment" consisted of five studies encompassing several exposure altitudes. The male subjects were decompressed, without prebreathing, to the exposure altitudes while breathing 50% oxygen and 50% nitrogen; the breathing gas planned for the next-generation Extravehicular Activity (EVA) pressure suit. The EVA exercises performed during these studies were as described by Dixon et al. (1986) and were similar, both quantitatively and qualitatively, to light workloads expected during actual EVA. The exercise workloads consisted of a different activity at each of three work stations and were designed to emphasize upper body stress. Inderbitzen and DeCarlis (1986) found that these exercises required a mean of 148 kcal/h (1.8 kcal/h/kg) for the male subjects. Results of this series of studies indicate increasing levels of Grade 3 and 4 bubbles at the higher altitudes. It is of interest that at least some bubbles were evident at exposure altitudes as low as 10,250 ft. The lack of Grade 3 or 4 bubbles at 11,500 ft resulted in the conclusion that 9.5 psia was the minimum safe pressure for a zero-prebreathe pressure suit (Krutz et al., 1988).

A later study (#7 in the annotated bibliography) demonstrated that oxygen toxicity was not apparent in individuals exposed to 100% oxygen at 9.5 psia (11,500 ft) for 8 h/day for 5 consecutive days (Webb et al., 1989b). The increased oxygen concentration in the breathing gas during the oxygen toxicity study may have been the reason no bubbles were detected throughout the exposures. This result led to a protocol designed to determine the lowest pressure to which subjects can be decompressed, while breathing 100% oxygen, with no bubble formation. This study may help to fill the large gap in our data from 16,500 ft to 22,500 ft and provide better information about denitrogenation rates at these altitudes.

Protocol:	Inflight Denitrogenation Study
Prebreathe:	1h or 2h, 100% Oxygen
Prebreathe Pressure:	Ground Level, 8,000', 12,000', or 16,000'
Exposure:	29,500', 4h, 100% Oxygen

PREBREATHE ALTITUDE (ft)	PREBREATHE TIME/h	EXPOSURES w/VGE	EXPOSURES w/DCS
16,000	1	91%	55%
12,000	1	82%	64%
8,000	1	85%	62%
0	1	75%	83%
MEAN		83%	66%
16,000	2	70%	40%
12,000	2	56%	44%
8,000	2	64%	43%
0	2	56%	44%
MEAN		61%	43%

The inflight denitrogenation study, currently underway, is providing information which may allow pilots to begin their missions with less prebreathing. Preliminary data show that prebreathing is just as effective at cabin pressures of up to 16,000 ft as it is at ground level. Dr. Pilmanis will present a paper on this study at the May 1991 annual scientific meeting of the Aerospace Medical Association.

An approved protocol, yet to begin, may provide an alternative to long prebreathe times or may increase the effectiveness of shorter periods of denitrogenation (Webb et al., 1989a). Exercise with prebreathe was tried in the 1940s, but was not operationally feasible due to long durations of strenuous effort assumed necessary for positive effects on denitrogenation efficiency. DCS rates during 30,000 ft exposures after a short-duration, strenuous exercise period while prebreathing will be compared to identical exposures after equal as well as longer periods of resting prebreathe.

The data contained in the USAFSAM Hypobaric Decompression Sickness Research Database and other past, present, and future results will aid in completion of the DCS model discussed by Dr. Pilmanis. The database can also serve as a standardized reference for similar exposures of operational concern which will be discussed throughout this workshop.

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MR GILBERT: Often this sort of data gets lost or is unavailable to others. I would like to propose that this group try to design a skeleton for a database or central repository. This skeleton would allow for the basic information that you wish to see from any particular experiment, and add on any particular fields that are thought to be relevant to the types of experiments being done.

DR. FRANCIS: I think that is an excellent idea. You have an end point of DCS. Could you elaborate and define that. Also, how it is audited prior to entry into database and what are the criteria?

DR. WEBB: It is audited by the principal investigators and the grading system we use is either Type I or Type II. If it is pain only DCS, then it is logged in intermittent or constant. Also, there is an intensity scale of 0 to 10. Typically, we do not see over about a 3 before we bring subjects down. Grade 1 would include the fullness of the joint. Grade 2 is a continuous pain at any level.

DR. FRANCIS: Have you had anything other than pain as the presenting symptom?

DR. WEBB: Yes, there have been a few instances of neurological symptoms.

MR GILBERT: We do a 12-hour observation after the postbreathe, just to make sure there are no recurrence of symptoms. If you terminate a test, and you bring somebody down regardless of whether they have a remission of symptoms on the way down, do you do a 12-hour observation after the postbreathe?

DR. OLSON: We do not use a 12-hour observation period. We make sure that they are clear of bubbles and symptoms and instruct them on what they should and should not do. After the 2-hour postbreathe, we send them home with a written set of instructions and phone numbers. In subjects who follow our instructions, we have not had any recurrence.

MR GILBERT: My question was related specifically to those subjects whose DCS symptoms are significant enough to cause a test termination.

DR. PILMANIS: We may have a problem of definition. You terminate your subjects with Grade 3 symptoms , and we terminate with Grade 2.

DR. PILMANIS: This is one of the inconsistencies in our databases. If you terminate a run at the first indication of a symptom, you will not know what happens further down the line. You do not have those data.

DR. BALLDIN: You are not giving them prophylactic hyperbaric oxygen treatment at anytime?

DR. WEBB: No, not without symptoms.

DR. BALLDIN: When I was provoking altitude decompression sickness, I usually gave them an hour hyperbaric treatment afterwards to make sure there would be no sequelae. That was just to avoid discussions afterwards that there could be some problems in the future.

COLONEL SHEFFIELD: Where do you put the individuals for that 12-hour observation? Are they hospitalized, sent home, kept at your unit?

MR GILBERT: We have a hyperbaric medicine trained EMT who observes them for 12 hours in a Holiday Inn.

COLONEL SHEFFIELD: The reason I asked the question is because there have been concerns about follow-up on treatment cases, who provides the observation? In some cases the individuals were hospitalized in one of our larger facilities, being observed by people who had absolutely no concept of why the subjects had been hospitalized. It is better to have them sent home to their spouse who would know what to do in case they had a problem.

DR. PILMANIS: We have found that at least one of our subjects was discouraged from reporting DCS because of what happens if they report it and have to be treated. If you report it, not only are you going to spend 6 hours in the chamber, but then you are going to be taken to a hospital and have IVs put into you, etc. It is easier just to keep your mouth shut. So here we have a situation of under-reporting in lab experiments.

MR GILBERT: NASA has a sort of a carrot here. Not only are our subjects paid their normal pay for the day, but they also get paid for the 12 hours of their "convalescence," and they also are allowed to order room service.

DR. VANN: Now you have got an over reporting problem. At Duke, we use a slightly different procedure. As the Air Force does, we terminate a flight with any symptoms. If the symptoms disappear on descent before we have reached 10,000 feet, then we put them on 100% oxygen for 1 hour at sea level. However, if we still have symptoms at a altitude lower than 10,000 feet, then we will generally give them a treatment Table 5. We have had four or five who were treated for either recurrence or incomplete resolution at ground level.

DR. WEBB: In the AF, we do have two distinctly different groups of subjects who participate in these studies. One group is the civilians, who are receiving $10 an hour for their services, including the time in the hospital and the treatment. The other group is the military subjects who receive $110 a month hazards and debts paid

regardless of how many times they are exposed. The motivations vary between these groups.

LTC WORKMAN: As Colonel Sheffield pointed out, it is often better to send the subjects back home to their spouses. However, in the case of someone who may be single, or someone living by themselves, we may choose not to send them home by themselves. If they live alone, then perhaps hospitalization is a better solution.

THE USAF CHAMBER TRAINING FLIGHT PROFILES

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The U.S. Air Force (USAF) uses chamber flight profiles to train aircrew members on the physiological hazards of altitude. There are approximately 10 training profiles which will be described. They will be referred to by Dr. Weien when he presents the data from his study. These chamber flight profiles, however, do not include the research profiles or the pressure suit profiles.

Type I Flight

The Type I training flight is routinely used for "original" students (Fig. 1). These are future aircrew members (undergraduate pilots, undergraduate navigators, loadmasters, Weapons Directors, boom operators, etc.) who need to learn their hypoxia symptoms, how the oxygen systems work, and to feel comfortable with these experiences. This Type I profile, as with all chamber flight profiles, starts with an excursion up to 5,000 ft and back down. This is called the ear and sinus check and is incorporated into the 30-minute denitrogenation period. When the 30 minutes has been completed, we start ascent. An ascent rate of 5,000 ft/min is maintained until the chamber reaches 35,000 ft. At 35,000 ft, one student removes his/her oxygen mask, turns off the oxygen supply system to his/her station and becomes a spectacle for the other students. Once this student sufficiently demonstrates the effects of hypoxia, as determined by the inside observers, we start to descend the chamber at a rate of 5,000 ft/minute, the normal rate of descent. We descend to 30,000 ft and have another volunteer demonstrate the effects of hypoxia. We usually ask the students to compare the demonstrators' time tolerance to hypoxia. Following this demonstration, we descend to 25,000 ft where all other students except the previous two demonstrators' experience hypoxia and learn to correct for it.

The next step down is to 18,000 ft where we demonstrate visual acuity by having the students drop their masks from the right hand side. We then distribute a color chart to them and turn down the lights. After 5 minutes, we instruct them to look at the card and put the mask on. Even with mild hypoxia, visual acuity is markedly effected. On the way to ground level the students practice using the emergency oxygen systems.

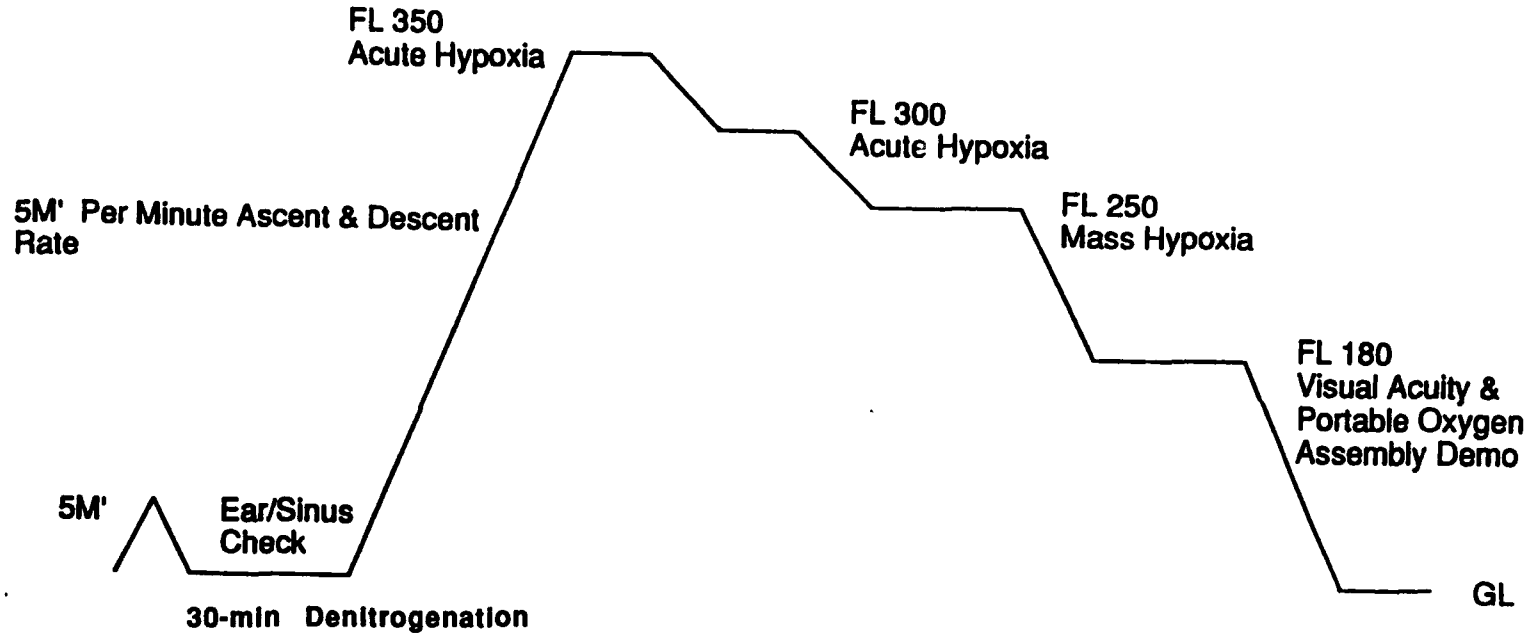


Figure 1. Type I chamber flight profile.

Type II Flight

The Type II chamber flight profile that a novice flyer would experience would be one to the equivalent altitude pressure of 43,000 ft (Fig. 2). The normal ear and sinus check and 30-minute denitrogenation precedes the flight to 43,000 ft. The overall objectives for a Type II chamber flight are the same as for Type I, with the addition that the students practice pressure breathing, experience communication problems during pressure breathing, and use the emergency and portable oxygen systems and equipment. These additional objectives give the students confidence in the equipment to function effectively during a possible decompression. After 43,000 ft is reached,we quickly descend to 25,000 ft and have the students experience and correct for hypoxia. Finally, we descend to ground level. On the way to ground level, we have students connect their oxygen systems to high pressure emergency cylinders. This is the same equipment one would find in a parachute pack. The idea is to experience what this system can do for you if needed.

Usually, following a Type II chamber flight, the "original" or novice trainee must experience a rapid decompression flight in which the main portion of the chamber is evacuated and depressurized to the equivalent pressure of 30,000 ft (Fig. 3). The students are seated in the lock portion of the chamber and are depressurized to a simulated altitude of 8,000 ft, roughly equivalent to the cabin pressure most airplanes use. The students are briefed on the proper procedures in the event of a rapid decompression. We then decompress the lock portion of the chamber until the lock and the main chamber equalize at approximately 22,000 ft. This decompression takes about 1.5 seconds. When the students say they feel "normal," we start the descent to ground level. During the excursion to ground level, we discuss with them the importance of proper treatment in case a decompression occurs in an aircraft.

Type III Flight

The Type III flight profile is used for training passengers who will fly on military aircraft (Fig 4). The initial portion of the flight is exactly the same as the flights described earlier. The major difference in this flight is the rapid descent from 35,000 ft to 25,000 ft. What we are trying to simulate is a parachute freefall. Once 25,000 ft is reached the hypoxia demonstration ensues and this demonstration is followed by a descent to 18,000 ft where the visual acuity demonstration takes place. From 18,000 ft we descend to ground level.

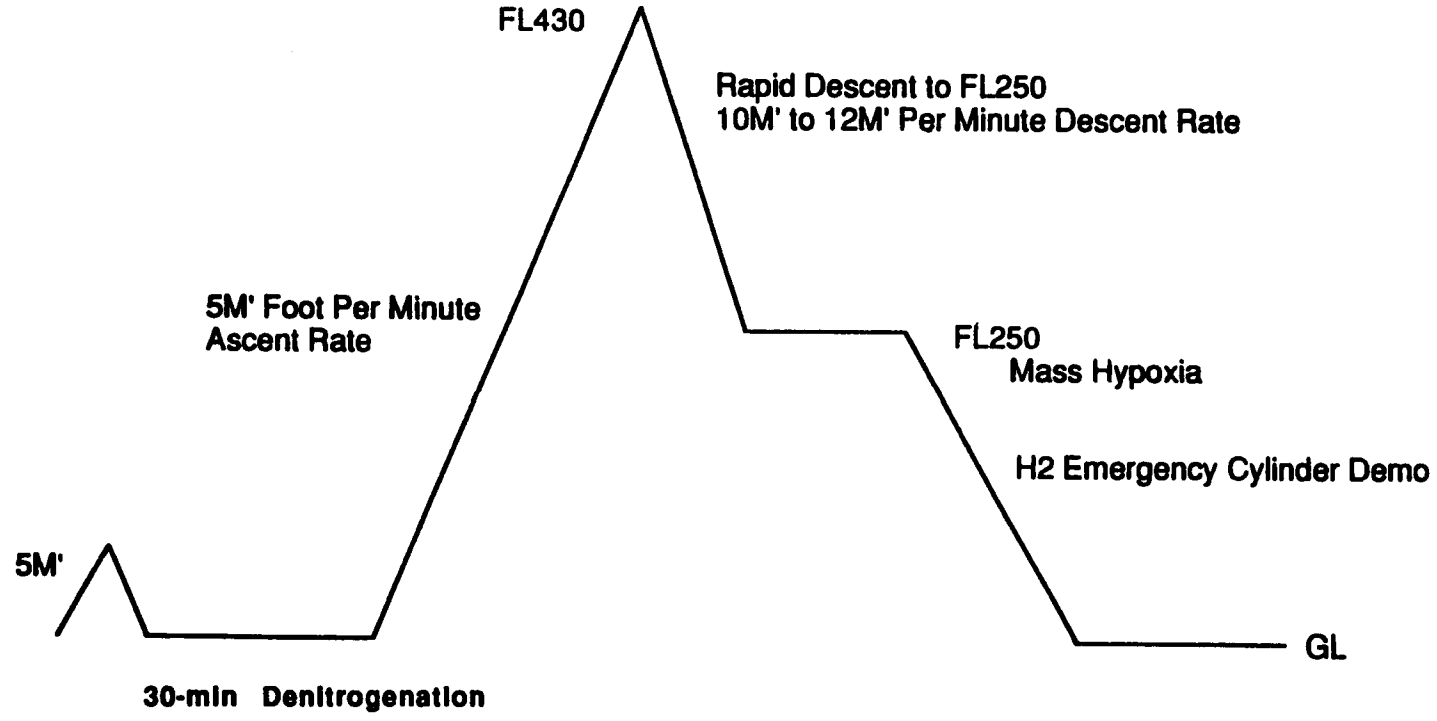


Figure 2. Type II chamber flight profile.

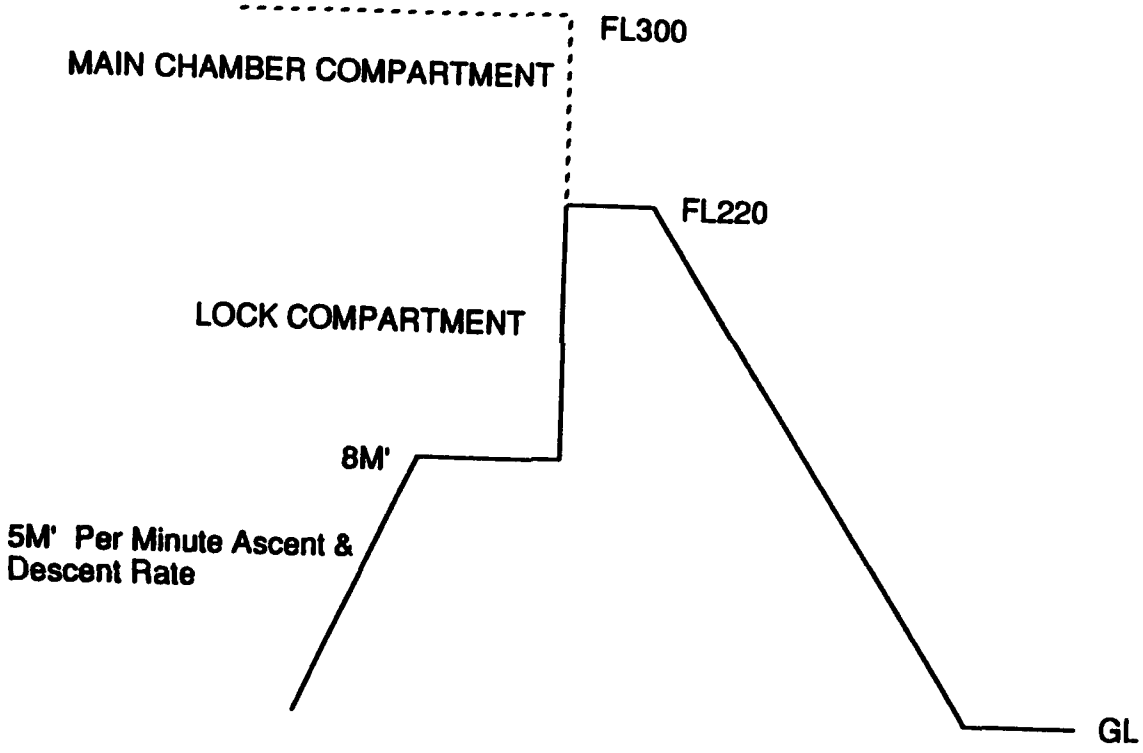


Figure 3. Rapid decompression flight profile.

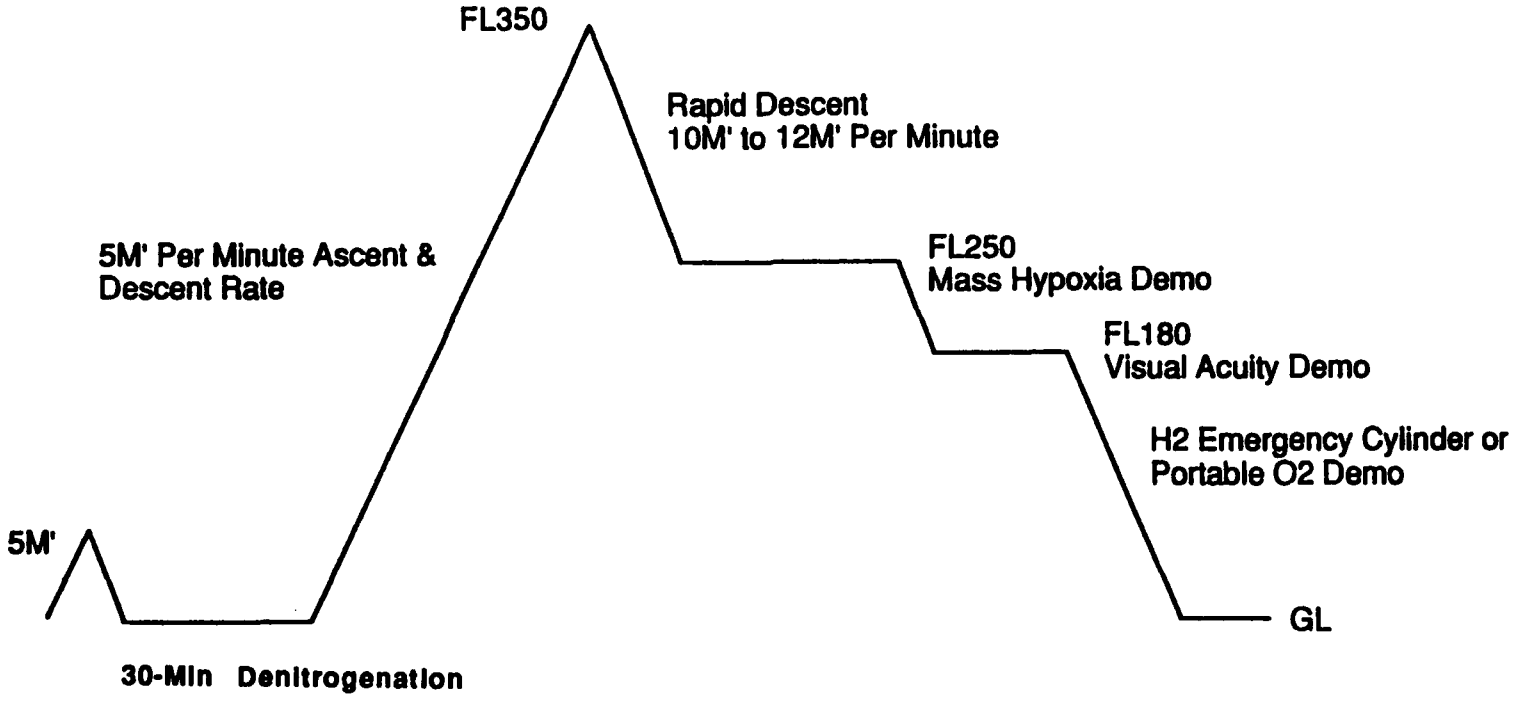


Figure 4. Type III chamber flight profile.

Type V Flight

The Type V flight profile is used for experienced aircrew (Fig. 5). The group consists of pilots, navigators, and other crewmembers who have been flying for a few years and who have been through the original training profiles. These people experience this profile once every 3 years unless they change weapon systems. This profile proceeds to the equivalent pressure of 35,000 ft and is immediately followed by a rapid descent to 8,000 ft. This descent simulates a freefall one would experience in an ejection scenario. On reaching 8,000 ft, we perform a rapid ascent to 25,000 ft which simulates a slow leak, canopy seal problem. At 25,000 ft, the students experience hypoxia symptoms. We then descend from 25,000 ft and level off at 18,000 ft where they experience the visual acuity demonstration. As with all other flights, we descend to ground level after the visual acuity demonstration.

Type VI Flight

The Type VI flight is designed to reacquaint helicopter pilots and pilots who fly slower moving aircraft with hypoxia symptoms. We commonly call this profile a low and slow flight. The rates of ascent basically mimic a typical flight of the aircraft. We only take the crewmember to 18,000 ft because that is the usual altitude to which these aircraft fly.

As with all chamber flights, we try to customize our flights to fit the customer.

Specialized Flights Conducted at USAFSAM Only

Since the USAF School of Aerospace Medicine is at Brooks AFB, special classes are conducted here only. One of these is the Flight Nurse class. This group, along with the Air Force Academy cadets and the pararescue students, experiences decompression sickness more than any other group of individuals we fly in the chamber. For that reason, we modified the Type I chamber flight to only reach an altitude of 25,000 ft. This profile is called the Flight Nurse Type I flight (Fig. 6). Here they experience hypoxia, descend to 18,000 ft for a night vision demonstration (visual acuity demonstration) and then return to ground level.

FN Type II Flight

In the FN Type II flight profile, flight nurses ascend to 30,000 ft where they pressure breathe (Fig. 7). One person demonstrates hypoxia. Next, they descend to 8,000 ft and start a slow decompression up to 22,000 ft. This decompression, which takes about 12 seconds, simulates the pressure changes and the physiological problems that may happen in an aeromedical evacuation aircraft. They then descend from 22,000 ft to ground level.

One other specialized type of flight is the Health Professions Scholarship program profile (Fig. 8). This flight is mainly for orientation to the altitude environment.

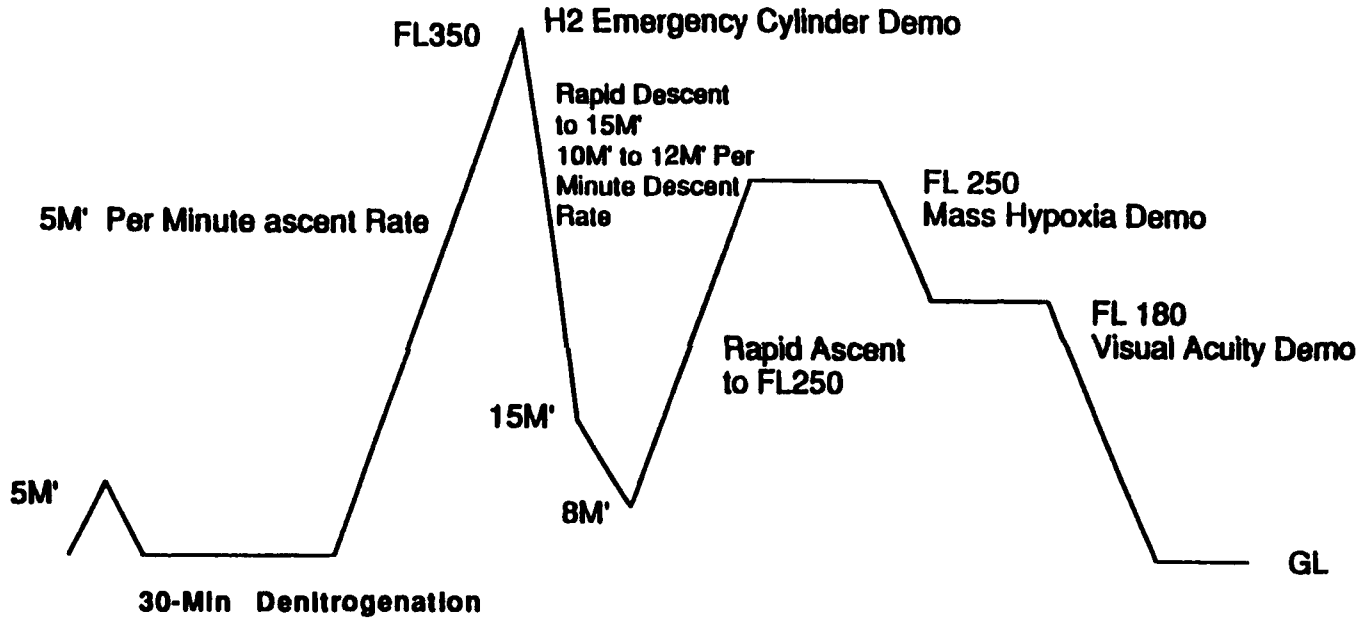


Figure 5. Type V chamber flight profile.

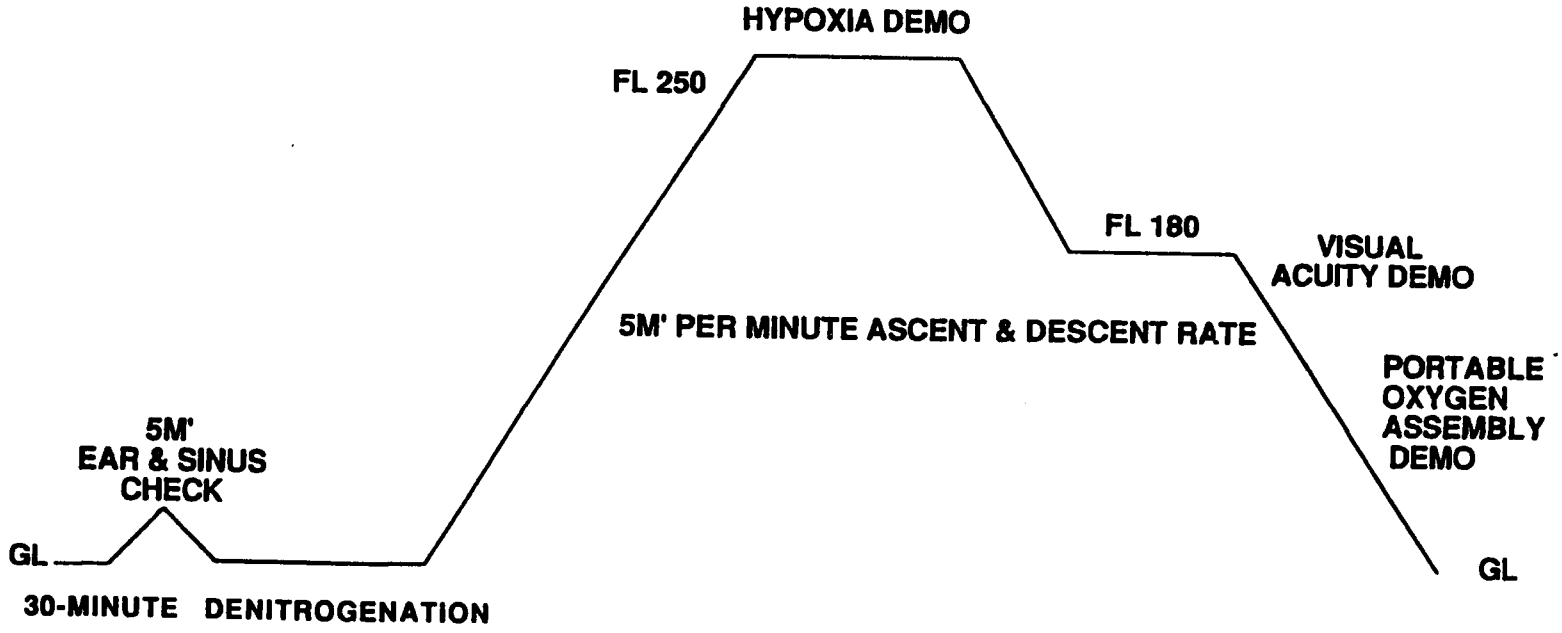
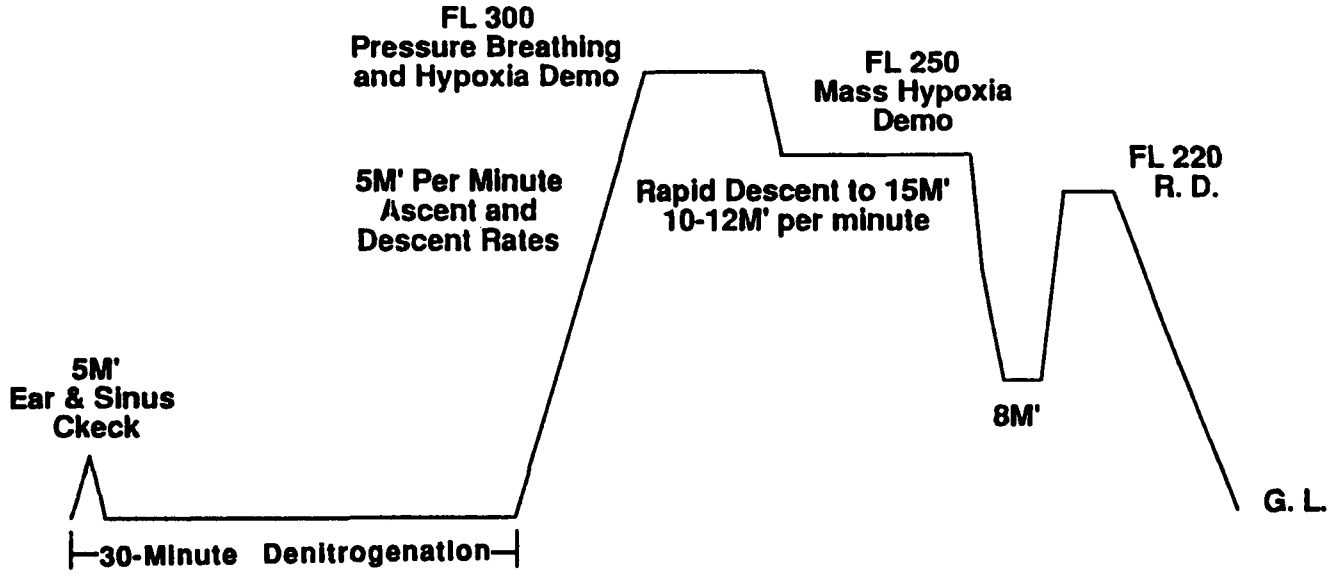


Figure 6. FN/AET Type I chamber flight profile.



1. All students (except the hypoxia demonstration student) will practice pressure breathing with the regulator in the ON, 100%, and Emergency setting during Pressure Breathing Demo.
2. All students will drop their masks at 8M' and use the MBU-8/P (passenger) mask after the R. D.

Figure 7. FN/AET Type II chamber flight profile.

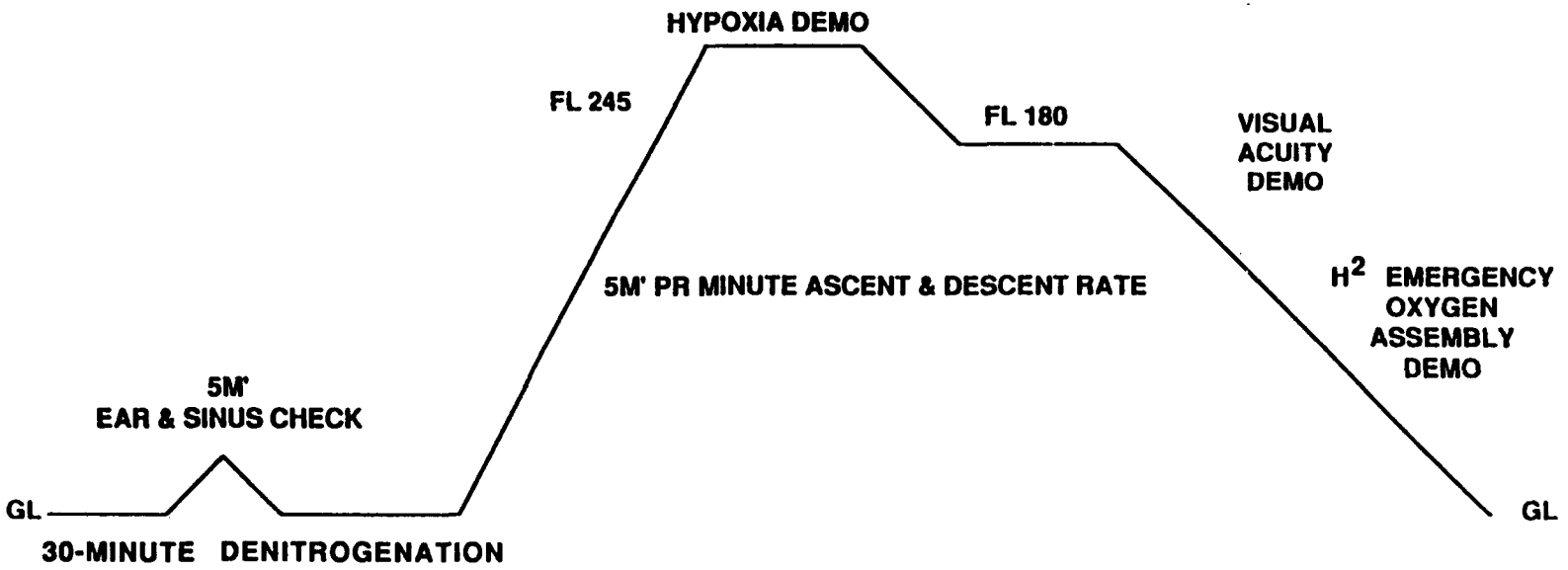


Figure 8. Health Profession Scholarship Program (HPSP) flight profile.

FAA Training profile

Occasionally, the USAF conducts training for the Federal Aviation Agency (FAA) (Fig. 9). The flight profile differs from the previous profiles in that there is a 15-minute denitrogenation period, rather than 30 minutes, and there is a 3,000 ft/minute ascent and descent rate. The profile goes to 25,000 ft for the hypoxia demonstration and descends to ground level. If the FAA requests, we will conduct a visual acuity demonstration for the passengers.

The data for the incidence of decompression sickness from these profiles will be presented later.

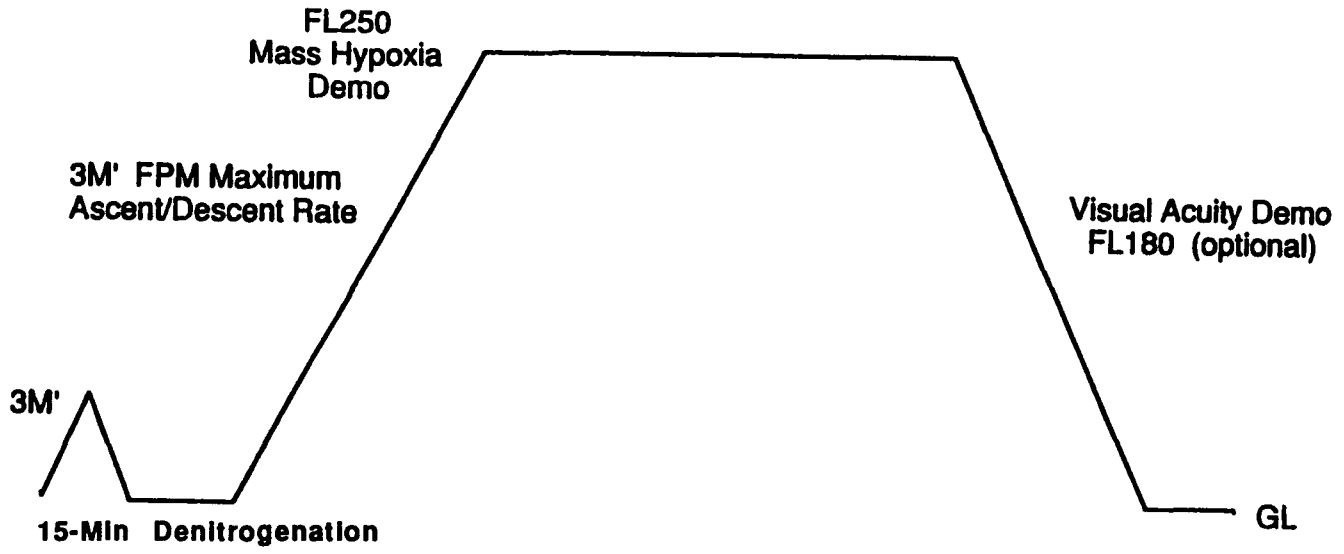


Figure 9. FAA flight profile.

DR. BAGIAN: Could you clarify the comments about the pararescue people?

