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DECOMPRESSION THEORY

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ELECTE

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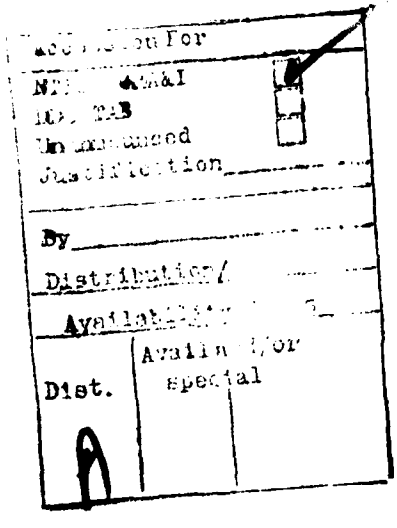

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INTRODUCTION

The initial letter that went out to Workshop participants outlined the background that led to the formulation of the workshop topic. It stated: "The median depth of diving in the U.S. Navy is only about 34 fsw. Because the great majority of fleet diving is only to shallow depths, the U.S. Navy has not yet really confronted the true magnitude of the decompression problem. There is every indication that if the U.S. Navy were to suddenly desire to start diving deeper, there would be a tremendous increase in the incidence of decompression sickness. The evidence to support this contention can be found in the fact that the U.S. Navy has never been able to conduct a "bends"-free deep saturation dive; the project to develop deep subsaturation decompression procedures for the MK 1 Deep Dive System was abandoned due to severe decompression sickness; the diver training program has its highest incidence of decompression sickness on its deep air narcosis demonstration chamber dive; and finally, the U.S. Navy Safety Center statistics show an inordinately high incidence of decompression sickness on dives deeper than 100 fsw."

Given this background, the chairman set as the Workshop objective the review and update of the assumptions incorporated in the Navy's neo-Haldanian decompression model. To accomplish this objective, the participants were asked to confine their discussions to empirically derived data that might impact on the 12 assumptions listed in the Workshop program.

The degree to which the Workshop participants complied with the Workshop outline varied greatly. Presentations ranged from those dealing specifically with the stated neo-Haldanian assumptions to those that ignored the stated objective. Despite the lack of continuity in the presentations, there were points of interest in most of the presentations. In an effort to give the reader the benefit of all the information presented, this report is divided into three sections.

The first section of the report presents a brief history of the development of the U.S. Navy's version of the Haldane model, along with a description of some of the diving environments in which the model has not worked. The second section deals with the 12 neo-Haldanian assumptions and presents the best current thinking with regard to their viability; it also serves to meet the objectives of the Workshop. The third section of the report contains the individual manuscripts presented by the Workshop attendees. This section provides amplifying remarks that can be used for interpretation of the comments made in the second section.

SECTION I

Historical Development of the U.S. Navy's Decompression Model and Its Operational Limits

HISTORY OF DECOMPRESSION THEORY

R. C. Bornmann

The organization of diving in the U.S. Navy at the turn of this century was very similar to that in the Royal Navy at the same time. The 1915 diving reforms of Warrant Gunner Stillson, USN, were considerably aided by the work of the Admiralty Deep Water Diving Committee of 1907 (8), as well as by Stillson's familiarity with foreign diving technology and his own inventiveness. All of the Workshop participants are familiar with John Scott Haldane and how he joined that Diving Committee. He had been asked previously to advise the Royal Navy on the condition of ventilation in ships, and it was a natural extension of this topic to consider how the question of ventilation related to CO_2 removal in diving helmets. His work on this problem permitted divers to go considerably deeper than previously thought possible. Because of the physical distress actually experienced by divers, it was believed that a "pressure barrier" existed that limited diving to 30 or 40 feet. Haldane showed that the problem was one of CO_2 buildup in the diver's helmet rather than pressure. With better ventilation, the CO_2 was removed and the so-called barrier disappeared. After Haldane had demonstrated that man could go deeper, he was faced with the need for effective decompression schedules for such dives. As a result of his work, stage decompression schedules were established, on a scientific basis, for the first time.

At that time, some very extensive, but shallow underwater construction had been underway for several years at the military base of Gibraltar. In his review of diving experiences, Haldane noted the complete absence of decompression sickness among the divers employed on the job in Gibraltar. He wrote, "There was abundant evidence that, when the excess of atmospheric pressure does not exceed about one-and-a-quarter atmospheres, there is a complete immunity from symptoms due to bubbles, however long the exposure may have been and however rapid the decompression. Hence it seemed to me probable [note the word *probable*] that it would be just as safe to diminish the pressure rapidly from 4 atmospheres to 2 or from 6 to 3 as from 2 atmospheres to 1. If this were the case [again note the conditional statement] a system of stage decompression would be possible and would enable the diver to get rid of excessive nitrogen through the lungs far more rapidly than if he came up at an even rate" (1). Haldane also added, "Whether the law holds good for pressures much exceeding 6 atmospheres is still doubtful as no experimental data exist" (1). "In spite of this characteristic caution," Sir Robert Davis said in 1951 (2), "later writers have mistakenly credited him with asserting that rapid decompression from any pressure 2N to N is safe." It is sad that the cautious Scot Haldane is still frequently cited as having said something that he did not say and that is well known now not to be true.

In 1914 Stillson had been asked to modernize U.S. Navy diving capabilities, then rather woeful. He fulfilled the assignment with dispatch and efficiency. He assembled a diving team, carried out diving trials, modernized diving equipment, wrote a diving report, published a diving manual, and established a diving school. The 1915 Stillson report (8) includes the Admiralty (Haldane) Diving Tables in the main text, and these tables are described again in the Medical Appendix written by Passed Assistant Surgeon G. R. W. French, USN. In the medical portion, Dr. French also refers extensively to the work of Haldane. In paragraph 10 of the main report is the statement, "References are also made throughout this report to the findings of the British Admiralty Deep Water Diving Committee and the works of the celebrated English scientist J. S. Haldane, M.D., F.R.S., and Leonard Hill, M.D., F.R.S., all of which are on file in the Office of Naval Intelligence." Remember that the year was 1915, before Information Exchange Projects and Memoranda of Understanding, before we had been allied with Britain in two World Wars. Also, in 1915 there was still some debate in the United States about whether to go to war on the side of Germany or on the side of England.

In the Haldane tables at depths greater than 10 fathoms, a thick black line is drawn below some schedule at each depth. This indicates a time limit that gets shorter as depth increases. The object of the limit is to prevent a time longer than 33 minutes being required to decompress the diver. The report states, "If the limit is greatly exceeded serious compressed air illness may develop in spite of the long decompression, because long decompressions can be so exhausting as to defeat their own end. But if the limit is only slightly exceeded there is not much danger, although bends which are painful though not dangerous to life may occur" (8). There is a note to the original Haldane air tables that states that repetitive diving is not to be done. The tables were not to be used for a second dive within four hours after a first dive. If, however, such a dive had to be made, the bottom times of both dives were added. The first half of the subsequent decompression was made in accordance with the schedule for the second dive alone, but the last half of the decompression was to be that required for a dive as long as the combined times of both dives.

The 1915 USN report published the Haldane tables for the United States Navy in only slightly edited form: the full range of schedules was given, but some of the intermediate ones were omitted, and the schedules below the black line were presented as a separate table, Table 2. Since French was so familiar with Haldane's published work, it is interesting to quote some sentences from his medical appendix (8): "While they are not guaranteed to protect from slight or moderate attacks of bends, a diver compressed according to them will be saved from any serious attack of caisson disease. . . . After complete saturation stage decompression becomes less efficient and it is questionable whether uniform or stage decompression has the greater advantage. With short exposures, however, a stage decompression is safe. . . . It is evident that if a diver saturates and desaturates at the same rate, decompression will be in proportion to compression and the length of exposure to high pressure." Sixty-three years ago, they were debating the same problems being considered here today.

In his book, Davis describes why Haldane stopped with schedules to 204 feet (2). Nothing deeper was attempted, he said, because the limit of what could be done using hand pumps had been reached. Three pumps were coupled up to each diver, each pump manned by six men who had to be relieved at five-minute intervals. Stillson, however, added a new technological feature to American diving when he made high-pressure, 2500-psi air flasks one of the alternate sources of diver breathing air. He got this idea from the Germans, but extended it considerably by putting the control valve (Stillson valve) at the diver's end of the supply hose rather than only at the high pressure air flask. This was the advance that made possible the successful salvage of the submarine F-4 in Hawaii in 1915, which required air dives as deep as 304 feet. This operation was a remarkable achievement, and it set a record that is not likely ever to be duplicated because helium-oxygen has so many advantages for diving to such depths. The decompression schedules were reported by Dr. French in 1916 (3).

In the USN Diving Manual of 1916 (one year after Stillson's Report), schedules for diving to 225 feet and 250 feet on air were added to the bottom of the original (Haldane) tables. It is also comforting to note that this Diving Manual states, "The cause of caisson disease has been explained. Its prevention consists of (1) limiting time of exposure to high pressure, (2) proper stage decompression, and (3) proper physical standards in the selection of divers."