COLONEL SHEFFIELD: In 1977, we looked at the Air Force Academy problem of increased decompression sickness incidence. We had a tenfold increase over other USAF groups. It turned out that three USAF groups, the flight nurses, the parascuba rescue people, and the Academy cadets had the highest DCS rates. We did not change the flight profile for the parascuba rescue. Rather, we tried to de-emphasize the amount of physical conditioning they were doing, both pre and post exposure. In the case of the Air Force Academy Cadets, we restricted their altitude to 25,000 feet, and let them stay at Peterson AFB for 12 hours after the exposure rather than reascend to the Academy. However, the ultimate solution to the problem was the restriction of freshman cadets from chamber training. We still have the flight nurse situation, and we have again restricted them to 25,000 feet with the exception of the second chamber flight which has a brief excursion to 35,000 ft. We are still training parascuba rescue people on the standard flight profile.

DR. BAGIAN: How do these high incidence situations relate to physical conditioning and exercise?

DR. VANN: Several years ago, there was a paper about a group of Army Special Forces undergoing HALO training, including altitude chamber exposures.

DR. VANN: Out of a group of nine students, there was a very high incidence of bends. All these people were very athletic. It is anecdotal but suggestive that there may be a correlation between susceptibility to decompression sickness and physical activity during your normal life. A control study is of course needed.

DR. HAMILTON: Could this have been a factor within the AF Academic freshmen? Were they really working them hard?

COLONEL SHEFFIELD: We think that the real problem with the freshmen was the high stress levels that the individuals were undergoing at that stage in their training.

DR. HAMILTON: Could these freshmen have faked DCS?

COLONEL SHEFFIELD: I do not think so. Such things as loss of anal sphincteral control are very difficult to fake. It is possible that some of them convinced themselves that they had DCS when they really did not, but most likely most of the cases were real. I think that we are talking about a very highly stressed group of people. The same thing applies to the flight nurse group, and the parascuba rescue group. I think that was the one thing that they all had in common.

DR. PILMANIS: Could we compare the training chamber profiles used by the Air Force, Army, Navy, Canadians, and British? Are these profiles pretty much universal?

COMMANDER BASON: No. Navy profiles are much less hazardous than what was just described. We have one profile. We go to 25,000 feet at 5,000 feet per minute, do our hypoxia demonstrations, and come back down. We have no 40,000 ft runs. Those were canceled in 1979. Our inside observers prebreathe for 30 minutes. That practice was started in 1982.

MAJOR WEIEN: The Army uses standard Air Force protocol, right down the line. We train a lot of Air Force students. Fort Rucker trains Air Force helicopter pilots, as well as our own helicopter pilots, and we use all those initial profiles including the 43,000 foot flights, as well as the standard Type 4 flights for retraining. We follow the standard Air Force rates of ascent and descent.

DR. HARDING: In Great Britain, the profiles vary between being rather like the United States Navy and more severe than the severest profiles that have just been described.

DR. PILMANIS: More severe in what way?

DR. HARDING: We do rapid decompressions to 45,000 feet or 56,000 feet routinely. The 56,000 ft flights require only preoxygenation for half an hour. No decompression sickness has ever occurred.

COLONEL SHERMAN: USAF Pressure Suit Training chamber flights go to 75,000 or 100,000 ft. However, since they are in a pressure suit, the men are not exposed to anything over 35,000 ft. They get 30-minute prebreathing and ear and sinus check to 5,000 ft and then go up to about 18,000 ft to level off and check the equipment one more time, and then on to 75,000 or higher. Assuming the pressure suit is working properly, the pilot goes to 35,000 ft, descends to 29,500, and then has a rapid decompression to 35,000 ft again. The pilot then goes back down to 25,000 for a hypoxia demonstration and then back down to ground level. There was some discussion about having the high-altitude pilots exposed to higher chamber flights. They have capability for up to 45,000 feet in the aircraft. If they had a loss of both aircraft and suit pressurization, they would have to use that regulator to bring them down to the lower altitude. Since it puts out a lot of pressure, there was some thought of letting them have an "eye opening experience" in the chamber of going to 45,000 and then starting a 2,000 ft per minute descent. This would definitely open the aircrew members' eyes to what they might be faced with from the pressure breathing standpoint. The RAF has a good pressure breathing scenario, at least they used to, in which they let the pilot sit and pressure breathe for about 30 minutes.

DECOMPRESSION SICKNESS DUE TO USAF ALTITUDE CHAMBER EXPOSURE (1985-1987)

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The primary purpose of this study was to report the incidence rate (DCS cases/altitude chamber exposures) for type V altitude chamber training flight.

The records of all USAF DCS cases due to hypobaric chamber exposures for the period Jan 1985 - Dec 1987 (282 cases) were reviewed.

We calculated incidence rates for total cases, gender, duty position, DCS diagnosis category and nine different altitude chamber training flights.

Table 1. Age Distribution.

AGE: Males (n=234) x = 26.9 yrs SD = 6.6 yrs

Females (n=48) x = 25.4 yrs SD = 5.0 yrs

Overall (n=282) x = 26.5 yrs SD = 6.5 yrs

Table 2. Total Number of Cases.

Year	Total	Type I	Type II
1985	95	83	12
1986	88	75	13
1987	99	92	7
85-87	282	250 (89%)	32 (11%)

Table 3. DCS Incidence Rates.

	Cases	Exposures	Inc Rate %
Student	228	192,623	.118
Observer	54	46,261	.117
Male	234	219,682	.107
Female	48	19,170	.250
Overall	282	239,343	.118

Table 4. Incidence Rates (%) - DCS Historical Comparison.

Author	Period	Students	Observers	Total
Furry	CY 59-68	.105	.058	.096
Bassett	CY 68-72	.102	-----	-----
Bason	CY 72-75	.029	.380	.089
Davis	CY 73-76	.072	.210	.100
Howard	CY 78-81	.063	.140	.078
Baumgartner	CY 85-87	.118	.117	.118

Table 5. Cases and Incidence Rates by Altitude Chamber Training Flight.

Alt Chm Flight*	DCS Cases Type I	Type II	Type III	Altitude Exposures	Incidence Rate %
I (350)	54	3	57	33,962	.167
II (430)	54	17	71	31,810	.223
III (350)	18	3	21	15,021	.140
IV (250)	3	0	3	8,486	.035
V (350)	83	6	89	69,995	.127
VI (180)	3	1	4	2,913	.137
FN I (250)	12	0	12	1,761	.681
FN II (300)	7	0	7	1,729	.405
USAFA (250)	7	1	8	4,126	.194
Other	9	1	10	--------	------
Overall	250	32	282	239,343	.118

*Maximum altitude chamber flight level (FL)

Table 6. Incidence Rates by Chamber Flight for Student-Observer and Male-Female.

Alt Chm Flt	Student	Observer	Male	Female	Overall
I	.133	.323	.147	.428	.167
II	.242	.138	.209	.417	.223
III	.132	.173	.137	.183	.140
IV	.029	.061	.037	0	.035
V	.132	.105	.123	.191	.127
VI	.147	.130	.111	.565	.137
FN I	.825	.306	.401	.919	.681
FN II	.426	.310	.137	.593	.405
USAFA	.234	0	.191	.211	.194
Overall	.118	.117	.107	.250	.118

Table 7. DCS Type II Cases and Incidence Rates by Chamber Flight.

Alt Chm Flt	Cases	Inc Rate %	
I	3	.009	
II	17	.053	STU: 32 (.017)
III	3	.020	OBS: 0 (0)
IV	0	-----	Male: 26 (.012)
V	6	.009	Female: 6 (.031)
VI	1	.034	
FN I & FN II	0	-----	
USAFA	1	.024	
Other	1	-----	
Overall	32	.013	

Table 8. Maximum Altitude of Exposure for DCS Cases.

Altitude of Exposure	Cases	Exposures	Inc Rate %
Less than or equal to 25,000 ft	34	80,048	.042
Greater than 25,000 ft	248	157,249	.158
Overall	282	239,343	.118

Table 9. Symptom Onset.

Altitude: 58 cases
Ground Level: 221 cases

Range: 0-36 hours
Mean: 5:06 hrs:mins
SD: 6:46 hrs:mins
Median: 2:00 hrs:mins

Greater than or equal to 12 hours - 38 cases
Greater than or equal to 24 hours - 9 cases

Table 10. Previous DCS.

21 of 282 (7.4%) had previous DCS

15 - this was 2nd incidence
5 - this was 3rd incidence
1 - this was 4th incidence

17 - repeated with TYPE I diagnosis

4 - repeated with TYPE II diagnosis

Table 11. DCS Treatment Disposition.

Treatment Disposition	Cases	Percent
100% Oxygen GL	78	27.7
HBO Table V	34	12.0
HBO Table VI	161	57.1
HBO Table VIII	7	2.5
HBO Other	2	0.7
Total	282	100.0

From HOWARD (1982)

100% Oxygen GL	132	45
HBO	160	55
Total	292	100

Conclusions

1. The TYPE V altitude chamber training flight has a risk of DCS significantly lower than that of other altitude chamber training flights.

2. The TYPE II altitude chamber training flight has a risk of DCS significantly higher than that of other altitude chamber flights.

3. Female risk of DCS is significantly higher than male risk of DCS (2.34 rel risk, $p<.0000001$).

4. There is no significant difference in risk of DCS between students and inside observers.

DR. FRANCIS: Can you describe what Type I and Type II mean to you.

MAJOR WEIEN: Type I is, by Air Force definition, skin bends and limb bends. Type II consists of neurologic and cardiovascular presentations. There is this entity called the Type I peripheral nervous system case, that we clarified yesterday was an administrative category used to avoid grounding these aviators for life. I know I saw plenty of cases that I would have called Type II if I were the doc making the diagnosis, that had been called Type I for those administrative purposes. Something else I will point out is that the flight nurse DCS incidence was well above those reported for other profiles. There have been speculations that the population exposed in those profiles is different than the population exposed in the other profiles. That is, they may be a significantly older population, they may not be in as good of physical condition, and there may be some supertentorial aspects that can contribute to the development of symptoms.

DR. FRANCIS: I have a question about the policy of treating decompression sickness. Is it the AF policy that if they recover within 2 hours on 100% O_2 at ground level that you do not treat them in a hyperbaric chamber, even if they have Type II symptoms?

MAJOR WEIEN: No, if it is Type II symptoms, you treat them in any event. It is only Type I symptoms that you treat on ground level oxygen.

DR. FRANCIS: What is a Treatment Table 8? I am not familiar with that.

MAJOR WEIEN: A Table 8 is an experimental table used at Brooks AFB. It is a much shorter table and it is only a 30 ft exposure. There have been 11 patients treated on it and only 1 treatment failure that required a Table 5 or Table 6.

DR. PILMANIS: I do not believe Table 8 is being used currently.

LT CMDR CLARK: In our experience, we are using less and less Table 5s for treating Type I DCS because we have found that invariably at the 10-minutes decision point if symptoms are not resolved, Table 8 seems even more of an extreme adventure. In some of our female DCS cases, we have been concerned about perimenstrual period association. Have you been able to establish whether that might be a factor?

MAJOR WEIEN: Dr. Rudge recently published an excellent study that showed that the incidence of DCS is highest immediately after the menstrual period, and is linear down to the next menstrual period. It remains above the male incidence throughout the course of the cycle.

DR. PILMANIS: The mean onset of DCS of 5 hours after the flight seems a bit long.

MAJOR WEIEN: It is a long tailed right distribution. That is why the median was posted up there as a more descriptive number.

DR. PILMANIS: But still, that was 2 hours post flight.

LT CMDR CLARK: Onset of symptoms and presentation for evaluation are two different things. Very often if you query them closely they will have had symptoms soon after their flight, but they do not come in for 12 to 24 hours later for the previously stated reasons of their flight status and career.

DR. VANN: Do you have any feeling for that correlation, whether some of your later occurrences were just late presentations?

MAJOR WEIEN: I looked very hard through the charts for exactly that point, symptom occurrence versus symptom presentation. I agree with Dr. Clark that many people delay coming in for that very reason. An example was an inside observer that simply hoped it would go away, and it did not, so he came in about 14 hours after symptom onset. He actually had an onset at altitude but just thought it would go away.

USAF AIRCRAFT OPERATIONS: Decompression Sickness Mishaps

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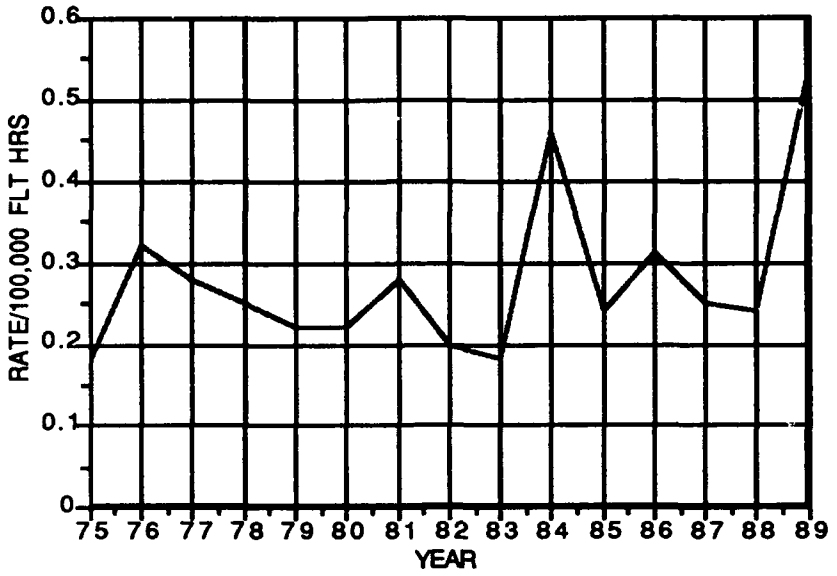
INTRODUCTION

This report presents the U.S. Air Force (USAF) Class C physiological mishap rate for decompression sickness (DCS) from 1975 through FY89. Data include cabin altitude and mishap occurrence per aircraft type. Mishaps resulted from either accidental or intentional exposure of aircrew members/jumpers to reduced pressure in the operational environment. Data were obtained from Safety Reports and the AF Form 711GC submitted by Wing Safety Offices to HQ AFISC/SEL, Norton AFB CA. Validity was (is) contingent upon thorough and complete reporting from field units and safety offices.

DISCUSSION

Since 1975, flying/jumping personnel have reported 135 DCS mishaps as a result of exposure to altitude. Severity of symptoms, as one would expect, ranged from mild to life threatening. Mild, or bends only, cases were referred to as Type I DCS, whereas severe cases were referred to as Type II DCS. Mild cases were treated by either monitoring the affected subject for several hours to days, or by monitoring the subject and administering 100% oxygen via an aviator's oxygen mask. Personnel diagnosed as having Type II DCS received recompression therapy in a hyperbaric chamber.

The DCS mishap rate, which includes both Type I and Type II DCS, from 1975 through 1988, was between 0.2 to 0.3 per 100,000 flying hours (Figure 1). Notably, in 1989 the DCS incidence rate more than doubled. This may be due, in part, to a 1988 change in a flying regulation regarding the treatment and wavering process for aircrew members having diagnosed Type I DCS. Aircrew members diagnosed and treated for the bends (Type I DCS) were now not "threatened" with permanent grounding and career termination. Furthermore, petitioning for a waiver from the Office of the Surgeon General was no longer required. Consequently, local Flight Surgeon Offices were able to approve or deny, on a case-by-case basis, an aircrew member waiver for such DCS mishaps. Type II DCS waiver requirements remained unchanged. Hypothetically, an aircrew member could have a marginally severe Type I DCS hit (in reality a Type II), receive recompression therapy, recover, be wavered by the local Flight Surgeon's Office, and continue flying for the Air Force.



135 MISHAPS (18 in FY89)

Figure 1. Decompression sickness mishap rate.

Also, word of the March 1987 DCS fatality may have spread throughout the flying community. Aircrew members previously downgrading the seriousness of bubbles in body tissues now realized their deadly potential. Type I DCS may be a safe, and perhaps prudent, confession on the part of the knowledgeable aviator.

Analysis of the Class C mishap cabin altitude showed that 15% occurred from 700 to 18,000 feet, 63% occurred between 18,000 and 25,000 feet, and 22% occurred above 25,000 (Figure 2). Except for the T-37 trainer and the parajumpers, cabin pressure decreased (increased cabin altitude) following a mechanical system failure. It is, therefore, unsurprising that most of the reported DCS incidents occurred between 18,000 and 25,000 feet - common operational altitude limits generally considered safe to maintain when an aircraft pressurization system fails. The T-37 trainer is an unpressurized aircraft, and many training sorties are flown within these altitude limits. Likewise parajumpers routinely exit aircraft at these altitudes for certain types of missions.

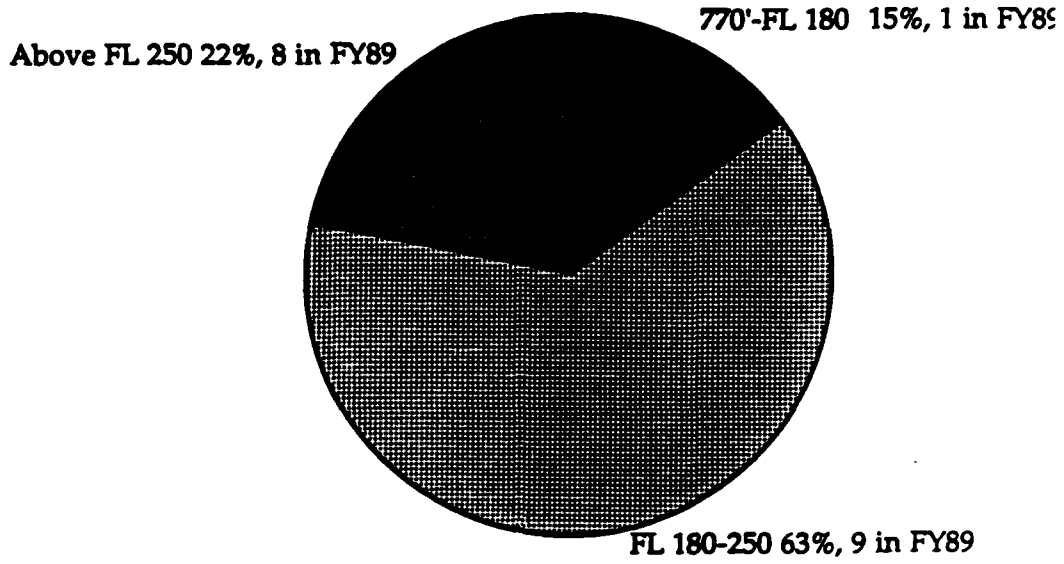


Figure 2. Mishaps by cabin altitude 1975-FY89.

The majority of personnel diagnosed as having experienced DCS, during or after a mission, fly in either trainers or cargo type aircraft (Figure 3). This is not surprising because one type of trainer aircraft is unpressurized, and cargo aircraft are used to transport jump personnel to their jumping location. Depending upon the mission, jumpers are sometimes exposed to extreme physiological limits. Aircrews in fighter aircraft generally fly mission sorties at lower altitudes, therefore one would not expect a large number of DCS mishaps in this group. It is noteworthy that four DCS mishaps were reported in this group for FY89. These incidents occurred in the F-16 (two) and A-10 (two). Aircrew members in the F-15, an aircraft notorious for losing cabin pressurization, reported no DCS incidents. Another notable fact is that only 2% of all DCS mishaps have occurred in aircrew members of high flying aircraft (TR-1). Remarkably, only 2 incidents were reported for FY89.

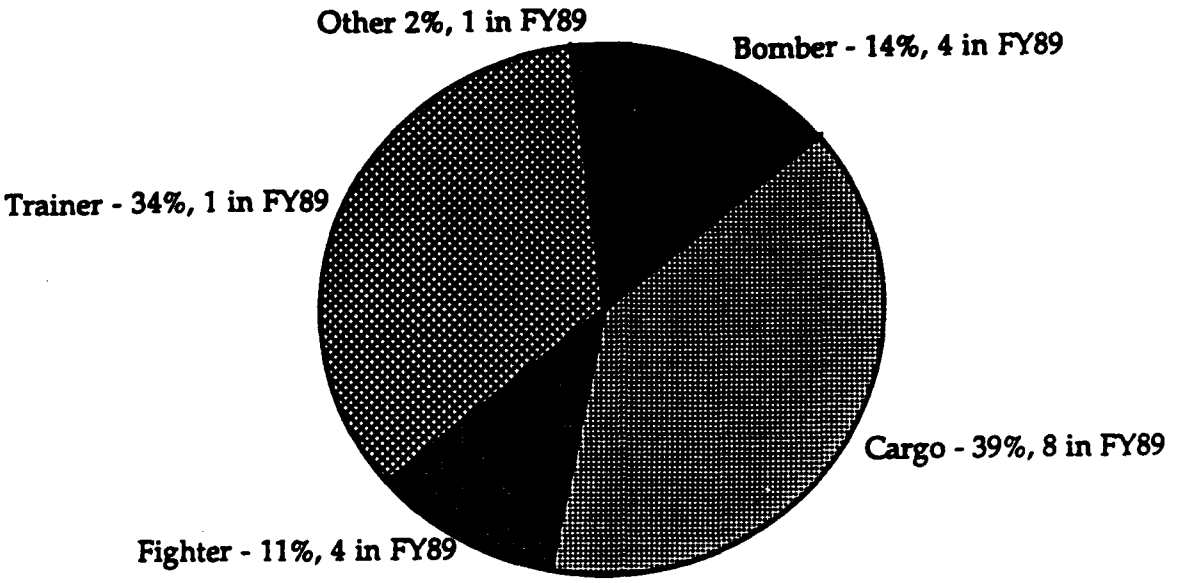


Figure 3. Percentage of DCS mishaps per aircraft type.

CONCLUSION

The number of DCS mishaps experienced by USAF aircrew members appears to be on the increase. It is unlikely that DCS mishaps have increased in the operational Air Force. It is likely that aircrew are more candidly reporting pain experienced during or after flights. The end result, based on the upward swing in reported DCS mishaps for FY89, may reflect a truer picture of operational "bubbling" with laboratory data for DCS incidents.

COLONEL SHEFFIELD: Do you have a feel for how many accidental decompressions (loss of aircraft pressurization) occurred prior to FY 89?

MAJOR KEMPER: No, I do not. I would comment that one of the worst culprits for accidental decompressing is the F-15. Yet, there is a gaping hole in the information that I received on the F-15.

COLONEL STORK: I can give a little insight on why it may be hard to answer your question. The requirements for reporting decompression changed a year or two prior to the '88-89 time frame. The reporting of accidental decompressions just went off the top of the chart for several years and overlapped the period that we are concerned about. That reporting requirement has been fine tuned by the safety center and the decompression rate has come back down to what we have seen previously. But, because of that overlap, it would be hard to dissect out the decompression history with the change in reporting years.

COMMANDER BASON: In the Navy, accidental decompressions are a common occurrence.

COLONEL SHEFFIELD: Most USAF aircraft are pressurized. The notable exceptions are the T-37 and the high flying reconnaissance aircraft. So, therefore, a loss of pressurization or exposure in the unpressurized aircraft would cause the decompression sickness mishaps. It would be interesting to know, as part of the denominator, not only how many total flying hours there were, but how much total decompression time these people put in.

MAJOR KEMPER: There are a lot of accidental decompressions in the F-15. Very few cases of DCS have been reported out of all those accidental decompressions.

LTC PARMET: There may be another reason why some cases are not reported to Norton. For flying class 3, the hospital commander can be the waiver authority for some items. When we get loadmasters and pararescue men who are flying in unpressurized aircraft and get bent, they may be waivered by the local hospital commander. That is not correct, but they can easily misinterpret the regulation. We saw a whole planeload in a C-141 have a decompression, and there were several injuries including CNS hits. I do not think any of those were reported because they were in the reserves. That was about 15 people. That would blow your chart right off the scale. Just one incident.

DR. HAMILTON: Regarding the accidental loss of cabin pressure in the F-15, when you lose cabin pressure you are supposed to go down below a certain altitude.

MAJOR KEMPER: Not in all cases. It is not an automatic descent. You pull out the book and you go by the book on the situation.

DR. HAMILTON: So there are some people who lose cabin pressure and stay at low pressure for considerable time?

MAJOR KEMPER: Yes, sir.

DR. HAMILTON: Okay.

DR. BAGIAN: But they are following the book.

MAJOR KEMPER: There are some descriptions from Norton that describe staying at altitude after decompression and following the book.

COMMANDER BASON: What types of mishaps are these?

MAJOR KEMPER: Class C.

COMMANDER BASON: There was no loss of aircraft or loss of life in any of these instances, correct?

MAJOR KEMPER: No, there was not. These are Class C physiological mishaps.

COMMANDER BASON: We do not call them class Cs in the Navy.

ALTITUDE DECOMPRESSION SICKNESS: THE U.S. ARMY EXPERIENCE

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Altitude decompression sickness is not as great an operational problem in the Army Aviation environment, due to the nature of Army operations. Nonetheless, it does occur in the Army and has been the source of some problems.

Altitude Exposures

The Army has aircraft which routinely fly at altitudes above 18,000 feet. These include the C-12, and the OV-1 Mohawk. The Mohawk is an unpressurized reconnaissance aircraft with an operational ceiling of 25,000 feet. The C-12 is a military version of the Beech King Air, and is pressurized.

Certain other aircraft operate at high altitude, but not on a routine basis. The UH-60A Blackhawk, for example, has reportedly been based in the Andes at 18,000 feet, and operated from there up to 27,000 feet. Other such unusual operations are rumored to have occurred in the Army, but data are difficult to confirm because many of these are in the realm of special operations.

The bulk of U.S. Army exposures to significant altitude have occurred in the altitude chamber based at the U.S. Army School of Aviation Medicine (USASAM), at Fort Rucker, AL. This is a standard altitude chamber of the type used by the U.S. Air Force (USAF); it seats 16 students and 2 observers in the main lock.

Altitude training profiles in use include all the standard flights used in the USAF. As Fort Rucker is the undergraduate pilot training (UPT) base for USAF helicopter flight training, consequently, Type II (43,000 ft) exposures occur. These Type II flights are relatively infrequent, however, as the bulk of the flights are the Type IV flights (25,000 feet) used for Army hypobaric exposure training. These flights are used for training in both the Initial Entry Rotary Wing Aviator Course, and as part of aeromedical refresher training in aircraft transition courses given to rated aviators.

Army Reporting Systems

Unlike the USAF, the Army has no organized system for reporting and investigating aviation oriented physiological incidents, such as altitude decompression sickness.

The closest equivalent is an **occupational injury reporting** system, which requires that certain types of on-the-job injuries be reported by health care facilities through US Army Health Services Command, and then to the US Army Safety Center. According to the Safety Center Surgeon, this would be the appropriate route for reporting altitude decompression sickness, but no cases have ever been recorded in this system.

Since there is no comprehensive reporting system, the only known cases of altitude DCS in the Army have originated in the altitude chamber at USASAM.

Previous Published Review

In 1986, Piwinski, et al., published the results of a retrospective review of altitude chamber operations at USASAM (3). Flights from January 1980 to March 1985 were reviewed and information was extracted and analyzed.

For flights that occurred during this 63-month period, only 25,000, 35,000, and 45,000 ft standard profile flights were analyzed. Other flights, such as high altitude, low opening (HALO) profiles and medical evaluation flights, were excluded.

The overall incidence rate was 20 cases out of 14,545 exposures, equal to a rate of 138/100,000. The data was further stratified by duty classification, and a statistically significant difference was found between inside observers (IO) and students (IO rate 616/100,000; Student rate 64/100,000). The magnitude of the difference between the two groups grew with increasing altitude. The rate ratio between IOs and students in 25,000 ft exposures was 4.64, while in the combined 35,000 and 43,000 ft exposures, it was 43.7. As the student rate remained the same, this large difference was exclusively due to increase in IO incidence.

The authors were unable to analyze gender-specific incidence, due to difficulties with the records.

When they looked at the altitude exposures of the cases, they found a significant increase in incidence between the 25,000-ft exposure, and the combined 35,000/43,000-ft group. There was no difference between the 35,000-ft and the 43,000-ft groups in incidence rates. Rapid decompression exposures were not found to be correlated with DCS, but the number of exposures in this particular chamber is too small to be statistically significant.

The authors' major conclusion was that the IOs have a greater incidence of altitude DCS. They attribute this to differences in the populations. First, IOs need only maintain the more lenient Class 3 flight physicals, whereas a large percentage of the students have recently passed a Class 1 physical. Most of the rest of the students maintain Class 2 standards. Second, the ages of the two groups are different. IOs have a 31-35 mean, while the students have a 21-25 mean. Third, the IOs are subject to repeated exposures (average 41 per year), while the students get just one.

Current Incidence Data

Records have been maintained at USASAM since 1978 on the numbers of flights, profiles, and number of person-exposures. Also included in these records are the number and type of chamber reactions, including decompression sickness. Beginning in 1981, the cases were divided into students versus chamber technicians, but the numbers of exposures were not stratified, thus making duty-specific rate calculations impossible. In 1984 the exposures were recorded by duty position, and rates have been calculated for these years. Since USASAM has not calculated their 1990 exposures, the analysis of the USASAM chamber data ends with 1989.

For the 6-year period 1984-1989, there were a total of 21,498 altitude chamber exposures, which generated 42 cases of DCS. The incidence rate for this period was 195.37 per 100,000 exposures. The rate for chamber technicians exceeded that for students by a rate ratio of 3.17. This is notably different from the rate ratio of 10 noted by Piwinski for the period January 1980 through March 1985. (The previous raw data on which Piwinski's calculations were based is not available to this researcher.)

USASAM does not record gender of students, and records names only by last name and first initial. Thus, not even an estimate of the gender-specific incidence rates can be determined.

Unusual Occurrence I: "A Blitz of Bends"

In 1985, a single altitude chamber flight resulted in 4 cases of DCS (three were Type 2, the other was Type 1). This flight was unusual in several ways, and resulted in multiple lessons learned.

The flight was a HALO profile, given to a group of nine elite Army airborne infantrymen. All nine were in superb physical condition, a byproduct of their rigorous military duties. None had any known predisposing factors for the development of DCS.

The altitude exposure was for approximately 45 minutes at a maximum altitude of 32,500 ft. It included a hypoxia demonstration, a freefall simulation, and a rapid decompression. No symptoms were reported at altitude. Approximately 2.5 hours after the flight, the students were flown by helicopter at 2,300 ft MSL, for about 1 hour.

The next morning four of the students reported for symptoms ranging from joint pain, to numbness in extremities. The patients were evacuated to Panama City, FL, and treated on USN Table 6, and all four resolved without sequelae.

The altitude chamber was thoroughly inspected; but no defects in equipment were found. The gas supplies were tested and found to be 100% oxygen.

Further investigation of the profile used discovered that it was a nonstandard profile of uncertain origin. As a result of this incident, the profile was abandoned in favor of the standard USAF HALO profile.

Flying after an altitude chamber exposure was believed to be a contributing factor to the incident. Therefore, procedures were changed to prohibit students from flying after an altitude chamber exposure, in accordance with AR 40-8 restrictions.

This experience was also used as a partial justification for the installation of the hyperbaric chamber at USASAM.

Unusual Occurrence II: An Epidemic of Altitude DCS

On 20 August 1990, the altitude chamber at USASAM was returned to operational service after an overhaul. From 21 August to 12 September, seven cases of altitude decompression sickness were recorded, including three Type 2 cases (two neuro and one chokes).

This was clearly an epidemic, with an incidence for this period of 1,351 per 100,000 (7 patients out of 518 exposures). There seemed to be little to correlate the cases, aside from all sharing an altitude chamber exposure. There were 2 IO s, and 5 students. The students were further classified as 1 commissioned officer flight student, 1 warrant officer candidate flight student, 1 USAF flight student, and 2 enlisted aero-scout observer students (interestingly, on the same flight). There was no unusual clustering of seat assignments in the cases (seats 5, 6, 7, 11, and ISO stations had cases.)

With one exception, the profiles followed were standard USAF altitude chamber profiles. The single exception was the last case, who was a USASAM chamber technician performing gas sampling at 25,000 feet as part of the investigation of this incident.

Investigation by USASAM personnel revealed no procedural errors, no significant equipment malfunctions, and no defect in gas quality or delivery. Consequently, a team from USAFSAM was requested to assist in the investigation.

Summary

Altitude decompression sickness is a serious disorder, which does affect Army personnel.

Analysis of the data reveals two major findings:

1) That the incidence in the USASAM chamber is higher than that experienced elsewhere. This increased rate has persisted over time.

2) That chamber technicians are at greater risk for developing altitude DCS, on a per exposure basis. This may be due to a number of factors, including age, frequency of exposure, and more lenient medical screening for chamber duties.

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COLONEL SHERMAN: Did you have the same instructors teaching these people?

MAJOR WEIEN: Yes. The chamber was only down for about 3 weeks. These guys that work in the altitude chamber all rotate around, giving the lectures and performing the various positions in the altitude chamber. The same group of people were doing the instructions. In summary, the Army experience with altitude DCS is that the incidence at USASAM appears to be higher than that reported by other people and that the technicians are at a higher risk than students.

MR GILBERT: Is there any career problem if the technicians report?

MAJOR WEIEN: Yes. If they report Type II, they do not get to be technicians anymore, and one of them did in fact get grounded.

MR GILBERT: You say you have basically 12 individuals who rotate through the chambers?

MAJOR WEIEN: Yes, about 12. There are a couple of other people that come in once a month to get their exposure pay, but the bulk of the exposures are in this group of enlisted guys that rotate through it pretty frequently.

DR. FRANCIS: What was the spectrum of symptoms of this epidemic?

MAJOR WEIEN: Four of the cases were limb bends. Two cases had headaches as their neurologic symptom; i.e., abrupt onset of severe headaches at altitude. The third one came in about an hour after his flight complaining of substernal chest pains, shortness of breath and diaphoresis. His blood pressure was actually dropping before we got into the chamber.

DR. FRANCIS: You said the chamber had been repainted. I was wondering what paints you had used and whether they had been allowed to dry because solvents can certainly cause headache.

MAJOR WEIEN: The students were on a mask the whole time.

DR. FRANCIS: They did not do a hypoxia run?

MAJOR WEIEN: They did do a hypoxia run, but one of the two headaches came on prior to the hypoxia run. He had not yet dropped his mask.

COLONEL SHAFFSTALL: That was pretty thoroughly investigated. Would the possibility that your inside observers are not full career aerospace physiologists but actually med techs who will go back to their regular jobs have anything to do with it?

MAJOR WEIEN: That factor may have something to do with it. These people do not choose to be chamber techs. They are what we call 91A and 91B, just medics that happen to be assigned to the School of Aviation Medicine. Theoretically, they could refuse that assignment and be sent to the hospital and pull shifts up on the ward, but most of them do not elect to do that. It is a persistent problem that we do not have a career field. Our physiologic training officers, for instance, are all Army helicopter pilots who make one tour, come down here for their training, and go back and become helicopter pilots again after that. We do not have a real live physiologist in the crowd.

COLONEL SHEFFIELD: Do you know what the interval ground level time was between the exposures?

MAJOR WEIEN: By USASAM regulation it is at least 24 hours, but they try to keep it more on the order of once a week.

LT CMDR CLARK: The Navy has had two of these blitzes, one in 1988 and one this year. Over a 2-week period, we had six patients the first time and five this most recent incident. We shut the chamber down the first time. We did gas analysis. We did a major epidemiologic investigation. Despite the fact that, if you assume that DCS is a randomly occurring event, to see that many in that short of time it should be a statistically significant event, we could find no common factors whatsoever. However, as a result of the second event, we came out with a new policy. Because a fair percentage of the patients were student aircrew under a lot of physical training, we restricted any major physical exertional activity for 24 hours before or after the chamber flight. We stuck by our rule of no drinking for 12 hours, which is our standard in aircraft operations, and we recommended that they get at least 8 hours of sleep, and that they be well hydrated prior to this. Traditionally, the school that is training air crewmen will have physical training right up till the chamber flight and work them to death as soon as they get out of it. Since then, we have still had cases, but because we have standardized the conditions, at least we do not have the confusion between musculoskeletal injury, dehydration, etc., before or after a chamber flight.

MAJOR WEIEN: I looked for something like that in this epidemic, and I could not find anything. It was a widely homogenous group.

LT CMDR CLARK: Two of our really serious cases occurred the day after a chamber flight. They were doing fairly aggressive physical exertion and came down acutely with severe DCS symptoms.

LTC DIXON: How about dehydration of these individuals, and specifically your technicians, is there any special effort that is taken to encourage these individuals to stay very well hydrated?

MAJOR WEIEN: To the best of my knowledge, there is no special effort. We do not watch them drink a liter right before they go in. But all the technicians do know that hydration is a factor. I think more importantly the question may apply to the students. This exposure was in the summertime. At least the aeroscout observer students and the warrant officers are involved in heavy physical training. Although we checked with their units, none of them had done physical training that morning because they were going to have an altitude chamber flight, and none of them did physical training afterwards. So I suspect that hydration was less of a problem in this particular group than what you might expect during August at Fort Rucker.

INFLIGHT DECOMPRESSION SICKNESS: USN EXPERIENCE 1969 - 1989

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In flight the Naval Aviator is subjected to many forms of stress, among them is decompression sickness. During the 20-year period from 1969 to 1989, decompression sickness in one of its forms was reported in twelve USN aircraft (Table 1) and involved fifteen aircrew. Of these twelve cases, six were due to malfunctions of the pressurization system, two were caused by intentional cockpit depressurization because of smoke/fumes in the cockpit, one as a result of rapid decompression, one as a result of lack of pressurization capability, and one as a result of scuba diving.

The cabin altitudes at which the decompressions occurred (Table 2) ranged from 1,000 ft to 38,000 ft with all but one case clustered between 29,000 ft and 38,000 ft. The one incidence at 1,000 ft occurred in a helicopter crewman who went scuba diving the day before the flight. On that day, the crewman made two dives. The first dive was to 90 ft for 15 minutes with a total of 40 minutes below 60 ft. The second dive was to 40 ft for 15 minutes. The crewman violated a Navy policy which restricts routine flying for 24 hours after scuba diving. The crew positions of the aircrew involved are shown in Table 3.

Decompression sickness can cause a variety of clinical symptoms ranging from minor to life-threatening. Table 4 shows the symptoms reported by the fifteen crewmen. The number of symptoms exceeds the number of crewmen involved because some crewmen had more than one manifestation of decompression sickness. The most commonly reported symptom by far was joint/limb pain which is consistent with other studies. Joint/limb pain accounted for 76% of all symptoms attributed to DCS. The shoulders and knees were the two most frequent sites of joint pain, which is consistent with other reported studies. A variety of neurological dysfunctions from paresthesia to chest pain were also reported. Chokes, which is considered a serious form of DCS, was reported once. None of the reported symptoms were incapacitating and none of the aircraft involved in these reported incidences crashed or incurred even minor damage. In all cases the afflicted crewmembers continued to perform their assigned duties without any degradation or compromise.

Following decompression sickness, most of the missions were immediately aborted, as they should have been, with a landing made as soon as possible. In a few cases, the afflicted crewman tried to continue the mission thinking the pain would go away, but it instead got worse. Of the fifteen aircrew afflicted, thirteen (87%) had

complete remission of symptoms with descent, or at least by the time they touched down. Two crewmen required recompression therapy for resolution of symptoms.

Once decompression sickness has occurred, the aviator has a required checklist. This checklist includes breathing 100 percent oxygen if not already on 100% oxygen, immediate descent and landing at the closest base, preferably one with a hyperbaric chamber, and immediate notification of the flight surgeon.

Regarding the aeromedical disposition of crewmen who have developed decompression sickness, the following is the Navy's policy:

(a) Type 1 DCS. The individual should be grounded for one week.

(b) Type 1 (recurrent). The individual should be found NPQ with information forwarded to the Naval Aerospace Medical Institute (NAMI), Pensacola, Florida, for review by the Hyperbaric Medicine Committee for waiver consideration.

(c) Type II DCS. The individual should be found NPQ with information forwarded to NAMI for review by the Hyperbaric Medicine Committee for waiver consideration. If a waiver is granted, the individual should be grounded for a minimum of 30 days following the incident.

To date, no designated Naval aviator, Naval flight officer, or crewman has been permanently disqualified from further flight duties.

Although loss of cabin pressurization is not a rare event in USN aircraft, only twelve incidents of DCS involving fifteen crewmen were reported to the Naval Safety Center over a twenty-year period from 1969 to 1989. This would tend to suggest that either inflight DCS is not a common occurrence in Naval aviation or that DCS is not being fully reported to the Naval Safety Center, as required by Navy instruction, to accurately ascertain the significance of the problem. It is this author's opinion, as a result of informal discussions with aircrew, that DCS is occurring at a higher rate than is being reported. However, even if fully reported, this author feels that DCS would not be a significant problem to Naval aviation. When DCS did occur, joint pain by far was the most commonly reported symptom usually involving the knees and shoulders. In most cases the pain was described by the affected crewman as mild to moderate and did not interfere with crew duties. All afflicted aircrew were returned to flight duties following treatment and recovery.