For a few more years, Dr. French was associated with diving, including duty on the *USS Falcon* in 1921 for the salvage of the S-5. His article "Remarks on Deep Sea Diving" was published in the Naval Medical Bulletin in 1922 (4). He then went on to more routine naval assignments. Beginning in 1922, sick leaves appear on his service record from time to time, and he finally was invalided out of the Navy in 1937 with diabetes and chronic myocarditis. He came back on duty in the San Francisco area during the years 1940 to 1945, but his health declined, and he was hospitalized and then sent home. He died in 1955. Dr. French was a graduate of the 1908 class of the Medical School of the University of Pennsylvania; he was an exemplary precursor of those doing the distinguished diving work at that medical center in recent years.

The first Navy diving school was established at Newport, Rhode Island. However, it was broken up and its staff was sent to France to serve as diving teams during World War I. The Navy's diving capability stagnated and diminished thereafter, and had to be reconstituted to salvage the submarine S-51, lost in 1925, and the submarine S-4, lost in 1927. Captain Ernest King was the naval commander for those jobs (5).* A Navy diving program was reestablished. The Deep Sea Diving School and the Experimental Diving Unit came into being in the Washington Navy Yard. Helium research, which led to the development of the helium-oxygen deep sea diving tables, began. New air decompression tables were also formulated. It was during this period, too, that the diving programs of the U.S. Navy and the Royal Navy diverged and really became nationally distinct. The two diving navies seemed not to be talking to each other too much in those years before the Second World War. U.S. Navy practice was either to decompress the diver in the water or to use surface decompression (so-called decanting), although the diver did breathe pure oxygen in the chamber during surface decompression. In contrast, the British developed a submersible decompression chamber, so that the diver's ascent was not broken by any surface interval as he was brought on board the diving vessel after his dive. The British also utilized oxygen breathing. In the end the tables of the two Navies were so different that it was not obvious that they stemmed from the same Haldane source.

At the same time, the U.S. Navy at the Experimental Diving Unit and the Bureau of Ships was working on the submarine rescue chamber, the McCann bell. In 1939 the *USS Squalus* sank. All survivors were rescued with the McCann chamber, and the ship was salvaged by using the helium diving tables. This successful operation led to the local transfer of the Deep Sea Diving School and the Experimental Diving Unit to spacious new quarters in Building 214 in the Washington Navy Yard. The 1000-foot pressure chambers built there in 1941 served the Navy well for 30 years. The Experimental Diving Unit, of course, moved to the sophisticated modern facilities in Panama City, Florida, in 1975, and the Naval School, Diving and Salvage, will also move to Panama City in 1980.

The interested scholar is referred to Workman's chapter on the development of American decompression theory and practice (10) for a description of the alteration of mathematical limits or ratios for air diving, as developed in the work of Hawkins, Shilling, Yarborough, Van der Aue, Behnke, and others from 1935 to 1951. Results of these studies allowed repeated reductions of ratios believed previously to be safe, separation of surfacing ratios and those for deeper decompression stops, and consideration of longer half-time tissues. The introduction of scuba into military use in the 1950's resulted in recalculation of the air decompression tables in a new format, which provided for repetitive diving to depths of 190 feet. We are in danger today of forgetting that the exceptional and extreme exposure tables were investigated during the same period, but were never developed and tested sufficiently to be considered completely safe. The cautions that were published in the past in the Diving Manual with regard to the use of these tables have slowly lost their impact and have even begun to disappear from print.

The development of the helium-oxygen deep sea diving decompression schedules was truly a remarkable achievement for the Experimental Diving Unit and the Navy. First reported by Momsen and Wheland in 1939 (7), the schedules were revised once by Molumphy in 1950 (6) in order to decrease the maximum depth of oxygen breathing from 60 to 50 feet. Assumptions used in the calculation of these schedules included:

(1) The minimum oxygen permitted was 16% in actual use, but helium was calculated to be 86%, to

*Captain Ernest J. King commanded the Submarine Base at New London from 1923 to 1926 and was therefore placed in charge of the salvage force which raised the S-51. For this he received the Navy's Distinguished Service Medal. In 1927 he had completed training as a naval aviator and was in command of the *USS Wright*, but was detached in order to lead the salvage of the S-4. He received a second DSM for that. There are those who say that the recognition King received for these accomplishments helped to propel him to further success in his career, which led to selection as our wartime Chief of Naval Operations and the rank of Fleet Admiral (5).

allow for a further 2% reduction in oxygen in the recirculating system.

(2) All dive exposures were calculated using twice the bottom time of the schedule to allow for increased helium uptake during exercise.
(3) Half-time tissues were 5, 10, 20, 30, 40, 60, and 70 minutes.
(4) A ratio of 1.7 to 1 to the absolute pressure of the decompression stop depth was used (equivalent to a 2.15 to 1 ratio calculated in the Haldanian manner).
(5) The efficiency of decompression with pure oxygen was considered to be equivalent to 80% oxygen, 20% helium.
(6) Rate of ascent varied from 20 to 75 feet per minute.
(7) Repetitive diving was not allowed for a 12-hour period after surfacing from a previous dive.

The published helium tables went far past what had been tested or used in the U.S. Navy. Many schedules have since been deleted, principally because dives past 300 feet without a submersible decompression chamber are extremely hazardous and there are better dive systems for diving operations to these depths. Schedules for other depths are also probably not particularly efficient for bottom times much past 30 minutes, again especially in the deeper ranges. The tables are still in general use throughout the world, although with modifications in many cases.

In more recent years the Experimental Diving Unit has developed decompression tables for diving with semi-closed-circuit mixed gas scuba and for 30- and 60-minute surface support dives to 500 feet. These were developed for use in support of SEALAB III, originally planned as a 450-foot saturation dive. When the depth of SEALAB III was extended to 600 feet, it was decided that long surface support dives to the new depth were not practicable. The saturation decompression schedules and the saturation-excursion schedule are two more examples of recent pioneering developments. Just now the Experimental Diving Unit has tested a decompression mini-computer designed to be worn as personal equipment by the diver. The Experimental Diving Unit formulated and tested the computer algorithms for this purpose, and the project is a success. However, if one reads the Experimental Diving Unit report (9), it can be seen that the computer is limited by the same constraints as the tables themselves, and that the computer had to be restricted in its use for longer dives and deeper depths because it does not produce schedules that test out well.

In the last paragraph of Workman's chapter (10) are the following words, "Mathematical models of inert gas transport and bubble formation in use today and developed through years of experience in diving differ mainly in their interpretation of three basic concepts. These are: the nature of the rate-limiting process in inert gas transport, the character of the body tissue as this affects gas transport and the process of gas phase separation leading to formation of gas bubbles in body fluids and tissues and thus to decompression sickness." A list of unanswered questions concerning these concepts in diving, decompression calculation, and the avoidance of decompression sickness would include these: Where is the bubble in decompression sickness, and what is the relationship between that specific trigger and all the gas bubble changes in decompression? How does one measure and describe the influence on the diver and his decompression of such factors as the inert gas or gases in his breathing mixture, the oxygen, the carbon dioxide, contaminant gases, exercise or work, heat, cold, position, etc., etc., etc.? If one is going to write a mathematical model of decompression, how are all these things to be described? What specific effects in gas exchange or bubble formation are precipitated by stage decompression for any given combination of riser and tread (to use a carpenter's phrase) or by continuous ascent/decompression at any given rate? How does one describe all the effects of time on the body? Why does one see evident differences in pragmatic decompression calculations or experiences between saturation and subsaturation exposures? What is the real basis for the evident expansion of decompression limits with increasing base depth in saturation-excursion situations? What is the physical difference between the diver making a first dive and the same diver making a repetitive dive, and what is the biophysical description of accommodation to decompression, assuming that that phenomenon truly exists?

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CURRENT PROBLEMS WITH THE NEO-HALDANIAN MODEL

T. E. Berghage

I would like to begin the discussion on current problems with the neo-Haldanian model by presenting data from the U.S. Navy Safety Center. This data may help to clarify why we are here today.

First, I want to set forth some of the reasons for dealing with the present theory:

1) Inert gas combinations and various oxygen partial pressures probably can be used to make our present decompression procedures more efficient.
2) Decompression risk associated with deep saturation diving is unacceptably high. The U.S. Navy to date has never done a bends-free deep dive, i.e., deeper than 1000 feet. The Navy's deepest chamber dive has been at a pressure equivalent to 1600 feet of seawater (fsw); subsequently, five of the six subjects were stricken with decompression sickness.
3) The excursion tables in the 1970 edition of the U.S. Navy *Diving Manual* were calculated using the neo-Haldanian method. There were over 300 clean dives on these schedules before they were put into operation. The empirical evaluation of excursion limits that the Experimental Diving Unit has embarked upon indicated that the 1970 values were ultraconservative for excursions from deep saturation levels and that we can greatly extend the depths and durations of these excursions safely.
4) Subsaturation decompression schedules are presently unavailable for deep dives of short duration. At the Experimental Diving Unit, the U.S. Navy tried to develop a series of schedules for the Mark I deep dive system. They finally abandoned the project because of the high incidence of vestibular decompression sickness. At present, we still do not have schedules for use with the deep dive systems; we do have existing hardware that cannot be fully utilized because of our lack of understanding of the decompression process.