Table 1. Decompression Sickness in USN Aircraft 1969 - 1989

Aircraft	Number
Attack	8
A-6 – 5	
EA-6 – 2	
A-3 – 1	
ASW	3
S-3 – 2	
E-2 – 1	
Helicopter	1
SH-3 – 1	

Table 2. Decompression Sickness and Cabin Altitude 1969 - 1989

Cabin Altitude in Feet	Number
1,000	1
29,000	1
31,000	2
32,500	2
33,000	5
38,000	1

Table 3. Decompression Sickness and Crew Position 1969 - 1989

Crew Position	Number
Pilot	3
Co-pilot	3
Naval Flight Officer	7
Aircrew	2

Table 4. Clinical Manifestations of Decompression Sickness 1969 - 1989

Clinical Manifestations		Number
Joint/Limb Pain		19
Shoulder	8	
Knee	8	
Wrist	1	
Elbow	1	
Arm	1	
Neurological		5
Paresthesias of arm	1	
Numbness of arm	1	
Loss of hand strength	1	
Headache	1	
Chest pain	1	
Pulmonary		1
Chokes	1	

ALTITUDE CHAMBER DCS: USN EXPERIENCE 1981 - 1988*

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In 1941, the U.S. Navy launched its program of altitude indoctrination of aircrew using low pressure chambers. It was not until 1953, however, when the Bureau of Medicine and Surgery established an Aviation Physiology Training Report, that information relevant to the incidence of decompression sickness became available. The first publication specifically reporting the incidence of decompression sickness (DCS) in Navy low pressure chambers did not appear until 1973.

Since 1973, several reports have been presented on the incidence of DCS in Navy low pressure chambers. In 1973, Furry (5) reported a 0.1% incidence of DCS among altitude indoctrination trainees for the 10-year period 1959 through 1968. Among inside observers, the reported incidence of DCS was 0.06%. Furry's data reflected maximum altitude exposures of 30,000, 35,000, and 43,000 ft. During this period, the Navy's standard operating procedure for inside observers limited them to one exposure to 30,000 ft in a 24-hr period, or one exposure to 43,000 ft in a 48-hr period.

Bason et al. (2) next reported the incidence of DCS in Navy low pressure chambers covering a 4-year period from 1 January 1972 to 31 December 1975. During this period, the primary hypoxia demonstration flight was lowered from 30,000 ft to 25,000 ft, and the pressure breathing flight was conducted at 40,000 ft. The Navy also started to provide low pressure chamber training for the FAA. A small number of general aviation pilots were given 29,000-ft indoctrination flights. During this period, there were 79 cases of DCS in 88,520 altitude chamber exposures, an incidence of 0.089%. Among trainees, there were 22 cases in 73,561 exposures, an incidence of 0.029%. Among inside observers, there were 57 cases in 14,959 exposures, a rate of 0.38%. This 13-fold greater incidence among inside observers was statistically significant and in a reverse direction from the results reported by Furry (5). Bason's report (2) marked the beginning of the end for 40,000-ft exposures in Navy altitude chambers because it clearly showed that the risk of DCS associated with 40,000-ft altitude was at least 3.5 times greater than that associated with 25,000-ft flights. Of the 79 cases of DCS, 18 cases (23%) occurred during the 25,000-ft chamber profile and 61 cases (77%) during the 40,000 ft chamber profile.

*The opinions and interpretations expressed are those of the author and should not be construed to be the official views, policies, endorsements, or decisions of the Department of the Navy.

In 1988, Furry (unpublished report) reported the incidence of DCS from 1 January 1976 to 31 December 1977. His data reflected no change in the incidence of DCS among trainees and inside observers from the 1972-1975 report. In 1982, Bason (unpublished report) reported the incidence of DCS for the 3-year period from 1 October 1978 to 30 September 1981. During this period, there were 47,380 student exposures and 10,020 inside observer exposures. There were 39 cases of DCS reported among the student population for an incidence of 0.08%. In contrast, there were 48 cases of DCS reported among inside observers, a rate of 0.48%. Of the 87 total reported cases of DCS, only three occurred following 40,000-ft exposures. Exposure to training altitudes of 40,000 ft were officially terminated in May 1979; however, a few training units continued to conduct 40,000 ft training until December 1979. As previously reported, there was a higher incidence of DCS among inside observers than trainees. What is perhaps more disconcerting was the fact that the incidence of DCS increased 66% and 55% for students and inside observers respectively, during the period when the 40,000-ft chamber exposures were terminated. The reason for this increased incidence could not be explained.

The present report provides an update on DCS in Navy low pressure chambers for the 7-year period 1 October 1981 to 30 September 1988.

METHODS AND PROCEDURES

Information for this report was obtained from the Aerospace Physiology Training Quarterly Report (NAVMED 6410/3), the Chamber Reaction Report (NAVMED 6410/4), and appropriate individual health record entries. Each Chamber Reaction Report was critically reviewed to ascertain the type of DCS manifested.

RESULTS

During this 6-year period, there were 140 cases of DCS in 136,696 exposures, an incidence of 0.10% (Table 1). Among students, there were 78 cases in 111,674 exposures, an incidence of 0.07%. Among inside observers, there were 62 cases in 25,022 exposures, an incidence of 0.25%.

Table 2 summarizes the types of reported DCS. Type 1 includes limb/joint pain and cutaneous manifestations of skin mottling (cutis marmorata), and pruritus (tingling, creeps, formication without any visible lesions). Type 2 includes chokes and the broad spectrum of neurological dysfunctions. Cutaneous manifestations of DCS were reported three times. One of the reported cases included skin mottling (cutis marmorata) in an inside observer. Limb/joint pain was reported 69 times. Decompression sickness did not present most commonly as limb/joint pain as is frequently reported. CNS dysfunctions were reported almost as frequently as limb/joint pain. Chokes was reported once.

Table 1. 1 October 1981 - 30 September 1988
Total Incidence of Decompression Sickness

	Total Man Exposures	Number of Cases of Decompression Sickness	Incidence (Percent)
Student	111,674	78	.07
Inside Observer	25,022	62	.25
Total	136,696	140	.10

Table 2. Types of Decompression Sickness

	Type I DCS		Type II DCS		Total
	Skin Bends	Limb Joint Pain	Chokes	CNS	
Inside Observer	1	31	0	30	62
Student	2	38	1	37	78
Total	3	69	1	67	140

A variety of symptoms occur in DCS. In this study the most common symptom was found to be limb/joint pain (Table 3). The next most frequent symptoms were extremity manifestations of paresthesia, numbness, and muscular weakness. Dizziness was fairly common, followed by headache, nausea/vomiting and visual disturbances. In addition to the one reported case of skin mottling, another individual developed skin mottling in the hands and forearms at depth while being treated on Treatment Table 6 for Type II CNS decompression sickness. One individual presented with unconsciousness which represents one of the more serious forms of DCS.

The sites of joint/limb pain are shown in Table 4. The elbows presented as the most common site, with the shoulders next, followed by the knees, arms, wrists, hips, legs and ankles, fingers, feet and hands, and heels. The lateral distribution favored a larger involvement of the right side. Likewise, the upper extremities were much more involved than the lower extremities.

The initial onset of DCS can occur either at altitude or at ground level following some asymptomatic period. In this study, 46% (65/140) of the cases of DCS occurred at altitude and 54% (75/140) occurred at ground level following an asymptomatic period (Table 5). Of the 65 cases occurring at altitude, 37% (24/65) resolved upon return to ground level. Three of these individuals still received hyperbaric treatment, however. Of the 24 individuals who were asymptomatic upon return to ground level, four had recurrence of symptoms. The latent period of recurrence ranged from one-half hour to six hours. All four of these individuals received hyperbaric therapy with complete resolution of symptoms. Of the 65 cases occurring at altitude, 63% (41/65) had symptoms which persisted upon return to ground level. All 41 individuals received hyperbaric therapy with complete resolution of initial symptoms. Three of these individuals, however, had recurrence of symptoms after resolution by hyperbaric treatment. In one individual, the latent period of recurrence was 15.5 hours and in the other two individuals, 24 hours. All three were squeezed a second time.

Seventy-five individuals had initial onset of DCS at ground level (Table 6). In eight of these individuals, the symptoms resolved without any treatment other than 100% oxygen at ground level. Three of these individuals had recurrence of symptoms and received recompression therapy. In 83% of the cases (62/75), complete resolution was achieved following initial hyperbaric treatment. Three of these individuals, however, had recurrence of symptoms following resolution and had to be squeezed a second time. In 7% of the cases (5/75), multiple treatment tables were required to achieve initial resolution.

Table 7 shows the times of initial onset of symptoms of DCS following return to ground level. The times ranged from less than one-half hour to more than twenty hours. In 61% of the cases, the initial onset of symptoms occurred within one hour.

Table 3. Clinical Manifestations of Decompression Sickness

Clinical Manifestations	Number of Patients	Percent
a. Joint and Limb Pain	99	70.7
b. Extremity Paresthesia	46	32.9
c. Numbness	33	23.5
d. Muscular Weakness	24	17.1
e. Dizziness	22	15.7
f. Headache	12	8.6
g. Nausea/Vomiting	11	7.8
h. Visual Disturbances	10	7.9
i. Fatigue/Malaise/Lethargy	8	5.7
j. Apprehension	7	5.0
k. Mental Confusion	7	5.0
l. Disorientation	7	5.0
m. Hyperventilation	5	3.6
n. Paralysis	4	2.9
o. Pruritus	3	2.1
p. Muscle Spasms	3	2.1
q. Skin Mottling	2	1.4
r. Ataxia	2	1.4
s. Chokes	1	0.7
t. Unconsciousness	1	0.7
u. Slurred Speech	1	0.7
v. Vertical Nystagmus	1	0.7
w. Abdominal Pain	1	0.7
x. Hot and Cold Flashes	1	0.7
y. Difficulty Forming Words	1	0.7

Table 4. Site of DCS

Site	Left Side	Right Side	Both Sides	Total
Elbows	14	16	4	34
Shoulders	15	12	3	30
Knees	6	20	3	29
Arms	3	6	1	10
Wrists	4	5	0	9
Hips	1	4	0	5
Legs	1	3	0	4
Ankles	0	4	0	4
Fingers	1	1	0	2
Feet	2	0	0	2
Hands	0	1	0	1
Heels	1	0	0	1

Table 5. Onset of DCS at Altitude

- Initial onset of Decompression Sickness at altitude 65
- Asymptomatic upon return to ground level 24
  - ➤ Hyperbaric Treatment 3
- Recurrence of symptoms at ground level after resolution 4

  Latent period of recurrence

  1/2 hour 2 hours

  1 hour 6 hours
  - ➤ Hyperbaric Treatment 4
- Symptoms persisting on return to ground level 41
  - ➤ Hyperbaric Treatment 41
- Recurrence of symptoms at ground level after recompression 3

  Latent period of recurrence

  15.5 hours

  24.0 hours (2)
  - ➤ Hyperbaric Treatment 3

Table 6. Onset of DCS at Ground Level

- Initial onset of Decompression Sickness at ground level 75

- Relieved at ground level 8
  Recurrence of symptoms after resolution 3
  ➤ Hyperbaric Treatment 3

- Relieved following initial recompression therapy 62
  Recurrence of symptoms after recompression 3
  ➤ Hyperbaric Treatment 3

- Relieved after multiple treatment tables 5

Table 7. Time of Initial Onset of Symptoms at Ground Level

Time in Hours	Frequency
Less than 1/2 hour	34
0.5 - 1.0	12
1.1 - 1.5	2
1.6 - 2.0	2
2.1 - 2.5	1
2.6 - 3.0	5
3.1 - 3.5	2
3.6 - 4.0	1
4.1 - 4.5	2
4.6 - 5.0	1
5.1 - 5.5	1
5.6 - 6.0	1
7.1 - 7.5	2
7.6 - 8.0	1
10.1 - 10.5	1
16.6 - 17.0	1
17.6 - 18.0	1
19.6 - 20.0	2
Greater than 20 hours	3
Total	75

Exercise, injury, obesity, dehydration, alcohol, hypoxia, and cold are the more commonly discussed predisposing factors in the development of DCS in an altitude environment. In this study, fifteen cases (10.7%) had predisposing factors (Table 8). The incidence might have been higher had the Chamber Reaction Reports specifically addressed these factors. The eight overweight individuals were judged so on the basis of the very liberal naval aviation height/weight standards. The excess weight ranged from 11 pounds to 31 pounds. Five of the individuals were trainees and three were inside observers.

In 13 cases (9.3%), there were one or more documented prior episodes of DCS (Table 9). These involved twelve inside observers and one trainee. The 12 inside observers accounted for 13 previous cases of Type I (joint pain only) and three previous cases of Type II (CNS) decompression sickness.

Eight inside observers were subsequently removed from further low pressure chamber exposures, either because of too many episodes of DCS, or due to the severity of a single episode.

The aim of treatment for decompression sickness is to halt the progression of the disease, accomplish quick resolution of the signs and symptoms, and avoid residual effects. In many instances, resolution of decompression sickness can be accomplished by just returning to ground level. In this study, 16% of the cases resolved completely upon returning to ground level. More frequently, however, the only definitive treatment is hyperbaric therapy. In this study, 118 cases (84%) of DCS required hyperbaric therapy (Table 10). Of these, 107 (91%) completely resolved after a single recompression treatment. Multiple treatment tables were employed in 11 (9%) cases. The USN Treatment Table 5 is the accepted mode of treatment for Type I pain only bends. Table 5 was used to treat 33 (28%) patients. USN Treatment Table 6 is the accepted mode of treatment for Type II DCS. This table was used for 60 single treatment cases (51%) and was the initial table in 4 cases of multiple treatments. For those individuals who do not show a rapid improvement during initial recompression, or who show no rebounding effects, Treatment Table 6 can be extended up to two additional oxygen breathing cycles at 60 ft of seawater (FSW) and two at 30 FSW. Extended Treatment Table 6 (Table 6E) was used 12 times in single treatment recompression therapy and four times for multiple therapy. In USN Treatment Table 6A, the patient breathes air at 165 FSW (6 ata) for 30 minutes and then is decompressed according to Table 6. This table was used twice in single treatment cases and twice again as the initial table in multiple treatment cases.

Treatment Table 4 is used for the treatment of serious symptoms of DCS, such as gas emboli, when oxygen cannot be used and when symptoms are not relieved within 30 minutes at 165 feet. This table was used once. The overwhelming majority of patients achieved full recovery after hyperbaric treatment. Only six patients had residual symptoms that involved mild residual joint pain, asymmetrical deep tendon reflexes, "chest fullness," and paresthesia.

Table 8. Predisposing Factors

Old Injury	6
History of Migraine	1
Overweight	8

Table 9. Prior Episodes of DCS

Students	1
Inside Observers	12
Type I	13
Type II	3

Table 10. Types of Treatment Tables

USN Treatment Tables	Number
Single Treatments	**107**
Table 5	33
Table 6	60
Table 6E	12
Table 6A	2
Multiple Treatments	**11**
Initial Treatment Table	
Table 6	4
Table 6E	4
Table 6A	2
Table 4	1

DISCUSSION

Exposure to reduced atmospheric pressure by individuals undergoing high altitude indoctrination or refresher training is not without risk. The development of altitude decompression sickness is a real and potentially life-threatening disorder. Since May 1979, U.S. Navy low pressure chambers have utilized one standard chamber training profile for thirteen training sites. This training profile consists of: (a) thirty minutes of denitrogenation for inside observers; (b) ground level positive pressure breathing/communication demonstration by all trainees; (c) ascent to 25,000 ft at 5,000 ft per minute; (d) student hypoxia participation at 25,000 ft; (e) descent to 18,000 ft at 5,000 feet per minute; and (f) slow the descent from 18,000 ft to ground level at a rate of 2,500 feet per minute. Table 11 summarizes the reported incidence of decompression sickness in Navy low pressure chambers since 1959. The data clearly show, especially from 1 January 1959 to 30 September 1981, a trend toward an increased incidence of decompression sickness. In all the studies, including this one, the major impact appears to be with the inside observers. They experienced an incidence ranging from 3.5 to 13 times higher than the student/trainee population rate. Davis et al. (3) reported a 3-fold increased incidence among U.S. Air Force (USAF) inside observers. More recently, USAF data reported a 1.7 times higher incidence among this group (9).

During the period 1959 to 1961, information concerning the total number of exposures for inside observers was missing from the Bureau of Medicine and Surgery (BuMed) Physiology Training Report. The descriptive statistics expressed exposure in terms of altitude exposure points. Furry (5), in his report, applied a mathematical conversion to altitude exposure points to obtain an equivalent expressed in terms of number of exposures. Since that study period, the number of exposures for inside observers at predetermined altitude, as well as the time in low pressure chambers, is reported. If we exclude the Furry data in Table 11, due to the use of a conversion factor, the incidence of decompression sickness among inside observers in this current study reached an all time low (0.25%). There was almost a two-fold reduction in decompression sickness among inside observers compared to the previous report of 1 October 1978 to 30 September 1981. Despite this reduction, however, the incidence of DCS among Navy inside observers is still almost three times higher than the incidence of 0.064% reported in 1977 by Davis et al. (3) for USAF inside observers.

Table 11. 1 January 1959 - 30 September 1988 Comparative Incidence of Decompression Sickness in Navy Low Pressure Chambers

	Total Exposure	No. Cases of DCS	Incidence (%)
FURRY (1 Jan 59-31 Dec 68)			
Students	252,564	266	0.100
Inside Observers	60,000	35	0.060
Total	312,564	301	0.096
BASON (1 Jan 72-31 Dec 75)			
Students	73,561	22	0.029
Inside Observers	14,959	57	0.380
Total	88,520	79	0.089
FURRY (1 Jan 76-31 Dec 77)			
Students	31,645	10	0.030
Inside Observers	6,470	20	0.310
Total	38,115	30	0.078
BASON (1 Oct 78-30 Sep 81)			
Students	47,380	39	0.080
Inside Observers	10,020	48	0.480
Total	57,400	87	0.150
BASON (1 Oct 81-30 Sep 88)			
Students	111,674	78	0.070
Inside Observers	25,022	62	0.250
Total	136,696	140	0.100

While the reasons for the reduction in decompression sickness among inside observers in the present study are not readily apparent, there are several plausible explanations. First, in July 1984, BuMed required, for the first time, that all inside observers denitrogenate for at least thirty minutes immediately prior to altitude exposure and then remain on 100% oxygen for the entire flight. Second, in July 1984, BuMed reduced the allowable exposure rate to what is current standard operating procedure. Inside observers can make no more than three chamber flights above 18,000 ft in a 7-day period with a minimum of 48 hours between exposures. Prior to July 1984, standard operating procedures mandated that no inside observer could make more than one exposure to 25,000 ft in any 24-hour period. Therefore, in a 7-day period, an inside observer could possibly have made a maximum of four chamber flights. A third explanation is better training, education, and awareness of decompression sickness by naval flight surgeons. Every little twinge, tingle or muscular-skeletal pain associated with altitude exposure was not erroneously being diagnosed as decompression sickness. Last, one can only speculate that perhaps the Navy's health promotion programs over the past five years have had a beneficial effect on those individuals who are frequently exposed in high altitude chambers. These programs, which include physical fitness training, weight reduction, smoking cessation, and zero drug and alcohol tolerance, all produce a healthier individual with perhaps a more efficient cardiorespiratory system for nitrogen elimination.

Fifty-four percent of the individuals (75/140) experienced initial onset of DCS at ground level. In sixty-one percent of these cases, the initial onset occurred within one hour post-flight. Standard operating procedures for Navy low pressure chambers requires all individuals to remain at the training site for at least one hour following a 25,000 ft exposure. The data from this study strongly support continuation of that policy.

Many studies (1,2,7,8) have reported the shoulders and knees as the two most frequent sites of joint pain. In this study, data showed the elbow presenting as the most common site. No explanation is readily apparent for the large number of elbow involvements.

Leaders in the aeromedical community strongly support the idea to educate and train aircrews to recognize and correct hypoxic hypoxia. Harding and Mills (6) stated that "the fall in total barometric atmospheric pressure and the consequent reduction in the partial pressure of oxygen, poses the greatest single threat to anyone who flies." Ernsting (4) declared that " the most important single hazard of flight at high altitude is hypoxia." He continues,"all aircrew should receive initial and refresher training in the effects of hypoxia." There is no doubt that hypoxia training is highly desirable; indeed it is mandatory for all involved in U.S. military aviation. We must ask ourselves, however, " Is the risk of altitude induced decompression sickness with its potential threat to life, worth the training achieved by low pressure chamber exposure to induce hypoxia?" We must also ask ourselves, " Is it really necessary to expose any member of the aviation community to even normal flight ambient operational altitudes, or can we develop a means to safely induce ground level hypoxia which would demonstrate

symptoms similar to those actually experienced at high altitude?" Even though the incidence of decompression sickness among inside observers in this report shows a marked decrease over previous Navy reports, it is much higher than USAF incidence and in this author's opinion, it is unacceptable. In 1977, Davies et al. (3) reported an incidence of 0.064% among USAF inside observers. More recently, USAF data showed an incidence of 0.14% among this group (Howard et al., 1982 unpublished). Just on chamber profiles alone, the USAF should theoretically have a higher incidence of decompression sickness. The primary training profile for the USAF is a 35,000-ft pressure breathing exposure, followed by a free fall to 15,000 ft, followed by a rapid descent to 8,000 ft. At 8,000 ft oxygen masks come off and the chamber is rapidly ascended to 25,000 ft for a hypoxia demonstration. After the hypoxia demonstration there is a descent to ground level. The Air Force also conducts a few 43,000-ft exposures.

The incidence of decompression sickness reported here suggests that present Navy altitude chamber profiles are once again in need of reexamination unless decompression sickness is to be dismissed as either a necessary risk or a mere quality of life judgment. Altitude chamber flights beyond minimum levels acceptable for teaching the effects of hypoxia cannot be defended as long as less dangerous avenues are available. Alternate means of inducing hypoxia, such as using hypoxic gas mixtures at ground level, should be explored.

SUMMARY AND CONCLUSIONS

The incidence of decompression sickness was determined for both students and inside observers for the 7-year period, 1 October 1981 to 30 September 1988.

Pensacola reported the highest number of chamber reactors (45); Brunswick, the highest incidence among inside observers (0.71%) and overall incidence (0.23%); and Cherry Point had the highest incidence among students.

On the average, approximately 15,590 trainees and 3,575 inside observers are exposed each fiscal year.

The overall Navy incidence for combined students and inside observers was 0.10%, for students alone 0.07%, and for inside observers alone, 0.25%.

The incidence of decompression sickness was the highest during the second and fourth quarters, during the coldest and hottest 3-month periods.

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COLONEL SHERMAN: I think it is clear at this conference that we really cannot compare data such as reported cases, because it is all based on whether or not someone wants to report DCS. In the Air Force, I think there is an implied situation that if you report something, then you may have something bad happen to you. If you try to compare what the Navy reports versus how they react to it, and what the Air Force reports and how they react to it, which in turn varies from base to base, I think it is almost impossible to compare data and come up with anything meaningful. I think it is a big problem. We need to address, from the flight surgeon level, how the philosophy should change in order to get our aircrews to be honest with reporting so that we can help them from the long-term health view. This in turn will help them do their job better.

COMMANDER BASON: When I looked at these data, especially those of our inside observers, I concluded it could be higher. I believe our guys are not reporting hits. There are probably two reasons. Of course, the incentive pay is one. But, even more important, with our enlisted techs, if these guys get permanently grounded, they will get orders, and can be out in the Persian Gulf, for 6 to 8 months deployment.

DR. PILMANIS: Do you have a breakdown of Type I vs. Type II?

COMMANDER BASON: Yes, I do. It is about 50/50.

DR. PILMANIS: I think this is an important difference in the Navy and Air Force numbers.

LT CMDR CLARK: It is also important to note that 65% of symptoms present while at altitude.

DR. BAGIAN: You wonder if when DCS presents at altitude, it is something victims cannot afford to ignore. They are probably more likely to report it when it happens than afterwards when there is more chance to look for other ways out.

USAF DECOMPRESSION SICKNESS DUE TO AIRCRAFT SYSTEM FAILURE

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As reported earlier in this workshop, the U.S. Air Force (USAF) has had a significant number of decompression incidents in the operational flying environment. With the exception of a few unique operational missions which will be covered in separate papers, the USAF does not ordinarily expose its flying population to an environment expected to elicit decompression sickness. USAF aircraft are either pressurized to maintain a safe cabin altitude or, as in the case of the T-37 aircraft, limited by altitude, rate of ascent and duration at altitude to maintain a safe environment. Here, I would like to address the cause of decompression sickness in normally pressurized aircraft.

Historically, the incidence of decompression sickness due to a malfunction of the pressurization system is approximately 38% of all non-high altitude reconnaissance (HAR) incidents (3). More recently, in 1989, the rate had increased to approximately 60% of the year's cases, including the HAR incidents (2).

Tables 1 and 2 list the incidents in the USAF from 1970-1980 (Table 1) and 1980-1990 (Table 2).

The 1989 incidents were analyzed to get a closer look at the specific causes of aircraft system failures which have led to decompression sickness. That year, there was a total of eleven incidents in normally pressurized aircraft (non-HAR); six in tanker/transport aircraft, two in bombers and three in fighter/attack aircraft. Of these, ten (91%) were caused by a malfunctioning pressurization system. Specific malfunctions can be grouped as: failure to establish pressurization (3 incidents), inflight loss of pressurization (6 incidents) and improper pressurization setting (1 incident). One incident was caused by a structural failure in the aircraft; a hatch was lost during severe turbulence.

Whereas the loss of effective pressurization may be the primary cause of these decompression sickness incidents, a possible secondary factor may be inappropriate or ineffective post decompression reactions.

Table 1. System Failure Decompression Sickness In USAF Aircraft 1970 - 1980*

AIRCRAFT	NUMBER OF DCS INCIDENTS by AIRCRAFT	by TYPE
Trainer		2
T-33	2	
Transport		5
C-135	3	
C-5	2	
Bomber		12
B-52	12	
Fighter		3
F-4	2	
F-101	1	
	TOTAL	22

TABLE 2. System Failure Decompression Sickness In USAF Aircraft 1980 - 1990 (part)**

AIRCRAFT	NUMBER OF DCS INCIDENTS by AIRCRAFT	by TYPE
Trainer		7
T-33	6	
T-38	1	
Transport		17
KC/C-135	6	
C-5	1	
C-130	4	
C-141	5	
E-3	1	
Bomber		15
B-52	15	
Fighter/Attack		10
F-4	3	
QF-100	1	
F-15	1	
F-16	2	
A-10	3	
	TOTAL	49

*From: Rayman, R.B., and G.B. McNaughton, *Decompression Sickness: USAF Experience 1970-80.* Aviat. Space Environ. Med. 54(3):258-260, 1983.

**1990 data were incomplete at time of publication.

Most pilots are well aware of the risks associated with exposure to high altitudes, especially above 25,000 feet. The basic regulation, AF Regulation 60-16, General Flight Rules, sets forth a sound framework for dealing with a loss of pressurization (1).

> **para 6-4c(1)** "If the aircraft loses cabin pressure, the pilot must initiate an immediate descent to the lowest practical altitude, but in no case must the pilot maintain the cabin altitude above 25,000 ft, unless the occupants are wearing functional pressure suits."
>
> **para 6-4c(4)** "If in any case an individual appears to be suffering decompression sickness, a crewmember should administer 100 percent oxygen as soon as practical and land at the nearest suitable installation if medical assistance is available."

The pilot is left with enough flexibility to deal with other operational and safety demands. However, the pilot must weigh the risks and demands and may at times impose an increased risk of decompression sickness on crew and passengers.

A closer look at the incidents in 1989 reveals that while there were no cases where AFR 60-16 was violated, six of the eleven incidents occurred after the occupant(s) of the aircraft had been exposed to reduced pressures for prolonged periods following the pressurization failure.

- 1 spent 5 minutes at 23,500 ft before descending.
- 1 spent 20 minutes at 18,000 ft and then another hour at or above 8,000 ft.
- 2 spent approximately 3 hours at 10,000 ft post decompression.
- 1 spent 30 minutes at 8,000 ft after exposure to FL320.
- 1 delayed at 8,000 ft to burn a whole load of fuel.

Continued efforts to educate the crews to the risk associated to reduced pressures, even pressures associated with 25,000 feet and lower, are indicated. And while we may never get the incident rate of decompression sickness to zero, we can work to keep the present rate as low as possible.

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CAPT GARDETTO: The C-141 at 37,000 feet that you mentioned had to be diverted to Antigua, and the crew was smart enough to stay on oxygen. The aircraft commander is to be commended. The B-52 cannot dump fuel. It must circle around and burn fuel before it can land. If it has a decompression, there is a real problem with the pressurization system and air conditioner. There was one recent mishap where the cabin was pumped to below sea level and then shot it up to 22,000 feet, resulting in DCS for a couple of people.

LTC RUSSELL: Yes. AF Regulation 60-16 gives the leeway to the crew who has to deal with these constraints in their environment. In all these 11 incidents, I believe every one of them went on 100% oxygen once symptoms were discovered. The procedures followed in all of the incidents I found were according to the book. They behaved as they are taught to behave and followed the regulation.

LTC DIXON: How many cases of bad DCS did we have?

LTC RUSSELL: There were three serious cases in fiscal year 89. Some do not say whether it was Type I or Type II. This report is only as good as what comes into the office.

LTC BISSON: Speaking for myself, as a prior B-52 pilot, most of the aircrew have enough experience and judgment to realize that they need to get on 100% oxygen and need to get below 25,000 ft. With the numbers you have presented, it is hard for me to say that we should change those requirements.

COLONEL SHEFFIELD: I review every mishap that occurs. I have been impressed with how our crew members have been trained to respond to these emergencies, and they have done it very well. The other thing I am impressed with is that as our fleet ages we continually get greater and greater risk for our crew members. It is an acceptable risk, apparently, to our line, and our crew members. This means that our training people have to have a greater burden to make sure that they are trained to handle that risk. We apparently are doing a good job, even though our number of events keep going up. However, we do not lose people from them.

DECOMPRESSION SICKNESS DURING C-130 HIGH ALTITUDE OPERATIONS

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Introduction

During the Vietnam War, a number of C-130 (Hercules Cargo Aircraft) combat and training missions evolved which required high altitude unpressurized flight operations. These unpressurized operations represented a unique mission from the aspect of exposing the aircrew to significant physiological hazards which are not routinely seen in modern aircraft. The combat missions involved the high altitude delivery of leaflets, equipment or bombs. In addition to the combat missions, unpressurized flight operations were conducted for freefall parachute training. To support these missions and provide life support/ aerospace physiology expertise, Aerospace Physiology Officers and Specialists from the 15th Physiological Training Flight, Kadena AB, Japan, were assigned to fly as aircrew members. The aerospace physiology personnel routinely kept records of all high altitude missions and recorded all significant physiological aspects of the flight.

Between 1967 and 1974, aerospace physiological personnel participated in and documented 1,638 high altitude unpressurized flights. The information gathered from these flights provides an opportunity to evaluate the significance of decompression sickness in an actual operational environment.

Table 1. Documented C-130 High Altitude Unpressurized Missions

Mission Type	Number of Flights
Parachutist Training (HALO)	176
Psychological Warfare (leaflet drops)	1,321
Equipment/Bomb Drop	141
Total	1,638

Mission Descriptions

Parachute Training. Parachute missions involved high altitude freefall training for US Army and US Air Force personnel to develop or maintain proficiency. This training required high altitude egress from the aircraft with low altitude deployment of the parachute and was commonly known by the acronym "HALO." To support these missions, Aerospace Physiology personnel installed and maintained oxygen systems, checked and repaired oxygen masks, supervised oxygen discipline and treated any altitude related problems.

HALO missions were characterized by a relatively high level of physical activity by five or six crew members (loadmasters, jumpmasters and aerospace physiology personnel). These individuals were constantly standing and moving around the aircraft to check equipment, oxygen systems and aircraft systems. The parachutists were less active. Once the aircraft was depressurized, they moved only to stand up, walk to the back of the aircraft, and jump. During the higher altitude missions, the personnel stationed near the rear of the aircraft (usually a loadmaster, the jumpmaster and an aerospace physiology specialist) were exposed to extreme cold and wind blast. The total unpressurized exposure varied according to the number of jumpers, the maximum altitude of the flight and the specific flight profile. During drops from above 18,000 ft, denitrogenation was generally accomplished prior to aircraft depressurization. The denitrogenation schedules for HALO missions required 30 minutes of oxygen prebreathing for exposures between 18,000 and 25,000 ft, 40 minutes oxygen prebreathing for exposures between 25,000 and 30,000 ft, and 60 minutes oxygen prebreathing for exposure above 30,000 ft. In unpressurized flights below 18,000 ft, denitrogenation prior to aircraft depressurization was not required. On flights below 18,000 ft, all personnel would breathe 100% oxygen for between 5 and 10 minutes prior to the start of depressurization and remain on 100% oxygen until the aircraft was repressurized to below 10,000 ft. From the standpoint of decompression sickness generating factors, the load masters, jumpmaster and the aerospace physiology personnel endured the most significant exposure.

Psychological Warfare (Leaflet Drops). The leaflet drop missions were initiated in 1966 at the request of the US Army 7th Psychological Operations Group and were primarily flown from South Vietnam and Thailand air bases. The mission targets, leaflets, desired leaflet distribution, and aircraft loads were specified by the Army Psychological Operations Group. Air Force crews were responsible for the determination of the appropriate drop altitude and mission planning. Potential enemy threat, leaflet aerodynamics, winds, fuel, and other flight characteristics were used to determine the drop altitude.

The average C-130 load consisted of 175-200 boxes of leaflets, each box weighing approximately 100 lbs. The boxes were palletized by aerial port personnel (freight handlers) prior to loading the aircraft. Each pallet consisted of approximately 60 boxes stacked 5-6 boxes high.

The average total mission time was approximately 3.5 hours. The cargo compartment crew was generally busy during the first 45 minutes of the mission securing equipment, rigging additional oxygen stations, checking equipment and preparing for the leaflet drops. If the drop altitude was below 18,000 ft, the crew would breathe 100% oxygen for 10-15 minutes prior to depressurization. On missions above 18,000 ft, the denitrogenation schedule shown in Table 2 was used.

Table 2. Denitrogenation Schedule for C-130 Leaflet Missions (1973 Pacific Air Force Policy)

Altitude	Drop time 12-20 second intervals	Drop times less than 12 seconds or more than 20 second intervals
Below 18M	Not required	Not required
Up to 25M	30 minutes	Additional 10 minutes
25M-30M	40 minutes	Additional 10 minutes
30M-35M	60 minutes	Additional 10 minutes

Prior to 1973, other various denitrogenation schedules were used. As in the HALO missions, the exact denitrogenation times were often not documented.

The process of dropping the boxes of leaflets was complex and required considerable teamwork. Two crew members (loaders) lifted the boxes from the pallets, placed the boxes an a roller ramp and pushed the boxes toward the rear of the aircraft. Two other crewmembers (hookers) who were stationed near the cargo ramp, pulled parachute lanyard out of the box, hooked the parachute lanyard on an overhead cable and pushed the box to the aft edge of the cargo ramp. Two more crew members (kickers), who were stationed at the rear edge of the cargo ramp, pushed the boxes out of the aircraft at a predetermined rate, usually 1 box every 10-20 seconds. The parachute lanyards were approximately 12 ft long and were rigged to pull the bottom out of the box when the lanyard reached full travel. This design ensured that when the boxes broke open, a uniform spread of leaflets was achieved. In addition to the regular aircrew, at least two physiological training specialists flew each mission. One physiological training specialist was usually stationed near the cargo ramp and another forward in the cargo bay. The physiological specialists were constantly moving around the cargo bay to monitor the aircrew and check oxygen systems.

Several passes over a target area were often required to attain the desired leaflet distribution. The average time to drop a full aircraft load was 40 to 50 minutes. When all of the boxes were unloaded, the "kickers" were required to pull the 175-200 lanyards out of the slip stream and back into the aircraft to allow the cargo ramp to be

closed. After pulling the flailing lanyards back into the aircraft and cleaning the cargo ramp, the ramp was closed and the aircraft repressurized. Total unpressurized exposure for all crewmembers averaged 70-80 minutes per load. After the aircraft was repressurized, the aircrew member would spend approximately 30-40 minutes cleaning up the cargo bay of the aircraft and securing equipment.

The drop rates determined the work load and the total unpressurized time. Fast drop rates minimized the unpressurized exposure but forced a very intense workload. Slower drop rates caused long exposure times but reduced the workload. The psychological warfare missions represented extremely hazardous missions from all depression sickness aspects. A total of 11 or 12 aircrew were exposed to high altitudes and 6 to 8 crew members were involved in very heavy work at altitude.

Equipment/Bomb Drop. Equipment drop and bomb drop missions were generally simpler than the leaflet missions and did not require high levels of physical activity or extended exposure at high altitude. Equipment drop missions involved parachuting heavy equipment into areas where no landing was possible. Crew activity during depressurization was relatively light and involved checking and releasing the load. Bomb drop missions were similar. During the Vietnam War, 15,000 and 20,000 lb bombs were dropped from C-130's to clear jungle areas for helicopter landing pads. On level ground, a well placed bomb would clear a 200-yard diameter area of all trees and brush. The bomb blast and shock waves would also tend to suppress enemy action in a wide area around the target.

The cargo drop and bomb drop missions used oxygen breathing schedules and denitrogenation procedures that were similar to the leaflet missions. The average bomb missions exposed nine crew members to altitude, but only 3 or 4 of the cargo compartment crew were involved in light to moderate activity (walking around the cargo compartment). The average altitude exposure for the bomb missions was 20 minutes and the average mission time was 3.5 hours.

Exposure Data

Records maintained for the C-130 high altitude missions provided the following information.

Table 3. Exposure Data

Mission Date

Type Mission

Total Flight Time

Total Aircrew

Number of Jumpers

Cargo Compartment Crew

Aerospace Physiology Crew

Denitrogenation time below 10,000 ft

Maximum Altitude

Time at Maximum Altitude

Total Time Unpressurized above 10,000 ft

Activity Level

Temperatures (cargo compartment and ramp)

Comments (equipment repairs/failure)

Physiological Incidents

These data were recorded by the Aerospace Physiology Specialists during or immediately following each mission. Records were maintained at the 15th Physiological Training Flight. A listing of all missions information was constructed manually and these data were computerized for analysis. Tables were constructed to summarize the number of missions during which reactions did or did not take place and as a function the variables documented. Due to the high number of variables and the low number of decompression sickness incidents in any given category, statistical analysis did not provide meaningful results and descriptive methods (mean and range) were used to present information.

Tables 4 through 6 present exposure information for each type of mission. These tables summarize the number of missions, number of personnel exposed to altitude, the average time spent unpressurized above 10,000 ft for each mission, and the range of the exposure times above 10,000 ft (range = minimum exposure time and maximum exposure time considering all missions in each altitude category).

Tables 4 through 6 also include the average exposure time at maximum altitude for all missions in each category and the range (minimum and maximum time considering all missions in each category). To facilitate evaluation, the mission exposure data are categorized by altitude range; for example, the 10,000 ft to 17,900 ft category includes all missions in which the maximum unpressurized exposure was between these altitudes.

Table 7 is a summary of information relative to the 14 cases of decompression sickness that were reported by the Aerospace Physiology Specialists. Due to the classified nature of these missions, the method of operations from remote bases, and the reluctance of the crewmembers, the majority of these cases were not reported to a flight surgeon or other medical authority. While verification of these reported cases of decompression sickness via medical records could not be accomplished, the documentation of this information by the Aerospace Physiology Specialists is considered to be reliable. Note that three of the reported incidents involved central nervous system (CNS) disorders, with two of these reported CNS incidents (Numbers 3 and 14) causing a mission abort and the other CNS incident (Number 11), requiring compression treatment. It is also of interest to note that three of the "bends pain" incidents (8, 9, and 10) occurred at 18,000 ft or below on missions where little or no denitrogenation was used.

Table 8 provides a comparison of the decompression sickness incidence rates for all missions combined. The decompression sickness rates, expressed as number of incidents per 1,000 exposures, demonstrates a sharply increased risk at 25,000 ft and higher.

Discussion

The high altitude unpressurized missions represent a combination of physiological stresses that are unique in modern aviation. In addition to the increased hazard of decompression sickness, these missions exposed crewmembers to an increased potential for hypoxia, trapped gas problems and thermal stress. The presence of aerospace physiology specialists and their actions to inspect and repair oxygen equipment and enforce oxygen discipline greatly reduced these risks; however, the incidence rate for decompression sickness was notable. In particular, the flights at or above 25,000 ft showed a DCS rate of 7.76/1000 exposures, nearly 10 times higher than the DCS rate for missions between 18,000 ft and 24,900 ft. While the actual number of DCS cases was low, the high rate of DCS at or above 25,000 ft seems probable.