If we look at U.S. Navy diving, we find that 99% of the diving is done to 200 feet or less, so we really aren't gaining the experience that is needed for evaluating decompression schedules at deeper depths (Fig. 1).

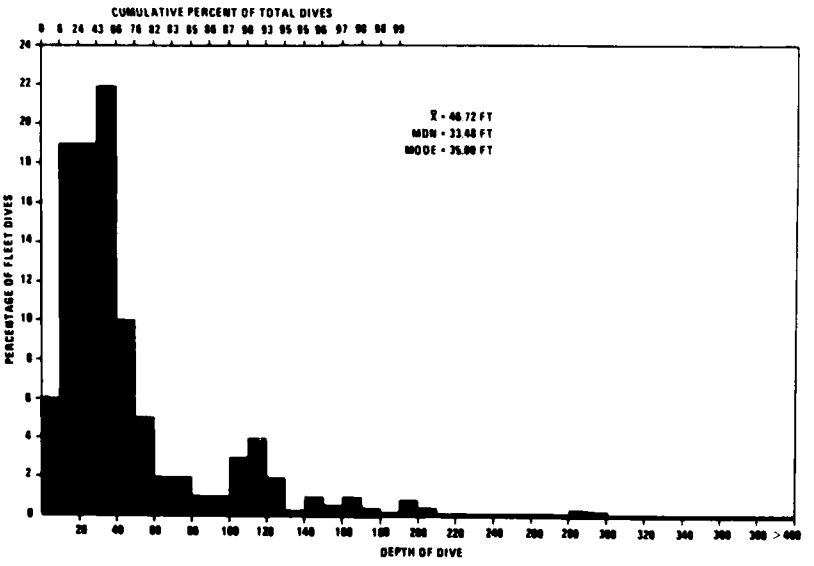

Fig. 1. Graph showing depth ranges of USN dives.

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With a median depth of about 34 feet, we just don't get into the decompression problem. If the Navy, however, chose to start diving deeper tomorrow or next week, there is the possibility of increasing numbers of decompression problems. In our analysis of Navy diving, we have divided the dives into various categories of shallow-short, shallow-medium, shallow-long; medium-short, medium, and long; deep-short, medium, and long; subsaturation; shallow saturation dives and deep saturation dives (Table 1). Section (a) of Fig. 2 gives

Table 1

Categories used in the analysis of Navy fleet dives

Abbreviation	Description	Definition	
		Depth, fsw	Bottom Time, min
	Standard Dive Categories		
SS	Shallow-short	<100	<30
SM	Shallow-medium	<100	30-60
SL	Shallow-long	<100	>60
MS	Medium-short	100-200	<30
MM	Medium-medium	100-200	30-60
ML	Medium-long	100-200	>60
DS	Deep-short	201-300	<30
DM	Deep-medium	201-300	30-60
DL	Deep-long	201-300	>60
	Special Dive Categories		
Sub. Sat.	Subsaturation	>300	720
S. Sat.	Shallow saturation	≤300	≥720
D. Sat.	Deep saturation	>300	≥720

an indication of where the Navy is diving—in the shallow range and across all lengths of time; very little diving is done in the deep range—less than 3/10 of 1% of Navy diving is done using saturation techniques. If one looks at the distribution of decompression sickness (Fig. 2, section (c)), one finds a grossly different profile than is found in the distribution of dives. Some 20% of the Navy's decompression sickness is occurring on the saturation dives. We also have an inordinately high incidence of bends in the medium-depth, short-duration dives. If we look at the time spent under pressure (Section (b) of Fig. 2), we again find that some 20% of the man-hours spent under pressure are in the saturation mode. We also find a different profile when we look only at the number of dives made. This type of information suggests that if we were going to move our diving to deeper depths, we would start having decompression problems. Presently, the only air diving the Navy does to a deep depth is the training dive at the Diving School, where the divers go to 285 feet for a short time (5-10 minutes) to gain experience with nitrogen narcosis. The incidence of bends on this particular dive is very high, another indication that if we went deeper, we would have severe decompression problems. In this Workshop, we want to deal with ways to extend the present decompres-

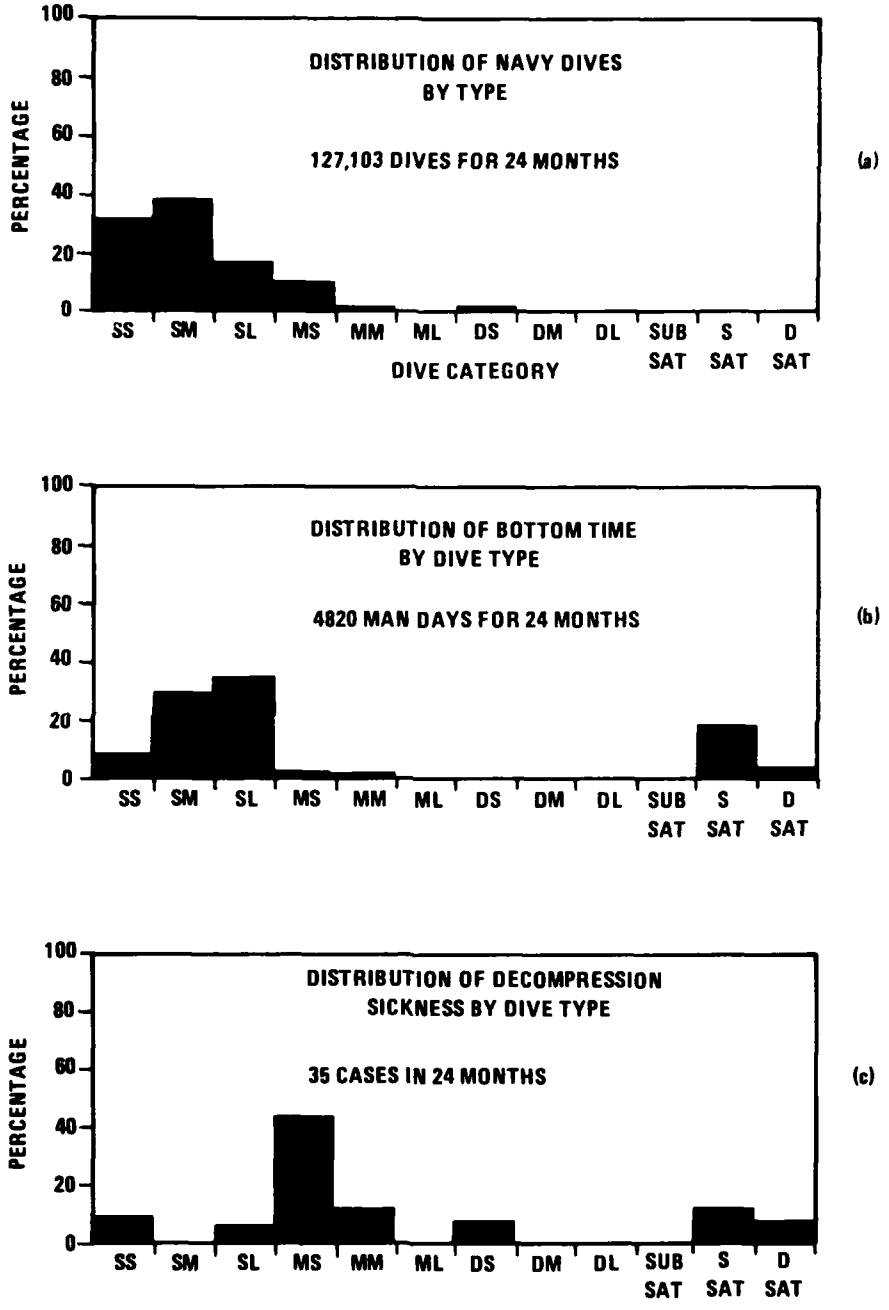

Fig. 2. Distribution of USN dives.

sion procedure. It is obvious that the procedure doesn't work for all of our present dive environments. We need a general model that can be applied across all dive situations.

Before dealing with the specifics of the present model, I would like Dr. Beckman to tell you about the attempt to use the present decompression procedures during a salvage job in the Pacific.