Table 4. Parachute Training (HALO) Exposure Summary

Maximum Altitudes

	10,000-17,900 ft	18,000-24,900 ft	25,000 ft and Higher
Number of Missions	112	22	11
Total Number Personnel Exposed	3,808	614	342
Average time unpressurized above 10,000 ft and time range of exposure $\bar{X}$ min./range (min.-max)	54 (10-130)	47 (12-90)	51 (20-95)
Average time unpressurized at maximum altitude and time range of exposure, $\bar{X}$ min./range (min.-max.)	38 (5-115)	19 (5-60)	16 (5-30)

Table 5. Psychological Warfare (Leaflet Drop Missions) Exposure Summary

	Maximum Altitudes		
	10,000-17,900 ft	18,000-24,900 ft	25,000 ft and Higher
Number of Missions	873	397	51
Total Number Personnel Exposed	10,476	4,565	553
Average time unpressurized above 10,000 ft and time range of exposure $\bar{X}$ min./range (min.-max.)	84 (15-210)	78 (25-280)	61 (10-130)
Average time unpressurized at maximum altitude and time range of exposure, $\bar{X}$ min./range (min.-max.)	68 (2-210)	57 (2-260)	31 (5-95)

Table 6. Equipment/Bomb Drop Missions Exposure Summary

	Maximum Altitudes		
	10,000-17,900 ft	18,000-24,900 ft	25,000 ft and Higher
Number of Missions	85	55	1
Total Number Personnel Exposed	729	468	7
Average time unpressurized above 10,000 ft and time range of exposure $\bar{X}$ min./range (min.-max.)	38 (15-190)	78 (25-130)	25 (N/A)
Average time unpressurized at maximum altitude and time range of exposure, $\bar{X}$ min./range (min.-max.)	26 (5-180)	35 (5-115)	15 (N/A)

Table 7. Decompression Sickness Incidents

Incident Case Num	Symptom	Crew Pos.	MAXIMUM Alt (ft)	TIME/MAX Alt (min)	TIME ABOVE 10,000ft (min)	DENITRO	OTHER FACTORS
1.	Bends pain, both knees	Loadmaster (feeder)	25,000	40	Unknown	40/<10,000	* Very heavy work (1 box/21 sec) *Inexperienced crew
2.	Bends pain, both knees	Loadmaster (killer)	25,000	40	Unknown	40/<10,000	* Same flight as above * Very heavy work
3.	Bends pain, right knee	Loadmaster (feeder)	25,000	25	50	20/5,000	* Very heavy work * Mission abort * Pain cleared 13,000ft
4.	Burning Sensation, both knees	Unknown	20,000	40	Unknown	40/<10,000	* Cargo section worker exact job not known * Flt Surgeon on board, diagnosed possible bends
5.	Bends pain,	Loadmaster	25,000	40	50	35/5,000	* Very heavy work * Pain cleared 12,000ft on descent
6.	Bends pain, Left ankle	Loadmaster	25,000	30	80	30/<10,000	* Denitrogenation during slow ascent to 10,000ft * Additional 20 min on 100% O_2 at 10,000ft * Hyperventilation during drop * Pain at end of drop
7.	Bends pain, Left elbow and shoulder	Loadmaster	22,000	80	105	25/5,000	* Denitrogenation includes 5 min between 5,000 and 10,000ft * Pain onset after 70 min at 22,000ft * Slow drop, activity, moderate
8.	Bends pain, knee	Loadmaster	17,000	180	155	None	* No denitrogenation * Pain onset 150 min at 17,500 * Activity moderate

Table 7. Decompression Sickness Incidents (Cont'd)

Incident Case Num	Symptom	Crew Pos.	MAXIMUM Alt (ft)	TIME/MAX Alt (min)	TIME ABOVE 10,000ft (min)	DENITRO	OTHER FACTORS
9.	Visual symptom bends pain	Flt Engineer	18,000	35	55	None	* Symptoms at 18,000 ft during drop * Disposition unknown * Activity light
10.	Bends pain, both knees	Loadmaster	17,900	145	160	None	* Heavy work * Pain onset 4 hrs post flights * Two missions previous day to 20,000 ft. Had unreduced bends pain following the second flt
11.	CNS, bends	Flt Engineer	24,900	40	50	35/grd level	* Older (35+) out of shape inexperienced crew members * Strenuous activity * Transported to Kadena for HBO * Successfully treated
12.	Bends pain, Left knee and ankle	Med. Tech	25,000	40	50	40/<10,000	* Light activity * Pain onset 30 min at 25,000ft * Pain cleared at 10,000ft on descent
CARGO/BOMB DROP							
13.	Bends pain, left knee	Loadmaster	23,000	15	35	36/3,000	* Pain onset at 23,000ft * Pain cleared on descent * Activity moderate
PARACHUTE DROP							
14.	Bends pain, possible CNS	AeroSp. Phys. Specialist	25,000	25	40	10/<10,000	* Activity moderate * Symptoms onset at 25,000ft near end of drop * Pain still present at ground level * Mission aport

Table 8. Decompression Sickness Rates for All Missions

Altitude (Ft)	Total Exposures	Number DCS	Rate/1,000
10,000-17,000	14,998	2	.133
18,000-24,900	5,647	5	.885
25,000 or higher	902	7	7.761

The combined effect of strenuous physical activity, repeated exposures, general fatigue and dehydration placed the crews for these missions at an increased risk. Of the 1,638 missions flown, only two were aborted for DCS reactions; nevertheless, the potential for altitude related problems was considered to be a limiting factor. The development of safety rules and procedures for these missions was an evolutionary process. In addition to DCS, some of the initial high altitude flights were fraught with physiological problems (hypoxia, hyperventilation, fatigue, etc.). The lessons learned concerning oxygen discipline, adherence to denitrogenation schedules, crew briefings, crew physical condition, and the requirement for physiological support should be reemphasized for all medical and aircrew personnel who may fly these missions.

HIGH ALTITUDE AIRDROP MISSION SUPPORT: DECOMPRESSION SICKNESS CONCERNS

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During the Vietnam War, the U.S. Army's 7th Psychological Operations Group and the U.S. Air Force's (USAF) 374th Tactical Airlift Wing conducted leaflet drops over North Vietnam. In order to assure a large dispersal and avert ground fire, bundles were dropped from very high altitudes. In the early years of the war, missions had to be altered and aircrew members hospitalized because of physiological incidents resulting from these high altitude exposures. Routine problems included hypoxia and oxygen equipment malfunctions along with the occasional sinus and ear block. The most serious problem encountered was decompression sickness (DCS). An episode of DCS could mean permanent grounding for an aircrew member, or at the very least short-term grounding. Within a short time, the tasking for inflight monitoring of aircrew members and support for oxygen systems was assigned to the Aerospace Physiology Unit at Kadena AB, Japan.

Following the Vietnam War, high altitude training increased dramatically as clandestine combat units recognized the covert possibilities available with a high altitude parachute insertion of small units. Today over fifty combat units worldwide are freefall parachute qualified and jump from 20,000 ft above mean sea level (MSL) and higher. These units consist of various conventional and unconventional special operations units from all three U.S. military services, as well as foreign military services. These groups employ two methods of parachuting. High altitude, low opening (HALO) involves a free fall from high altitude with low altitude parachute opening. High altitude, high opening (HAHO) incorporates a high altitude jump with high altitude parachute deployment and subsequent glide toward the objective. Parachute drops are conducted from C-130 and C-141 tactical aircraft, and may also consist of equipment drops from high altitude which may be impossible from lower altitudes due to hostile fire.

Forty-five officer and enlisted Physiological Technicians, called Phys Techs by the user groups, provide medical and oxygen systems expertise for worldwide support of these high altitude airdrops. The Phys Techs are stationed around the world at nine USAF Aerospace Physiology Units: Andrews AFB, Edwards AFB, Shaw AFB, Fairchild AFB, Wright-Patterson AFB, MacDill AFB, Little Rock AFB, Kadena AB Japan and Weisbaden AB Germany. Figure 1 depicts actual numbers of missions and airdrops (sorties) flown during the 1987 to 1990 time frame. Taskings for High Altitude Airdrop Mission Support (HAAMS) are directed by Military Airlift Command (MAC) regulations 55-130 and 55-141 which require physiological technicians on all airdrops above 18,000 ft MSL.

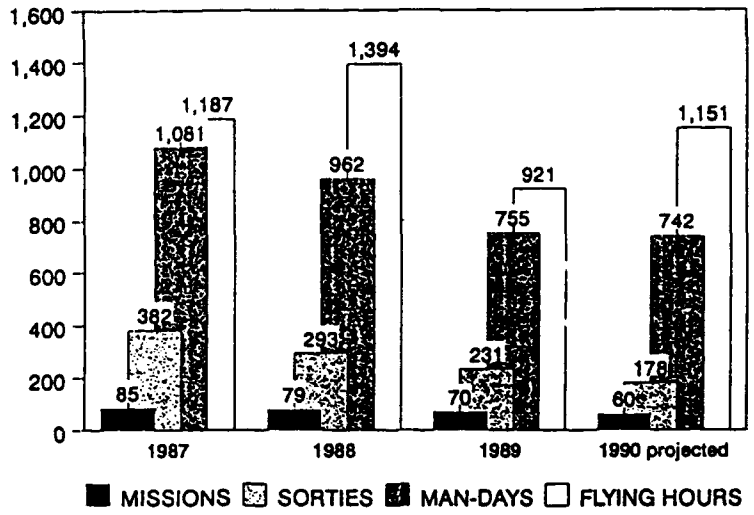

Figure 1. High altitude airdrop mission support.

The duties of a Phys Tech normally begin a week prior to the airdrop. During preflight planning the Phys Tech will coordinate with the parachuting organization on such things as number of jumpers, location of drop zone and altitude of the drops. Once the mission location is established, the flight surgeon at the nearest military facility is notified to be ready in case of a serious physiologic incident. The Phys Tech will also locate the nearest hyperbaric chamber for decompression sickness treatment and have that information available so that the aircrew can pre-plan for a diversion in case a medical evacuation becomes necessary. Prior to flight, the Phys Tech will give jumpers and aircrew members a threat briefing on physiologic concerns unique to the airdrop. Discussions include aircraft depressurization schedules, inflight medical problems, communication procedures, and schedule of events regarding disconnect from aircraft oxygen systems. Another important responsibility is as an advisor to the aircraft commander as problems arise with drop altitudes or equipment malfunctions. The majority of aircrews are unfamiliar with high altitude airdrops and the Phys Techs will often assist in rigging the aircraft's oxygen systems. A large part of any Phys Tech's job is to insure that proper prebreathing of oxygen is conducted prior to cabin depressurization. During the airdrop Phys Techs will monitor the aircrew and jumpers for symptoms of hypoxia and treat any medical problems encountered. They will also disconnect jumpers from the aircraft oxygen supply and insure that individuals are breathing off of their portable oxygen supply before departing the aircraft. That may sound obvious, but we have had cases of people leaving the aircraft without any oxygen or passing out in the airplane on their way out.

Incidence of Decompression Sickness

A primary responsibility of any physiology technician is the avoidance and (if necessary) treatment of decompression sickness (DCS). A great deal of effort is placed on educating aircrews and parachutists on the importance of proper pre-

breathing and oxygen discipline necessary to avoid DCS. Parachutists and aircrew alike are required to be current in physiological training, especially the block of instruction dedicated to decompression sickness. The number of reported cases of DCS during high altitude airdrops is relatively low when compared to other physiological problems encountered. It can be assumed that a very large number of potential DCS cases go unreported because of the nature of the population we are working with.

The rest of this article will discuss our experience with DCS on high altitude missions during the time period from 1987 to 1989. During this 3-year period, a total of 148 airdrops were performed at altitudes of 18,000 ft above mean sea level (MSL) up to but not including 25,000 ft MSL. Only one parachute drop was conducted above 25,000 ft from a Marine Corps KC-130 by Army Special Forces. The Air Force Physiology Technicians were along at the request of the jumpers. Table 1 is a presentation of the 9 DCS cases which occurred during or immediately following these 149 high altitude exposures.

Table 1. Incidence of DCS During High Altitude Operation Supported by Air Force Physiology Technicians During 1987 to 1989.

Date	DCS	Position	Location	Peak Alt/ Duration	# Sorties/ AVC Duration (min)
Aug 89	Type I	Loadmaster	Yuma AZ	FL250/90min	3/78
Aug 89	Type II	Navigator	Yuma AZ	FL250/64min	3/78
Dec 88	Type II	Jumpmaster	El Paso TX	FL230/53min	1/53
Dec 88	Type I	Marine Corps	(classified)	FL372/34min	3/32
Dec 88	Type I	Marine Corps	(classified)	same as above	
Dec 88	Type I	Marine Corps	(classified)	same as above	
Nov 88	Type I	Jumper	Pope AFB NC	FL250/36min	2/35
May 88	Type I	Loadmaster	Yakima WA	FL250/19min	2/15
Oct 87	Type I	Jumper	Yuma AZ	FL250/32min	2/29

Physical Activity

Physical activity at altitude or immediately following the high altitude exposure has been described previously as a major factor in the occurrence of DCS. This activity may accelerate the amount of evolved gas or perhaps the coagulation of trapped bubbles. In the majority of high altitude unpressurized flying, physical activity is minimal. Two exceptions to this are space shuttle extra vehicular activities and high altitude airdrops. During a typical airdrop mission, the cockpit crew remains rather stationary except for the navigator who checks the view out both side windows while calculating run-in headings and the release point. The personnel most active during the airdrop are those in the cargo compartment at the rear of the aircraft. Responsible for changing aircraft configuration such as seating, ramp door openings, and special airdrop equipment, the loadmaster has authority over actions taken in the cargo compart-

ment. The loadmaster's exertion at altitude can at times be extreme if cargo loads or equipment are moved or if a side door must be opened and closed repeatedly. Very active during all stages of the operation, the Phys Tech moves about the aircraft checking oxygen quantities, condition of personnel, and rigging of the oxygen equipment. The individuals most active on any high altitude airdrop are the parachutists and in particular the jumpmaster who is responsible for the safety of the drop. Jumpers must move about the back of the aircraft while wearing a parachute, and under some conditions, a packed rucksack. Their exertion is readily apparent; they sweat while wearing the thermal clothing required to protect them from the extreme cold at altitude. Following the airdrop, only minimal activity is required at the rear of the aircraft involving reconfiguration for landing. However, once departing the aircraft, the jumpers continue physical exertion throughout freefall, and once on the ground are required to form up with the rest of the team; after securing their gear, they go on to accomplish their mission. Clearly the population most susceptible to DCS are the jumpers and jumpmaster (Table 1).

Location of Operations

The Federal Aviation Administration controls all airspace within the United States above 10,000 ft MSL. With the increase in air travel throughout the country, especially around metropolitan areas, it is exceedingly difficult for parachuting organizations to get authorization, called block times, for high altitude airspace. This restriction necessitates training at remote locations. The availability of treatment facilities is very limited in these locations and 100% oxygen therapy is virtually nonexistent at many facilities. Because of their clandestine nature and frequently classified locations, real world scenarios also present a particular problem for the immediate treatment of any DCS episode. Because a large amount of training is accomplished in the desert southwest, a certain degree of dehydration often accompanies any mission in that region. The implication of dehydration as a factor in DCS has been reported by several researchers, and further adds to the problem. These potential problems require an increased awareness by the Phys Tech and further exemplifies their requirement on high altitude airdrops.

Altitudes Flown and Duration

The presence of Phys Techs is required on any airdrop mission conducted at or above 18,000 ft MSL. However, there are times when Phys Techs are requested on board the aircraft by the user group or are brought along as additional crewmembers during an exercise conducted below 18,000 feet. Statistics compiled from Phys Tech mission completion reports show that from 1987 to 1989 a total of 148 airdrops were conducted between 18,000 ft and 24,999 ft. The time duration spent unpressurized at these altitudes ranged from 6 to 144 minutes with an average of 36 minutes. Duration of time during airdrops at altitudes between 16,500 ft and 17,999 ft ranged from 3 to 108 minutes, with an average of 39 minutes. Because Phys Techs are only present on a small percentage of drops conducted below 18,000 ft, the exact number of drops conducted is unknown.

Oxygen Prebreathing Requirements

Tissue denitrogenation through the prebreathing of 100% oxygen has proven itself a necessary requirement prior to planned cabin depressurizations above 18,000 ft. MAC regulations 55-130 and 55-141 describe the prebreathing requirements for various altitude exposures and exposure limitations for aircrew and jumpers (Table 2). A primary responsibility of the attached Phys Tech is to insure that proper prebreathing is accomplished prior to cabin ascent. Preplanning oxygen quantities and quick visual inspections of equipment insure uninterrupted denitrogenation of all personnel. In order to avert oxygen prebreathing requirements, jumping organizations frequently perform airdrops at 17,999 ft MSL. This is an unfortunate situation that has little chance for change. Prebreathing is very uncomfortable in the aircraft because of the long hose lengths (25 ft is normal). Also the extreme cold at high altitude dictates that warm clothing be worn, a mask and helmet can become unbearable at times, especially when operating in the desert southwest. Aircrew and jumpers are knowledgeable about DCS, but will often find any excuse possible to avoid oxygen prebreathing requirements.

Table 2. Prebreathe Requirements and Exposure Limitations for Aircrew Members and Parachutists as Depicted in MACR 55-141.

Drop Altitude (MSL)	Prebreathing Times Aircrew/Jumpers	Exposure Time Per Sortie	Sortie Per 24 Hours
From FL 180 to FL 249	30 Min/30 Min	2 Hours	2
From FL 250 to FL 299	45 Min/30 Min HALO/ 45 Min HAHO	1 Hour	1
From FL 300 to FL 349	60 Min/60 Min	30 Min	1
FL 350 or above	75 Min/75 Min	30 Min	1

Summary

USAF Physiology Technicians are required by regulation to fly as additional crewmembers on all airdrops conducted above 18,000 ft. An important reason for this requirement is the possibility of decompression sickness in an aircrew member or parachutist.

Increased awareness and proper preventive measures have significantly decreased the number of decompression sickness episodes on operational high altitude missions. However because of the small number of cases, some aircrew

members and jumping organizations become complacent. As education in this area increases and solutions are more defined, operational situations which could lead to DCS will be minimized. The example that comes to mind involved the Marine Corps KC-130 airdropping Army Special Forces complemented by USAF Physiology Technicians. During this mission, the Marine Corps aircrew failed to heed repeated warnings by the Phys Techs about wearing a fitted mask during prebreathe rather than the quick-don mask they were accustomed to using. The result was DCS in three aircrew members.

The treatment of DCS is well described in the literature and has proven effective for many cases. However, there still remains a large number of military clinics without the equipment necessary to deliver 100% oxygen to an individual awaiting air evacuation to a treatment facility. Increased promotion of possible solutions may address this problem for remote operations.

Considering the dangerous environment in which we conduct these high altitude airdrops, it is indeed surprising that the number of DCS cases is so low. It can be reasonably assumed that a very large number of simple cases of DCS are unreported. In fact, on several occasions I have had individuals come to me and mention a particular DCS symptom they had while on a mission some time in the past, but were afraid to reveal because of possible loss of flight privileges. Continued definition by practitioners and researches about the treatment, potential for grounding, and need for flight physical waivers would aid in reducing the reluctance of flyers to report DCS symptoms.

In recent years, Special Operations has gained popularity as an effective way to conduct limited operations while containing costs and the potential for casualties. High altitude airdrops are an altitude tactic favored by units requiring clandestine insertion of small units. As the number of units requiring this ability increases, the propensity for problems will also increase. However, with increased knowledge by the units and medical community, the unavoidable risks can be minimized.

HIGH ALTITUDE RECONNAISSANCE DECOMPRESSION SICKNESS: STRATEGIC AIR COMMAND EXPERIENCE

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Introduction

The United States has been conducting high altitude reconnaissance and/or air sampling flights at or above 60,000 feet altitude since about 1956 to present. The aircraft that were used to support these missions, to include the trainers, consisted of: RB-57D, RB-57F, U-2A (WU-2), U-2C, U2CT, U-2R, U-2RT, TR-I, TR-IB, ER-2 (NASA), SR-71, and SR-71B. Management of most of these aircraft was initially conducted by the Central Intelligence Agency (CIA) and later by the USAF Weather Service and the Strategic Air Command (SAC). Due to the design characteristics of these aircraft and the high flight altitudes, aircrew members have been exposed to relatively high cabin pressure altitudes.

The RB-57D and the RB-57F, stationed out of Kirkland AFB, New Mexico, mainly used for air sampling and weather reconnaissance, flew slightly in excess of 60,000 feet altitude. NASA still flies two of these aircraft. No records of decompression sickness were found on this aircraft, therefore it is unknown whether there were documented problems of decompression sickness.

For simplification, reference to U-2 in the rest of this paper refers to the U-2A (WU-2), U-2C, U-2CT, U-2R, U-2RT, TR-I, TR-IB, and ER-2 airframes. The first flight of the prototype U-2 took place on 1 August 1955. The early U-2's were managed by the CIA and were based out of Davis-Monthan AFB, Arizona; Laughlin AFB, Texas; and other United States bases; and overseas locations from the late l950s till 1974. SAC took over the management of the U-2 in September 1974. The larger U-2R had its first flight on 28 August 1967. The TR-I, an internally modified U-2R, took to the air for the first time on 1 August 1981. As of September 1990, the U-2R and the TR-I (Figure I) were still deployed by SAC worldwide, conducting daily reconnaissance missions. NASA is still flying the smaller U-2C and the larger U-2R called the ER-2.

The first SR-71 came into the Air Force inventory on 7 January 1966 and was formally retired on 26 January 1990. The SR-71 flew at approximately 80,000 feet altitude and had a maximum cockpit pressure altitude of approximately 26,000 feet (5.22 psi, 269.8 mmHg). The typical mission length was about four to six hours, but could be much longer if required. The SR-71 crews prebreathed 100% oxygen for approximately 30 minutes prior to takeoff. They closed their pressure suit helmet visors and started breathing 100 percent oxygen prior to engine start. Counting this time plus time to taxi the aircraft, perform the engine runup, and wait to take off on the exact take off time

accounted for the 30 minutes. After takeoff, the SR-71 climbed to an altitude of approximately 25,000 feet, cabin altitude of 9,000 feet, where it was refueled. After approximately 45 minutes at this cabin altitude, the SR-71 (Figure 2) climbed to maximum altitude of approximately 80,000 feet, resulting in the cabin altitude rising to about 26,000 feet. Therefore, before reaching a cabin altitude of about 26,000 feet, the SR-71 aircrew member had been prebreathing oxygen for approximately 85 minutes. Depending upon the mission length, the SR-71 crewmember descended several times from a cabin altitude of 26,000 feet to 9,000 feet for the refueling stage of the mission and returned to a cabin altitude of about 26,000 feet when continuing at operational speed and altitude. Figure 3 indicates the pressure altitude the pilot and reconnaissance systems officer were exposed to versus time during a typical 5-five hour mission.

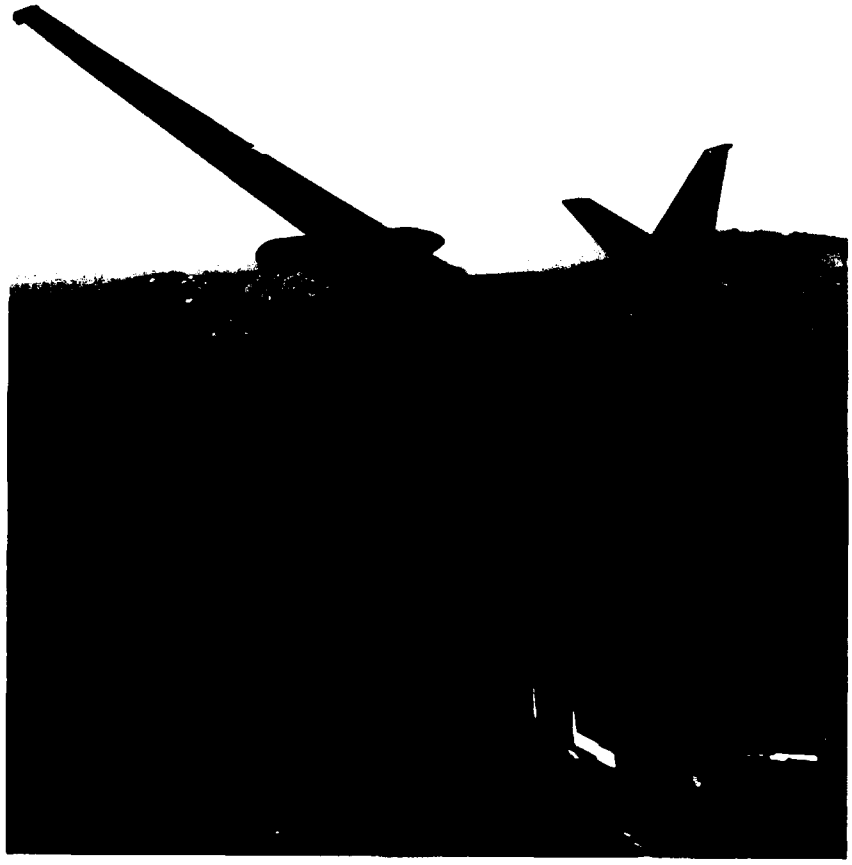

Figure 1. TR-1 .

Figure 2. SR-71.

The U-2 flies in excess of 60,000 feet altitude and has a cabin altitude during most of the mission of about 29,500 feet (4.46 psi, 230.6 mmHg). The typical length of the operational mission is over nine hours. In accordance with SAC Regulations, the pilot prebreathes 100 percent oxygen for 60 minutes prior to takeoff. In the late l950s, the 1960s, and the 1970s the prebreathing time varied from four hours, two hours, 75, 60 to 30 minutes, depending on the mission and what organization was managing flight operations. The U-2 climbs straight to maximum altitude immediately after takeoff and does not descend until preparing to land at the end of the mission. It takes about 30 minutes to climb to maximum altitude. The pressure altitude the pilot is exposed to in relation to time during a typical 9- hour mission is portrayed in Figure 3.

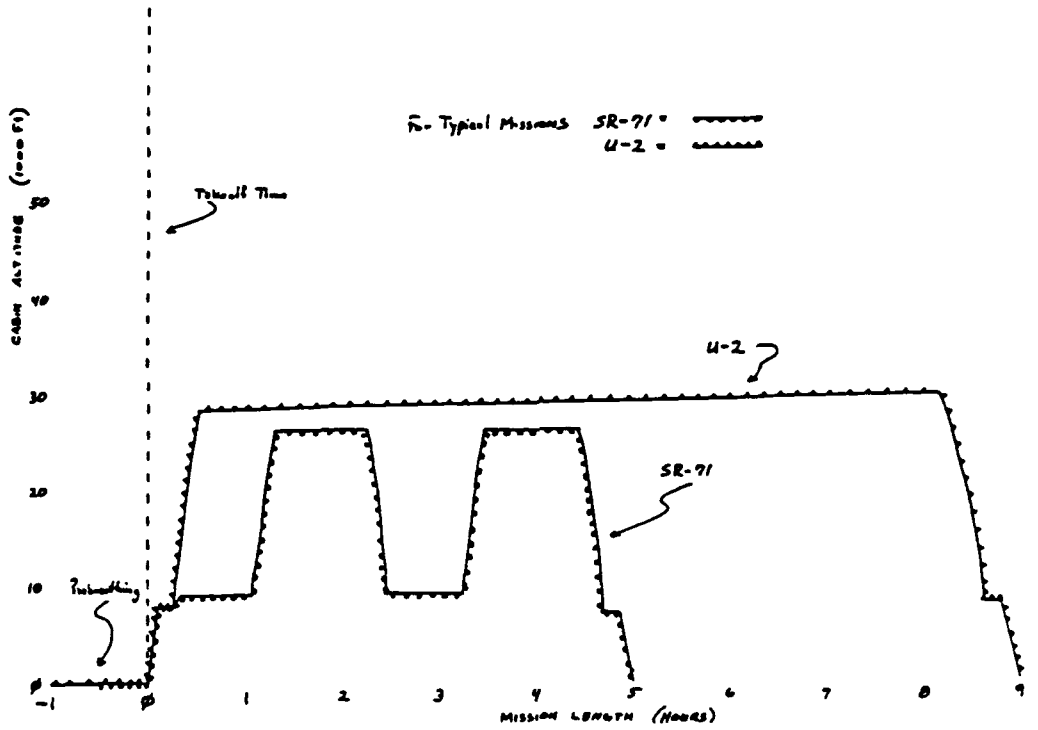

Figure 3. Cabin altitude versus flight time.

The early, smaller U-2s, due to a small cockpit size, used the less bulky partial pressure suit or modifications of this suit which maintained the pilot at a pressure altitude of 40,000 feet (2.72 psi, 140.7 mmHg) in the event of aircraft pressurization failure or bailout. In the event of an aircraft decompression or bailout, the full pressure suit used in the SR-71, U-2R, TR-I, and ER-2 maintain the crew member at approximately 35,000 feet pressure altitude (3.5 psi, 178.7 mmHg). A decompression in the SR-71 and U-2/TR-I was and is an extremely rare event and does not account for any incidents of decompression sickness since at least 1976.

Methods

Very limited data are available on decompression sickness among high altitude reconnaissance aircrews. An explanation may be due to the physiological mishaps in the SR-71 being classified during its operation and the U-2/TR-I physiological mishaps being declassified only in the last three years. Much of the information used to describe various cases of inflight decompression sickness is based on the memory of individuals that worked with the high altitude reconnaissance program or based on informal data maintained at the Beale AFB or RAF Alconbury Physiological Support Divisions (PSDs). Meader's 1967 Aerospace Medicine article titled: Decompression Sickness in High-Altitude Flight (1) is the main source of formal written data on past decompression sickness incidences in high flight operations.

Informal records of U-2/SR-71 operational decompression sickness over the last 14 years were investigated. Previous episodes of decompression sickness as recalled by Mr Thomas Bowen are listed. Mr Bowen has directly supported U-2 operations for the last 32 years and SR-71 operations for the last 14 years. Also, several decompression sickness episodes are recalled by Col Robert E. Sherman (author) who has directly supported U-2 flight operations for 8 of the last 14 years and SR-71 flight operations for about 6 of the last 14 years.

Informal comments from SR-71 and U-2 aircrew members on the prevalence of unreported decompression sickness are included in this paper.

Results

Meader's 1967 report (1) indicated 11 WU-2 pilots with a total exposure of 958 high altitude flights had reported 36 cases of decompression sickness. However, 19 of these 36 episodes involved one crewmember. Only flights to an altitude of 45,000 feet or higher were considered. This would equate to a cabin altitude of 23,000 feet or higher, up to a maximum of about 29,500 feet. The symptoms of these decompression sickness cases were reported to be entirely confined to symptoms of bends pain. This information came from September 1959 to December 1964 flight data.

The pilots wore an MC-3A partial pressure suit with an MA-2 helmet, inflatable gloves, and unpressurized boots which closely duplicated the pressure altitude of 40,000 feet for the torso and lungs during emergency cockpit decompression or bailout exposures to a pressure altitude higher than 40,000 feet. The feet, fingers, and some small areas of the body where there are seams from the suit were not pressurized to 40,000 feet. This sometimes resulted in blisters forming in these areas after prolonged exposure to operational flight altitudes during operational decompressions or training altitude chamber flights. The cockpit of the WU-2, like the U2A and U-2C, was the size of most Century series fighter aircraft (F-100, F101, F-102, F104, etc.) resulting in very little room in which to move around. The U-2R and TR-I cockpits are slightly larger than the early U-2A (WU-2) and U-2C, thus enabling the use of more bulky but more protective full pressure suits.

Of the 11 pilots reported on, the one with 19 episodes of decompression sickness wore glasses. It was speculated that he could have opened his face-plate to adjust his glasses on some of the longer flights. Meader speculated this break in his oxygen prebreathing, increased the potential for decompression sickness. If this pilot is excluded, the total operational incidence of decompression sickness among all pilots who flew the WU-2 during this time period was 0.17%. If you count the 11 pilots that had decompression sickness problems, their incidence rate was an average 3.92 percent of all high flights. For the 10 pilots, excluding the one that had 19 incidences, the decompression sickness rate was 2.96 percent of all high flights. None of the missions were aborted due to the bends pain and only one resulted in a descent to a lower altitude in order to continue the mission. This reflects a difference in procedures followed today which require descending as soon as practical and landing where the pilot can be seen by a flight surgeon when symptoms of decompression sickness are encountered. In analyzing the flights of each individual, Meader could make no correlation between bends and body temperature, pulse, blood pressure or weight.

Of interest is the difference in the incidence rate of decompression sickness symptoms among the WU-2 pilots when compared with the 100 percent oxygen prebreathing times. Prebreathing time varied from 30 to 60 minutes.

The relationship of time of preoxygenation to incidence of bends was as follows: a 0.9% incidence rate in 114 flights for those that prebreathed 50 to 60 minutes; a 2.0% incidence rate in 556 flights for those that prebreathed 40 to 49 minutes; a 3.6% incidence rate in 139 flights for those that prebreathed 30 to 39 minutes.

Based on informal records at Beale AFB and RAF Alconbury, and the memory of Mr Tom Bowen and the author, there have been at least 18 total reported cases of decompression sickness among the U-2/TR-I pilots and none in the SR-71 crew force from about August 1976 to September 1990. The U-2s moved from Davis-Monthan AFB, Arizona, to Beale AFB, California, in August 1976. Based on a total of approximately 12,320 flights of 3 to 8 hours duration (training flights) and 18,030 high flights of 9 hours or longer (operational missions) for a total of 30,350 high flights, the reported incidence rate of decompression sickness was 0.06%. The SR-71 flew about 9550 high flights during its 24-year history with no recorded cases of decompression sickness.

Possible contributing factors to these 18 U-2 decompression sickness incidences were listed by flight surgeons and aerospace physiologists as: excessive activity in the cockpit (four pilots reported bends trying to retrieve items they had dropped on the floor of the cockpit, one pilot reported bends problems after considerable effort to loosen a spur); cockpit temperature of 26 degrees F; stress due to lack of sleep, poor nutrition, and recent illness; exercising prior to flight (two separate cases of bends); dehydration.

Immediately following are 7 examples of decompression sickness that occurred in the U-2/TR-I over the last 14 years:

(1) One case in a TR-I occurred at the 5-hour point in the mission at a cabin altitude of about 28,000 feet. The pilot had prebreathed 60 minutes prior to takeoff. He experienced a gradual onset of a mild pain in his right knee. The pain did not radiate, but was localized and was not relieved with movement. He aborted his mission and while descending through a cabin altitude of about 20,000 to 15,000 feet, the pain completely resolved with no return. He continued on 100 percent oxygen until landing. For safety's sake, he was subsequently treated with 100 percent oxygen for 2 hours after landing. No recurrence of symptoms were reported.

(2) The author recalls a pilot that had three cases of Type I (bends only) decompression sickness in the TR-I while at maximum altitude at a cabin altitude of approximately 29,000 feet. The first two episodes occurred with the pilot prebreathing at least the required 60 minutes prior to takeoff. The third episode occurred even though he prebreathed for approximately 75 minutes prior to takeoff. In two of the three cases, the pilot stated he exerted considerable effort and movement trying to get a pencil off of the floor of the aircraft. In all cases, this pilot resolved all his pain during the descent phase of flight and was subsequently treated by being administered 100 percent oxygen at ground level for about 2 hours with no reoccurrence of symptoms. This pilot eventually was deemed to be a bender and was returned to flying an aircraft with a cabin altitude below 10,000 feet.

(3) One TR-I pilot reported neck pain similar to that of a pulled muscle and experienced mild nausea. The pilot had preoxygenated for 61 minutes prior to takeoff. He was flying at a cabin altitude of about 29,000 feet. He stated the pain increased considerably during the approximate 30 minute descent. Upon landing, with the neck pain still present, the pilot was immediately taken to the local hyperbaric treatment chamber and treated on a USAF Table 5 and experienced total relief from discomfort.

(4) There were at least two incidents of bends in the late 1970s that occurred to one U2 pilot who had been running for several miles just prior to his high flights. The bends occurred at a cabin altitude of about 29,000 feet. In both of these cases the pilot was prebreathing for at least 60 minutes prior to takeoff. After this preflight exercise was stopped, he indicated no further bends incidences.

(5) A U-2 pilot denitrogenated for 71 minutes prior to takeoff. He developed minor pain in the right knee at a cabin altitude of 29,600 feet after 4 hours and 45 minutes into the mission. He declared an emergency and began his descent. At 34,000 feet flight altitude, with a cabin of 20,000 feet, the pain subsided. Upon reducing power in the aircraft, resulting in the cabin altitude increasing to 23,000 feet, the pain returned. After descending through a cabin

altitude of 18,000 feet the pain disappeared again. The pilot was treated by remaining on 100 percent oxygen for two hours after landing. The pilot was placed on a 90-minute prebreathing schedule prior to all high flights for 120 days. No further decompression sickness episodes were reported by this pilot for the next 7 years of flying the U-2, even though he returned to 60 to 75 minutes of prebreathing prior to takeoff.

(6) A 35-year-old pilot with 200 hours in the U-2 had a bends incidence. On the day of this physiological mishap, he prebreathed 68 minutes prior to take off. Two hours and 52 minutes after takeoff, while flying at operational altitude at a cabin altitude of 28,000 feet, he noticed the arch of his left foot began to hurt. With some difficulty, he was able to loosen the spur on the left foot. The pain subsided. About four hours and 37 minutes into the mission both knees felt tight, slowly worsening. During this time the pilot flew with the suit partially inflated. This was a normal procedure for this pilot. When he deflated the suit to perform some cockpit duties, the pain became worse. At about 5 hours into the mission the pain in both knees and ankles was described to be about 75 percent distracting. The mission was then aborted and a descent was started. During the descent, he fully inflated the 3.5 psi suit. The pain began decreasing immediately with the descent, finally disappearing at 28,000 feet flight altitude or about 14,000 feet cabin altitude. With the suit fully inflated, his pressure altitude was 5500 feet. At ground level, he was kept on 100 percent oxygen for 4 hours with no return of pain.

(7) A U-2 pilot took off after prebreathing for 62 minutes. He encountered tingling in the hands, chest area, and a slight dizziness about 1 hour and 30 minutes into a high flight at a cabin altitude of 29,500 feet. The pilot added a little pressure to the suit and decided to continue the mission. Three hours later he had a slight itching in the shoulder and chest area. He completed his 7-hour 30-minute mission. About 10 minutes before landing the pilot vomited. After landing, the pilot was kept on 100 percent oxygen and transported to the hospital. Upon removal of the pressure suit it was noted the pilot had red petechia blotches on his chest. After 2 hours of breathing 100 percent oxygen, he was hospitalized overnight and subsequently released with no further problems.

Discussion

There are no accurate records of decompression sickness incidents of the U-2R, TR-I, or SR-71 aircrew during the life of these aircraft. Data on these incidences were scarce and of little detail and point out a need to maintain a better historical record of operational decompression sickness episodes. A key factor in determining the operational incidence of decompression sickness is that SR-71 and current U-2 aircrew were and are still reluctant to report symptoms of decompression sickness unless symptoms are severe. Based on conversations with SR-71 and U-2 aircrew members, incidences of unreported decompression sickness in the SR-71 appeared

to be very rare or nonexistent while there appears to be a significant number of unreported cases of minor decompression sickness in the U-2 crew force.

Based on actual reported cases of decompression sickness and the informal indications from the crew members of unreported symptoms that could relate to decompression sickness, it is safe to assume that the incidence of decompression sickness in the U-2 crew force is significant while among the SR-71 force it was no different than among fighter aircrew members, i.e., very rare. The cause of this obvious difference in decompression sickness episodes, many of which may not have been reported, becomes fairly obvious when comparing the pressure altitudes and normal mission length of the U-2 and SR-71. See Figure 3. It could be speculated that the past and possibly current U-2 crew force's reluctance to report decompression sickness during high flights could imply that Meader's study of decompression sickness in high altitude flight of the WU-2 did not indicate the true picture of decompression sickness incidences. The number may have been higher than reported.

The reduction of reported decompression sickness among U-2 pilots during the last 14 years when compared to the WU-2 pilots in Meader's study may be due to close adherence to the minimum 60 minutes prebreathing and more guidance toward limiting exercise prior to and immediately following high flights. The development of an efficient and easy to use urine collection device (UCD) in the full pressure suit versus no UCD in the partial pressure suit resulted in the maintenance of good hydration that may have contributed to a reduction of reported decompression sickness. The SR-71 aircrews always used the full pressure suit. U-2R and TR-I pilots have been using the full pressure suit since about July 1967. However, the number of cases of U-2 decompression sickness that has occurred that was not reported may be fairly significant.

The primary reason for a lack of reporting of decompression sickness symptoms by aircrew members is due to the post and sometimes current management of these reports by the USAF medical community. Until decompression sickness is recognized as a normal reaction to high altitude stress and the symptomatic crewmember is managed so as to be quickly placed back on flying status without extensive grounding or medical testing, aircrew will continue to not report decompression sickness. During many of the early CIA managed U-2 flights, which were flown in areas where the pilot could not freely descend after a cockpit decompression emergency, the prebreathing time was initially four hours, then 2 hours, then finally 75 minutes. This preoxygenation policy was designed to enable the pilot to complete the flight at a suit pressure altitude of 40,000 feet with minimal physiological consequences. During some flight operations, the pilot was allowed to select his prebreathing time. Therefore, you have the varied prebreathing times reported in Meader's 1967 article.