SAFER AIR DECOMPRESSION SCHEDULES DEVELOPED DURING A TWO-YEAR SALVAGE DIVING OPERATION

Hisashi Yano and E. L. Beckman

Diving salvage operations have been carried out by commercial diving industries under various conditions and in many parts of the world. These industries may have attempted to improve their decompression schedules for air diving and to decrease the incidence of decompression sickness during their actual diving operations, but such experiences have seldom if ever been reported in the open literature. It is our purpose to record the experiences of one diving salvage company in carrying out what were reputed to be "simple standard diving operations" and in developing safer air decompression tables for the occupational health and safety of its divers.

THE INCIDENT

On 12 August, 1974, the German tugboat *Hamburg*, while towing the passenger ship, *M.V. Caribia* (no passengers aboard), tried to enter Apra Harbor, Guam, to escape from tropical storm Mary. At that time, the *Hamburg's* generator failed under the very rough sea conditions, and the tugboat captain ordered the towing line to be cut to avoid a double disaster. Without the towing line, the *Caribia* (25,794 gross tons, length: 715 feet, breadth: 91 feet) went aground at the tip of Glass Breakwater at the entrance of Apra Harbor, Guam. Several hours later, she sank completely, except for part of her bow. The *Caribia* landed at the sea bottom in about 100-150 feet of water. Her bow was headed toward the North and she was listing approximately 75 degrees to port. She formed an underwater obstruction that blocked half of the entrance channel into the harbor.

THE SALVAGE OPERATION

In the Spring of 1975, on the basis of competitive bidding, the U.S. Army Corps of Engineers awarded a contract to Nippon Salvage Company, Ltd. (NSC), to remove the sunken passenger ship from the entrance of Apra Harbor. The salvage operation was planned by NSC technical staff in such a way that divers would divide the vessel's body, by oxy-arc and explosive cutting, into 400 ton-weight blocks that a floating crane of 500 tons' lifting capacity would then lift and move to a breaking yard.

For two years, beginning 13 June 1975, NSC conducted a salvage operation of the 25,794 ton *Caribia*. The divers worked daily from sunrise to sunset, except on days when adverse sea conditions prevented diving.

THE DIVING OPERATION - EQUIPMENT

In the earlier part of this salvage operation, two barges of identical size (length: 20 meters, breadth: 9 meters) were used as the diving platform. The diving barge contained the usual diving support equipment, including a large double-lock decompression chamber. Unfortunately, the salvage operation was interrupted by a devastating typhoon, Pamela, which struck Guam on May 21, 1976, and sank the diving barges with the loss of all diving equipment. A new diving barge, the *Masakuni* (length: 50 meters, breadth: 18 meters), was then built in Japan and put into operation in July 1976. In addition to a double-lock deck decompression chamber, this new diving barge had a large built-in (10-fsw maximum pressure) decompression chamber (length: 10 meters, breadth: 5 meters, height: 3 meters) in which 5 oxygen masks were installed. A 1.8-meter diameter diving tube extended down 3 meters into the water from the diving room for divers to enter the chamber from under water during decompression. The schematic of the chamber

is shown in Fig. 1. Divers could perform the final 10-fsw decompression and breathe oxygen by mask under dry circumstances in this chamber. Diving gear used for this diving salvage operation was principally surface-supplied air gear with either hard hat helmets or masks. Approximately half of the divers wore standard Yokohama hard hats and dry suits and the others wore wet suits with hookah masks with communications.

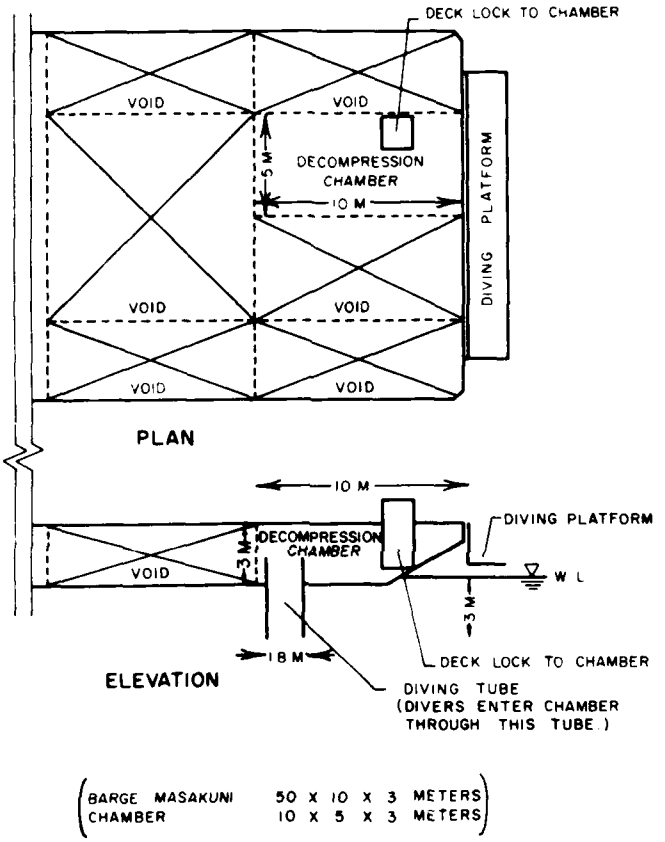

Fig. 1. Schematic of diving barge, showing decompression room.

PERSONNEL

The Divers

The divers in this project were professional divers. The number of divers in the crew varied from day to day and month to month, but in general there were 20-25 divers working daily. The ages of these men varied from 18-55 years at the beginning of the project, with a mean age of 34.0.

OPERATION

The divers began work at 0700. The work was rigorous, and the water temperature was 26° ± 2°C. The divers dived twice a day; one dive period lasted 3-4 hours. The divers were therefore exposed to pressure for approximately 6-8 hours/day. In general, divers made two dives per day, with a surface interval during which they had lunch. More than 90% of the dives required in-water decompression. Surface decompression diving tables were not used.

The depths of the dives carried out varied from 10 to 180 fsw, depending upon the purpose of the dive and the part of the ship involved. However, 73% of the dives were to depths between 90 and 150 fsw, i.e., 10528 of the 14358 dives. A more detailed breakdown of the depths of the dives during each period in which different decompression programs were used is shown in Tables 1A, 2A, 3A, and 4A.

Typical dive schedules for the members of the dive teams who made two dives per day were:

TYPICAL SCHEDULE FOR DIVER

(Surface Interval From Previous Dive Greater Than 12 Hours)

0700	Divers leave pier by boat for diving barge
0800	Diver leaves surface Dive, 120 fsw x 90 min
0930	Diver leaves bottom Diver begins decompression in water
1200	Diver arrives surface Surface interval and lunch
1400	Diver enters water and leaves surface Dive, 120 fsw x 55 min
1455	Diver leaves bottom and starts decompression in water on 120 fsw x (90 min + 10 min) table
1745	Diver arrives surface
1845	Divers return to pier by boat

The decompression tables selected were obviously those which would provide the longest working time on bottom and still permit the completion of the decompression in time to catch the last boat back at sunset.

RESULTS

DECOMPRESSION SCHEDULES

On the basis of the recommendation of the contracting agency (U.S. Army Corps of Engineers) to the contractor (NSC), the U.S. Navy Standard Air Decompression schedules (*U.S. Navy Diving Manual* 1973) were adopted as the fundamental schedules for this diving-salvage operation. However, the contractor, on the basis of his experience, elected from the beginning to extend and modify the U.S. Navy standard air decompression tables (1973) in an effort 1) to decrease the incidence of decompression sickness, 2) to prevent the occurrence of decompression sickness during decompression, and 3) to increase the efficiency and comfort of the divers. The diving schedule modifications used during each successive period of time were designated NSC air decompression tables Mark I, Mark II, Mark III, and Mark IV.

A. NSC Tables Mark I – Period 1 (June 13, 1975 - March 18, 1976)

U.S. Navy standard air decompression tables were used, but the schedules were modified by using the decompression table for a depth 10 feet deeper than the actual dive depth. The commercial diving industry has had reservations for some time about whether the U.S. Navy standard air decompression schedules are suitable for commercial diving operations that require longer times on the bottom, more strenuous work, and more continuous operation than required in normal military usage.

From January, 1976, half of the divers (mask divers) used the diving schedules with bottom times 10 minutes longer than the actual bottom time, so results could be compared with those of other divers who used schedules for dives 10 feet deeper than the actual dive depth. No significant difference in decompression sickness incidence was observed for the two groups. In NSC experience, as the working depth increased below 90 fsw, the incidence of decompression sickness increased, and by March 1977, 9 of the 22 cases of decompression sickness had occurred during decompression. In February, an unacceptably high incidence of decompression sickness occurred (1.13%), indicating the need for a change in decompression schedules. The number of dives accomplished, together with the depths of dives, are shown in Table 1A, and the incidence of decompression sickness for this period of the operation appears in Table 1B.