There are several SAC regulations/directives that address the decompression sickness problem in high flying reconnaissance aircraft. SAC Regulation 161-3, para 16c, 4 December 1989 (U-2/TR-I/SR-71 Physiological Support Program) states the following: "Physiological support personnel will ensure that aircrews are dressed, tested, and denitrogenated on 100 percent oxygen for a minimum of 60 minutes before

scheduled takeoff time for U-2/TR-I high flights. Denitrogenation times will be closely observed. Deviations in the start of prebreathing resulting in conflict between prebreathing requirements and scheduled takeoff times will be logged on the flight data sheet and reported to the mobile officer before takeoff. Deviations from these prebreathing requirements due to an early takeoff will be reported to the Chief, PSD, as soon as possible who will then brief the Deputy Commander for Operations. At forward operating locations, the PSD supervisor will notify the operating location operations officer of deviations from prebreathing requirements." (2)

SAC Supplement 1, AFR 161-33, para 2-8g(2)(f) 1 November 1989 (The Aerospace Medicine Program) states the following: "Dehydration. Unless compensated by the ingestion of plentiful fluids while aloft, the aviator quickly dehydrates through exposure to breathing dry oxygen and the evaporation effects of the ventilation system. Hydration is important in order to reduce risk of decompression sickness." (3) Para 2-8g(3) of the same regulation states: "It is recommended that 48 hours recovery time be allowed between consecutive high altitude pressure suit missions when the first flight is greater than 6.5 hours in duration. It is recommended that 36 hours recovery time be allowed between high altitude pressure suit missions of 6.5 hours or longer duration and training low altitude flights of less than 3 hours duration in U-2R, TR-I, T-38 or similar aircraft. ... When an airborne mission aborts, resumption of the flight by the original mission pilot/aircrew is not recommended. However, when operational requirements dictate that the same pilot/aircrew be relaunched to resume the mission, the following guidelines should be observed.

[a] Ground Abort: As long as the pilot/aircrew can be assured of a full and proper prebreathing period, they may discontinue 100 percent oxygen prebreathing while awaiting launch. [b] Airborne Abort: Resumption of flight by the original mission aircrew after mission abort should be permitted only if abort occurs within the first 2 hours following takeoff and there is not interruption of 100 percent oxygen pre-breathing between abort and relaunch for all cases where relaunch is appropriate/ required.

...Adequate hydration with appropriate fluids before flight is mandatory. ...Fluids are furnished for the flyer to maintain hydration while in flight. ...Exercise is encouraged; however, no strenuous exercise should be performed during the 12-hour period following high altitude flights because of the increased chance of decompression sickness." (3)

Conclusions and Recommendations

A reliable record of operational decompression sickness problems in high altitude reconnaissance aircrew members is seriously lacking. A means to track occurrences of decompression sickness needs to be developed. Currently Strategic Air Command Surgeon's office requires a quarterly summary of physiological problems or symptoms reported by high altitude reconnaissance pilots from the PSDs at Beale AFB, California, and all the worldwide detachments that it supports, and RAF

Alconbury, United Kingdom. This data will be compiled by the SAC Coordinator for Aerospace Physiology (HQ SAC/SGPAT). A copy of this information will be forwarded to the SAC Surgeon and the Chief SAC Flight Surgeon.

To fully understand the real needs and stresses placed on the current U-2R and TR-I crew force, a confidential questionnaire needs to be developed and administered to the current U-2 crew force to determine the basic incidence rate of decompression sickness symptoms that have and are occurring during normal operational high flight missions. Also, use of a questionnaire to those separating from the USAF may be a source of accurate decompression sickness problems while flying this aircraft. The Black Bird organization of former SR-71 and U-2/TR-I aircrew members would also be a source of individuals that could add very informative data.

Publication of the study demonstrating the reduction of bubbling and reported decompression sickness symptoms by prebreathing 100 percent oxygen at various altitudes and for 1 and 2 hours could provide some flexibility to the operational support of the current U-2/TR-I mission and provide information that could lead to an increase in decompression sickness protection for the aircrew member.

All training of aircrew with a high probability of encountering decompression sickness should be conducted from the point of view that the pilot probably will not report most incidents of decompression sickness. Therefore, all the data must be provided to the pilot pertinent to dealing with this phenomenon which will have minimal impact on the mission and the aircrew member's long-term health.

An increase in the current 60-minute prebreathing requirement for U-2 pilots is not thought to be a requirement at this time. If a future study indicates a significant number of hidden or unreported decompression sickness symptoms among the current crew force, then an increase in prebreathing time will be evaluated.

A lack of documentation of decompression sickness by aircrew subjected to 9 hours at 29,000 feet pressure altitude prevents any change in aircraft design to lower the cabin altitude. It is perceived and backed by a lack of documentation that this issue is a nonproblem.

References

1. Meader, W.L. Decompression sickness in high-altitude flight. Clinical Aviation and Aerospace Medicine, Vol 38, No. 3, pp 301-303, March 1967.

2. Strategic Air Command Regulation 161-3, Aerospace Medicine - U-2/TR-1/SR-71 Physiological Support Program, p. 4, 4 December 1989.

3. Strategic Air Command Supplement 1, Air Force Regulation 161-33, Aerospace Medicine - The Aerospace Medicine Program, pp. 3-4, 1 November 1989.

DR. HAMILTON: In industry, very often when somebody leaves they get an exit interview. I do not know whether the government does this, the military or NASA or anybody, but it would certainly be useful, if you have such a thing you could add a couple of questions on DCS that would give you this information. It would be retrospective, it would be anecdotal, but it would be better than what we are getting now.

COLONEL SHERMAN: As long as they are not getting jobs with the airlines.

DR. HAMILTON: I am saying at some point when they do have impunity. You cannot give them impunity now. We've determined you cannot.

DR. BAGIAN: In aviation, if there is a safety violation, you have 7 days to put in a report of the incident. NASA also does it, and it's kept anonymous, that way if you do get cited, you do not get penalized. There is real motivation for everybody to make sure they get the report right because if they do not, then they will be cited. I do not know how you can do that in this environment, but there could be some thought put into motivating reporting where it's kept anonymously, the stick being that anybody that does not report it and gets caught is out.

COLONEL SHEFFIELD: One of the problems of getting some of these old data was the fact that those missions were classified, and, as a result, there was an attempt to insure that all classified information was retained within the group. Therefore, Norton never received any of the mishap report data.

DR. BUTLER: With respect to exit interviewing and reporting, in the diving field it's now pretty much known that retired divers always have delayed symptoms years later. In other words, if they've had multiple minor cerebral infarcts that do not manifest themselves for years, there is an occupational hazard involved with decompression sickness. Even with dysbaric osteonecrosis, it may not manifest itself for years to come. Therefore, if there is a conspiracy of silence, the potential to catching up down the road may occur. That's where education may get these people to start talking, even if it's anonymous. If you tell them that in the long haul, 10 years later, you may start to get symptoms. On the other hand, if we know it, we could avoid it earlier and you would not have the problem.

DR. PILMANIS: The USAF Air Training Command uses the T-37, an unpressurized trainer, and the T-38, a pressurized trainer. They see approximately 3 to 4 reported DCS cases per year; half from the T-37 and half from the T-38. The maximum altitude of the T-37 is 25,000 ft. Most of the cases occur during cross-country flights. The T-38 seems to have a habit of losing pressurization. Would anyone like to comment on these DCS cases?

COLONEL SHEFFIELD: The T-37 cases, as you have stated, are primarily from cross country flights, multiple hops, involving perhaps three or four ascents, coming back down for refueling, or overnight layovers. We are looking at an individual who would be then exposed to two, three, perhaps four exposures to 25,000 feet, and then present for decompression sickness.

COLONEL SHAFFSTALL: The T-38 cases involve loss of cabin pressurization, a malfunction.

LT COL BISSON: Typically in the T-38, when you lose cabin pressure, it's on the way up because the canopy seal does not inflate.

DR. BAGIAN: We had a big rash of these about 3 years ago. I had two within a 2-week period at Flight Level 410.

LT CDR CLARK: I will give you the Navy's perspective. We only have one aircraft in the training command that's unpressurized, the T-34C. It is a turboprop single engine trainer. Because of the training environment, they have limited altitude. Its service ceiling is 25,000, but they've limited flights above 20,000 to 30 minutes. Basically, they've tried to restrict it to flights below 20,000 feet.

COLONEL SHAFFSTALL: I think in actual practice the T-37 will seldom spend any more than 30 minutes above 20,000 ft. By the time you get up there, you've got 30 minutes of fuel, and you have to come down.

DR. WEBB: That's part of it. The other part is that they do not have enough oxygen to do any real prebreathe. They do not carry much oxygen, and when they go cross country, it takes quite a while on the ground to get oxygen, because it is a separate conveyance.

COLONEL SHEFFIELD: By going higher they conserve oxygen and, as a result, extend their flight time. At the higher altitude, fuel also is conserved.

THE FAA ALTITUDE CHAMBER TRAINING FLIGHT PROFILES: A SURVEY OF ALTITUDE REACTIONS - 1965-1989

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Introduction

Since 1962, physiological training has been available to the aviation populace at the Federal Aviation Administration's (FAA) Civil Aeromedical Institute (CAMI) in Oklahoma City, Oklahoma. The curriculum consists of five to six hours of classroom instruction in aviation physiology subjects and an altitude chamber flight with rapid decompression. Students may consist of flight attendants, engineers, student pilots, flight examiners, and airline flight crews.

Participants in the altitude chamber must meet the following requirements:

1. Minimum age of 18 years (no maximum age limit).

2. Students may not wear beards.

3. Students must be free from colds or allergies and have no afflictions that could be aggravated by atmospheric pressure changes.

4. Students must hold a valid FM Medical Certificate.

5. An Assumption of Risk and Treatment statement must be signed.

Three classes of medical certificates are issued by the FAA based on medical standards established by law and contained in the Federal Aviation Regulation (FAR) Part 67 (14 CFR 76) (2). First class certificates are required for airline transport pilots, second class certificates for commercial pilots, and third class certificates for private and student pilots.

First class medical certificates require an electrocardiograph evaluation (EKG) at age 35 and annually after age 40. EKGs are not standard requirements for second and third class certificates. Except when additional medical information is required, serum chemistry tests are not required for certification in any of the three classes.

Altitude Chamber Instructors

Four instructors are required to operate an FM altitude chamber flight: a chamber operator, a flight recorder, and two inside observers; also, a flight surgeon must be on telephone standby. The inside observers who participated in these series of training flights are all former U.S. Air Force chamber technicians with many years of altitude chamber experience. Three of the four instructors have been participating for over 30 years in exposures to reduced atmospheric pressure. All inside observers possess a current FAA second class medical certificate, which is valid for one year. Maximum exposures are in accordance with U.S. Air Force Regulation (AFR) 50-27 (3) which permits two rapid decompressions in a 7-day period with at least 24 hours between exposures to rapid decompression. Routinely, all inside observers received the maximum exposure permitted during this 23-year period. On every training flight, inside observers are subjected to an altitude chamber flight and rapid decompression.

Procedures

During the 23 years cited in this report, the following profiles were conducted:

Altitude Chamber Flight Profile Type A (1965-1971): After a routine medical inquiry of each student's physical condition, the students take assigned seats in the altitude chamber. An evacuation to 7,000 ft (2,133m) at a rate of 3,000 ft (914m) per minute begins. The chamber operator levels the chamber on reaching 7,000 ft (2,133m) and lowers the chamber to 2,000 ft (610m) at a rate of 2,000 n (610m) per minute. On reaching ground level, any student suspected of being a candidate for sinusitis or aerotitis media is removed from the chamber. The chamber run is then continued, at a rate of 3,000 ft (914m) per minute, to 29,000 ft (8,839m), where the students experience symptoms of hypoxia. Exposure to 29,000 ft (8,839m) averages about 8 minutes. After the demonstration, the chamber returns to 8,000 ft (2,438m) at a rate of 2,000 ft (610m) per minute. The students next experience a decompression from 8,000 ft (2,438m) to 29,000 ft (8,839m) in 20-24 seconds. On arriving at 29,000 ft (8,839m), the chamber descends to ground level at a rate of 2,000 ft (610m) per minute. If pressure breathing equipment is used during the flight, pressure breathing and communication techniques are demonstrated. The total time of the chamber flight averages about 45 minutes. Figure 1 illustrates the Type A Profile.

Altitude Chamber Flight Profile Type B (1973-1989): After being medically screened for any predisposing factors that could be aggravated by altitude, acceptable students were seated in the training chamber and taken to 6,000 ft (1,829m) at 3,000 ft (914m) per minute. The chamber was returned to 2,000 ft (610m) at a rate of 3,000 ft (914m) per minute. Students suspected of having trapped gas problems or exhibiting unsuitable psychological manifestations were removed from the flight and these reactions are included in Table 1. The chamber was next evacuated to 8,000 ft (2,438m) at a rate of 3,000 ft (914m) per minute. Quick don oxygen masks were in the hanging position next to each student. Decompression was initiated to 18,000 ft (5,486m) during a 5-second time period. After students donned their masks, the chamber

continued to 25,000 ft (7,620m) for the hypoxia demonstration. Students experiencing hypoxia at 25,000 ft (7,620m) were restricted to a maximum time of 5 minutes without supplemental oxygen. After the hypoxia demonstration, the chamber was returned to ground level at a rate of 3,000 ft (914m) per minute. Pressure breathing and voice communication against positive pressure were practiced by each student on the descent. Average flight time was 34 minutes. Denitrogenation was not a prerequisite for these flights. Figure 2 illustrates the Altitude Chamber Training Profile Type B.

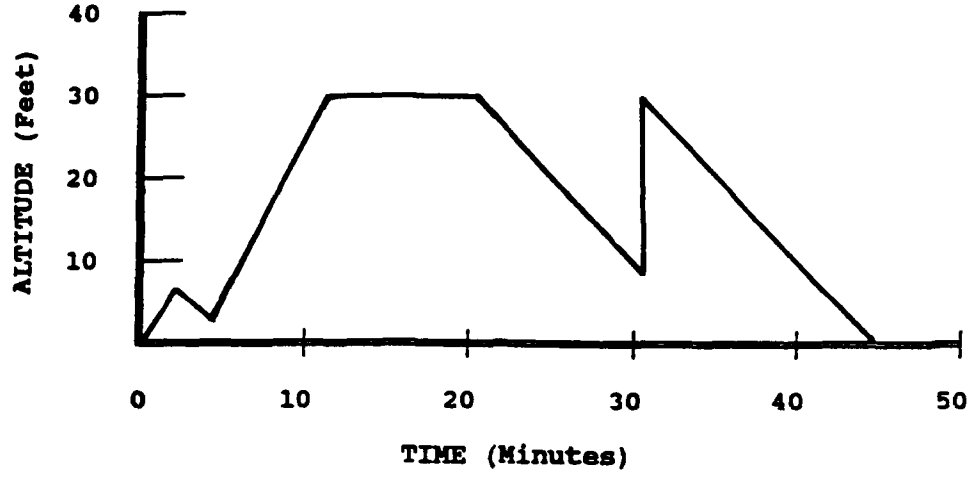

Figure 1. FAA altitude chamber profile type A. (1965-1971)

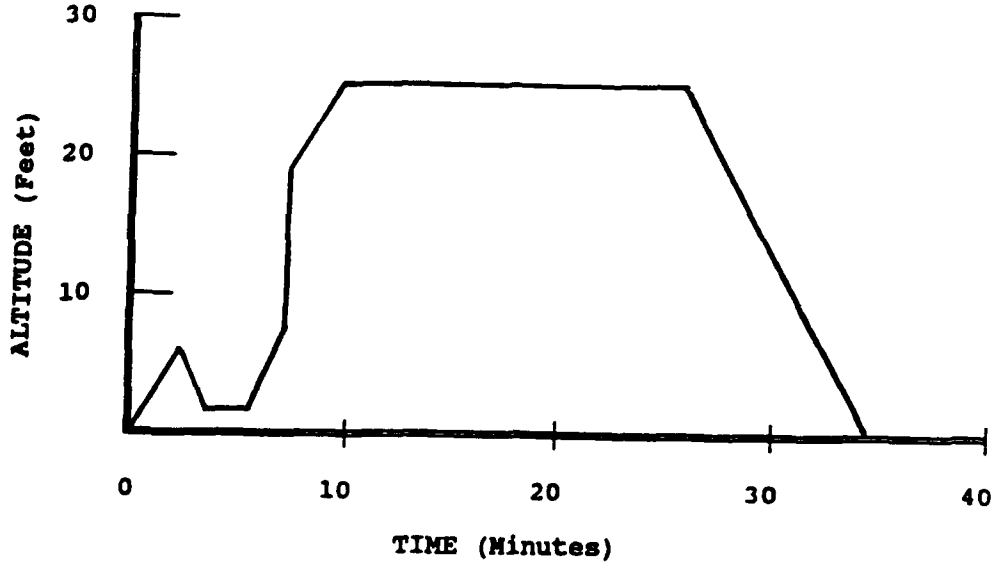

Figure 2. FAA altitude chamber profile type B. (1973-1989)

Results and Discussion

During the 23-year reporting perioa, 12,759 students were exposed to these training profiles. A total of 1,503 training flights were conducted. Table 1 shows the altitude chamber reactions.

Table 1. FAA Altitude Chamber Reactions

Symptom	Profile Type A (1965-1971)* 479 Flights 3.034 Students	Profile Type B (1973-1989)* 1,024 Flights 9.725 Students	TOTAL
Aerotitis Media	248	634	882
Aerosinusitis	81	119	200
Aerodontalgia	6	14	20
Hyperventilation	3	12	15
Abdominal Distress	5	14	19
Claustrophobia	2	0	2
Decompression Sickness	2	8	10
Apprehension	0	9	9
Tingling	0	3	3
Unconsciousness	0	1	1
			1,161

*Data from the years 1972 and 1985 were not available.

The three reported cases of tingling occurred following pressure breathing and may have been related to hyperventilation. During a hypoxia demonstration at 22,500 ft (6,858m), one student lost consciousness one minute and 54 seconds into the demonstration. He remained unconscious during the emergency descent until the chamber reached an altitude of 8,000 ft (2,43m). Decompression sickness reactions

were suspected on ten flights with inside observers accounting for most of the reactions. Table 2 illustrates the reactions.

Table 2 - FAA Decompression Sickness Reactions

Suspected Decompression Sickness

Students

SYMPTOM	ALTITUDE	RESULTS
Elbow Pain	25,000 ft (7,620m)	Relieved at 24,000 ft (7,315m)
Ankle Pain	23,000ft (7,010m)	Relieved at G.L.
Shoulder Pain	18,000 ft (5,486m)	Relieved at 10,000 ft (3,048m)

Inside Observers

SYMPTOM	ALTITUDE	RESULTS
Joint Pain	29,000 ft (8,991m)	Relieved at 27,000 ft (8,229m)
Wrist Pain	28,000 ft (8,534m)	Relieved at 22,000 ft (6,705m)
Knee	25,000 ft (7,620m)	Relieved on descent
Paresthesia (foot)	23,000 ft (7,010m)	Relieved at 13,000 ft (3,962m)
Shoulder	25,000 ft (7,620m)	Grounded
Neck, Arm and Shoulder Pain (several episodes)	25,000 ft (7,620m)	Grounded

One inside observer with over 30 years of participation in altitude chamber exposures experienced two reported and several undocumented episodes of neck, shoulder, and arm pain over a two-year period. Dull chronic pain over these areas of the body intensified with increased frequency of exposure. Paresthesia and elevated levels of pain would persist for 2 or 3 days following each exposure to altitude. Orthopedic and neurologic evaluation of this individual resulted in these findings:

1. 75% flexion, extension and rotation of his neck with pain on forced neck rotation.

2. 1 + hypoactive left biceps reflex.

3. Plain X-ray evidence of marked narrowing of the disc space at C6-C7 and narrowing of the C7 root foramen.

4. Plain X-ray evidence of narrowing of the L5-51 disc space.

5. Magnetic Resonance Imaging scan showed no nerve root compression in the cervical spine. Slight bulging of the L5-51 disc was not considered severe enough to warrant surgical treatment.

6. Neurological examination was normal except for intermittently depressed left biceps tendon reflex.

7. Nerve conduction velocities and electromyogram were normal, showing no significant neuromuscular damage.

This inside observer has been restricted from any future altitude chamber flight on the basis of cervical radiculopathy with clear cut symptomatology aggravated by altitude.

Another inside observer with over 30 years of participation in altitude chamber flights experienced shoulder pain at 25,000 ft (7,620m) during a research chamber flight. The chamber profile for this flight was similar to the training flight, except for a 3-minute stop at 15,000 ft (457m) on the way to 25,000 ft (7,620m) and a 3-minute stop at 15,000 ft (4,572m) on the way to ground level. Denitrogenation was not included in the protocol and total flight time was 46 minutes including 4 minutes at 25,000 ft (7,620m). Pain persisted after the flight and skin mottling was evident the following morning. This inside observer continued to have soreness and pain for the next two weeks and was referred to the aviation medicine experts for evaluation. Bone necrosis was not found, but he was diagnosed as having tendonitis and bursitis with the impression that years of repeated episodes of untreated limb bends had contributed to the development of a painful shoulder. He also was restricted from any future altitude chamber flights.

Examination of the data suggests that although some students did experience mild and expected altitude reactions, these training profiles appear to provide a safe learning environment while meeting the training needs of the aviation public. Considering the nominal medical requirement, unknown physical condition of students, different levels of student experience, and the minor reactions cited, continuation of the profiles is indicated.

To this author, the data compiled in this report tend to render more questions than answers. Three major concerns which immediately come to mind are:

1. Does age contribute to the susceptibility of decompression sickness?

2. On a weekly basis and over a long period of time, do rapid decompressions and altitude exposures to 25,000 ft (7,620m) and above predispose an individual to orthopedic or neurological problems during the latter years of life?

3. Do frequency and duration of altitude exposure, as defined in this report, have adverse long-term effects on respiration and circulation, hearing, the digestive system, and the various other systems in the body?

If future research should support the premise that age and long-term exposure to altitude have a deleterious effect on the body, then different safeguards may be needed for senior inside observers. Some safeguards to be considered may include:

1. An age limit or years of exposure limit for inside observers. At present, no age limit exists for inside observers. Conceivably, a 70-year-old inside observer could be repeatedly exposed to 43,000 ft (13,106m) and rapid decompression. In this study, because of physical problems, two of four inside observers with long-term exposure to reduced atmospheric pressure have been grounded.

2. A weekly maximum exposure level for inside observers considering the long-term exposure and upper age range of FAA inside observers. At the present time, FM inside observers are being maximally exposed to reduced atmospheric pressure in accordance with AFR 50-27. Is this military exposure level, which cautions against maximum exposures except in extraordinary, appropriate for older, long-term observers, such as those employed by the FM?

3. The desirability of requiring biannual or annual physical examination including an EKG, X-ray, and serum chemistry. The data obtained would not only provide information in approving or disapproving inside observers for continued exposure to altitude, but would also provide medical data on the effects of long-term exposure to reduced atmospheric pressure.

References

1. Valdez, Charles D. Ten-Year Survey of Altitude Chamber Reactions Using The FM Training Chamber Flight Profile. FM Office of Aviation Medicine Report FM AM-774, 1977.

2. Federal Aviation Regulation Part 67 Medical Standards and Certification, September, 1974.

3. Air Force Regulation 50-27, Air Force Aerospace Physiological Training Program, June, 1987.

DR. VANN: I have one comment regarding the neck pain and the cervical myelin degeneration. The cervical spine is a common site for the vacuum phenomenon. It occurs with greater frequency as people age. This may be an isolated occurrence of vacuum phenomena perhaps associated with the incident.

DR. HAMILTON: I would like to, I think, address a question to Colonel Shaffstall regarding his paper on wartime experience. You mentioned that they might do two or three missions a day for three days, and then take a couple of days off, and then they would do it again. In diving, there is a phenomenon called "workup" in which people who are diving regularly seem to have a higher resistance to decompression sickness up to a point. This is also true in tunnel work. Whenever they change the depth or change the construction job, the tunnel workers all get the bends at the start of the job. Has anybody given any thought to the application of this phenomenon for altitude? Has acclimation or accommodation to exposure been observed?

COLONEL SHAFFSTALL: With respect to the wartime high altitude missions, I could not track an individual from day to day over a period of time. However, in the 14 cases of bends that are documented, none of them were on multiple missions, they were all on single missions. However, I could not tell how many times they had flown the previous week.

CAPT GARDETTO: When we do the same thing currently, usually the high drops are saved for the end. They practice it at higher and higher altitudes until the final one is the highest. I do not know whether that is good or bad.

DR. HAMILTON: It might be favorable. Several of the people have mentioned one or another kind of rule that actually prohibits frequent exposure. One could make a case that frequent exposure might actually be to the advantage of the individual, rather than a disadvantage.

COLONEL SHERMAN: The 48 hours I mentioned for U-2/TR-1 pilots is based on fatigue, not DCS.

DR. BALLDIN: This phenomenon of decreased rate of decompression sickness during the course of the week for caisson workers could be due to a consumption of bubble nuclei. You consume those nuclei that are needed for bubble formation. Another observation is that if you work with a diving and flying situation, then you can find that even 24 hours afterwards a subject will have sudden bubbles at altitude. Immediately, when you come up to the altitude you will have precordial bubbles. If there was no dive prior to the flight, the subject usually does not have bubbles. This finding might be due to silent bubbles expanding again during the altitude exposure if the ground level internal is short.

DCS EXPERIENCE OUTSIDE NORTH AMERICA

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Introduction

This paper will necessarily be somewhat brief and parochial since my queries concerning the problem of Decompression Sickness (DCS) in the military aviation community beyond North America have met with the consistent response: "What problem?" This response is also that of the United Kingdom, so I feel it is appropriate to describe in some detail the experience of the Royal Air Force (RAF) over the past decade or so and to use that as a representative European outlook, particularly since the RAF fields the largest air force in the European arm of NATO, before considering reports from farther afield. It is convenient to consider the topic under the headings of Operational, Training and Research experience.

Operational Experience

Operationally, the RAF is largely a low-level air force, and has been for some while. The only aircraft in which cases of DCS have been officially and predictably reported are the venerable Canberras, of 1950s vintage, which have poor cabin pressurization systems and leaky cockpit seals. Even so, I am aware of just two such cases being reported in the past ten years: both victims had joint pain, both recovered during descent, and neither required active treatment. As is policy in the RAF, subsequent disposal of aircrew so affected is re-assignment to an even lower level role, usually helicopters.

In addition, a high altitude parachutist was diagnosed, whilst on detachment in the US, as suffering from mild "bends" pain following several high altitude exposures during a jumping session. And an RAF exchange officer, again in the US, was likewise diagnosed as having DCS in flight. This diagnosis was considered unlikely upon review in the UK, mainly because other more significant factors were operating.

There have been several anecdotal incidents of unreported "bends" pain in Canberra crew members and in the civilian instructors of now obsolete unpressurized training aircraft (Jet Provost Mk 4).

Training Experience

All aviation medicine training in the RAF is centralized at the Aviation Medicine Training Centre (AMTC) in Leicestershire. There, all RAF aircrew (and indeed all passengers in RAF military aircraft) undergo aviation medicine training courses (1).

This training always includes exposure to hypoxia at 25,000 feet and may include experience of rapid decompression (RD) over a representative (i.e., appropriate to aircraft type) pressure-altitude range.

These standard RDs have the following profiles:

8,000 - 25,000 feet in 3 sec (low differential cabin)
8,000 - 25,000 feet in 12 sec (high differential cabin)
17,000 - 25,000 feet in 3-5 sec (Jet Provost Mk 5 basic jet trainer)
25,000 - 45,000 feet in 3-5 sec (all fast jets, Canberra, Victor)

Decompression to altitudes at which pressure breathing is required is preceded (at least 16 hours beforehand) by formalized ground level training in the technique. Pre-oxygenation for (not less than) 30 minutes is only required if the final altitude is above 25,000 feet; and no special ascent for an ear and sinus check or for gastro-intestinal gas is ever carried out, reliance instead being placed on the preflight medical screen for the former and adequate pre-flight dietary briefing for the latter. Whatever the profile, the rate of ascent to base altitude is always 4,000 $ft.min^{-1}$.

During the five-year period 1983 - 1987, just five cases of simple DCS in students were reported by AMTC in over 12,000 exposures; giving a rate per 1,000 exposures of 0.41 (2). Onset in four of the five cases was at 25,000 feet, while the fifth occurred at 22,000 feet. Descent was initiated immediately symptoms were reported (upper limb pain in four, "minor creeps" in one) and all had cleared by ground level. No recurrence was reported and no further treatment was required, although one subject was observed overnight. In 1988, advice on suspected DCS was sought from Institute of Aviation Medicine (IAM) by AMTC on three occasions, and in 1989 no cases were reported at all. The hyperbaric support facility at AMTC has never been called into clinical use: AMTC celebrated its 25th anniversary last year.

At AMTC, a Medical Officer Instructor is the inside observer for all hypoxia exposures. Accordingly, these staff members have the highest exposure rate of all RAF personnel and it is not surprising that there are anecdotal reports of repeated joint pains in these doctors. Formal reporting of such incidents has not occurred despite the lack of career implications for these officers: perhaps the possibility of grounding and subsequent posting has not been appreciated as a means of fore-shortening what is widely regarded as an unpopular tour!

A fifth RD profile is undertaken by crews of our high altitude reconnaissance aircraft (Canberra PR9), who undergo decompression at RAF IAM from 25,000 - 56,000 feet in 3 sec in the one-man chamber and on an as required basis. These subjects receive a strict regime of pressure breathing training on the day before decompression, and also pre-oxygenate for 30 minutes. A similar training program is established at Farnborough for NASA Test Pilots from the Dryden Flight Research Facility at Edwards AFB. No problems even remotely related to DCS have been noted during these courses, although the number of subjects is low.

Research Experience

No specific research into DCS has been carried out at the IAM for many years. A fully operational hyperbaric chamber is, however, maintained at the facility in support of other altitude research programmes, which currently include RDs to 60,000 feet in support of the European Fighter Aircraft (EFA) program, and has recently involved prolonged (overnight) exposures to moderate altitudes (15,000 feet). No cases of DCS have been reported during these studies: indeed, the hyperbaric chamber was last used in anger over ten years ago when a middle-aged research medical officer suffered joint pain at altitude which did not remit during descent.

Discussion

In view of these negative findings, we should not be surprised that DCS is not regarded as a major problem in the RAF, or indeed in the national Air Forces of our European partners whose experience is apparently similar to that of the UK. We simply see neither objective evidence of widespread DCS nor have any suspicion that it is occurring but not being reported, in either the operational or the training field.

But something is clearly going on. The USAF, USN and USA experiences have been reported elsewhere and reveal an alarming incidence of DCS, especially in the training environment. And Canadian figures are believed to be similar. Last year, the RAAF reported (3) a total of six cases of DCS (four of which required hyperbaric treatment) during training profiles at 25,000 feet: an incidence similar to that of the USA hypobaric chamber operations (i.e., 0.64 per 1,000 student exposures(4)). From other sources, I believe the RAAF has had two further cases in that period. In 1988, the RNZAF too joined the club and reported two cases of DCS during chamber flights: one was mild but the other, which followed an RD (subsequently described as "inappropriate") from 8,000 to 35,000 feet in 2.4 sec, was an arterial gas embolism. The victim recovered, but with residual neurological deficits.

The forum into which the concerns of these nations have been brought is Working Party 61 (WP 61) of the Aircrew Standardization Coordinating Committee (ASCC). No mention of DCS as a problem was reported by any of the services represented at the annual meetings of WP 61 until the 25th meeting in 1984 when a single case during training was mentioned by Australia. By the following year, there was apparently sufficient concern within the United States for the USN to suggest that hypobaric training should cease. This proposal was rejected because the high incidence of DCS was not shared by other nations. Since then, however, some nations have reported a steady incidence of problems to the WP and an ASCC Advisory Publication (Number 61/101F), entitled Reporting of Hypobaric Chamber Physiological Incidents, has reached the draft stage. This document is designed to help fill the knowledge gap for all untoward chamber events, including DCS.

	AS	CA	NZ	UK	US		
					USAF	USN	USA
1989	1	-	-	-	NB A	NB B	-
1988	2	-	2	-	-	-	-
1986	1	-	-	-	-	-	-
1985	3	-	-	-	-	-	-
1984	1	-	-	-	-	-	-
1983	1	-	-	-	-	-	-
1982	-	-	-	-	-	-	-
1981	-	-	-	-	-	-	-

NB A. USAF presented detailed summary of in-flight DCS for 1975-1988 inclusive, but made no mention of training incidents or incidence.

NB B. USN reported treating 34 cases of DCS during training since 1986 and regarded this as unacceptable: use of hypoxic breathing gases at ground level was being considered.

Cases of DCS reported to ASCC WP 61, 1981-1989
[Note that nil returns ≠ nil incidents]

Conclusion

In the operational world, the low reported incidence will continue for as long as either the incidence is truly low (!) or until the serious career implications attached to admitting to suffering from DCS are removed or equitably modified. Until that time, it is likely that the "so what" attitude will prevail.

On the ground, whatever the outcome of laudable but retrospective surveys, there already appears to be a dramatic change in the occurrence of DCS, particularly in the North American training environment. It is here that questions of differences in definitions, education, reporting criteria, chamber procedures, and equipment must all be examined in an effort to clarify the conundrum.

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DR. FRANCIS: It seems to me impossible that we are dealing with the same condition. What sort of briefing do people have on what to expect if they were getting bends? Are they told what to look for and to report it?

DR. HARDING: In our training environment anybody who goes into an altitude chamber is briefed on the hazards of going to altitude. They have a briefing on hypoxia, cold, gas containing cavity problems, and decompression sickness. It is my belief, having taught it for three and a quarter years, that we neither emphasize it overly nor dismiss it. We in the United Kingdom do tell people about it. I agree with you, I simply cannot believe that, in our 12,000 subjects in the 5-year period, not one of them had a severe case of decompression sickness to match the incidence that we have seen reported this afternoon. I do not believe that they can hide it. Something strange is going on.

DR. PILMANIS: It is a North American illness.

DR. BALLDIN: The Swedish experience is that we have not treated any case of decompression sickness in the hyperbaric chamber since we decreased maximum altitude from 12,000 to 9000 meters and short exposure. That will explain why we have no apparent cases of decompression sickness. Maybe there are some unreported, I do not know, but I think that our short exposures to 30,000 feet are the explanation of the results in Sweden.

DR. HARDING: Do you preoxygenate for those exposures?

DR. BALLDIN: No.

DR. HARDING: Nor do we.

COLONEL SHEFFIELD: In looking at reports on altitude decompression sickness we have a tendency, if we are in doubt, to call it decompression sickness, rather than something else. So, if we are in doubt, we tend to err on the side of being conservative, and indeed treat that person. Is that a correct assessment?

LT CDR CLARK: For the Navy it is.

COLONEL SHEFFIELD: As a result we will see perhaps a much larger incidence, "reported incidence," of decompression sickness, whereas perhaps in your case you might have called it a muscle strain rather than decompression sickness, since you have a choice between the two.

DR. VANN: Was the time at altitude comparable to the U.S. profiles?

DR. HARDING: I think so. In a routine hypoxia demonstration, we have between 8 and 12 subjects in the chamber, and we minimize their time of oxygen to less than four or five minutes.

DR. VANN: How about age?

DR. HARDING: I cannot tell you exact figures, but it is almost overwhelmingly student pilot population, with very few people over age 40-45.

DR. VANN: I presume that is comparable to U.S. experience.

DR. HARDING: What I really find intriguing is the fact that we do not preoxygenate for these excursions. Although in the USAF chambers they do bounce up to 30-35,000 feet for a short while. Nevertheless, they do preoxygenate, and we do not.

DR. FRANCIS: I offer an anecdote to illustrate a point. Recently, I talked to a senior naval medical officer who has spent a long time in aviation medicine, and was involved in chamber training back in the early 1970's very similar to what you have described. He reckoned to get limb bends once a week. He thought nothing of it, did not report it, thought it was absolutely routine. I was astonished. He even had difficulty with intermittent urination, which again he did not report. He was married during this time and had intermittent difficulty with sexual function. Again he did not report it because he did not link it to his hypobaric decompressions. I find it amazing that people can experience this and just not relate it at all. This is a medical officer, someone who should have recognized this as being something that is possibly related to his hypobaric decompressions.

DR. HARDING: Our medical officers in the Royal Air Force know what it is and accept it for what it is. We do limit the exposure of the medical officer to once a day, unless there are absolutely dire circumstances.

DR. BALLDIN: The only time we preoxygenate is when we do a decompression from 9,000 meters to 20,000 meters in half a second, which is not routine. Then we preoxygenate for an hour and we stay there for four minutes with positive pressure breathing and go down at 3,000 meters per minute. I studied the amount of intercardiac bubbles during that type of exposure. In just one case, I found Grade 1 bubbles. There do not seem to be many bubbles during that short period of time. We have not had any problems with decompression sickness during that exposure of decompression. But then we had one hour of preoxygenation. That is the only time we use preoxygenation.

COLONEL SHEFFIELD: When I first came into the USAF program in the early 1960s, we had just started the denitrogenation requirement, 15 minutes of denitrogenation. The troops, of course, were grumbling about this, and the word spread around our unit that we did not have a bends problem until we started denitrogenating. Prior to the denitrogenation requirement, there were fatal cases of

DCS. However, we certainly did not have a large number of cases reported. Maybe denitrogentration made us more aware of it.

LT COL DIXON: During my time spent here at the USAF School of Aerospace Medicine, we have had a number of foreign senior medical officers from all over the world come through for various kinds of courses, indoctrination, tours, etc. In discussing the issue of decompression sickness, they were surprised that it existed. There appeared to be very little interest in decompression sickness even though I could show considerable data on it.

DR. PILMANIS: Most DCS symptoms are subjective. However, we also have objective information. The tape I showed you demonstrates gas emboli in vivo. DCS reporting is a very soft subject, but objective data are another matter. These data are from 30,000-ft exposures with 1- or 2-hours prebreathing, and, as you saw, there were a lot of bubbles. Day after day we watch both bubbles and bends. That is the other side of the discrepancy.

DR. NORFLEET: Have there been any Doppler studies in the UK during your exposures?

DR. HARDING: I am sure we would see bubbles. I am convinced about the bubbles. However, I saw my first case of decompression sickness ever here at Brooks three weeks ago.

DR. BUTLER: British divers certainly get the bends.

DR. FRANCIS: Oh, we are world leaders.

DR. BALLDIN: In Sweden, I have provoked decompression sickness in hundreds of subjects. We have done studies with the Doppler device, and also looked at ways to decrease the risk of decompression sickness. Actually, we are aware that it really exists. But we do not see it in the hypobaric chambers for those short exposures I mentioned.

DR. STEGMANN: Do you have people that present to the flight surgeon's office after a flight complaining of pain after a flight?

DR. HARDING: No, very rarely.

COLONEL SHAFFSTALL: We have always viewed severe decompression sickness, i.e., CNS, as being a continuum from simple bends. Is there any evidence to show that perhaps there is not as strong a link as we thought, or that something else is intervening here that is preventing them from purely subjective symptoms, which can be ignored, into something like central nervous system disorders which cannot be ignored?

DR. VAN LIEW: A better question is why you did not get any answers to the questions in the past.

COLONEL SHAFFSTALL: With 12,000 exposures, if you really had something going on, you would think at least one person would have come up with a CNS problem. You can ignore a bunch of bends, but at least somebody should have gone blind.

DR. FRANCIS: In the case I mentioned earlier, he clearly had spinal cord involvement and was ignoring it. He had difficulty urinating, and he had sexual problems, but he ignored them.

DR. PILMANIS: The wartime data showed the highest DCS field incidence reported today. I would think that under these circumstances there would be the least amount of reporting because they had other more serious things to worry about.

COLONEL SHAFFSTALL: Except for the two cases of CNS, these were unoffical reports. It was absolutely no-threat reporting and everybody knew it.

LT CDR CLARK: The following are some interesting observations from a review of Fryer's Subatmospheric Decompression Sickness in Man. This was prior to the advent of recompression therapy as a standard treatment modality. In Adler's series in 1950, there were 150 cases of serious cardiovascular collapse in a million exposures. These were altitude chamber exposures that occurred in various centers in the United States to evaluate why our bomber pilots were having trouble in bailout situations. These were fairly significant exposures; e.g., 35,000 feet for four to six hours. Their incidence of death was estimated to be seven in a million. Thus, even prior to the advent of recompression therapy, the incidence of serious complications was relatively uncommon. In that whole series there were 17,000 cases of DCS. But, once again, some of them could have been considered quite trivial; pain, numbness, paresthesias that could have easily been misdiagnosed or just ignored. So, when you are talking about 12,000 exposures, it is conceivable with the British resolve that they could just very well ignore some of these symptoms. We just have not had enough cases that have been serious enough that have been brought to light. I bet if you thoroughly briefed subjects that if their low pressure chamber flight if they have any symptoms whatsoever for the next 2 days to come back in, you might find that in fact their incidence is actually there. The fact that they have not come back in, they have not required recompression, is not incompatible with subclinical or inapparent DCS. Based on the studies before the advent of hyperbaric recompression adverse outcomes were relatively uncommon. Seven in a million deaths and approximately a hundred in a million collapses occurred.

DR. VANN: That is a good point. Fryer was British and obsessed with decompression sickness. It seemed that post-Fryer the problem stopped.

COLONEL SHEFFIELD: In the meantime, in the same time period, in this country there was a report published in the Aviation Physiologist Bulletin which described a survey of all the physicals that were performed on altitude chamber technicians (all male technicians at the time). They had 1,100 completed physicals on 461 technicians and they did an analysis of all the different problems these people had, including the sinuses and the ears and the eyes and so on. They mentioned that two of those technicians who had susceptibility to bends were restricted to flights of 30,000 feet or below.

COLONEL SHERMAN: There is a tendency for doctors to cover themselves by always going for the worst case scenario. As soon as anybody presents with possible DCS we dive them, even when I am convinced that they do not have the bends. I think that the medical community has to do that from a protection point of view. If they call Brooks they are always going to go to the conservative. That is why there is maybe a hesitation at 2:30 in the morning to call and say should we go on a Table 6 or 5. He is not going to go to Table 5 because it is always safest to go to Table 6.

DR. FRANCIS: In the hyperbaric environment, we are faced with this all the time. Is it musculoskeletal injury or is it a bend? Basically, if someone does not respond to recompression you say it is musculoskeletal injury. You do not leave them with DCS as a diagnosis.

DR. BALLDIN: You can always do a test of personnel, not a therapeutic but diagnostic hyperbaric treatment to see if the symptoms disappeared.

COLONEL SHERMAN: The other side of that is that at the 10-minute decision point, they know that if they do not report complete resolution of the pain, that they are faced with an additional two or three hours. They may at that point make a decision to report resolution no matter what their condition.

COLONEL SHEFFIELD: A classic case is one that Col. Sherman presented. The U-2 pilot who vomited in his mask and had visual problems, but was not treated in a hyperbaric facility. There was a hyperbaric facility 30 minutes away. He chose not to go. He did not want the problem of having to go for that treatment. He went to the hospital for observation. That was a crew member's call.

DR. NORFLEET: Col. Sherman, I found it interesting that the pilots had a passion for Gatorade. Do you think that they found out among themselves that it was helpful?

COLONEL SHERMAN: I think they drank a lot of Gatorade out of boredom. If you are up in orbit for nine or ten hours, you take along food and drink, something to pass the time besides reading a pocket book. But I think some of it is hydration. Some of it is just something to do. Drinking and urinating beats doing nothing.

LT CDR CLARK: Decompression sickness or decompression illness is a physiologic response to an environmental situation, similar to hypoxia, and that with the proper use of protective ensembles you can prevent or eliminate it, and as such should be treated like a physiologic response with respect to aeromedical disposition. We should totally take away the incentive or disincentive of reporting it by our aircrews by removing it as a disqualifying condition.

DR. PILMANIS: Can you take that a step further and describe what the diving Navy does, or the attitude that exists there?

LT CDR CLARK: The undersea environment is actually a quite harsh environment. The Navy diving operations treat decompression sickness as, in fact, a physiologic response to an environmental situation. It is not treated like a diagnosis of exclusion, like it is in the aviation side. The dive supervisors are very keen about querying the divers as they come to surface for any symptoms at all. In fact, they are kept in front of the dive supervisor for ten minutes after reaching the surface and closely watched and closely questioned by personnel who are familiar with that person's individual psyche, and if there is any question at all they are evaluated. If they have any symptoms or signs of any significance, they are immediately placed in the chamber and treated. Disposition wise, the procedures are quite well established. If you have Type I DCS that resolves with a Table 5, you are grounded or you are kept out of diving duties for a week. If you have Type I DCS that required a Table 6, as opposed to a Table 5, then you are grounded for two weeks. If you have Type II DCS that resolves, peripheral nervous system, or patchy peripheral paresthesia, you are grounded for two weeks. If you have any other type of Type II DCS, central nervous system DCS, either spinal or cerebral, chokes, cardiovascular symptoms, you are kept off of diving duties for a month. If you have any residual sequelae you are kept off of diving duties for up to five months. After six months, if your symptoms and findings have resolved, and your residual sequelae have resolved, then you are put back on dive status. If they are not resolved, then you are disqualified from dive status. There are many divers that I know have had bends upwards of five, six, ten times and are still on diving status. Their main emphasis is early recognition and treatment and prevention using a variety of health modification programs.

DR. PILMANIS: There is no penalty for getting DCS?

LT CMDR CLARK: Right, there is no penalty.

DR. STEGMANN: Is a wavering process required or is it automatic?

LT CDR CLARK: The Navy's diving community and flying community, both of which I am a member, are quite different. In the diving community, there are virtually no disqualifying features unless it impacts operational or performance capabilities. On the flying side, any diagnosis whatsoever seems to disqualify. Because of our insecurities, we automatically disqualify flyers and then, depending on the person's

qualifications, and our instincts or fears, we give them a waiver. The diving and flying communities have very different systems.

DR. STEGMANN: The divers do not require waivers but fliers do?

LT CDR CLARK: Right. Generally you are disqualified in the flying community.

COLONEL SHAFFSTALL: A diver usually can only kill himself.

DR. BUTLER: A diver may not take an aircraft full of people down with him.

DR. PILMANIS: However, what can an aviator in a chamber do?

LT CDR CLARK: I would submit to you that with the majority of altitude DCS cases coming from training situations in low pressure chambers, divers are more vulnerable and are more susceptible to the probabilities and chance of having DCS than our fliers are, yet we treat a flier like he has got some kind of contagion if he gets DCS. The risk of DCS for Naval aviators, the window of vulnerability, is once every four years in the low pressure chamber. Divers are in the window of vulnerability every time they dive, and that is quite frequently. I am convinced that DCS is a physiologic response that sooner or later anybody, given the proper pressure profile and time profile, would get. We take protective measures to prevent hypoxia. We should take protective measures for DCS and somebody who gets it does not necessarily represent somebody who is excessively susceptible or vulnerable to it. It is a physiologic response.

COLONEL SHAFFSTALL: The diver is only evaluated in terms of whether he can continue to do his job or not. The flier, since we cannot measure completely what type of neurological damage may be done, or what further impact that damage may on his capability to do the job, and because he has the capability of doing damage to much more than himself, should be looked at somewhat differently than the diver.

LT CDR CLARK: We do, and our policy in the Navy has become quite liberal. We will base things primarily on a performance capability. We run a series of very sophisticated tests and then we recommend an evaluation in the flight environment, in a simulator. They do flying profiles, and their emergency procedures. They get everything thrown at them, as a step back into the flying realm. You can assess their functional capability, their performance capability, anything that is compatible with their flight crew duties. I think that is an entirely reasonable approach, rather than just arbitrarily disqualifying them because they have this DCS condition.

DR. PILMANIS: Would it be fair to say that in the Navy the divers (through their education) have developed an attitude that getting bent is just part of their job?

LT CDR CLARK: Very much so. Actually, most guys are not really in the Navy diving family until they have bent, it is kind of a rite of passage almost. If they have been at it long enough they will have been bent.

DR. BALLDIN: In the diving situation you also have the risk of the possibility of dysbaric osteonecrosis coming up six months later. But, as far as I know, there is no reported osteonecrosis due to decompression in the altitude field.

DR. PILMANIS: There has been one study and it was inconclusive.

LT CDR CLARK: The concern is a very good one. Are long-term sequelae going to be evident? The diving community restricts the divers from certain operational dives. For example, they would not be experimental divers after age 45. They are then restricted to supervisory dives only. In other words, you are not going to be doing hard-core working dives. You can still do your qualifying dive in a dry chamber so you can still maintain your dive qualifications and still maintain your dive pay. Because in reality, what you are dealing with here, not unlike the aviation community, is the fact that your experience is more valuable than some of your actual skills in the environment. In the diving environment, your supervisory skills are infinitely more important by the time you have gotten to that age. In the flying community, most of the time you are going to be flying a desk, you are going to be doing mission planning, policy making or whatever. To take somebody off flight status is a tremendous insult to one's ego, and it is also a financial loss.

COLONEL SHEFFIELD: In the USAF waiver process for our crew members, our senior physicians agonize over which one should be returned to duty and which one should not. They go through very careful analysis of the events that caused the decompression sickness to occur and what the symptomatology was. They dig in the literature and pore over data to determine whether or not it is possible for this to recur in the same anatomical location. They go through a very lengthy process before perhaps finally deciding to cancel this person's flying career. Since I have been in the surgeon's office now for over two years, I have not seen any aviators removed from flying status because of decompression sickness. However, we have had a physiologist removed from flying status, and we have had some noncrew members prevented from going on flying status because they developed decompression sickness in their first training chamber flight.

LT CDR CLARK: How you treat your undesignated people or students and how you treat your designated or qualified aircrew may be different. I think that is an area you can use a little bit of perhaps arbitrary judgment. You may say, " We have not invested a lot of money in him, we would just as soon not take that risk." But once somebody is qualified and designated, I think that it is a tremendous waste not to take advantage of their experience and training, and to just arbitrarily disqualify them.

DR. PILMANIS: I would like to pursue the attitude issue. If you send aircrew members through a decompression procedure, that is their mission, and you accept

that decompression sickness is a statistical event, then you should not punish them when it happens. It is not a matter of "if," it is a matter of "when." They should be treated and sent back to work, with impunity!

MR GILBERT: Decompression sickness, it seems to me, is being treated as sort of a mystical process. We do not know precisely how it occurs. The symptoms and the sequelae are ill defined in many cases. Yet, if we take away this mysticism, we are looking at something that is truly a physiological process. We can compare DCS with motion sickness. In the Navy, we take aircrew members with intransigent motion sickness and actually train them how not to be motion sick. They can be more debilitated in aircraft being motion sick than they could being bent. Yet, we treat motion sickness as if it is something that can be handled. They have about a 90% success rate putting them back to work.

DR. PILMANIS: This is an important point. We heard that hyperbaric therapy of decompression sickness of aviators is 99% effective. However, in aviation we are perpetuating medical management. By maintaining an attitude of not reporting,we are, in effect, withholding treatment. Those people that do not report, particularly chamber techs who do it day after day after day, are injuring themselves and are not getting the medical treatment that the diver does after every dive.

DR. FRANCIS: I wonder whether there is also a psychological problem here with this waiver that results as an act of some god, miles away, over which process you have no control. What you are really after is determining whether a pilot or member of an aircrew is fit to return to duty in their functional capability. I wonder if there might be some functional test that every individual knew they would have to go through, which is directly relevant to their capabilities in the air. This would prove to be an acceptable way of making them fit to return to flying without the hands-off god effect, which could become a devil effect and instill a certain amount of fear.

LT COL BISSON: When I was a flight surgeon at Beale AFB, I was convinced that almost half the cases that I diagnosed as bends probably were not bends, although the people did seem to get better with treatment. About half of the cases, I felt were over-treated. Another point regarding the U-2 community is that the pilots do generally accept bends as a hazard of their profession. I would be willing to venture a guess that about 50% of the U-2 aircrew over the span of their career will admit to having had at least one case of bends. I personally am aware of several aircrew members who admitted that they had repeated episodes of bends. One of the ways you could detect that pilots have had bends is that you would see them coming in early for prebreathe. Instead of doing the one hour prebreathe, they will come in half an hour to an hour early.

The third thing I wanted to mention briefly is that it is getting to be a worse problem rather than a better problem in the USAF. There is a financial disincentive for reporting any inflight sickness. It was always a dent in one's pocket book. But because of recent USAF policies, now there is a substantial dent in the pocket book, if

an aircrew member disqualified, depending on the stage of one's career. Not reporting to the flight surgeon is becoming more and more of a problem. If it is something that is serious, aircrew members become their own physicians, or go downtown and get the first opinion. There are a number of aircrew members who do just exactly that.

DR. BALLDIN: This is a problem similar to the problem with the G-induced loss of consciousness. Today, most accept that there are incidents of G-induced loss of consciousness, but you will not be grounded if you report it.

DR. PILMANIS: This is a very similar situation. The change, I believe, came as a result of an anonymous survey given to pilots. Up to that point, there were few reports of GLOC. Because you were automatically grounded if you reported a loss of consciousness. After the door was opened, then there was a flood of reports.

DR. BALLDIN: Thirty percent incidence.

LT COL BISSON: I would strongly echo the concept of removing DCS as a disqualifying condition.

DR. BAGIAN: DCS is a normal physiologic response to an environmental stress. Suppose you have an incident of inflight hypoxia due to equipment malfunction. We do not ground anybody for that. They go back on status; they are never taken off status. It is just an incident. Does anybody disagree that a pilot with DCS, with no residua, should be returned to flight, no penalty, or qualifications?

DR. BUTLER: I think if it is a serious neurological complication, it needs to be handled more on a case-by-case basis.

DR. BAGIAN: I indicated "no residua."

LT CDR CLARK: You can do an evaluation, a functional evaluation, and an anatomic and physiologic evaluation.

DR. BUTLER: I still would not call it a normal situation. Part of it may be nomenclature. I do not know that I could call a serious CNS complication as a normal physiological response to decompression stress. I agree with you in principle, but I would put numerous qualifiers on what you are describing as decompression illness.

DR. BAGIAN: One of the important questions is, do we think that if somebody has had an episode of decompression sickness that they are now a bad risk? Do we think that their symptoms are more likely to recur next week?

DR. PILMANIS: As was mentioned yesterday, the study done at EDU about the Type II DCS showed that there was no correlation between an event today and what happens tomorrow.

DR. BUTLER: I am only taking the serious CNS complications and separating those out. Otherwise, I would agree with you that DCS could probably be construed as a normal physiologic response, given some sort of pressure/time violation.

MR WALIGORA: It seems to me that there are two things going on here. We are saying that DCS is a normal response to a situation. We are also saying perhaps that it is not such a big deal to have DCS. I think those are two different things, and the latter bothers me. We have shown some very dramatic differences between what we see when we ask people who have nothing to lose, i.e., research subjects reporting 80% symptoms, and operational situations reporting less than 1% with the same kind of exposures.

DR. BAGIAN: If an incident is reported, and the person has no performance deficit and is still qualified for flight, they should not have any black mark against them.

MR WALIGORA: I think we should take away that black mark, but if we took away that black mark and then 70% of the people reported that they had symptoms, what would we do then? Would we change the environment?

DR. FRANCIS: I do not think we should leave this room in the belief that this is just a physiological response, because there is demonstrable pathology in cases of serious CNS decompression sickness. However, the fact is that the functional recovery from these insults is generally extremely good, and particularly for cerebral decompression sickness. So I think it is the functional side that one needs to approach, and if somebody is functionally normal to any test that we can throw at them, I do not see why they should not go back to flying.

LT CDR CLARK: There are some analogies here. One is the hypoxia situation. Somebody at altitude who is deprived of supplemental oxygen will develop substantial neurologic and cardiovascular sequelae, which can be permanent if long enough. In addition, there is a range of susceptibility in the time-of-useful consciousness. Yet, we do not say "This jock who did not do well on time-of-useful consciousness will have to be disqualified." Perhaps it is not a physiologic response in the sense that untreated, undiagnosed it can lead to permanent residual sequelae. No question that it can. But it is incumbent on us to find the solutions of treating and preventing it, rather than disqualifying people who subsequently get it. We know that the susceptibility factor is there. However, the fact that they had gone through the risk prevention screening programs is an acceptable element of the population. In the diving community, we have not disqualified people for it unless they had residual sequelae or abnormalities in their evaluation. We have returned them to duty. Diving is a much harsher environment than flying, in which they are at a higher probability of re-exposure and, basically, re-injury. An analogy to the DCS problem in flying is the oxygen toxicity problem in diving. Oxygen at one atmosphere is physiological normal, but in a hyperbaric environment it can be toxic. The Navy used to do what was called oxygen tolerance testing based on the belief that somebody who developed oxygen CNS toxicity in the form of seizures at 60 feet on 100% O_2 was more susceptible, or at

an increased risk, for it in the future. Based on a substantial clinical input, people have now said that test is not a way of predicting who will get oxygen toxicity, and we have now discontinued its use. I think the analogy could apply here. Oxygen toxicity is another one of those physiologic responses that under certain situations everybody, sooner or later, would succumb to.

DR. FRANCIS: There is a diver/flyer difference we should remember. Although, the underwater environment is harsher, generally speaking the diver's job is normally a fairly simply sort of function. It is generally physical, and does not require the same concentration and higher function skills that piloting an aircraft does. I think that one needs to bear that in mind when thinking about fitness to return to duty.

DR. PILMANIS: I would like to point out a serious contradiction. We all agree that DCS should be treated with hyperbaric therapy. Yet, by maintaining administrative penalties of grown diving pilots, thereby forcing nonreporting of DCS, we are, in effect, withholding proper medical care. If there are any long-term courses of untreated DCS, we are perpetuating them.

DR. HAMILTON: To get people to report DCS matter of factly, we will have to tighten up our environmental procedures. We are going to know when it is putting people at risk, better than we do now.

DR. PILMANIS: Another consequence will be having accurate databases to work from. We will know if we're dealing with an ice cube on the top of an iceberg. In addition, the treatment chamber will be busier.

LT CDR CLARK: The answer is not the disqualification of people who get DCS, but rather to understand that we need to get an idea of the incidence rate, and also to approach it from a prevention and treatment standpoint.

DR. VAN LIEW: You are assuming that everybody will respond the same to decompressions. We have seen data that this is not true. There is a lot of variability. Is the variability associated with day-to-day changes or is it constant in an individual? I suspect that the variability is because of both factors. I tend to agree with your point of view, but it seems to me that you are stating it a little stronger than I like to hear. I suspect that there are truly some people that are at higher risk. The other point I would like to make is that people that get altitude DCS get it while they are on their missions, and they may be much more alone than a diver. Divers get DCS after it is all over, and are brought back up and they are in the hands of their tenders and all the people that can look after them. Divers are not apt to wreck the equipment.

LT COL BISSON: If the aviator gets DCS during the mission, it is too late. You are disqualifying him after he has already experienced it, it is after the fact. He gets the bends when he is performing his mission.

DR. VAN LIEW: That makes aviators more special than divers who get DCS after it is all over and people are taking care of them.

DR. BAGIAN: But you are assuming that we are selecting out only the pilots, and that is patently not true.

DR. VANN: There seems to be no question that decompression sickness has both random and systematic components to it. The random one is apparently greater than the systematic one. In tunnel workers, 6% of the workers were responsible for a much higher incidence than they should have been if it was simply random. But you do find people who are occasionally susceptible. Not many, but they do occur. Recently, one of our tenders bent on four out of roughly six to eight flights. That means that we lose data from this all day experiment. Certainly you have to worry about the individual's needs. I think you have to take it on a case-by-case basis, and you cannot say automatically that decompression sickness should be disqualifying, because it is so random. On the other hand, if you do have somebody who is a repeater, then you do need to think about the repeater's particular safety.

DR. BAGIAN: You can do that the way Dr. Clark has suggested. That is the way many other medical problems in the flight environment are handled. Motion sickness is a classic example. Unless it is intractable and interferes with flying duties, pilots return to flying status. It does not require a waiver.

DR. BAGIAN: However, you need to develop the ability to see functionally if there are residual effects that affect performance, even in subtle ways that are meaningful.

DR. BUTLER: Performance is the first decision to be made. However, you may be able to perform and still have permanent damage.

DR. BUTLER: For example, insurance companies that insure commercial divers may not cover a diver anymore that has had a CNS hit because that person now is at higher risk for problems down the road. There is validity to that attitude.

1990 Hypobaric Decompression Sickness Workshop

Session Five: WORKSHOP CONCLUSIONS

Andrew A. Pilmanis, PhD, Chairman

THE CLASSIFICATION OF DECOMPRESSION ILLNESS

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Over the years, I have reviewed literally thousands of diving accident case histories from around the world. It is fair to say that the quality of these reports has varied enormously from comprehensive narrative histories to entirely worthless, one liners such as "Type II DCS."

It is sad to relate that the epidemiology of diving accidents is a much neglected subject. Having recognised this, it has for many years been a goal at the Institute of Naval Medicine (INM) to try to create a diving accident database. By this, I mean the design of a formalized means of collecting data on each diving accident we see and entering these into a programme which is capable of manipulating the data in such a way that worthwhile epidemiological information about the decompression illnesses can be accrued. To date, this has proved to be an impossible target.

Part of the problem has been persuading our diving medicine physicians to collect adequate data. To this end, I found when I arrived at the Institute about a year ago that the diving accident reporting form ran to 14 pages. It required the collection of data in enormous detail. This proved to be unworkable, because few, if any, of the physicians completed even a modest proportion of the form. Before this form was developed, there was a free-style approach to data collection. While I accept that a well written narrative history is an ideal way to describe a case, without some form of guidance, the quality and extent of the data recorded in such a narrative varies so greatly that the collation of information is extremely difficult and is invariably degraded by numerous missing data points. What is required is a core of information which is considered to be essential, around which more detailed information can be recorded.

There is another disquieting aspect to the way in which we currently collect our information. This is our method of diagnostic labelling. For a database to be valuable, all the data entered must undergo an auditing process to ensure its validity. This includes the diagnosis. When David Smith and I were considering how to construct the INM diving accident database, we wondered about which diagnostic criteria we should employ. We ran a pilot study to test the adequacy of the current classification of the decompression illnesses. One hundred case histories were prepared, with all the identifying information removed. These were given to 3 diving medical officers to review independently and to allocate one of 10 diagnostic labels. Despite the fact that these three physicians had worked closely together, their concordance with respect to agreement on the diagnosis of these cases was just under 60%. I was one of those physicians. One month later, the cases were shuffled and out of interest I repeated the exercise. I am ashamed to report that I agreed with myself in just under 80% of the

cases! I could only conclude that either I am a useless diving medical officer or there is something wrong with our technique of diagnostic labelling. I expect both apply!

Let us look at a current classification of decompression illnesses and assess it dispassionately. A distinction is drawn between decompression sickness and barotrauma. Decompression sickness has been classified as:

Type I" (mild)

- Pain-only (Bends, "Niggles" etc.)
- Skin
- Lymphatic

"Type II" (serious)

- Cerebral
- Spinal Cord
- Vestibular ("Staggers")
- Cardiopulmonary ("Chokes")

There are additional manifestations of decompression sickness, such as anorexia, malaise and excessive fatigue which are generally allocated to the "Type II" category. Some authorities classify pain other than that which is in, or adjacent to, a joint as "Type II."

Barotrauma is classified as:

- Pulmonary
  - Arterial Gas Embolism (AGE)
  - Interstitial Emphysema
  - Pneumothorax
- Sinus
- Inner ear
- Middle ear
- Outer ear
- Dental
- Gastrointestinal

There are substantial problems with this classification. First of all, "Type I" and "Type II" are not mutually exclusive categories - a patient may have conditions in both categories at the same time. Second, the mechanisms involved in the decompression illnesses are poorly understood. However, given what we do know, each category contains conditions with aetiologies and mechanisms which probably differ, and thus they are not homogeneous from that point of view. Third, it has been recognised for

years that DCS is a dynamic condition and "Type I" may progress to "Type II." Thus, the potential benefit of having some loose index of therapeutic urgency built into the terminology is invalid as currently practiced. Fourth, with respect to neurological involvement, it is often difficult to identify the location of *the* lesion, because decompression sickness is a multifocal disease. The current USAF practice of describing some cases as "Type I" where there is evidence of peripheral nerve injury is therefore suspect. Finally, in practice, it is sometimes very difficult to distinguish between Arterial Gas Embolism and Cerebral Decompression Sickness. In the diving environment, it is widely recognised that unless the illness occurred in a swimming pool or during submarine escape training, where the patient's gas burden is unlikely to be adequate to cause decompression sickness or there is overt evidence of pulmonary barotrauma, it can be very difficult to distinguish between the two conditions. This is particularly the case when there is a relatively rapid onset after surfacing. Since the mechanism of injury in the two conditions - arterial bubble embolism, is highly likely to be the same, it makes little sense to "label" them differently.

In essence, what the current categorization does is to require interpretive steps between what the physician detects is wrong with the patient and how the condition is labelled. These interpretive steps involve the identification of a presumed site of injury and a guess at the mechanism of the disease. I submit that it is these interpretive steps which, in part, result in the poor concordance between and within physicians.

The inadequacy of the way in which we label the decompression illnesses has important consequences. I have mentioned the problems involved in designing databases. Despite these difficulties, many such databases have been put together, however, because of poorly reproducible diagnostic labelling it is not possible to share information between them. Another consequence is that it has made multicentre therapeutic trials very difficult to plan. If the patients entered into a trial have a dishomogeneity of illness it will be difficult to draw valid conclusions from the study. It may be for this reason, at least in part, that no multicentre trial of DCS therapy has ever been published. Finally, perhaps the greatest folly of the present system is that in many instances the decision on which recompression table to use is based on the diagnostic label. If the distinction between "Type I" and "Type II" is occasionally confused and if there is difficulty in distinguishing between Cerebral DCS and Arterial Gas Embolism (AGE), should treatment decisions be based upon such labelling? This is not to say that every case should be treated identically. Rather, if different groups of illness are found to respond differently to recompression, it should be possible for any trained physician to define, reproducibly, to which group a patient belongs.

Just a week ago, a Workshop was held at the Institute of Naval Medicine in England. The purpose of the Workshop was to try to develop a descriptive definition of the decompression illnesses and within that, attempt to classify the neurological disorders associated with decompression. We were privileged to have some of the recognised world authorities in diving medicine and neurology at the Workshop.

The problems with the current classification which I have outlined above were agreed to and the need to abandon the "Type I", "Type II" classification of DCS was accepted. Furthermore, it was agreed that since AGE is frequently difficult to distinguish from Cerebral DCS, these conditions should be brought under the same "umbrella" description of Decompression Illness. For the purpose of a decompression illness database, core descriptive information was identified which should be recorded in each case.

Evolution

This term describes the course of the condition. When describing a case, the term which is used may change from time to time because decompression illness is a dynamic condition. However, this term should only be used to describe the progress of the condition prior to instituting recompression therapy. Decompression illness may be:

- Spontaneously Resolving
- Static
- Relapsing
- Progressive

The final piece of information required prior to recompression (if this is undertaken) is **the time interval from the onset of symptoms to the commencement of recompression.** Again, this should be recorded as accurately as possible. For the purpose of a database, there are two other categories of information which are desirable to collect following treatment. First is **the response to recompression,** which can crudely be described as:

- Complete recovery
- Incomplete recovery
- None

Last, it is important to record the **results of any investigations,** being sure to note when the investigations were performed.

Clearly, it is not possible to incorporate all of these descriptive elements into a user-friendly label. It would be hopelessly unwieldy. The Workshop concluded that a considerable improvement on the current terminology would result if just the first two descriptive terms were incorporated into the title *"Acute Decompression Illness."* The term "acute" being used to distinguish these illnesses from any possible chronic consequences of decompression such as osteonecrosis or retinal degeneration. The term "decompression illness" being used to avoid confusion between decompression sickness and arterial gas embolism.

The proposal would result in terminology with the following format:

Acute /Evolution Term/, [Manifestation Term] Decompression Illness

Examples would include:

*Acute **Spontaneously Resolving, Cutaneous** Decompression Illness*

or: *Acute Static, Limb-Pain Decompression Illness*

or: Acute **Relapsing, Neurological** Decompression Illness

or: *Acute **Progressive, Multisystem** Decompression Illness*

Conditions which are obviously the result of barotrauma would continue to be diagnosed and described as such. Where it is difficult to ascribe descriptive terms to a case, the optional qualifying terms may be omitted and so long as the other core data are collected, the case may be referred for expert evaluation.

The advantage of this system is that by using readily understood descriptive terminology and few interpretive steps, consistent "diagnosis" should be possible between and within physicians. If the other core data are routinely collected, the epidemiology of the decompression disorders may, at last, be studied. If sufficient, consistent data are collected, it may be possible to identify prevalent patterns of disease which can be studied and which may eventually be given their own names. This may shed some light on the likely disease mechanisms involved. It is considered that such an approach offers advantages over the current system in which patients are "classified" (sometimes with much difficulty) into preconceived diagnostic categories. It is this process which has resulted in heated debate, often at cross-purposes, with respect to the pathophysiological mechanisms of decompression sickness--a good example being spinal cord DCS. Equally, debate has raged over therapeutic options with claims for outcome for one protocol which may have little relevance to competing therapeutic regimens due to the dissimilarity of the study populations. The challenge before us is to put such issues to one side and collect better information on the condition with which we are dealing - Acute decompression illness.

DR. PILMANIS: I am going to ask Dick Vann to describe a case that occurred to him and have you classify it.

DR. FRANCIS: I will describe it for you. I am not sure I can classify it.

DR. PILMANIS: It is rare that you get a case where the patient is a trained observer who can detail the case.

DR. VANN: I am a little reluctant to do this because it is an anecdote, and we are supposed to be getting away from anecdotes. Sometimes at least they are interesting stories, if not useful.

This occurred on one of our chamber flights for NASA to 30,000 feet. In the last four years I have made about 30 of these as an inside observer to do Doppler monitoring of subjects. The IOs normally breathe oxygen for three and a half or four hours. We were well into the flight, an hour or two hours, and I started noticing extreme fatigue, beyond the normal fatigue that one has when one is tired. It was very hard to get out of the chair to do the Doppler monitoring. These are all-day experiments, and I wanted to get through the experiment and not abort it. Next I started noticing sensations in my knee, on and off, very mild pain, but, once again, I did not want to abort the flight. Next I noticed a dry sore throat, very much like you feel the day before you start to get a cold. At that point, I did a Doppler on myself and found I had fulminating bubbles from the knee that had the niggles.

Recently, I was at the experimental Diving Unit reviewing all the flying after diving data that was available. One of the symptoms that kept coming up in their flights after these dives was a sore throat, the same kind of dry sore throat I was experiencing.

Having been sensitized to this finding, we recompressed. The knee pain went away, the sore throat went away and the fatigue went away by about 20,000 feet. They were completely gone. We ran a Table 5, just prophylactically, when we got back to the surface. There were no sequelae.

I had made several flights within the previous month. What was different about this one? The only thing that appeared to be different was that I recently had two weeks of Navy Reserve training, being quite active physically, for a SEAL team field exercise. Perhaps it was related, perhaps it was not.

We had not been briefing our subjects to look for symptoms of this nature. Because of that, it is a possibility that such symptoms might be overlooked. I would not call it prime chokes, but I would call it incipient chokes. That was my interpretation. It

is probably worthwhile for altitude chamber questions to include a description of potential symptoms to the trainees or subjects.

DR. FRANCIS: If I had to describe this cord, it would be acute progressive multisystem decompression illness.

DR. BUTLER: If we were still relying on the old system would you call this cord a Type I?

DR. FRANCIS: The old language is inadequate to describe this, as it is with so many of the things that one sees.

DR. BUTLER: Would you treat it as a Type I with a Table 5? Were there any neurological symptoms involved?

DR. VANN: There were no neurological symptoms.

DR. FRANCIS: How do you describe fatigue?

DR. VANN: Well, that is a constitutional.

LT CMDR CLARK: How do you handle someone whose symptoms are fairly substantial but then recover with return to site level?

DR. FRANCIS: I ought to make it clear that this particular descriptive process was designed specifically for the diving setting. Normally, one requires intervention such as return to depth. One would use this terminology, and particularly the evolutionary part of it, to describe what happened prior to intervention, i.e., recompression.

LT CMDR CLARK: So you consider return to site level or ground level intervention?

DR. FRANCIS: That is recompression. That is intervention.

LT CMDR CLARK: Unfortunately, sometimes peoples' symptoms return after returning to sea level.

DR. FRANCIS: In which case, you can refer to it as relapsing.

DR. VANN: That is why we did a Table 5, just to avoid that potential problem.

LT CMDR CLARK: There is also the judgment issue if you pursued this a little bit more with mental status testing. This gets back into this flight performance problem. You knew you were having symptoms. You were tending to a norm like many of us do for operational considerations. However, as it progressed, you finally overcame that tending. One of the things we found in our evaluations is that judgment

and cognitive dysfunction can be very subtle, You may have had a judgment issue as well. It is hard to, retrospectively, identify.

DR. FRANCIS: Part of the problem with the way we have done things in the past is that we have imposed on this very, very complex illness a mechanistic structure. We have tried to fit in the manifestations of the disease into our preconceived mechanistic structure. If we can get away from that and describe what we actually see, we have a fighting chance of coming up with commonly appearing syndromes and give these names which may be meaningful when we discuss them with each other.

DR. HAMILTON: What do you mean by acute?

DR. FRANCIS: I mean acute as opposed to chronic. Chronic we will reserve for things like dysbaric osteonecrosis. It is the acute illness as it presents to you following a dive or flight.

DR. HAMILTON: What do you mean by relapsing?

DR. FRANCIS: Relapsing is, for example, and again strictly with reference to diving, where there is a rapid onset of a problem, often neurological, after reaching the surface. This gets progressively better and the divers may even be asymptomatic when they reach you. But within an hour or two they have a recurrence of symptoms.

DR. HAMILTON: Then this case would have been progressive.

DR. FRANCIS: It was progressive until intervention.

DR. HAMILTON: Rather than relapsing.

DR. FRANCIS: Well, it is progressive until intervention. I described it as progressive.

LT CMDR CLARK: I think for such a classification system to work, and I think it is an excellent idea to do this, you need to have some specifics so that there is very little room for misinterpretation by the person who's going to be filling out the evaluation form.

DR. VANN: In our altitude experience, all of our symptoms have been pain only. We have attempted a similar domification process in order to better quantitate the pain. What is the description of the pain: steady, transient or increasing. What is the character of the pain: sensation only, dull, sharp. Where is the location, obviously. Is it position sensitive. The intensity is graded on a zero to ten scale where zero is nothing, ten is excruciating. It is subjective of course but it gives you a feel.

DR. FRANCIS: I think that is great. Please understand that this is an irreducible minimum information that we require. I have no problem at all with people elaborating on this. This is merely a foundation from which to add further detail, and I think what you propose for pain is wonderful. What I propose is the core information that one must have in order to get some sort of idea as to what is going on, and the more information the better.

LT COL BISSON: The concordance of 60% for presenting symptoms is not all that terrible when you look at things like radiological films. Thus, the disconcordance, depending on how specific the presentation is, is really not that bad.

DR. FRANCIS: This was a group of three people who had known each other for a long period of time and have worked together.

LT COL BISSON: But it depends on just what kind of information you had.

DR. FRANCIS: If I was to take three people from around the world who never met each other, and give them the same list, 60% may be a very optimistic target. But when you want to start getting multi-center trials going, you have got to do better than that.

COLONEL SHEFFIELD: Recently we have had two cases reported in which they were listed as Type II decompression sickness, and yet they were pain only bends cases. In contacting the physicians, we found that they recorded it as Type II because more than one limb was involved.

LT CMDR CLARK: That is the Navy's policy.

COLONEL SHEFFIELD: That explains it because, in each of these cases, the individual had been trained in a Navy facility.

LT CMDR CLARK: Your guys have taken what we have already decided is an outmoded and inefficient or unacceptable classification system, and modified it. That is the problem. We need to come up with some standardized approach.

DR. PILMANIS: One of the most dramatic and important lessons we learned early in the operation of a hyperbaric facility for treatment of emergency diving accidents was the simple fact that you must do a thorough neurological exam on patients. If you relied on what most people rely on, on patient testimony, 80% of the cases were Type I. If you did a thorough, competent neurological exam prior to compression, 80-90% of the cases were Type II. Relying on patients as witnesses is one of the problems inherent in this field. In many cases, patients are simply not aware of their Type II symptoms.

LT CMDR CLARK: That has been our experience too. If you compare the Air Force and the Navy incidence, you will find that the Air Force's ratio of Type I/II is about

90%/10% and the Navy's ratio is about 50%/50%. Part of the explanation for this is a difference in classification. However, I think the primary reason is that the Navy trains its people to be very thorough in neurological examinations of DCS patients.

THE NEUROLOGICAL EVALUATION OF DECOMPRESSION SICKNESS

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Introduction

Neurologic sequelae are common in decompression sickness following exposure to hypobaric and hyperbaric environments (4, 8, 19). The purpose of this presentation is to review basic neurologic assessment techniques used to evaluate personnel following exposure to a hypobaric environment where decompression sickness (DCS) is suspected. Since neurological consultation and sophisticated neurodiagnostic testing are usually not immediately available and may delay therapy, emphasis will be placed on the initial history and examination in the diagnosis and management of DCS. A standardized format for neurologic evaluation will aid in the diagnosis, treatment, and epidemiologic assessment of DCS. Neurobehavioral screening examinations have been recommended for saturation divers at greater risk for DCS (16).

Methodology

The goals and characteristics of the DCS neurological evaluation are outlined in Table 1. The indications for neurologic evaluation of DCS are outlined in Table 2.

Table 1. The Neurological Evaluation

Goals:	1) Identify differential diagnosis/pathology
	2) Establish baseline
	3) Support therapeutic decision
	4) Monitor therapy
	5) Provide epidemiologic database
Characteristics:	1) Comprehensive
	2) Expeditious

Table 2. Complete Neurologic Examination Prior to Hyperbaric Recompression

Not Essential:	1) Substantial neurologic deficit
	2) Cardiovascular collapse
Indicated:	1) Symptoms of DCS in a stable patient
	2) Following hyperbaric recompression
	3) Post treatment recurrence
	4) Prior to or during transportation

The primary purpose of the neurological history and physical examination is 1) to identify the level of the lesion in the neuraxis and 2) to establish a pertinent differential diagnosis. The neurologic evaluation of DCS should be thorough but brief. The detection of neurologic deficits is essential to the management of decompression sickness. Neurologic DCS is often multifocal, and a comprehensive examination is necessary to determine the extent of involvement. The presence of subtle but definite findings would dictate extensions or additional hyperbaric treatments (2).

The neurologic evaluation should be able to identify the region of the neuraxis affected at the level of:

1. The cerebral hemispheres (i.e., supratentorial)
2. The brain stem or cerebellum (i.e., intratentorial)
3. The spinal cord level
4. Peripheral nerve/nerve root
5. Muscle/musculoskeletal

A number of conditions may mimic altitude DCS. A differential diagnosis of Altitude DCS is outlined in Table 3. A format for the neurologic evaluation of decompression sickness is presented in Table 4.

The initial step is to obtain a brief but thorough history with specific emphasis on the patient's complaints. A number of excellent books on the neurologic history and examination are available (3, 12, 13). It is helpful to establish the temporal relationship of the patient's symptom or sign, including onset, course, and resolution of complaint or neurological deficit with respect to time. A severe or sudden onset of a neurological deficit may indicate an early presentation of potentially life or limb threatening decompression sickness and more urgent referral for recompression indicated. Under such circumstances, a neurologic examination should not delay therapy, as the

decision to treat has already been made, although an examination should be performed enroute or during recompression, if possible. An assessment of risk factors for DCS may also be helpful in the history.

Table 3. Differential Diagnosis of Altitude Decompression Sickness

1) Musculoskeletal (Non DCS) limb pain

2) Hypoxia

3) Hyperventilation

4) Carbon Monoxide poisoning

5) Spatial disorientation

6) Air sickness

7) Trapped gas syndromes

    a) abdominal distention

    b) barotitis

    c) aerodontalgia

    d) aerosinusitis

8) Alternobaric Vertigo

9) Perilymph fistula

10) Acceleration atelectasis

11) Spontaneous pneumothorax

12) Migraine syndrome

13) Entrapment neuropathy

14) Cervical radiculopathy

15) Factitious DCS

Table 4. The Neurologic Evaluation of Decompression Sickness

Neurological history
Neurological examination
Mental status testing
Cranial nerve testing
Motor evaluation
Sensory evaluation
Reflex testing
Coordination testing
Gait and station
Diagnostic evaluation tests
Formal neuropsychological testing
Electroencephalography
Cortical evoked potentials
Cranial computerized tomography
Magnetic resonance imaging
Single photon emission computed tomography

The mnemonic LEARNIT can be applied in the taking of a history of neurological complaints, particularly headache and pain syndromes:

L - location
E - exacerbating factors
A - alleviating factors
R - radiation (where it starts, where it goes)
N - nature (pressure, sharp, throbbing)
I - intensity (scale of 1 to 10)
T - timing (frequency and duration)

The DCS examination should include an overall general physical examination with attention directed to the cardiopulmonary system. The neurological examination traditionally begins with the mental status examination. Generally this is a part of the overall response of the patient to the evaluator. The DCS patient may have subtle cognitive dysfunction, although not specifically complaining of problems with thinking, memory, or performance. Significant symptoms of DCS may be present without objective neurologic findings (17). Subtle personality changes noted by friends or work associates may be a sign of cerebral DCS. Fatigue, lassitude, dizziness, or heaviness may also be subtle signs of DCS. A screening mental status examination may detect subtle higher cortical dysfunction, indicating need for hyperbaric recompression therapy, even in the absence of other neurologic deficits (2, 5, 15). Screening mental status tests include the Mini Mental Status Exam (MMSE), the Neurobehavioral Cognitive Status Examination (NCSE),and Halstead Reitan Test Battery (Appendix A). There is an outline for the neurologic examination in Appendix B.