B. NSC Tables Mark II – Period 2 (March 19, 1976 - July 19, 1976)

In an attempt to decrease the incidence of decompression sickness, the USN decompression schedules were further modified to include a stop for 10 minutes at a depth 10 feet deeper than the first decompression stop recommended by U.S. Navy schedules. This modification alone was inadequate. The effect of this modification upon the incidence of decompression sickness for the dives carried out is shown in Tables 2A and 2B.

Modification of the USN tables by use of the 10-fsw deeper table and the additional 10-minute decompression stop was also insufficient to decrease the incidence of decompression sickness to a satisfactory level for deeper working dives.

The diving-salvage operation was severely hampered during this period by a devastating typhoon, Pamela, which struck Guam on May 21, 1976. The contractor suffered equipment damage, including the loss of the diving barge and all equipment on board. After this date, what little diving there was was directed to search and recovery of lost equipment and survey of the hull of the *Caribia* to determine how much she had been moved and heeled over. Much salvage time was lost in replacing the diving barge and its equipment. The salvage-diving operation did not regain its former momentum until mid-July.

C. NSC Tables Mark III – Period 3 (July 20, 1976 - November 18, 1976)

A much slower rate of ascent and oxygen and air breathing during decompression, such as are used in the Royal Navy Physiological Laboratory (RNPL) air diving tables, were then initiated (RNPL 1968). Divers were instructed to come up at 60 feet/minute for the first 30 feet and then at a rate between 2 and 4 feet/minute, in accordance with University of Hawaii recommendations.

The University of Hawaii diving consultant had computed decompression tables not only to optimize the bottom time of the divers but also to minimize the probability that decompression sickness would occur (see Appendix). Among other recommendations was one to use a very much slower ascent rate, i.e., 4 feet/minute from 100 to 70 ft, and then 2 ft/min from 70 to 40 ft, after 120 to 130 foot dives. NSC operational experience showed that the divers ascended at a rate of approximately 6 fsw/min. Therefore, the NSC Diving Officer instructed the tenders to ensure that their divers had received a total of 15 minutes

Table 1A. Period 1, 6-13-75 to 3-19-76, Summary of Dives

Year and Month	Dive Depth, fsw 10-30	30-60	60-90	90-120	120-150	150-180	Total Dives
1975, 6/13	26	13	42	6	4		91
7	44	157	344	12	1		558
8	7	174	185	0	2		368
9	0	87	320	30	99		536
10	25	144	176	56	126		527
11	1	13	88	39	19	1 (180 ft)	161
12	5	120	320	226	0	2 (160 ft)	673
1976, 1	1	66	200	319	0		586
2	0	65	191	349	16		621
3/1-3/18	11	46	132	290	126		605

Table 1B. Summary of Decompression Sickness Data

Year and Month	Total Dives	Number of Bends Type II	Type I	Incidence of DCS/Mo., %	Incidence of DCS for Period	Cases of DCS during Decompression
1975, 6/13	91	0	0	0		
7	558	0	0	0		
8	368	0	0	0	22/4,726	9/22
9	536	0	1	0.19	(0.47%)	(41%)
10	527	0	0	0		
11	161	0	1	0.62		
12	673	1	3	0.59		
1976, 1	586	1	4	0.85		
2	621	0	7	1.13		
3/1-3/19	605	0	4	0.66		

Table 2A. Period 2, 3-19-76 to 7-20-76, Summary of Dives

Year and Month	Dive Depth, fsw 10-30	30-60	60-90	90-120	120-150	150-180	Total Dives
1976, 3/19-3/31	0	4	29	279	85		397
4	17	6	34	552	157		766
5*	3	4	26	349	0		382
6			38	($\bar{M}$ Depth = 75 fsw)			38
7/1-7/9	6	18	15	3	2		44

*Typhoon Pamela struck Guam; dive barge and equipment destroyed.

Table 2B. Summary of Decompression Sickness Data

Year and Month	Total Dives	Number of Bends Type II	Type I	Incidence of DCS/Mo., %	Incidence of DCS for Period	Cases of DCS during Decompression
1976, 3/19-3/31	397	0	2	0.5		
4	766	2	8	1.31		
5*	382	0	1	0.26	13/1,627	6/13
6	38	0	0	0.0	0.80%	46%
7/1-7/19	44	0	0	0.0		

*Guam hit by typhoon Pamela May 21, 1976.

of decompression time before arriving at the first USN scheduled decompression stop. This decompression time consisted of ascent at 60 fsw/min for 30 ft, ascent at 6 ± 2 fsw to 40 fsw, and additional stop time at 40 fsw to complete 15 minutes of decompression time before ascending to the first scheduled USN stop at 30 fsw. Divers also breathed 100% oxygen during the first 15 minutes at the final 10-fsw decompression stop in the decompression chamber of the barge. After October 1976, a policy was adopted that applied to the second dive of the day only, i.e., using a decompression schedule for a bottom time 10 minutes longer than the actual bottom time plus the repetitive dive time for selecting the proper depth-time decompression table. Although the dives carried out were deeper than those of the previous period, the incidence of decompression sickness remained unacceptably high and the incidence of decompression sickness that occurred during decompression was also unacceptable. The dive depths and the incidence of decompression while using this modification of the USN diving tables are shown in Tables 3A and B.

D. NSC Tables Mark IV - Period 4 (Nov. 19, 1976 - June 30, 1977)

Divers and tenders were now instructed to use a modification of the USN tables that included an ascent rate of exactly 3 feet/minute, similar to the rate proposed at the beginning of period 3. By marking the ascent line at 3-foot intervals, an ascent rate of 3 ± 0.5 feet/minute was achieved.

The final modified USN air decompression schedule for air diving to 100-140 fsw used in this period was: (a) ascend from the bottom for the first 30 feet at the rate of 60 feet/minute; (b) ascend above first 30 feet to the first decompression stop, i.e., stop recommended in U.S. Navy schedules at a rate of 3 feet/minute; (c) follow U.S. Navy schedules after the first decompression stop; (d) breathe 100% oxygen during the first 15 minutes of the final 10-fsw decompression stop; (e) apply decompression schedules for 10 minutes longer than the actual bottom time plus repetitive dive time only for the second dive of the day.

The number of dives and depth of dives carried out on this schedule are tabulated in Table 4A. The effect these modifications to the U.S. Navy Standard Air Decompression schedules had upon the incidence of decompression sickness is shown in Table 4B. Using this modification to the USN Air Decompression tables reduced the incidence of decompression sickness to 0.36%, i.e., number of cases/number of dives, for a total of 5,611 dives at a mean depth of 115 feet. The divers were able to work on the bottom for up to 145 minutes per day by making two dives per day, for a total of approximately 8 hours under pressure daily. In Fig. 2, the NSC Mark IV decompression profile for a 120-fsw dive for 90 minutes is compared with the RNPL air diving tables with air and oxygen breathing during decompression.

DECOMPRESSION SICKNESS

A review of the data on the 86 cases of decompression sickness reveals that 31 divers were afflicted. The ages of the afflicted divers varied from 20-52 years. There was no apparent correlation between the diver's age and the frequency with which the diver experienced decompression sickness. One diver was afflicted 6 times and 9 divers were afflicted 4-5 times, but 15 divers (of the 46 divers engaged in the salvage operation) did not suffer from decompression sickness although they used the same decompression schedules as their fellow divers.

The types and symptoms of decompression sickness fell within the standard classifications. There were 73 cases of type I (pain-only) decompression sickness, and 13 cases of type II: 8 cases of limb weakness or loss of sensation, 4 cases of vertigo and nausea with difficulty in seeing and with balance, and one case of severe coughing, choking, difficulty in breathing and pain in the chest. The types and symptoms of disease are tabulated in Table 5. No dysbaric osteonecrosis investigations were carried out.

Table 3A. Period 3, 7-20-76 to 11-18-76, Summary of Dives

Year and Month	Dive Depth, fsw 10-30	30-60	60-90	90-120	120-150	150-180	Total Dives
1976, 7/20-7/31	0	0	2	210	0		212
8	9	6	6	372	194	5 (170 ft)	592
9	31		4	12	399		446
10	5	0	2	17	626		650
11/1-11/18	28	0	0	146	320		494

Table 3B. Summary of Decompression Sickness Data

Year and Month	Total Dives	Number of Bends Type II	Type I	Incidence of DCS/Mo., %	Incidence of DCS for Period	Cases of DCS during Decompression
1976, 7/20-7/31	212	0	1	0.47		
8	592	2	7	1.52	31/2,394	11/31
9	446	2	4	1.35	1.29%	35%
10	650	1	8	1.39		
11/1-11/18	494	0	6	1.21		

Table 4A. Period 4, 11-19-76 to 6-29-76, Summary of Dives

Year and Month	Dive Depth, fsw 10-30	30-60	60-90	90-120	120-150	150-180	Total Dives
1976, 11/19-11/30		0	0	28	274	0	302
12	7	0	[illegible]	0	870		877
1977, 1		4	0	74	683		761
2		6	0	401	251		658
3	8		0	785	32	1 (160 ft)	826
4	0	0	173	614	121		908
5	2	1	282	361	130		776
6	24	2	5	259	202	11 (160 ft)	503

Table 4B. Summary of Decompression Sickness Data, Period 4

Year and Month	Total Dives	Number of Bends Type II	Type I	Incidence of DCS/Mo., %	Incidence of DCS for Period	Cases of DCS during Decompression
1976, 11/19-11/30	302	0	2	0.66	Begin Period 4	
12	877	1	4	0.57		
1977, 1	761	0	2	0.26	20/5,611	6/20
2	658	0	3	0.46	0.36%	30%
3	826	1	1	0.24		
4	908	1	2	0.33		
5	776	0	0	0		
6	503	1	2	0.60	Completed on 6/29/77	

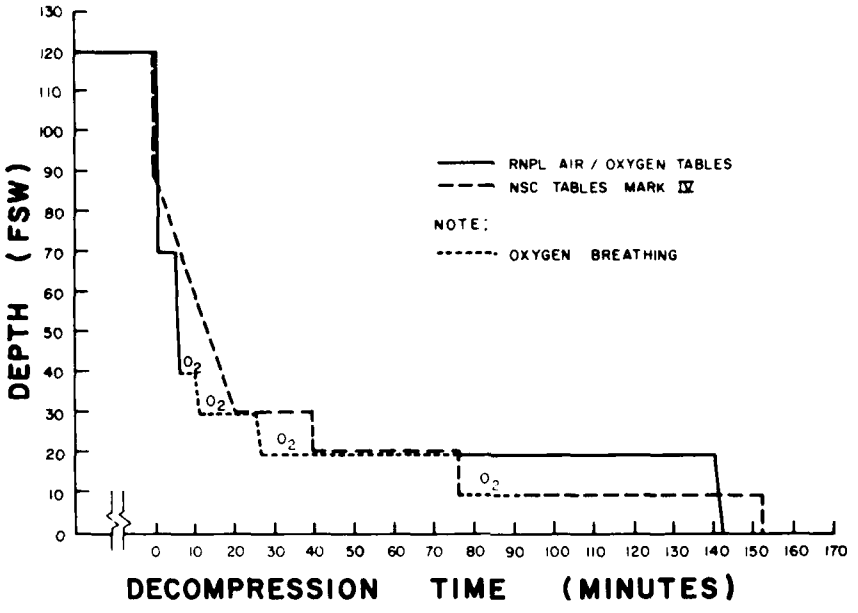

Fig. 2. Comparison of RNPL (1968) and NSC Mark IV decompression schedules for 90 minutes at 120 fsw.

Table 5. Types and Symptoms of Decompression Sickness

Symptoms	No. Cases	Percent of Total
	Type I	
Arms, legs, shoulders	73	84.9
	Type II	
CNS	8	9.3
Vestibular	4	4.65
Chokes	1	1.15
Type II Total	13	15.1
Total, Types I and II	86	100

Table 6. Comparative Air Decompression Schedules for USN and Those Used by NSC for 120 fsw Dive for 90 Minutes

Table Source	Rate of Ascent	Time to First Stop	40 fsw	30 fsw	20 fsw	10 fsw	Total Decompression Time, min
USN	60′/min	1.5		19	37	74	132 (Z)
NSC MK I	60′/min	1.3	8	19	45	80	154
NSC MK II	60′/min	1.3	10	19	37	74	142
NSC MK III	60′/min (From 120 to 90 FSW) 0.5 6′/min (From 90 to 30 FSW) 10.	10.5	4.5	19	37	74 (Including 15′ 100% O_2 breathing)	146 (161 air equivalent)*
NSC MK IV	60′/min (From 120 to 90 FSW) 0.5 3′/min (From 90 to 30 FSW) 20.0			19	37	74 (Including 15′ 100% O_2)	151 (166 air equivalent)*

*Air equivalent time is based upon the theory that air decompression time is one-half as effective as an equal period of breathing 100% oxygen during decompression, i.e., 15 minutes of breathing 100% oxygen is as effective in ridding the body of nitrogen as 30 minutes of air breathing at the same depth.

DISCUSSION

In accordance with the recommendations of the contracting agency, the NSC used the U.S. Navy standard air decompression schedules, with extensions of decompression time, to decompress the divers. The NSC progressively modified the basic USN tables in an effort not only to decrease the incidence of decompression sickness, but also to prevent its occurrence during decompression and to increase the efficiency of the salvage operation (Table 6).

Although the RNPL air diving tables were recommended by the Diving Medicine Consultant in February 1975, the increase in decompression time required over USN tables and the need to provide oxygen breathing facilities delayed acceptance of this system. Instead, several modifications of the USN tables were successively introduced into operational use (NSC Decompression Schedules Mark I-IV). As can be seen in Fig. 2, there is very little difference in the decompression time required by the RNPL air decompression table using oxygen and that required by the NSC Mark IV table, which evolved from theory, to test, to evaluation, to acceptance. However, the oxygen breathing time required by the RNPL (1968) air-oxygen decompression table is both longer and occurs at greater depths (5 minutes at 40 fsw, 15 minutes at 30 fsw, and 50 minutes at 20 fsw) than the NSC Mark IV table (15 minutes at 10 fsw).

The relative effectiveness in preventing decompression sickness of the modifications to the U.S. Navy standard air decompression tables (1973) developed by NSC can be evaluated from the data in Table 7. Since decompression sickness did not become a problem until the diving depths reached 90 fsw when the NSC Mark I table was being used, it may be inferred that this table was effective for working dives to 90 fsw, and therefore dives to this depth or less need not be included in effectiveness analyses.

Table 7. Effectiveness of Decompression Procedures Used During Different Periods Compared With the Number and Depths of Dives

NSC Period	Number of Dives/Depth			Total No. Dives 90 fsw	No. of DCS Cases	Incidence of DCS/100 Dives, %
	90 fsw-120 fsw	120 fsw-150 fsw	150 fsw-180 fsw			
1	1327	393	3	1720	21	1.22
2	1183	244	0	1427	13	0.9
3	757	1539	5	2301	31	1.35
4	2522	2442	12	4976	20	0.4

The effectiveness of the tables (NSC Mark I-IV) in preventing decompression sickness for working dives at depths deeper than 90 fsw can be assessed from the data shown in Table 7. The number of deeper dives and the type of work done in periods 3 and 4 were similar. However, the incidence of decompression sickness for deeper dives in period 4 is significantly lower than that for period 3 (as well as for periods 1 and 2), which implies that the NSC Mark IV decompression schedule is significantly more effective than the other schedules. Nevertheless, even with this decompression schedule, 30% of the divers who developed decompression sickness were afflicted while still decompressing under pressure. This fact suggests that decompression time at depth was still inadequate even in the NSC Mark IV schedule.

The experience gained in diving during this two-year diving-salvage operation reinforces the belief that longer decompression times at deeper depths than those programmed in the U.S. Navy decompression schedules are necessary for working-salvage diving. Similarly, the experience gained in this diving-salvage operation indicates that oxygen breathing during decompression provides a significant advantage. These observations are more apparent when the different schedules used for a 120 foot, 90 minute dive are compared (Table 6). In addition, the use of the decompression room in the barge during the last (10 fsw) stop was found to increase the safety and comfort of the divers significantly during decompression, and is strongly recommended.

CONCLUSION

On the basis of the experience gained in this diving-salvage operation, strenuous working dives on air to 100-140 fsw should use a modification of the USN standard air decompression table to provide more time at deeper depths during decompression to decrease the incidence of decompression sickness, thereby increasing the efficiency of the diving operation. The NSC modifications to the USN tables described as NSC Mark IV tables were effective in decreasing the incidence of DCS and are therefore recommended for working dives under these conditions.

APPENDIX*

THE RATIONALE BEHIND THE MODIFICATIONS MADE BY NSC TO THE USN AIR DIVING TABLES FOR USE IN THE DIVING-SALVAGE OPERATION IN APRA HARBOR, GUAM

Edward L. Beckman

The contractor (NSC) first consulted with the University of Hawaii Department of Physiology Diving Research Group (Hu'a hui) in mid-February 1976 regarding the high incidence of decompression sickness their divers were experiencing using the U.S. Navy standard air decompression tables. NSC reported that as their working depth increased toward 100 fsw, their incidence of decompression sickness increased from 00% to over 1%, even using the USN standard air decompression tables properly and using tables for depths 10 feet deeper than required.