Mental Status Examination

The mental status examination includes assessment of level of alertness, orientation to person, place and time, effect, and appearance. An evaluation of memory function would include immediate recall-digit span (forward and reverse), short-term memory - object recall after three minutes or the ability to recall a previously told short story, and remote memory tests, such as points of history (past presidents). The patient's level of education should be estimated. Speech and language function may be tested by having the patient repeat words or phrases, name objects, read and comprehend and perform simple commands. Judgment, insight, and abstracting ability may be tested by asking the patient to make comparisons between similar objects. Calculations may be tested by having the patient subtract seven from 100 and each successive number (Serial 7's) or by calculating how many nickels are in a $1.35 or some other coin exchange problem. An impaired mental status may hamper other portions of the neurologic examination requiring patient feedback, such as the sensory and motor assessment.

Cranial Nerve Examination

The next section of the neurological examination is an evaluation of the cranial nerves. General visual acuity and visual fields should be tested. The pupils size and response to direct light and accommodation should be noted. The pupils should react to the same degree with the same light source and the patient can be asked if the exam light appears the same brightness in either eye. An unequal response or subjective difference in light intensity is suggestive of an afferent pupillary defect (APD). Visual fields may be tested by finger count confrontation, with stationary fingers placed in the central 30 degrees of vision and the patient asked to give the total number of fingers. All four quadrants should be tested in each eye separately. The fundiscopic examination should include a brief look at the optic disk, macula, blood vessels, and nerve fiber layer.

In the extraocular muscle examination, the eye muscles should be tested in the six cardinal fields of gaze and taken to their endpoints. Subtle clues of eye muscle imbalance include an asymmetric corneal light reflection, or more scleral margin seen in one direction than another with the the eyes in an eccentric field of gaze. The eyes are tested in the cardinal fields by having the patient pursue a slowly moving finger. Eye muscle imbalance may be detected using the cover/uncover (tropia) or alternate cover (phoria) method. The patient is directed to stare at the examiner's finger and then the eye is covered and uncovered and movement of the eye from the covered to uncovered position is noted. An eye that moves from inward to outward on the cover/ uncover method would be indicative of esotropia. On alternate cover testing one eye is always occluded and any latent deviation of the eye is noted, deviation from inward to outward would be an example of an exophoria.

The sensory Trigeminal (V) nerve is tested by eliciting the cornea reflex or the sternutatory reflex. The corneal reflex may be tested by gently blowing on each open eye separately. The sternutatory reflex is tested by sticking a small stick up the nose and looking for a blink or cough. Trigeminal motor function tests the muscles of mastication (masseter, temporalis, and pterygoids which open, close, and move the jaw to the front, back and side to side). Facial nerve testing evaluates the muscles of facial expression (forehead wrinkles, eye closure, smiling and pursing of the lips), and should include a comment on eyelid symmetry. The Glossopharyngeal (IX) and Vagus (X) nerves are tested by the gag reflex, and testing phonation (saying consonants ba, da, fa, la, ga). Although the following are technically cranial nerves, they are usually tested as part of the motor examination. The Spinal Accessory (XI) cranial nerve is tested during the muscle exam by having the patient turn the head to either side and by shrugging the shoulders (trapezius muscles). Hypoglossal (XII) nerve function is tested by having the patient protrude the tongue forward and to either side.

Motor Examination

The motor examination is designed to detect muscle weakness in a pattern which localizes the level of involvement (central nervous system, spinal cord, peripheral nerve, or muscle). The motor examination begins proximally and goes distally starting with neck flexion, extension, and rotation, then shoulder abduction, adduction, internal and external rotation then shoulder shrugging. The elbow is tested for flexion, extension, pronation, and supination. Flexion and extension of the wrist is followed by finger flexion and extension, then spreading of the fingers. In the lower extremities, hip flexion, extension, abduction and adduction are tested. Knee flexion and extension, ankle dorsi-flexion, plantar-flexion, then toe flexion and extension are tested. Motor strength is graded according to a 0-5 point scale, 0) being no movement, 1) being a flicker, 2) being movement of the muscle with gravity removed, 3) movement overcoming gravity, but not against resistance, 4) being able to move against resistance, and 5) being normal strength (Appendix C). Tone of the muscle should be noted for stiffness, rigidity, cogwheeling and the presence of action or resting tremor.

Sensory Testing

The sensory system is divided into fine sensation (carried in the posterior column of the spinal cord) or course sensation (carried in the spinothalamic tract). The fine sensation includes vibration, proprioception, and two point discrimination. Cortical sensation, processed from signals from the fine sensory system, can be tested by having the patient identify numbers written on the palms and soles (graphesthesia), or identifying objects placed in the palm, such as coins (stereognosis). Double simultaneous stimulation, tested by applying stimuli on one side, the other, or together simultaneously, is another test of cortical sensory function. Cortical sensation should not be tested unless fine sensation is intact and CNS DCS is suspected. Crude sensory function, carried in the spinothalamic tracts, is tested by light touch, temperature, and pin prick. Sensory deficits should be recorded on a sensory chart, with type sensation, such as pinprick, and an estimation by the patient in percent of normal sensation. The sensory exam, like the motor exam starts at the head and progresses down the trunk and extremities proximal to distal. Sensory deficits may be outlined by marking the patient's skin with ink, noting percent and type of sensory deficit as well as time and date. This is particularly useful if the examination is done enroute, or in the recompression chamber and a sensory chart is not available.

Reflex Testing

Reflex testing is divided into muscle stretch or deep tendon reflexes, frontal release reflexes, and cutaneous reflexes. Frontal lobe reflexes include the glabellar sign, elicited by tapping on the forehead and observing whether the eyes continually blink, and the root or snout reflex which is tested by having the patient look straight ahead and tapping on or above the lips, or scratching the side of the mouth and looking for a rooting contraction of the mouth. The palmomental sign is elicited by scratching the palm and observing for twitching of the mentalis muscle, just underneath the lower lip. The positive Wartenberg reflex is elicited by having the patient very gently flex the fingers against resistance and observing the thumb crossing over into the palm of the hand.

Reflex assessment of the upper extremities should include at least the biceps tendon and triceps tendon reflexes. Other reflexes that can be tested are the superficial radial (brachioradialis) elicited by tapping over the radial aspect of the forearm and the deltoid and pectoral reflexes, tested by tapping over the deltoid and pectoralis muscles. The finger flexion reflexes seen with normal brisk reflexes, include Hoffman and Tromner signs. The Hoffman reflex is triggered by taking the middle finger and flicking away from the palm and observing a pincher movement between the thumb and index finger; the Tromner sign is elicited by elevating the middle finger from the rest of the hand and flicking it toward the palm again looking for the pincher movement between thumb and index finger. These two reflexes are not necessarily a sign of pathology but rather a sign of a brisk muscle stretch reflexes. Asymmetry may be significant.

Reflexes in the lower extremity include the quadriceps reflex (knee jerk) and the gastrocnemius reflex (ankle jerk). In addition, reflexes of the hamstring muscles (biceps femoris) can also be tested. In the lower extremity, the plantar response, commonly called the Babinski sign, should also be tested. This extensor plantar reflex or positive Babinski sign, refers to the initial dorsiflexion of the great toe upward and spreading of the other toes and is indicative of corticospinal tract dysfunction. This is elicited by a gentle stimulus applied to the lateral aspect of the sole in a fashion starting over the heel and extending upwards to the base of the little toe. This can also be applied to the side of the foot in a similar manner, which is called Chaddock's sign. Other reflexes similar to the Babinski sign can be tested by laterally abducting the little toe in a brisk manner and allowing it to slap back against the foot again looking for dorsiflexion of the great toe, or flicking the third or fourth toe down in a rapid manner, again looking for the great toe dorsiflexion which is abnormal (positive sign).

Cutaneous abdominal reflexes may detect subtle spinal cord dysfunction. Superficial abdominals are tested by scratching from the margins toward the umbilicus and observing a quivering motion of the abdominal muscles. The deep abdominal reflex is elicited by tapping over the anterior rectus abdominal muscle sheath and observing a contraction of the abdominal muscles. Asymmetry of the reflex or presence of deep without superficial reflex would be significant. Other superficial cutaneous reflexes are the cremasteric reflex (in males), tested by stroking the thigh and observing ascent of the testicles, the anal wink reflex (anus contraction to light pin prick), and the Bulbocavernosis reflex (contraction of the anal sphincter by stretching the penis). These reflexes are usually only tested if spinal cord DCS is suspected.

Cerebellar Station and Gait Testing

Cerebellar testing includes finger to nose, heal to shin, and rapid alternating movements as well as rebound (ability to hold extremity with changing loads). Gait testing combines cerebellar, motor, and sensory function. Normal gait is tested by having the patient walk up and down the hallway, and doing rapid turns. Stress gait is tested by having the patient walk on the outsides and insides of the feet, then duck walking. This may enhance the detection of reduced arm swing or hand posturing (subtle paresis). Tandem gait testing (axial cerebellar function) is performed having the patient walk heel to toe (like a tightrope walker with eyes open/then closed). Station is tested by having the patient stand with feet together (Romberg position) with the eyes opened or closed. If done without difficulty, test next in the tandem position, with one foot in front of the other and the eyes open and then closed (Tandem Romberg). Finally, test in the sharpened Romberg position with the one foot in front of the other, head tilted back toward the ceiling, eyes opened then closed. A healthy adult should be able to stand in the Sharpened Romberg position with eyes closed for 20-30 seconds on the third trial.

The neurological examination only represents one point in time. In a DCS patient with a rapidly evolving neurologic syndrome, the initial effort should be stabilization and emergent referral for hyperbaric recompression. One of the most

important features of the neurological examination is defining the course of DCS by reevaluation and reassessment.

Neurodiagnostic Tests

Several neurodiagnostic tests are available in the evaluation of DCS (Table 4). They are rarely indicated in the acute management prior to recompression, unless signs and symptoms have resolved or the diagnosis of DCS is in question. They may be useful in the evaluation following recompression when further recompression is considered, or when determination for fitness for duty or aeromedical disposition is required. Formal neuropyschologic tests are time consuming and require skill in administering and interpreting; however, they may detect neurocognitive deficits in an otherwise normal appearing individual (2, 15). Electrodiagnostic techniques, such as evoked potentials and electroencephalography, may be helpful in differentiating between spinal and cerebral pathology, and monitoring recovery following recompression therapy (21). Computerized tomography may not be as sensitive as Magnetic Resonance Imaging in detecting CNS lesions (10, 11, 18). Single Photon Emission Computed Tomography (SPECT) has been reported to be a sensitive test of cerebral perfusion deficits associated with Type II CNS DCS and cerebral arterial gas embolism (1).

Discussion

The early literature of DCS contained a variety of descriptive terms, such as bends, chokes, staggers, and niggles. The aeromedical literature also contains a wide spectrum of terminology (Table 5). Classification schemes, outlined in Table 6, have evolved to assist in diagnosis, prognosis, and treatment using clinical, pathological, or outcome criteria (6, 7, 9, 20). Currently, the U.S. Navy is using the Type I/Type II classification scheme, as outlined in Table 7 (14).

Table 5. Terminology of Bubble Related Disease Associated with Altitude

1907 - Aeropathy (Erdman)

1939 - Aeroembolism (Armstrong)

1939 - Decompression Sickness ? (Mathews)

1942 - Drückfallerkrankheit (Hornberger and Benzinger)

1950 - Dysbarism (Adler)

1955 - Hypobarogenic pneumatosis, aerebullosis, pompholyhemia (Hall)

1965 - Mechanicobaropathy (Andersen)

1966 - Aeroarthrosis (Fryer)

Table 6. Altitude Decompression Sickness Classification Schemes

Clinical Classification

1936 - (Garsaux) Mal des Altitudes Classification

1) Euphoric
2) Convulsive
3) Arthralgic
4) Syncopal

1944 - (Swann and Rosenthal)
1945 - (Motley, Chin, and Odell)

Bearable vs. Unbearable Bends

1950 - Adler

1) Bends
2) Chokes
3) Abdominal pain
4) Neurologic
5) Collapse

1960 (Golding)

TYPE I Decompression Sickness - Simple

TYPE II Decompression Sickness - Serious/complicated

Pathologic Classification Clinical

Experimental

Outcome classification

Selection tests

RAF direct selection test

WW II - Three exposures of two hours at 37,000 feet on alternate days

Post World War II - One hour at 37,000 feet

Table 6 (Continued)

Classification System

No symptoms - Class A/no restrictions

Mild/moderate symptoms - Class B/low altitude only

Severe symptoms - Class C/excluded from aviation

High Altitude Selection Test

1954 - USAF

28,000 feet/2 hours, repeated 2 to 30 days later

Flight performance capability

Wirjosemito (1989)

Class 1: Symptoms would not have interfered with flying duties

Class 2: Conscious, would have been able to land safely

Class 3: Conscious, would not have been able to land safely

Class 4: Unconscious/unresponsive

Table 7. U.S. Navy Decompression Sickness Classification System

Type I Decompression Sickness

1) Limb pain (musculoskeletal symptoms)

2) Skin bends (cutaneous symptoms)

3) Lymphatic bends (lymph node swelling and pain)

Type II DCS

1) Neurologic DCS

2) Pain (systemic)

3) Inner Ear DCS

4) Fatigue

5) Cardiopulmonary DCS

Type II Decompression Sickness

1. Neurological DCS

a) Peripheral nervous system

Patchy peripheral paresthesias (burning or tingling) dermatomal numbness or weakness, confined to one extremity

b) Spinal cord

Numbness, weakness, quadriplegia/paraplegia, or urinary dysfunction

c) Cerebral DCS

Disturbance of higher cortical function, including personality changes, confusion, or inappropriate behavior

Hemiplegia, hemisensory loss, incoordination, ataxia, tremor

Migraine-like, unilateral headache and visual scotoma

Table 7 (Continued)

2. Pain (Systemic)

Bilateral pain, truncal pain, radicular (dermatomal) pain, thoracic, abdominal pain, tingling or burning pain (paresthesias), pain that moves from one area to another, or hip pain

3. Inner Ear DCS - vertigo, dizziness, tinnitus, and hearing loss

4. Fatigue

5. Cardiopulmonary DCS

   a) Pulmonary DCS or "chokes" is manifested by

      1) Burning substernal chest pain, often aggravated by breathing

      2) Cough

      3) Shortness of breath (dyspnea)

   b) Cardiovascular collapse

Conclusions

A systematic neurologic evaluation scheme is essential in the diagnosis, management and treatment of DCS. A standardized format would also aid in the epidemiologic assessment of DCS. Classification of DCS should be standardized to provide optimal management and monitoring of this condition.

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Appendix A

Cognitive Capacity Screening Examination
Mini Mental Status Exam

Examiner Date

Instructions: Check items answered correctly. Write incorrect or unusual answers in space provided. If necessary, urge patient to complete task. Introduction to patient: "I would like to ask you a few questions. Some you will find very easy and others may be very hard. Just do your best."

Trial Date/Time: _____

1. What day of the week is this? _____

2. What month? _____

3. What day of month? _____

4. What year? _____

5. What place is this? _____

6. Repeat the numbers 8 7 2. _____

7. Say them backwards. _____

8. Repeat the numbers 6 3 7 1. _____

9. Remember these numbers 6 9 4.
Count 1 through 10 out loud, then
repeat the numbers (6 9 4)if help
needed use numbers 5 7 3. _____

10. Remember these numbers 8 1 4 3.
Count 1 through 10 out loud, then
repeat the numbers (8 1 4 3). _____

11. Beginning with Sunday, say the
days of the week backward. _____

12. 9 + 3 is _____

13. Add 6 (to the previous answer or "to 12." _____

14. Take away 5 ("from 18"). Repeat these words after me and remember them. I will ask for them later: HAT, CAR, TREE, TWENTY-SIX _____

15. The opposite of fast is slow. The opposite of up is _____

16. The opposite of large is _____

17. The opposite of hard is _____

18. An orange and a banana are both fruit. Red and blue are both _____

19. A penny and a dime are both _____

20. What were those words I asked you to remember? (HAT) _____

21. (CAR) _____

22. (TREE) _____

23. (TWENTY-SIX) _____

24. Take away 7 from 100, then take away 7 from what is left and keep going: 100-7 is (93) _____

25. Minus 7 (86) _____

26. Minus 7 (79) _____

27. Minus 7 (72) _____

28. Minus 7 (65) _____

29. Minus 7 (58) _____

30. Minus 7 (51) _____

TOTAL CORRECT (Maximum score 30) _____

Patient's occupation _____ Education _____ Age _____ Sex _____

Estimated intelligence (based on education, occupation, and history, not on test score): Below average, Average, Above average

Patient: Cooperative Uncooperative Depressed Lethargic Other ____

Temp _____ B.P. _____ HR _____ Hct _____ WBC _____

Clinical History: _____

Medical History: _____

Drugs/Medication: _____

Focal neurological signs: _____

Signature & Title Date

Patient Identification:

NAME:

SSN:

RANK:

DUTY STATION:

Appendix B

Neurologic Examination

1. General: Head Sinuses

   Spine ROM

   Extremities ROM Pain

   Cardiovascular

   Pulmonary

   Skin

2. Cranial nerves:

   Eyelid

   Visual Acuity

   Pupil Size in Light in Dark Light Acc Shape

   Right.....

   Left .....

   Extraocular Muscles Ductions

   Visual Fields (finger count)

   Fundoscopy Disc NFB Macula Vessels

   Trigeminal motor

   Facial Motor

   Corneal Reflex

   Stennutory Reflex

   Gag Reflex

Whispered Voice A.D. A.S.

Phonation

Tongue

Trapezius

Sternocleidomastoid

3. Motor

Muscle Status (tenderness)

Strength (drift)

Tone (mirroring)

Character

Tremor

Gegenhalten

4. Sensory

Coarse sensation

Light Touch (LT)

Pinprick (PP)

Temperature

Fine sensation

Vibration

Proprioception

Cortical sensation

Stereognosis

Graphesthesia

Double Simultaneous Stimulation

5. Cerebellum

   FTN

   HTS

   RAM

   Check/ Rebound

6. Gait

   Regular

   Stress (posturing)

   Tandem (instability)

   Station

   Standard Romberg Eyes Open/Closed (Stable)

   Tandem Romberg Eyes Open/Closed (Stable)

   Sharpened Romberg Eyes Open/Closed (Stable)

7. Cutaneous Reflexes

   Glabellar

   Snout

   Root

   Palmomental

   Jaw

   Abdominal

   Cremasteric

8. Muscle Stretch Reflexes R L REFLEXES

Pectorals.....

Deltoids..... + more than other side

Biceps ..... - less than other side

Brachioradialis..... 0 Absent

Triceps ..... 1 Present with reinforcement

Patellar..... 2 Present

Prepatellar ..... 3 Brisk/Transient Clonus

Adductor ..... 4 Increased/sustained clonus

Crossed Adductor.....

Biceps femoris (hamstring)..

Gastrocnemius (Achilles).... NL = Normal

Hoffman ..... NT = Not Tested

Tromner.....

Finger Flexion.....

Wartenberg.....

9. Plantar Responses R L

Babinski..... + = extensor (abnormal)

Chaddock ..... - = flexor (normal)

0 = mute

Signature and Title Date

Patient Identification:

NAME:

SSN:

RANK:

DUTY STATION:

Appendix C

Motor Evaluation

MOTOR FUNCTION

5 = NORMAL

4+ = SLIGHTLY LESS THAN NORMAL

4 = MODEST WEAKNESS

4- = MODERATE WEAKNESS (BETTER THAN JUST ANTI-GRAVITY)

3 = OVERCOMES GRAVITY (ANTI-GRAVITY)

2 = MOVES WITH GRAVITY REMOVED (PARALLEL TO GROUND)

1 = FLICKER OF MOVEMENT ONLY

0 = NO MOVEMENT

UPPER EXTREMITY EVALUATION

	RIGHT	LEFT
SHOULDER ELEVATORS	5 4+ 4 4- 3 2 1	5 4+ 4 4- 3 2 1
SHOULDER DEPRESSORS	5 4+ 4 4- 3 2 1	5 4+ 4 4- 3 2 1
SHOULDER ABDUCTORS	5 4+ 4 4- 3 2 1	5 4+ 4 4- 3 2 1
SHOULDER ADDUCTORS	5 4+ 4 4- 3 2 1	5 4+ 4 4- 3 2 1
EXTERNAL ROTATORS	5 4+ 4 4- 3 2 1	5 4+ 4 4- 3 2 1
INTERNAL ROTATORS	5 4+ 4 4- 3 2 1	5 4+ 4 4- 3 2 1
ELBOW FLEXOR (SUPINATED)	5 4+ 4 4- 3 2 1	5 4+ 4 4- 3 2 1
ELBOW FLEXOR (PRONATED)	5 4+ 4 4- 3 2 1	5 4+ 4 4- 3 2 1
ELBOW EXTENSORS	5 4+ 4 4- 3 2 1	5 4+ 4 4- 3 2 1

FOREARM PRONATORS	5 4+ 4 4- 3 2 1	5 4+ 4 4- 3 2 1
FOREARM SUPINATORS	5 4+ 4 4- 3 2 1	5 4+ 4 4- 3 2 1
WRIST EXTENSORS	5 4+ 4 4- 3 2 1	5 4+ 4 4- 3 2 1
WRIST FLEXORS (ULNAR)	5 4+ 4 4- 3 2 1	5 4+ 4 4- 3 2 1
WRIST FLEXORS (RADIAL)	5 4+ 4 4- 3 2 1	5 4+ 4 4- 3 2 1
FINGER FLEXORS (DISTAL)	5 4+ 4 4- 3 2 1	5 4+ 4 4- 3 2 1
FINGER FLEXORS (PROXIMAL)	5 4+ 4 4- 3 2 1	5 4+ 4 4- 3 2 1
FINGER EXTENSORS	5 4+ 4 4- 3 2 1	5 4+ 4 4- 3 2 1
THUMB FLEXOR	5 4+ 4 4- 3 2 1	5 4+ 4 4- 3 2 1
THUMB ABDUCTOR	5 4+ 4 4- 3 2 1	5 4+ 4 4- 3 2 1
THUMB OPPOSER	5 4+ 4 4- 3 2 1	5 4+ 4 4- 3 2 1
FINGER INTRINSICS	5 4+ 4 4- 3 2 1	5 4+ 4 4- 3 2 1

MOTOR FUNCTION

5 = NORMAL

4+ = SLIGHTLY LESS THAN NORMAL

4 = MODEST WEAKNESS

4- = MODERATE WEAKNESS (BETTER THAN JUST ANTI-GRAVITY)

3 = OVERCOMES GRAVITY (ANTI-GRAVITY)

2 = MOVES WITH GRAVITY REMOVED (PARALLEL TO GROUND)

1 = FLICKER OF MOVEMENT ONLY

0 = NO MOVEMENT

LOWER EXTREMITY EVALUATION

	RIGHT	LEFT
HIP FLEXORS	5 4+ 4 4- 3 2 1	5 4+ 4 4- 3 2 1
HIP EXTENSORS	5 4+ 4 4- 3 2 1	5 4+ 4 4- 3 2 1
HIP ADDUCTORS	5 4+ 4 4- 3 2 1	5 4+ 4 4- 3 2 1
HIP ABDUCTORS	5 4+ 4 4- 3 2 1	5 4+ 4 4- 3 2 1
KNEE FLEXORS	5 4+ 4 4- 3 2 1	5 4+ 4 4- 3 2 1
KNEE EXTENSORS	5 4+ 4 4- 3 2 1	5 4+ 4 4- 3 2 1
ANKLE DORSIFLEXORS	5 4+ 4 4- 3 2 1	5 4+ 4 4- 3 2 1
ANKLE PLANTAR FLEXORS	5 4+ 4 4- 3 2 1	5 4+ 4 4- 3 2 1
ANKLE INVERTORS	5 4+ 4 4- 3 2 1	5 4+ 4 4- 3 2 1
ANKLE EVERTORS	5 4+ 4 4- 3 2 1	5 4+ 4 4- 3 2 1
GREAT TOE EXTENSOR	5 4+ 4 4- 3 2 1	5 4+ 4 4- 3 2 1
TOE EXTENSORS	5 4+ 4 4- 3 2 1	5 4+ 4 4- 3 2 1
TOE FLEXORS	5 4+ 4 4- 3 2 1	5 4+ 4 4- 3 2 1
TOE INTRINSICS (SPREAD)	5 4+ 4 4- 3 2 1	5 4+ 4 4- 3 2 1

Signature: Date

Patient Identification:

NAME:

SSN:

RANK:

DUTY STATION:

Additional Comments by CDR Clark:

The problem with decompression sickness, as I see it, is an extremely dynamic process. It is not unlike, in its manifestations, an occupational variant of multiple sclerosis. That is, it is a condition or illness manifested by lesions in space and time. I have seen peoples' examinations literally change before my eyes, without recompression, just back at site level. It makes the evaluation extremely difficult because now you see it, now you do not; now it is somewhere, now it is not. In the altitude realm it has been my experience that generally symptoms are a little more subtle and they may be quite evasive. But if you do have someone in the altitude environment who has a substantial neurologic deficit, or cardiovascular collapse, loss of consciousness, get them in the chamber as fast as possible or arrange for transportation, even if their symptoms resolve. I really firmly believe that if you have a condition where you have a substantial deficit or loss of consciousness, even if the symptoms resolve at ground level or after returning to ground level, that you still recompress them. In decompression sickness, decompression illness, you often have multifocal lesions, and it is extremely difficult, even for trained neurologists who are familiar with the hyperbaric environment, to even localize it at as simple a level as the five categories listed. But, nonetheless, it is still important to document, as best you can, the symptom complex and the pathology. I am a fairly ardent believer that DCS is not to be treated as a diagnosis of exclusion. That is, if you have somebody following an ambient pressure reduction who has any symptoms whatsoever, you should do your best to thoroughly evaluate them and exclude all the other things, but treat DCS as the prime culprit until proven otherwise. Decompression sickness, as a neurologic disease or as a multisystem disease, is potentially fatal. It certainly has potential complications of residual neurologic deficits. But more importantly, it is extremely treatable. I think by not treating it and not treating our aviators and aircrew who may be suffering from even subtle or subclinical DCS, we may be doing them a disservice by allowing long-term progressive processes to evolve.

From the standpoint of the neurologic examination, it has been my experience that mental status testing is very helpful if it is done appropriately, because very often, even though somebody may have just presented with limb bends, they may have subtle changes in mental status testing. I will read a very quick case concerning a 23-year-old physiology technician on our standard 25,000 foot altitude chamber flight, just to illustrate that point. He developed a mild headache after the flight and then developed extreme fatigue that evening. The next morning while he was teaching lectures as a physiology technician, he was noticed to have difficulty maintaining his train of thought, nodding off on occasion. He was also noted to be quite inappropriate and jocular, compared to his normal mature self. He was urgently evaluated, was found to have substantial memory difficulty, calculation difficulty, right-sided hyperreflexia, poor tandem gait and balance. He was treated urgently on a Table 6 and had prompt resolution of symptoms in ten minutes at depth. From my perspective, mental status, motor sensory and reflex testing, as well as a very cursory gait and station, can be performed within approximately 10-15 minutes with a standardized protocol and can be quite helpful in identifying further areas of involvement. I cannot tell you how much

I have been impressed with very subtle findings in someone who is presenting purely with a pain only syndrome. It has been my experience that, when you really look carefully it may very well be that someone presenting with pain only may actually have substantial deficits and should be classified by the present system as Type II. Deficits resulting from an ambient pressure reduction can be subtle, transient, and quite elusive to detection. An interesting point to make is that symptoms and signs do not always correlate. That is, someone may have symptoms, headache, classic unilateral headache with throbbing, visual component to it, classic migraine headache with no other findings--clearly a sign of DCS. An important point to make here is that any evaluation and examination represents only a point in time. This condition is a disease that is separated by lesions in space and time, so bear in mind a normal examination right now does not insure that five minutes from now that person may not have a substantial deficit. A systematic evaluation is absolutely crucial in the diagnosis, management, treatment and epidemiologic assessment of this condition. There is certainly a need for a review of our classification system and a standardization.

DR. VANN: I think you are absolutely right about doing the neurological evaluation if you do not have some serious or life threatening condition. There is one potential problem that can confuse it if somebody has a, normally for them, preexisting neurological problem. If a good baseline neurological exam is possible, it occasionally would pick up a reflex that is abnormal.

LT CMDR CLARK: That is an excellent point. In the diving community most of the divers will undergo a rather extensive neurological examination by a diving medical officer and that is documented in their chart to hopefully pick up any of these subtle abnormalities. To do screening exams on all of our candidates would not be possible. Some people have proposed doing them on our physiology technicians and inside observers, and we are currently debating the merits of that. Unfortunately, as a neurologist, I have seen inter- and intra-observer variability, and that is a problem as well. There are some neurologists that can find abnormalities in a rock. They are what I call microneurologists who can nit-pick about things and find subtle focal deficits. One of the things I have found that is a useful criteria is resolution with recompression. I have seen that some of these things that I would have possibly attributed to subtleties that might have existed premorbidly, promptly resolve with recompression. I usually go in the chamber and evaluate everybody so I will have a baseline. If you see a finding that does not go away with the recompression, then you are a little bit better equipped to say that it probably was a preexisting condition. But if you see a prompt change, it can be taken as evidence of pathology. I think for some people who are under an increased risk, such as inside observers, particularly if you are doing some provocative profiles at high altitudes and long durations, that a screening evaluation is warranted.

DR. HAMILTON: Would you comment on doing this exam by nonphysicians?

LT CMDR CLARK: All of our diving med techs are trained to do it. The diving med techs in the Navy are the inside tenders in the hyperbaric therapeutic recompression chambers and all of them are trained to do neurologic evaluations. We have several standardized mental status tests, and they are trained to do the baseline tests. They are not as good as higher levels of trained observers; however, I will trust a senior diving med tech over a junior hyperbaric medical officer any day.

DR. PILMANIS: The University of Southern California's Catalina hyperbaric chamber operates with a volunteer chamber crew who come from every walk in life you can imagine. Most of them have no formal medical training at all. They are all trained to do a 15-minute neurological, and they frequently find neurological deficits in divers.

DR. FRANCIS: We train our divers in the Royal Navy to do this as well. Only two weeks ago after a training course, a group of divers was doing some diving and one of them came to the surface not feeling particularly well. Another diver detected on his colleague a visual field defect and the loss of hearing in one ear.

LT CMDR CLARK: Divers are attuned to these kinds of problems, especially as they gain some experience and see a lot of cases. I think the utility lies in their experience level. They pay attention to details. You can't ignore anything, and subtle neurologic findings are very easy to ignore. When florid presentations occur, you have little question about it. It is the subtle findings that are often difficult. For example, somebody with fatigue at altitude, I would consider that as a manifestation of serious DCS. Overwhelming fatigue, never had experienced such bad fatigue, fatigue unassociated with any other predisposing factors (like sleep deprivation or substantial exercise, or dehydration) I would consider such fatigue a manifestation of DCS; no question about it. I can support that with the fact that if you treat them, you notice a prompt resolution of symptoms. That is a very rewarding feeling, and I have seen it on quite a number of occasions.

COLONEL SHEFFIELD: Since we at this time do not have a better classification system than the universally accepted Type I and Type II, would you share with us how the Navy currently defines those two categories, because it is clearly different than some of the USAF definitions?

LT CMDR CLARK: The Navy system has evolved over time, and actually we in the air arm of things have taken this from our diving community.

DR. FRANCIS: It is interesting that the origins of this Type I, Type II classification actually date back to a paper by Golding et al. in 1960, resulting from the digging of the Dartford, the tunnel in London. They actually made it very, very simple. It has grown out of control since then.

COLONEL SHEFFIELD: What was the original definition?

DR. FRANCIS: Type I is mild and Type II is serious.

COLONEL SHEFFIELD: Regardless whether it is a joint pain or other symptoms?

DR. FRANCIS: They reckoned that neurological symptoms were serious and pain was mild.

LT CMDR CLARK: The Navy's version of the classification is substantially modified from the original article. We have three types of Type I: 1) Limb pain (and I will distinguish this type of pain versus Type II pain), 2) Skin bends, (two categories). There is the very subtle formication type sensation that is very common after exposure to a hyperbaric dry chamber operations where the nitrogen diffuses in the skin and

then on ascent back to the surface diffuses back out. If you put somebody's arm in a pool of water who's got these sensations you can see bubbles coming out through the skin. Second, the more serious type of skin bends, which is cutis marmorata. In my opinion, this is usually a sign of a more overwhelming systemic bubbling, yet it is classified as Type I. Once again it emphasizes the lack of utility in this particular classification scheme. Finally, 3) lymphatic bends, which is, I think, quite uncommon in the hypobaric high altitude environment. Type II we break down into five different categories: 1) neurological, 2) pain (systemic), 3) inner ear DCS, 4) fatique, 5) cardiopulmonary DCS. Notice we do have fatigue in here. Inner ear DCS, vertigo, hearing loss, we generally do not see in the high altitude environment. It is more common in saturation divers. We do see a fair amount of systemic pain, neurologic and cardiopulmonary. The neurologic classification we try to break down into one of three categories. 1. Peripheral nervous system: This would be the patchy purple paresthesia, the dermatomal burning sensation. You could also see weakness or numbness, but it is clearly localized to a nerve or nerve root. 2. Spinal cord: Usually something that you are going to see in a truncal pattern, bilateral sensory motor loss, quadriplegic or quadraparesis, paraparesis, bowel, bladder, and sexual dysfunction. 3. Cerebral DCS: This includes higher cortical dysfunction, visual field abnormalities, etc. Mental status changes are not at all uncommon. And we also put in there migraine headache, or the migraine headache-like syndrome. Systemic pain we classified as Type II. It is pain that is in a dermatomal pattern that is not confined to one joint space. Any pain that is multiple, and that includes pain that starts in one area and then migrates to another, i.e., you could still have one source of pain, but if it started in one area and then went to another, that would be considered multiple. Next is truncal, and truncal is everything in the trunk excluding the shoulders. It does include hip and axial skeletal pain. Fatigue, I think is a common manifestation in serious type DCS.

DR. BALLDIN: What is the mechanism behind fatigue, if you will differ that from the neurological symptoms?

LT CMDR CLARK: I classify it in the neurologic realm. It may be immuniologic, it could be cardiovascular. I do not know what the cause of it is. I am speaking from a clinician's standpoint, not a researcher's standpoint. I see it quite commonly. And that is another thing, you have to ask them about it. You have to ask them about their symptoms.

DR. BALLDIN: But you differentiate that from the neurologic symptoms?

LT CMDR CLARK: This is what would be classified under the proposed scheme as constitutional.

DR. NORFLEET: Is this the scheme that is taught at the diving medical officer's curriculum now?

LT CMDR CLARK: Right, and the Navy's flight surgeon program as well.

DR. VANN: I would like to ask about headache at altitude. I have generally just passed this as an oxygen reaction or not related to decompression sickness. It generally has gone away in our subjects. I notice that this has been classified on occasion by some people as a manifestation of decompression sickness. How do you deal with a headache?

LT CMDR CLARK: First, let me comment on migraine. Migraine is a very, very over prescribed term. It is a unilateral throbbing, a severe headache, on a scale of 10, it is at least a five over ten or worse, associated with nausea, vomiting, stomach awareness and classic visual scintillations, a kind of wavering, flickering, jagged whitish yellow lines in their peripheral vision, or what looks like heat on pavement, things in your vision look fuzzy. Somebody who develops symptoms like that at altitude, who has never had a history of migraine before, I would treat. Somebody who has had a history of migraine, we might consider excluding from high altitude duties because of the conflict in trying to distinguish those two conditions, or some people may feel they are more predisposed. Bear in mind though that some recent studies have shown that migraine headaches respond to hyperbaric oxygen. Once again, muddling the issue.

DR. VANN: What about a standard headache, not one of these terribly severe ones, in somebody who has been breathing oxygen for several hours at sea level before a flight?

LT CMDR CLARK: That is a judgment call. I'd do a fairly thorough exam on them. That is a tough call, it really is.

MR WALIGORA: I just want to add that some of the tests we do involve wearing an oxygen mask five or six hours in many cases. This can cause "hat spots" on the head.

FUTURE HIGH ALTITUDE OPERATIONS

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Introduction

Ascent to altitude is clearly a hazardous undertaking: indeed, the World Health Organization defines the whole purpose of aviation medicine as "The safe passage of an aircraft through a physiologically and physically hostile environment." Not surprisingly, therefore, no self-respecting text on the subject is without a list of the risks associated with that "hostile" milieu. Such a list usually includes: altitude, disorientation, visual impairment, and acceleration. The last is particularly in vogue at present and, from the other contenders' viewpoint, enjoys a disproportionately high profile and share of resources.

My own, expanded, version of the 'altitude' component of the list looks like this:

On Ascent	Reduction in total pressure Decompression Sickness
	Effects on gas-containing cavities
	Reduction in PO_2 Hypoxia
	Reduction in temperature Cold injury
On Descent	Increase in total pressure

Protection against these threats is what the design of aircraft life support systems is all about; and any clinico-physiological description of the requirements for a life support system will rightly include a consideration of each.

We have heard already of some of the shortcomings in the protection offered against decompression sickness (DCS) to current high altitude aircraft crews, and it is probable that, with the exception of modifications to pre-breathing profiles, little improvement will be forthcoming for this population. But what of DCS protection in future high altitude operations?

The Next Generation

The next generation of fighter aircraft, on both sides of the Atlantic, has been designed to fly routinely faster and higher, and at higher sustained accelerations, than ever before. Thus, it is no surprise to learn that specifications for the European Fighter Aircraft (EFA) and the Advanced Tactical Fighter (ATF) call for life support protection up to 60,000 feet. At that aircraft altitude, the cabin altitude in the ATF and the EFA will be about 22,500 feet. Great reliance will therefore be placed on the oxygen system for protection against hypoxia in the normal way. Similarly, it is the normal function of the oxygen system to provide support following rapid decompression: so pressure breathing for altitude protection (PBA) is specified. Pressure breathing for G protection (PBG) will also be provided. A final (but significant) complication is that the normal source of breathing gas will be a molecular sieve which is only able to provide a maximum of 94% oxygen. The performance of the life support system in all these areas is specified in great detail in the design documents. But nowhere, in any of the material I have seen or heard of, is DCS addressed specifically: nor have I or my colleagues heard it mentioned by name in design review or technical meetings.

Extensive studies have been undertaken in support of these programs (and specifically of oxygen system performance), and it is fair to say that DCS has not occurred during any of these controlled experiments. But the dwell time at peak altitudes has been short, and the normal pre-oxygenation precautions have been taken. It is not difficult, however, to imagine a scenario where an extended dwell time at high altitudes following decompression is necessary for operational reasons; and it is in this area that we may be placing our airmen at risk.

Finally, the National AeroSpace Plane (NASP) program clearly also has implications for high altitude operations. Provisional plans for one version of the life support system in the NASP include the use of open-looped full pressure suits of the S-1030/S1031 (SR71) type (i.e., 3.5 psi), using established pre-breathing protocols. The cabin environment will be helium at a pressure of 5.5 psi, and the same gas will be used for suit ventilation. Within the USAF, a new pressure suit effort would be advocated with beneficial effects on comfort, mobility, weight and bulk, and glove performance (dexterity and tactility). There would also be a reduced pre-breathing requirement.

Conclusion

In discussions about the life support systems for future high altitude operations, DCS has retained its now traditional back-seat role. Simply put, DCS is not regarded as a problem for such operations (as they apply to the EFA and ATF programs), and any risk of its occurrence is clearly accepted as an operational hazard, despite the fact that our knowledge of DCS at such extreme altitudes is almost non-existent. On the other hand, this very lack of knowledge makes it difficult to know quite what else could or should be done about it even if we were asked!

DR. PILMANIS: I would like to expand on one area of your presentation. DCS at very high altitudes (above 30,000 feet). Symptoms onset times decrease with increasing altitude. It has generally been assured that above 40,000 feet you have 5-10 minutes before DCS onset. However, since essentially no hard DCS data for these altitudes exists, the real onset times are unknown. Therefore, there may be significant DCS risk in the high altitude "get-me-down" scenarios for future aircraft even with very short exposure times.

DR. BALLDIN: I would like to comment about future research projects. There is need for better preoxygenation for space EVAs. We need to have some methods to decrease the times of preoxygenation. Another is the combination of stress factors, as for instance in decompression and acceleration. I agree that the cabin pressurization is not as big of a problem as is acceleration and spatial disorientation which you cannot avoid.