Similar problems had been studied by the University of Hawaii Diving Research Group, and Beckman (1976) had reported the results of the investigation that same year. On the basis of these investigations, the following recommendations, in the order of importance, were made:

1) Use saturation diving. (This was not feasible because the contractor did not have proper equipment and it was not practical to delay the project to obtain it), or
2) Use the RNPL air diving tables (1968) using air and oxygen decompression (RNPL 1968), or
3) Use the RNPL air diving tables (1968) using air decompression, or
4) Have the University of Hawaii Diving Research Group compute diving tables adapted to the specific diving conditions encountered by NSC in their salvage operation, or
5) If the USN standard air decompression tables had to be used:
   a) Use only dive schedules with a residual nitrogen time of an O group dive

*This discussion of the recommendation made, accepted, modified or rejected is included in an effort to emphasize the very real problems which occur when the idealists from a university encounter the real world of operations as limited by management and labor.

b) Do no more than 2 O group dives/day

c) Use repetitive dive table procedures

d) Enter USN diving tables at a depth 20 fsw greater than the actual dive depth.

These recommendations were based upon methods to optimize the health and safety of divers being decompressed. However, three factors worked against the adoption of these recommendations:

1) The contractee (U.S. Army Corps of Engineers) had recommended the USN standard air decompression tables, so the contractor believed that any change in decompression procedure to improve safety should involve only a modification of the basic USN table

2) Any increase in the total decompression time, such as would have been required by the adoption of the RNPL air diving tables or the use of only O residual nitrogen time schedules of the USN tables, would have resulted either in an increase in the duration of the project, which the contractor could not accept, or more divers, which would have increased the cost of the project, an increase none would accept. Any change in the decompression procedures had to improve diving efficiency without increasing contract duration or costs!

3) The limited facilities precluded saturation diving. Also, the contractor did not have facilities for oxygen breathing under pressure during decompression, and this made it impossible to use the RNPL air diving tables using oxygen/air decompression.

In April, 1976 I visited the diving site to evaluate the decompression problems. As a result, the previous recommendations were reinforced. In addition, I learned that the diving barge contained a decompression room that was pressurized to 10 fsw during operations. Divers entered this room through the diving tube and took their last decompression stop while they took off their diving gear and cleaned up. Scott oxygen overboard dump masks had been ordered, but the long time before delivery had delayed the introduction of oxygen breathing in the decompression room during the 10-fsw stop.

It became apparent through conferences with contractor and contractee that the RNPL tables would not be adopted any more than would saturation diving.

Two additional recommendations were therefore made:

1) The divers should be monitored by Doppler bubble detector for venous gas emboli (VGE) during decompression while the divers were in the 10-fsw decompression room, and they should not be decompressed further as long as VGE were detected. The equipment for monitoring was supplied by the University of Hawaii.

2) The divers should intermittently breathe oxygen and air while decompressing in the decompression room for the entire 10-fsw stop time, i.e., 20 minutes of O_2 and 5 minutes of air, repeated until the stop time at 10 fsw was completed, and the decompression room should be modified to permit oxygen breathing. The University of Hawaii was also commissioned to calculate decompression tables for the NSC divers.

The arrival of typhoon Pamela on May 21/22 had two significant effects upon the decompression program:

1) The Doppler VGE monitoring program was abruptly stopped when the detector equipment sank with the diving barge, never to be recovered or replaced

2) The new replacement diving barge was equipped with a decompression room with Scott overboard dump oxygen masks so that oxygen could be breathed at 10 fsw.

However, the new diving barge was not a panacea. The recommendation to use intermittent oxygen/air breathing throughout the decompression stop in the decompression room was difficult to implement because:

1) The overlap of the decompression periods of successive dive teams limited the use of the oxygen breathing equipment by each team
2) The contractor was afraid that the divers would suffer from oxygen poisoning if oxygen was used during extensive twice-daily decompressions and was also used to treat decompression sickness. Therefore, each diver only breathed oxygen for 15 minutes at 10 fsw in the decompression room after each dive.

The University of Hawaii Diving Research Group prepared many decompression schedules for NSC for both single and repetitive dives. However, since they all required either a significant increase in the length of decompression or a reduction in the working time on the bottom, these recommendations were not acceptable to the contractor.

However, the contractor believed one proposed modification had merit–the recommendation about rate of ascent to the first stop of the USN standard air decompression table. The rates of ascent calculated and recommended were based upon the Hawaiian concept of decompression sickness prevention, which holds that the critical ΔP for bubble formation is similar for all gases, for all tissues, and at all pressures if nuclear crushing has been avoided. According to this theory, the rate of ascent should be slow enough to keep the ΔP in all half-time tissues at less than the critical value for bubble growth. This value was taken as 24 fsw for these computations, i.e., equivalent to a constant M value of 50 fsw for all tissues (Yount, Strauss, Beckman, and Moore 1975).

In the USN air decompression schedules for the depths and durations used by the contractor before Pamela, divers would ascend from working depths to their first stop at a rate of 60 fsw/min. Decompression schedules computed according to other concepts (Beckman 1976) required a decompression stop at much deeper depths, i.e., 50-80 fsw, although they used rapid rates of ascent.

Computation of decompression schedules using the University of Hawaii Diving Research Group concepts of a constant critical value for bubble formation regardless of the rate of gas uptake (T ½) of the tissues resulted in a much slower rate of ascent after dives of the type being done by NSC. The ascent from the bottom at 60 fsw/min was limited to a distance approximating that of the partial pressure of oxygen at that depth, i.e., for 130 fsw = 163 fsw x 21% O_2 = 33 fsw).

For a working dive to 130 fsw for 90 minutes:

1) Ascend at 60 fsw/min for first 30 feet (i.e., to 100 ft)
2) Ascend half of the distance to the first stop, i.e., 30 ft, at a rate of 4 fsw/min
3) Ascend the remainder of the distance to the first stop at a rate of 2 fsw/min
4) Take stops according to the USN air decompression tables
5) Breathe alternately 100% O_2 (20 min) and air (5 min) for duration of the stop at 10 fsw.

This recommended change in the rate of ascent was acceptable to the contractor. Although this recommendation was put into effect in a modified form, i.e., ascent rate = 3 ft/min, the modifications adopted certainly decreased the incidence of decompression sickness significantly.

The similarity between the decompression schedule evolved by NSC and the RNPL air/oxygen decompression tables is striking (Fig. 2).

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SECTION II

Current Status of Neo-Haldanian Assumptions

GAS UPTAKE ASSUMPTIONS

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ASSUMPTION NUMBER 1

Multiple tissues or tissue compartments must be used to describe whole-body gas exchange.

The first assumption in the Haldanian model that we would like to deal with is whether or not we need multiple tissues or tissue compartments to describe whole-body gas uptake. Opinion differs concerning this. The historical development of the concepts regarding the number of tissue compartments is shown in Fig. 1.

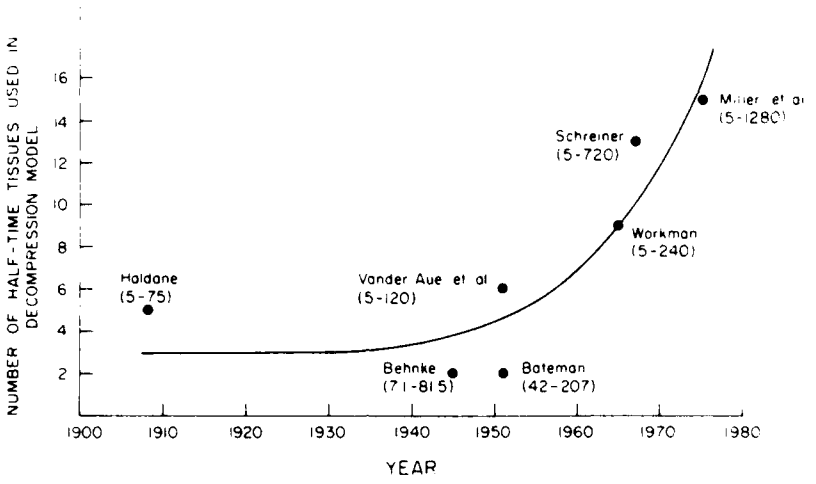

Fig. 1. Historical development in concepts of theoretical tissue compartments.

Haldane started with five, and as we progressed there was almost an exponential growth in the number of tissues postulated. This growth in the number of hypothetical tissues has been caused by the problems that arise every time we start diving a little deeper. We have been adding theoretical tissue compartments with longer half times in an effort to keep ourselves out of trouble. Whether or not one has to keep adding theoretical tissues indefinitely to stay out of trouble is open to question. The results shown in Fig. 2 are based upon a mouse study that we did, in which we exposed mice at 13 ATA on nitrox for various durations and abruptly decompressed them to the surface. We found that the incidence of decompression sickness reached an asymptotic value in about 40 min, which probably indicates that the animals reached equilibrium with the ambient pressure. We've since done the same thing with rats at 7 ATA with nitrogen-oxygen and helium-oxygen gas mixtures (Fig. 3). In all instances our results have shown a curvilinear relationship that reached an asymptotic level. So the basic question is, How are we going to describe this gas uptake curve in our model? For our mouse and rat data, a *single* exponential or half time describes the results adequately. For a larger, more complex organism, a single exponential may not be adequate.