DR. PILMANIS: You made an interesting point yesterday, that positive pressure breathing decreased nitrogen elimination by 40%. Positive pressure breathing is inherent in high altitude exposures. However, the times are short.

COLONEL SHEFFIELD: Wonder if you could expand on the concept of using helium in the NASP?

DR. HAMILTON: Helium would be fine in a spacecraft because of its lower solubility, but it has a lot of other problems. It is probably going to be detrimental with regard to decompression sickness.

DR. WENZEL: You can communicate. The voice will be just a little bit higher tone, but it is not a problem.

DR. HAMILTON: There are plenty of other problems. I think it is going to be a disadvantage in the space scenario.

CMDR CLARK: In trying to shorten your EVA times for the space station, would it be helpful to breathe hyperbaric oxygen at 10 or 20 feet. Would that enhance your washout and shorten your one atmosphere denitrogenation times?

DR. WENZEL: No. The only advantage would be if there were bubbles still resident from a previous EVA, then you would have a better protection for the next EVA. But it would not be helpful for a first EVA, 100% oxygen is 100% oxygen, no matter what the pressure is.

DR. NORFLEET: In fact, hyperbaric oxygen may slow inert gas washout due to vasoconstriction.

DR. VANN: We did an experiment to test a hypothesis that with hyperbaric oxygen you could eliminate gas nuclei or vacuum phenomena. We exposed rats to 240 feet for two hours on air. In our previous experiments this gave us around an 80% risk of bends. The experimental procedure added 60 minutes of oxygen at 60 feet prior to the exposure. There was no difference.

COMMENT MADE DURING THE BREAK: If the Air Force were to adopt the Navy's way of evaluating decompression sickness, especially the emphasis on expanding the neurological exam, and continue the Air Force current policy on dealing with neurological problems from decompression sickness, we would become just like the RAF and the problem would totally disappear.

DR. PILMANIS: Colonel Sheffield,could you kick off a discussion on acceptable DCS risk?

COLONEL SHEFFIELD: It is a tough assignment to come up with a concept of what acceptable risk might be. We had a very good introduction yesterday about the need for a medical research group to be a recommending group only and the operators themselves to actually determine what the acceptable risk is.

Clearly the optimum level of DCS risk, should be zero. If we chose that as our acceptable risk level it would result in a severe limitation in our operations today. Air Training Command would not be able to fly their unpressurized aircraft to 25,000 feet. They would be restricted to 12,000-15,000 feet, or would have to initiate very lengthy prebreathing.

Our high altitude airdrop mission support operations, which now are deliberately depressurized above 25,000 feet, would have to cease flying at those high altitudes.

The denitrogenation period for our U-2 flights, which currently pressurize, at a cabin altitude of 29,000 feet, would have to be extended to about four hours.

Glider training at the Air Force Academy that currently goes up to 25,000 feet in unpressurized gliders would have to be restricted. Parascuba rescue operations that currently fly to home base or to another location after a dive, would have to be severely restricted in the altitude of flight.

Currently, if we lose aircraft pressurization at high altitude, we descend to 25,000 feet. That altitude would have to be reduced to below 15,000 feet or perhaps even lower.

Traditionally, our operators have not been willing to accept these kind of restrictions, and the reason we do business the way we do today is because there is a compromise between those who have recommended the altitude ceilings and the operators who choose to fly in the mode that they are doing now. Case in point, when the U-2 program was established, the recommended denitrogenation period was four hours. The operators would not accept a 4-hour denitrogenation period, but they

finally bought off on one hour. Not because of the value that was placed on bends risk assessment, but because they felt that that was all that they would be willing to tolerate.

No one has, at this time, that I know of, put a risk assessment on those operators. Perhaps that is our shortfall, and we perhaps should be looking at the risk assessment.

The final point is concerning our training program. Our current philosophy is that if a person is going to be exposed to a hazard at altitude, that individual should have been trained to be able to cope with that situation. Should we go to a zero bends operation, there would be severe restrictions on the type of training that could be done.

I would suggest that a zero bends situation would not be practical in terms of the operational aircraft, and therefore, we should be talking in terms greater than a zero bends risk.

COLONEL SHAFFSTALL: I would point out that in our current mode of operation there is or has been very little documented mission impact. So we are operating at or very close to zero impact as far as mission abort.

COLONEL SHEFFIELD: You mean the number of bends cases that resulted in mission abort have been so few that it is considered by the line as a non-problem?

COLONEL SHAFFSTALL: Yes, it is a non-problem. So, it depends on whether you look at it from the medical standpoint or the operational standpoint.

COLONEL SHEFFIELD: Furthermore, in those cases that have occurred, the success of treatment has been so good that medical assessment would indicate that it would also be a very low risk.

LT COL WORKMAN: Yesterday Dr. Wenzel pointed out that if you have no treatment capability available then you are forced into a zero risk acceptability level. However, when treatment capability is available, two factors are important in deciding acceptable risks. First, asymptomatic bends upon reaching ground level is very high. Second, the effectiveness of hyperbaric oxygen therapy is close to 100% effective.

DR. VANN: As a point of departure in our discussion of acceptable risk; why not start with the current DCS incidence. This seems to be fairly small, around a tenth of a percent. That seems to be manageable in terms of morbidity and sequelae.

DR. FRANCIS: Do you want to take that as a real rate or the rate that is currently reported?

DR. VANN: It is the best rate we can come up with from reports we have. However, as an alternative, we could start with the combined NASA/Air Force DCS database from the ongoing studies. These are well controlled studies and well described.

DR. PILMANIS: If we were to do that as a basetime, then the high altitude reconnaissance flights should be running an 80-90% risk level.

DR. VANN: In that case, let's look at the severity of the symptoms. Are you willing to accept a higher incidence of mild limb pain? NASA uses 6% as the acceptable risk for shuttle operations. For the space station, where, however, people are less accessible for medical treatment, 1% has been proposed.

MR WALIGORA: The 5% was performance impact and that is related to an estimate of mission performance. Thus, severity of symptoms was a factor in our limits.

DR. VANN: Another relevant factor is what the people are doing at altitude. There is a big difference between reading pocket books and doing an EVA simulated workload.

MR WALIGORA: I think we have not stressed that enough. The effect of exercise is definitely dramatic.

DR. VANN: I have attempted to indicate that these factors need to be considered. I am not saying one or another is dominant, but rather that all these factors are important in describing acceptable risks.

LT COL BISSON: When you are talking risk and you attach the term acceptable to it, you are now talking more of who is deciding that acceptable risk. The hypobaric community of researchers has to define what that risk is. But the operators are going to make a different value judgment of what risk is acceptable. The task of researchers is to define the risk in terms of ratios and rates, and determine the validity of their studies and their databases. But if you are looking at trying to define acceptable risk for the operators, it will not work. The operators are going to define the acceptable risk for themselves, based on what you have provided them.

DR. PILMANIS: You are saying the same thing Dr. Bagian said yesterday. The research community should provide the data; it is the operational management that will draw the line as to what is acceptable.

LT COL BISSON: Right. A lot of people will not fly airplanes because they feel it is too risky.

DR. BUTLER: I would suggest another item to put in the equation of acceptable risk. The point was made that if you have no mission impact you can look at it as a low incidence rate. However, if there is a residual neurological impact, that

person may no longer be available to fly future missions. That is a loss that needs to be taken into consideration, not just the success or failure of an individual mission, but the loss of a pilot in the future.

DR. BAGIAN: I think that factor fits well within the overall tenet that the data should be supplied to the operational commanders. A commander must decide not only to get the current mission completed, but also decide what resources will be available and conserved for future missions. That does not just mean tomorrow or next week, but also over the coming years. There should not be a turnover in pilots every three years because no one is fit to fly after three years of high altitude exposure. All of this goes into the database and the decision. Another factor is that the environment in which the decision was made may change.

Depending on the mission and the circumstances, what is an acceptable risk today may not be tomorrow. Obviously, there is the difference between the training environment and a combat scenario. Operators do not always have the luxury of consulting with specialists at some central repository of knowledge on what to do on a mission. They have to be provided with information ahead of time so that they can make an on-the-spot decision.

COLONEL SHERMAN: For those of you not in the military, I would like to emphasize the situation from the operator's point of view. It is basically understood in the high altitude reconnaissance situation that in a "wartime" scenario if they had a mission to fly, and I have heard many pilots say this, if they had to they would fly in a shirt-sleeve environment with only an oxygen mask at max altitude, because that is what they are getting paid to do. They know that if they lose cabin pressurization they would probably die. If 80% of them get a few joint pains, which apparently they should from your research data, they have elected to say that is just part of the ballgame and would decide to press on. On the other hand, if it gets serious enough, they report it.

From a mission point of view in the high altitude reconnaissance community, it appears we are not being affected much by decompression sickness. But we do not know the long-term problem. Are these pilots going to have problems 10-15 years down the road? Currently, of more concern to us is the high altitude radiation situation. They are more concerned with that than decompression sickness.

CAPT GARDETTO: Unlike the situations with fixed altitudes, such as the EVAs, the parachutists may have a choice of exposures. They can assess the other factors such as winds and weather and may not necessarily choose to go to 35,000 feet. They can vary their exposures. In addition, their acceptance of risk is very high. They expect people to break legs and otherwise to hurt themselves on these parachute drops. There are some situations such as remote operations or training scenarios in which no treatment capability is available. What these operators would like to see from us is answers to questions such as: what increase of risk am I going to have in jumping from 18,000 feet versus 25,000 feet? O.K., I am twice as likely to get bent going to 25,000 feet? The winds will have to be twice as bad at altitude for me to

make that judgment call. There is a lot of variability in these operations. Unfortunately, decompression sickness risk usually is not involved because we (the physiology community) are not giving them useful information.

DR. PILMANIS: That is exactly why we are attempting to develop a DCS risk assessment computer. This computer will be able to quickly respond to rapidly changing situations. The way to currently respond is to either make a best guess or, dig through the literature and pick out a study that might approximate the situation, or do a new study. This inaccurate, time-consuming, often expensive procedure repeats itself with each new situation. Thus, the objective is to produce a software package that will provide both real-time and predictive readout in percent DCS risk. For mission planning, some may accept 80% DCS risk, while other may not want to accept more than 10%. It depends on the circumstances.

LT COL DIXON: In summary, there are multiple levels of DCS risk that we can choose to deal with, depending on the kinds of operation that we are addressing. I would suggest, based on what we have seen this week, that flying operations have a lower risk than chamber operations because we go higher and with many more people in our chamber training operations than in flying operations. We have said that we should leave it to management to make the decision as to what is the acceptable risk. I think that our present operations from a training standpoint are very "safe." On the other hand, if we were to go to operations in which we were going to accept a higher risk, then I would want my chamber technicians and our aerospace physiologists trained more along the lines of med techs you would see in the Navy. Currently, we are not trained along those lines. We have evolved over the years more along the lines of life support individuals. We would have to change the way we are training.

COLONEL SHEFFIELD: I think we are focusing on two points in this discussion. First, we want to decriminalize DCS. Second, we have a reporting problem. There is such a wide difference in reporting that I am not sure that I could accept Lt Col Dixon's statement that we have a higher risk in chamber operations than we do operationally. The risk is perhaps the same since the environment is the same. However, we have a distinctively different reporting process such that we are able to collect more data from our controlled chamber environment than we can from the line operations.

CAPT GARDETTO: Because the exposure durations at altitude in chambers are five minutes, whereas most of our HALO exposures at altitude are longer than half an hour, the numbers may be greater in the training situation but the propensity for bends is much higher in the flying operations.

DR. PILMANIS: One of the DCS characteristics of altitude exposure is that you can get away with a lot by shortening your exposure. That fact has been extensively and successfully used.

DR. HAMILTON: If we are going to dicuss risk we have to include the consequences as part of the equation. The risk of getting decompression sickness may be the same, but apparently the consequences are not devastating. That is to say, the missions go on, the people get the pain in their knees but it does not compromise the mission. However, you have to consider what all the costs of getting decompression sickness are. Depending on how bad it is, the consequences vary a great deal. In some cases, the probability of getting DCS may be rather high, but the consequences may be minimal. On the other hand, there are some cases, as with Russian roulette, in which the consequences may be very serious.

MR WALIGORA: We have talked about limb bends as being something you can deal with easily. However, it may become more serious if it happens during EVA and the individual has to transit back to the spacecraft. I think there's a tendency to think that if you provide a certain level of protection all you are going to get is the mild one. I do not really think that is true.

DR. PILMANIS: The serious case will happen eventually. Is not the real concern in EVA that statistically you can say sooner or later there is going to be a "Type II"?

MR WALIGORA: Yes, but we are also concerned with the possibility that limb bends may severely impact the mission to the point where it is just as bad.

DR. FRANCIS: It may be that overt symptomatology is just one end of the spectrum of illness that is a consequence of repeated decompression. In the diving world, there is now concern about the long-term health effects of repeated decompression, the changes to the retina, psychological changes, the punch-drunk diver syndrome. And I wonder whether or not this is something which should be looked at as well. Are there long-term consequences that do not necessarily relate to acute episodes of decompression illness that are caused by repeated altitude decompressions?

DR. PILMANIS: To my knowledge nothing like that has been done in the aviation field.

LT CMDR CLARK: The AFIP fatalities could be looked at retrospectively for gross pathology. The problem is that much of the damage may be cellular and microscopic.

DR. BUTLER: This gets back to the question of acceptable risk. Do you lose the pilot in the end, maybe not on that particular mission, but does your population decrease if you have an acceptable risk of say 5% for acute problems? What are you doing to your general population of pilots over time?

MR WALIGORA: Dr. Francis, is there any testing, performance testing or cognitive testing, used in the diving world, that could be applied to the altitude community?

DR. FRANCIS: I should be able to tell you in about two years time because the Department of Energy in the UK has just commissioned a large study of commercial divers in the North Sea, to try to find out if there are any long-term health effects of diving. In two years we may be able to give you some answers. They are going to be looking at a huge spectrum of tests to be performed on these divers and compared with very closely matched nondiving controls.

LT COL BISSON: When discussing risk, there are definite problems with definition. Risk is generally defined as the probability of developing a given disease or experiencing some change in health over a specified period of time. It is dimensionless and risk always has some reference to time. When discussing the risk of DCS as a whole versus the risk of some subtle subclinical change, you need to define what the numerator is. I do not see that we have defined what the numerator is, and we even have problems with what the denominator is.

DR. NORFLEET: In helping to define the long-term risks, I think the exit interview along with the careful neurologic exam is an excellent idea.

LT CMDR CLARK: Risk assessment is a commonly used technique in a number of different industries. It is used in the nuclear industry. It is used in the civil aviation fleet, and in the manufacturing of aircraft, and a number of other situations. In classic risk assessment there are two components of it and both of them have been alluded to today. One is the probability component, and the second is the outcome component. Either of those without the other is meaningless.

Now there are a number of techniques that are used in these various risk assessment applications. In some of the medical areas, there is what they called decision analysis. It uses those two components, probability and outcome, as the variables applied to all the different scenarios. Part of this method is based on materiel that was obtained from the Nuclear Regulatory Agency on how they predict fault failures in their reactors. There is literature available on this technique. The heralding features of this technique are the probability of an occurrence and the outcome of an occurrence.

I applied this method to old data from Fryer's book "Subatmospheric DCS in Man." I chose a worst case scenario, data from long-term flights above 30,000 feet for four to six hours with no prebreathing. A general estimate of the probability is roughly seven deaths in a million flights. With today's procedures there is probably a tenfold increase in protection, and the probability would be less than one in a million. I then looked at the number of DCS cases, again with no denitrogenation. The probability is that for every 1,000 DCS cases you would expect to see one death. In every 20 DCS

cases one would be serious, involving neurologic dysfunction or loss of consciousness.

Next, I looked at the outcome side. I used data from an article by Wirjosemito in 1988 on about 140 Type II altitude DCS cases in the Air Force. If 20% of all AF DCS cases were Type II, then of those who had Type II DCS, approximately 30% would not have been able to land the plane. Obviously that is a flight critical issue.

So now we have a worst case scenario probability and worst case scenario from an outcome standpoint. Next, let me relate this to what I've done in other industries. I was on a panel specifically looking at risk assessment in the airline industry. They design their equipment to have a failure rate in flight critical components such as flight controls, from between one in a million to one in ten million. In other words, that is their acceptable risk in a flight critical component.

Human factors is a major component in aircraft accidents. They expect an acceptable risk of a flight critical human error factor of one in a thousand, and in a non-flight critical area of one in a hundred.

The point of all this is that there are techniques available from a number of different industries for doing risk assessment. The method is called the Monte Carlo Process. It actually allows you to computationally figure what your risk is, after you have inputted your best estimate probability and best estimate outcome.

I think clearly there is application for this type of technique in the DCS arena. If we look at our basic chamber incident rate of one in a thousand, which is well within the range of flight critical safety errors, and of that number, how many result in serious sequelae, we are talking about one in a million. That is certainly well within the acceptable range.

DR. PILMANIS: However, because of this lack of reporting issue, we really do not know whether that incidence number is correct or not.

LT CMDR CLARK: Right. But the numbers I gave are based on the worst case statistics, in the era prior to denitrogenation.

COLONEL SHAFFSTALL: In response to the lack of reporting issue, I think if you look at the consequence end of it, those that would have been unable to land the aircraft, I believe that in most cases the severe mishaps would have been reported.

COLONEL SHEFFIELD: If we want to improve the plight of our crew members, we must provide assessment of risk in order to convince the designers of new systems to improve the pressure suits, or the pressurization system, or the flight profiles. Without that assessment we will not be able to improve any of our future systems. We have not done that in the past. It is now time to start it. We have, today, a

DCS database. We need to use what we have and plan in the future to improve that database by improving our data collection methods.

DR. BAGIAN: In providing risk assessment, you have to operate with what database you have, with all its potential limitations. Providing that assessment is an educational process. Along with that process, you need to improve the source of your data, which might involve decriminalizing DCS. This would then give the users better assessment capability and allow them to make better operational decisions.

I think if you put it in that kind of context you provide a very honest appraisal to them of what is going on. Then maybe you will be able to pull them in to support you in trying to really learn what the truth is, not just what we happen to know.

LT COL DIXON: The way in which the USAF handles chamber reactions and reports them, has not changed for a good long while. The ways in which we put the terminology down on paper leaves a lot to be desired. Part of that happens to be the form itself. My suggestion is that we need to modify the Air Force Form 361.

MR GILBERT: It seems to me we have three separate items we are talking about here, and they seem to be getting muddled together. What risk do we feel is acceptable risk at the present time? How are we going to decriminalize or to reduce the professional impact of reporting DCS by an individual? And, how are we going to have them report it?

LT COL WOLF: I would like to emphasize some of the points that we have discussed. Decompression sickness is an occupational illness. Our first job is to minimize the environment that will precipitate that illness. In addition, we need to be able to handle the individuals who happen to, in spite of our preventive measures, come down with the illness. We have that aspect fairly well under control as far as treatment is concerned. The big problem we have is the fact that there is a perception that if you come down with decompression sickness two or maybe three times, you are kicked out of the career field. People get DCS but do not report it. We do not know what the long-term effects of DCS are. I think we are doing a disservice, from a medical standpoint, of perpetuating that philosophy. I think we should treat decompression sickness as an occupational illness, recognizing that it is going to occur at times. Treat it as such. After it is treated, if there are no residual problems let them go back to their job, just like any other occupational illness. Finally, continue to try to prevent subsequent recurrences. I think that will hopefully get more accurate estimates of how many people are actually having the problem. From the long-term standpoint, this will also help these people who may develop long-term sequelae. It may even prevent them from developing something that may be coming down the line, particularly if they are repeatedly getting bent and not treated.

DR. STEGMANN: If you ask chamber techs off the record if they have ever bent, I would say 80% of them have and they have not reported it. This can be said for both research and training chamber operations.

DR. BUTLER: Is career impact the only cause of this?

COLONEL SHAFFSTALL: No, Dr. Pilmanis showed a list earlier that had a number of other reasons. Some people just do not want to mess with it at the time.

DR. PILMANIS: There are other reasons than career impact. But career impact is probably the primary stumbling block.

COLONEL SHAFFSTALL: It may just be the most convenient one. If you eliminate career impact they may just come up with another reason to replace it. Career impact is macho so they use that one first.

DR. PILMANIS: Col Sheffield, you said yesterday that in reality there was an extremely small number of people that have actually been taken out of the career field. If so, why then is there this fear of career impact?

COLONEL SHEFFIELD: We have had one who has been removed within the last year for migraine. I am not sure that it was related to the chamber flight or not. We have one that is in the process now of altitude restrictions. Then going back, during my whole career I remember maybe one or two that have been removed from the career field.

LT COL RUSSELL: I know of only two cases in which chamber techs have reported DCS and been removed from the career field. One was a possible CNS hit. He was removed automatically and immediately without recourse. The second case reported two DCS incidents. The first one was because of a diving incident that he related to the flight doc. The flight doc removed him from the career field because of multiple hits.

DR. BAGIAN: It is more than just how many have lost their career. It is knowing what will happen to an individual during the process. I have known pilots that have gone through the medical evaluation and it is no fun, even if you beat the rap. It is like being indicted for murder and you finally get off, but who wants to go through that? That might sound ridiculous but it is not the exam itself that is a problem. It is that your whole career is screwed up for a long period of time, you do not know where you are going. Nobody that I know would allow that to happen to themselves.

LT COL PARMET: I come out of the operational field, and I can confirm that there are a lot of unreported DCS incidents out there. It is partly because so many of the events are subjective, and they do not volunteer to be run through a meat grinder. People have some degree of self preservation. If they do not, they are extremely naive or fools. Most of our aviators are neither. ATC gets some reported, and we probably have three chamber reports for every field report, and I think those are both seriously under reported. I know our USAF DCS database has bad, bad problems. It is all we have, but it is not very good. I personally know of three neurologic cases in flying

class 3's who were medically retired because of DCS. However, they are apparently not in the database.

We obviously cannot accept zero risk. So we need to better establish what the real risk is. We need to decriminalize DCS. A few years ago a military person who developed gonorrhea received a court martial. Our rate of gonorrhea dropped to an extraordinarily low level. We do not do that anymore, and at some Air Force bases our current reported rate of gonorrhea is very high. But, at least we can deal with it medically now.

Unreported DCS is definitely out there. How much of the iceberg are we seeing? I do not know. However, as an operator, am I real, real concerned about decompression sickness? I am not concerned in the Air Force. We are not losing airplanes, and we are not losing very many missions to DCS. If the high altitude reconnaissance group were to start losing 10% of their missions due to DCS, I assure you they would start accepting four hours of prebreathing. However, since they do not appear to be losing a significant number, they will continue with one hour. The operator is going to determine that.

DR. VANN: Three things stand out in describing decompression sickness: it is fatal, it is preventable, and it is treatable. We know it is not a big problem. But we know it can be. We know it can be fatal. So we are trying to determine the outside of the envelope.

We are defining what the extent of the problem is, what are the pressure and time boundaries. That is what we have to find out in order to be able to define the outside of the envelope. Then we have to make value judgments. We can use all the statistics and the quantitative techniques that are available but, in addition, have to consider the effects on an individual's career, we have to consider the effects on the NASA program if somebody in the space station gets DCS on national television.

It all boils down to improving our ability to make these decisions by knowing what the outside of the envelope is. DCS can be a problem. It can be fatal. But it is preventable and it is treatable. It is all of those things, and all of those need to be recognized, and they are not incompatible either.

COLONEL SHEFFIELD: I had an occasion a couple of years ago to get a call from a group who wanted to do a research project. They were going to make a couple of excursions up to 60,000 plus feet to test a piece of equipment. The plan was to make one flight for an individual on the given day and then a week later to come back and do it again. However, they were only able to have the airplane one day, so they wanted to make three flights on this excursion for that same individual on the same day. How I wish I would have been able to take a "Formula" and show them what the risk assessment would be for that kind of profile. Such a model was not available so we had to tell them the best thing we knew, which was chances are you are going to bend them pretty badly with that kind of profile.

We need to have a method which we are able to determine what the risk assessment is for a variety of profiles. I hope that the need for such a method is one of the things that will come out of this meeting.

COLONEL SHERMAN: To summarize the situation in the high altitude reconnaissance area, I do not think there is a DCS problem affecting missions. However, I definitely think there is a need to enlighten our aircrews by enhancing our training with the assumption that probably 80% of them are going to get repeated minor problems. I also feel very, very responsible for providing them with available information that may affect their long-term health. There is a lot of resentment, and probably out and out anger, with the aircrews on the radiation issue. They feel that the medical people had the responsibility for finding out the facts and warning the aircrews. The same thing is true with decompression sickness. If there are long-term effects from DCS, then they should be educated about them. This, in turn, may help in the reporting and decriminalizing of DCS. However, I do not see an operational effect currently. I do not see missions being canceled. I do not think our pilots are so distracted by pain at altitude that they are not doing their jobs correctly.

COLONEL SHEFFIELD: Colonel Sherman, as new systems are designed, would you not be interested in reducing DCS risk in the new design?

COLONEL SHERMAN: Good point. When the earlier U-2 was redesigned and rebuilt by Lockheed, if DCS had been documented as a problem, at that time they would not have redesigned it so the cabin pressure would have been changed from 29,5000 ft to say 25,000 ft. But at that time DCS was a non-problem so why spend the money, why add the weight, why lower the performance of the aircraft by increasing the cabin pressure? If at that time the DCS incidence had been accurately reported we may not have even had our current problem in the U-2. That is a very good point.

With respect to the potential for long-term injury from DCS, a tremendous opportunity for research exists with the Blackbird community. I suggest that these 55 and 65-year-old men who were flying the U-2 in the early stages of the program could be studied. If long-term effects could be documented, even though DCS was not being reported, even though they did not cancel missions, then we would need to redesign the airplane. The ops people would put pressure on the designer.

COLONEL STORK: If we do not educate the users better, then they do not know what the risk is. They are the ones who drive the design of the future systems, the future life support equipment. In the equipment development business, we need requirements, which translate into funding, before we build new systems. It is the operators who generate those requirements and generate that need. If we cannot define the problem for them, convince them that there is a risk, then the circle stops there. Education is a critical portion of this cycle.

DR. FRANCIS: I think the point here is that DCS is an occupational illness, and it is incumbent upon us to understand the condition and to make sure its incidence

is as low as reasonably practical. Very little is known about the pathology of altitude DCS. This is one of the problems, I think. In humans, fortunately, it is very rare, it is very rarely fatal. So there is not very much material around to study. The stuff that has been studied in the past has not been subjected to modern techniques. Altitude decompression sickness in the human, when it is fatal, is complicated by shock, the return to one atmosphere, and any other therapeutic interventions. Normally, it takes a long time for the person to die and there may be a considerable delay before the tissue is examined. So in fact the quality of our understanding of the pathology of the disease in humans is very, very poor. And we have no good animal model.

DR. PILMANIS: In the hyperbaric field, I think it is fair to say that there is solid evidence of long-term pathology from decompression sickness. Since it is the same disease, but we have no information in the hypobaric environment, some people argue that we should assure altitude DCS results in long-term effects. Any comments?

DR. FRANCIS: We heard from Dr. Lambertsen the first day how dangerous it is to extrapolate from the hyperbaric to the hypobaric field. I think there's a very good argument for understanding hypobaric decompression sickness better in itself.

DR. BUTLER: As Colonel Sherman mentioned, there are probably good study populations that can be utilized to try to get a handle on whether there are long-term complications, and research on them should be encouraged.

In addition, studies on long-term effects from DCS in animals should be encouraged. In ongoing human DCS research, better neurological exams and follow-up should be done.

LT CMDR CLARK: If you look at residual sequelae after initial treatment, Weien's results show 1.52%, Davis' show 2.06%, Wirjosemito's show 2% plus, and the Navy studies show 4% residual sequelae. These sequelae are not significant, they range from persistent numbness to chronic pain syndrome to mild hemiparesis. But they could be classified as permanent or chronic sequelae of the altitude. They are basically treatment failures. Generally, if you have residual sequelae, you are retreated until your sequelae are resolved or stabilized.

DR. PILMANIS: There is a belief that if you follow these patients for six months that an awful lot of those sequelae will disappear.

LT CMDR CLARK: Probably.

DR. FRANCIS: Your incidence rate of sequelae will depend entirely on how hard you look. In Gorman's study, for example, in Australia, they did an elaborate battery of tests on hyperbaric decompression sickness cases. They showed that there was a residual illness in 70% of these people one month after recompression. That is because they looked really hard for it.

COLONEL SHAFFSTALL: I would point out that if there was any serious long-term effects we would have probably seen it appearing on some of us old guys who have been in the chamber for 20 plus years.

DR. FRANCIS: Has anyone looked?

LT COL RUSSELL: I think everybody agrees that certain forms of DCS with residual effects we cannot treat with impunity. They just must be grounded. But, as soon as you do that to one person, the entire community is going to know.

The problem is in the reporting. In recent years, there has been some loosening up of the waiver requirements. There is more evaluation on a case by case basis. However, no matter how much we talk about the kinder, gentler flight surgeon, there will be some who will not sign off. Simple bends, is often ignored because it usually resolves on its own and it may not even be recognized. Serious decompression sickness on the other hand is taken very seriously. With this separation, we can put the impunity on the simple bends situation. But then we must unfortunately live with the problems of grounding in the more serious case.

DR. BAGIAN: So then your operators know which ones not to report.

DR. PILMANIS: That is exactly what happens. They know they are going to be treated the same whether they have a Type II or Type I, so they report a Type I on the record.

DR. HAMILTON: But they get examined. You do not put them in the chamber without examining them.

DR. PILMANIS: A lot of these things are very subjective.

DR. FRANCIS: If you stick to describing the disease, as opposed to some arbitrary dichotomy between Type I and Type II, then you will avoid the problem entirely.

MAJOR WHITE: Short of telemetry of total body function, you cannot know for sure what they have because there is so much subjectiveness with DCS. The flying environment is different from sitting in a chamber. DCS is just another one of those physiological effects that is accepted.

I think we would be surprised if we surveyed the people sitting in this room. We would get DCS results from one extreme to the other. There would be individuals who have never experienced any DCS complaint. Others may have had ten hits and consider that part of the job.

Because of this, I think gathering more data with a survey is going to be interesting, and somewhat helpful, but it is going to only go this way. It is not going to draw us a straight line and say we have got to avoid this situation. That is not going to happen.

COLONEL SHEFFIELD: The lack of reporting is not necessarily fear of the flight doc or career threatening. Our peers consider us wimps if we go around telling every time we have got a hurting ankle or elbow.

DR. PILMANIS: Can we compare a DCS with what happened with GLOC?

COLONEL SHAFFSTALL: We killed people with GLOC.

DR. PILMANIS: Yes, so there is an order of magnitude difference. However, as far as the impunity aspect, is it successful, is it working?

MAJOR WHITE: I think that survey really changed how we look at GLOC. I think it was a super survey, and I think such a survey is appropriate for decompression sickness too. However, I do not think it is going to make a big difference to our pilots. When our pilots are looking over their shoulders worried about somebody fixing to shoot them in the rear end, they are not going to be checking their right elbow for pain.

LT COL PARMET: GLOC was harder to hide, and we had it on video. There is no way to document DCS, short of total body monitoring, unless we can get them to give us the information without risk to their career and without a lot of paperwork. It is a whole lot of trouble for the pilots to fill out forms.

DR. HAMILTON: Do we have any suggestions on how to get started with decriminalizing DCS.

DR. PILMANIS: My first suggestion is to hit the problem at the ground level through education. My second suggestion is to use the proceedings of this workshop in the educational process.

COLONEL SHEFFIELD: As I indicated earlier, at HQ it is a real soul search on the part of the physicians who make those DCS decisions. They do it as a group, as a panel. They are torn between the willingness to put that crew member back into the cockpit with a chance of losing an airplane and the airplane full of people, versus what is best for the crew member's career. They walk a tightrope every time they make that decision. The only way to change that situation is to change the view of our docs on whether or not it is a risk to the aircraft.

DR. PILMANIS: Considering that most of the altitude DCS cases are altitude chamber operations, not aircraft, are these cases handled differently? Chamber cases are different because there is nobody else at risk except that individual person.

COLONEL SHEFFIELD: In the recent case I mentioned earlier of a physiologist who had something like 3 cases of bends in only 12 hours of chamber exposure, the feeling was that it was an undue risk to the individual to allow that person to go back into the chamber.

DR. PILMANIS: So, in that case the decision was based on risk to the individual rather than risk to other occupants of an aircraft.

DR. HAMILTON: As with GLOC, you have two things to consider. There is experiencing GLOC, and there is having a low G tolerance. Similarly with DCS. Experiencing it should not be prejudicial. Having damage from it, or having a low tolerance for it, is something else that should be looked at on a case-by-case basis.

COLONEL SHAFFSTALL: I would point out that the investment in the individual also is taken into account. There is a huge investment in an aircrew. The government does not have much money invested in physiologists or chamber technicians.

DR. PILMANIS: The least investment is in chamber technicians, and they are at the highest risk since they go to altitude the most. They also have perhaps the largest risk of long-term effects, if such effects exist.

COMMANDER BASON: There are examples such as the aircraft mishaps boards that can be used for patterns in decriminalizing DCS. The accident information that is gathered for a class A mishap is privileged, to allow the aviator to talk without impunity.

COLONEL SHEFFIELD: In neurological DCS cases I see our Air Staff panel agonizing over the question about whether they are willing to put that person back at risk again by allowing them to continue to go in the environment. Can this group pass on to our docs information that will permit them to clear such DCS cases to return to duty without penalty.

DR. PILMANIS: Turn that around. Is there any evidence to suggest that a neurological hit from altitude exposure will be followed by another one resulting in a higher probability of being?

LT COL PARMET: The doctrine that we in the Air Force operate under is that for any medical condition, if there is no greater increase of risk to that individual's personal health, or to the completion of the mission, or to flying safety, then we let that individual go fly. On the other hand, if we can't determine what the risk is, then the tendency is to try to and protect the individual and not put them at risk. If the gospel has always been one CNS hit means that another one will inevitably come, what other decision can we make? Information is needed that will prove or not prove that you are going to be at greater risk or not be at greater risk of a second occurrence with possible permanent injury or death or loss of aircraft.

DR. PILMANIS: Colonel Workman, is it correct that the 1989 regulation change regarding DCS waivers came as a result of that EDU study done by your division?

LT COL WORKMAN: The change in the regulation did result from a briefing that was given to the Air Staff. The results of the study showed that there was no correlation to indicate that Type II DCS recurs in an individual. There were Type II diving decompression sickness cases, as defined by the Navy, I think there were about 3,000 cases. Unfortunately the study has yet to be published.

DR. BAGIAN: If you look at the disparity in DCS reporting rates, between operations and the laboratory, even with relaxation of the waiver rules it is obvious we are not getting anywhere near full reporting. Overall we are still looking at the tip of the iceberg. Obviously, paralysis or death gets reported. But short of that, we need to do more to decriminalize DCS. There will always be a mechanism by which the flight surgeon can recommend a person for evaluation or take them off status. That is within their prerogative. If you really want to deal with the DCS reporting problem, I think the waiver process needs to be further liberalized. Otherwise, you are still going to have a lot of people who will be reticent to report because even if they are returned to status, why should they subject themselves to that process.

DR. STEGMANN: I understand that if inside observers report three Type I hits spread out over two years, they are grounded. That has always been the answer in training. I think we have an education problem. The inside observers I have worked with believe that any three hits, and they do not even specify a time frame, will result in grounding.

COLONEL SHEFFIELD: I do not think that would be the case with the physicians that are currently in the Surgeon General's Office who would be reviewing the case.

DR. STEGMANN: Would they still have to go for a waiver?

COLONEL SHEFFIELD: No, with a simple Type I bends case, the local flight doc can return them to status in 72 hours. Now a person who has had three cases of bends in the last year would certainly be looked at more carefully. On the other hand, the person who has had three cases of bends over 12 years would not be looked at.

LT COL WOLF: I would like to see the current classification of decompression scrubbed altogether. No Type I or Type II, just decompression sickness and describe the symptomatology. Then describe the resolution of the symptoms or the non-resolution of the symptoms. In the USAF, we have a tracking mechanism on at least the cases that are reported. If the symptoms do totally resolve, with or without treatment, they go back to work after 72 hours. If they have symptoms that recur, you continue treatment. If they continue to have problems and stabilize, you treat them like any other illness. They are evaluated and would probably be grounded.

COLONEL SHEFFIELD: The local flight surgeon on the local base has a lot of discretion as to how a case is going to be handled. In fact, that is part of the problem with our classification of Type I and Type II decompression sickness. Generally, flight docs do not, despite common opinion, try to ground people, but rather try to return them to duty. Since they try to return them to duty there's a greater tendency to call it Type I decompression sickness, even though there may be some neurologic involvement.

DR. PILMANIS: It is interesting that the Navy reports about 50% Type II DCS while the Air Force reports typically around 10%.

LT COL WOLF: If someone had decompression sickness with a neurological involvement, a serious one, they are not going to be returned to flying status unless they get a neurology consult anyway. If we just throw out the current classification and call it decompression sickness, look at the symptomatology, see if it resolves or not, and treat it accordingly.

DR. PILMANIS: On another topic, how do we use the Doppler data? There is disbelief on the operational side that we are dealing with anything real. We are convinced we are dealing with something real.

DR. WENZEL: The Doppler objective measurement. In the hyperbaric field, Doppler data have been used to produce safer tables, just by titrating dives to a lesser degree of bubbles. These are the Canadian tables. We have not yet done this kind of thing in the hypobaric exposures. Although, we see from the NASA/USAF data for EVA procedures, that the amount of bubbles is much higher than the amount of DCS. What could be done is to go to a lesser amount of bubbles, and perhaps we would also get a lower degree of DCS. That would inevitably mean safer, but longer decompression procedures, as we had in the diving scenario.

COLONEL SHERMAN: Things like that video tape that was showed at the beginning of this workshop, in the proper setting could be used to educate the operators. Show them exactly what is going on. Impress on them the fact that just because there are bubbles does not mean you are going to get the bends. This may be kind of a revelation to them.

DR. PILMANIS: Colonel Sheffield's training tape perhaps should be updated.

MR WALIGORA: When we have talked about the development of in-suit Dopplers, many people have the impression that we are talking about a monitoring system that we would use operationally. I certainly do not think that is in any way appropriate, and have certainly told everyone that, but I think that the best use of the Doppler is as a research tool to give you, an objective measure of what is going on. You would not want to use it operationally because it does not tell you anything. You anticipate the incidence of bubbles is going to be high.

I should add that the relationship between bubbles and symptoms is dependent on the severity of the exposure. If the exposure is severe enough, the relationship will be close.

DR. PILMANIS: I concur, Dopplers should not be used operationally. My first research project using Doppler in the diving field was in 1971. One of the objectives was whether or not it had operational relevance. The answer was a solid no, and it still is today. It is a research tool, and it should not be considered an operational tool.

DR. BUTLER: I do not think the jury is in on whether it would be useful as a diagnostic or a screening tool. It is an obvious desire whenever we work with new methods. It is attractive to think that you may be able to screen and hence prevent a negative outcome.

DR. PILMANIS: It should be remembered that the Doppler gives you a very small window into the body. It does not tell you the full story.

In summary, the Doppler should not be used operationally, but it is a valuable research tool.

COLONEL SHEFFIELD: If we had a 5-minute tape on Doppler, we could use it in our teaching to help educate our crew members.

DR. OLSON: I do not think it will be long before there will be improvements in the technology. I think bubble sizing is on the horizon. It is not a static thing.

DR. PILMANIS: Changing topics, is the development of a decompression computer, a worthwhile objective?

COLONEL SHEFFIELD: I think it would be very useful for our special missions. The HAAMS program, in particular could use it to help answer questions about DCS risk. In the reconnaissance program, we could use it to try to determine what our new pressure suit capability should be for example. Yes, I think that a decompression computer would be useful in guiding our operations.

COLONEL SHERMAN: I can give you an example. On a 10-hour mission, one hour or two hours into the flight, the plane loses pressurization and the pilot goes from a 29,000 ft to a 35,000 ft pressure suit. Should the pilot continue to fly at altitude for one, two, three, or four hours, or should he come down? What is the DCS risk?

COLONEL SHEFFIELD: Another example is with the rapid deployment of the fleet when you may not have denitrogenation time available. What would be the risk of that particular kind of scenario? So I think those are all very useful reasons to develop that decompression model.

COLONEL SHAFFSTALL: I think it would also be useful in research and development by projecting the level of DCS risk For example, if I am doing a hypoxia study, I still need to know the risk of DCS.

DR. PILMANIS: I can relate another good example. We recently received a call from Edwards AFB. They were planning to take a new aircraft up and shut the engine down five times in one flight. This meant cabin depressurization 5 times. They wanted to know what the DCS risk was under these circumstances. That is a non-standard flight profile, and the only thing we could give them was a best guess answer. If we had an operational decompression computer, we could have responded with reasonable accuracy in a matter of seconds.

That ends this workshop. Thank you all for coming.

*U.S. GOVERNMENT PRINTING OFFICE 1992-0-761-052/60042