It is important to point out at the start that there is nothing magical about the half time tissue concept. This concept is only one of several convenient ways of describing an exponential rate of change (Fig. 4), and it is not the most common way of describing this type of phenomenon. In the physiological literature, a rate constant is usually used. In his classical work on gas exchange, Behnke used a combination of two rate constants, and he had a very good mathematical description of the empirically gathered data. I make this point to emphasize that we are not wed to the concept of half time tissues.

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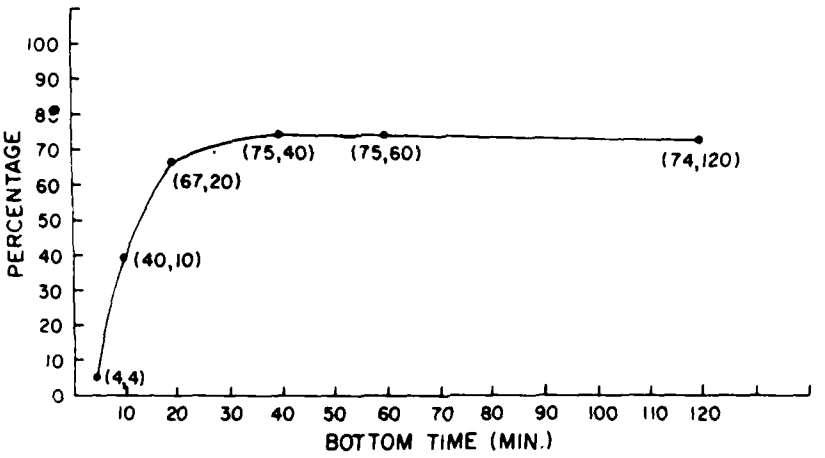

Fig. 2. Change in incidence of d compression sickness in mice across time (nitrox breathing mixture).

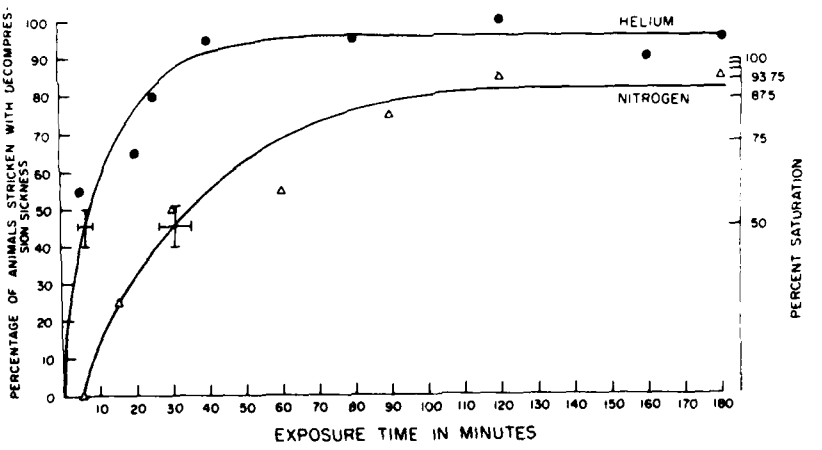

Fig. 3. Decompression sickness incidence in rats across time (N_2-O_2 and He-O_2 breathing mixtures).

The number of hypothetical tissues recommended by attendees at the workshop ranged from 1 (Hills) to 16 (Bühlmann). Hills suggested that for most cases of decompression sickness the same tissue type is involved, namely, tendon. Hills states that "... the same symptoms seem to occur whether we violate the 10-min tissue or the 40-min tissue and none of the advocates of the Haldane calculation method have ever shown any correlation between the hypothetical 'tissue' violated and the nature of the symptoms." These ideas have led Hills to suggest that decompression schedules calculated to avoid limb bends will be adequate for avoiding most decompression problems. He does hedge this position somewhat by saying that knowledge

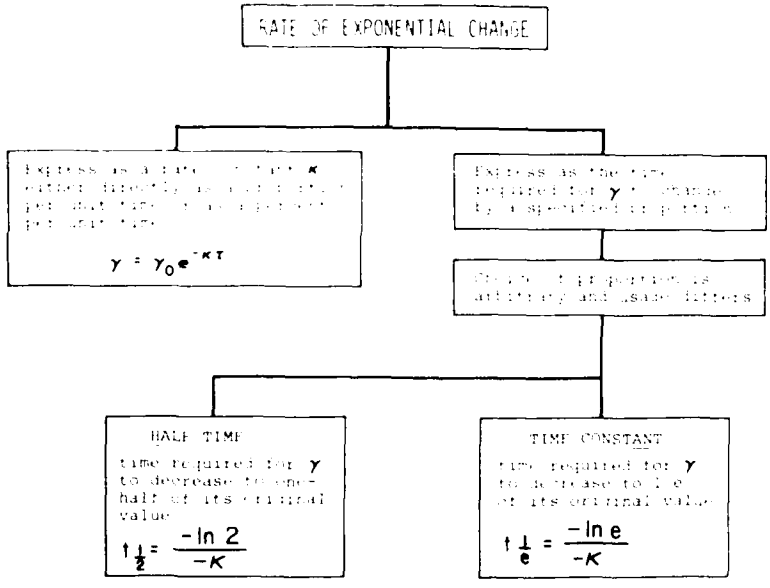

Fig. 4. Rate of exponential change.

of the gas-exchange characteristics of tendon probably will not help in avoiding the decompression sickness associated with the vestibular system or in handling the massive central nervous system symptoms associated with a "blowup."

Brian D'Aoust agrees that ". . . a completely empirical single 'tissue' approach would work but [he] would suggest the use of a multiple tissue model simply because the human body cannot be considered homogeneous as concerns gas saturation, and in fact, since nonsaturation dives constitute the real difficulty in estimating gas uptake, multiple tissues are logical."

Behnke concurs with the idea of multiple tissues for the decompression model, but believes that they should be related to our knowledge of human physiology. He suggests that a model with as many as 15 tissues ranging from a tissue half time of 5 to one equal to 1280 min is probably not needed. The longest half time empirically estimated for nitrogen uptake is about half as long as the 1280-min half time suggested in one model.

Bühlmann uses 16 half times in his model and believes that as long as one has a computer for calculation it doesn't matter how many tissue compartments are used. Keller agreed with Bühlmann and indicated that he used eight tissue half times in his model, mainly because he had a smaller computer than Professor Bühlmann. Bühlmann also suggested that he was able to relate the typical symptoms of decompression sickness to different half time tissue compartments. He provided the following table to show how they might be related (Table 1).

D'Aoust rejects the idea of a continuum approach, i.e., 10 tissues or more, because the model does not lend itself to interpreting the reasons for its failure. Hempleman also rejects the idea of many tissue compartments, but for different reasons. Hempleman points out that to use five or more exponentials for

describing the curvilinear gas uptake relationship implies significance to eight figures and he doubts that there are experimental data to support this level of significance.

Table 1. Tissue half times and compartments (Bühlmann)

Compartment, Tissue and No.	Helium half times, min
Blood/CNS	
1	1.5
2	3.8
3	9
Skin	
4	14
5	20
6	30
Inner Ear	
7	45
8	60
9	75
10	90
Joints/Bones	
11	110
12	130
13	150
14	175
15	205
16	240

Conversion factors for half times of other gases are $N_2/He = 2.6455$, $H_2/He = 0.7097$.

SUMMARY

The Chairman has concluded that body gas uptake can probably be adequately described with two or three exponentials. It would be nice if we could relate the mathematics of the model to actual anatomical tissues, but this correlation has not been demonstrated satisfactorily. Until these data are developed, the diving community is best served by pursuing a model limited to two or three exponentials. Behnke's 1937 model of

$$\text{Total } N_2 \text{ at time (t)} = \text{water } N_2\ (1-e^{-kt}) + \text{Fat}\ (1-e^{-kt})$$

could be expanded to a three-component model such as that proposed by Hennessy

$$\emptyset = 1 - A_1e^{-k1t} - A_2e^{-k2t} - A_3e^{-k3t}$$

The adequacy of this mathematical description is probably sufficient for the data that are presently available. This does not mean that we should not continue to try to improve our understanding of the gas exchange process and its corresponding mathematical description. It simply means that our present understanding of the process does not justify the use of anything more sophisticated than two or three time constants.

ASSUMPTION NUMBER 2.

Tissue gas uptake is described by the following items: exposure time, pressure differentials, and tissue half times.

The second Haldanian assumption to be addressed concerns the components in the gas uptake equation: What factors should be included and what is their interrelationship?

The equation shown in Fig. 5 describes the gas uptake process of the Haldanian model. The final tissue pressure (P_F) is a function of the starting tissue pressure (P_T) plus the differential pressure between tissues and the ambient environment (P_A-P_T) times the change associated with time ($1-\frac{1}{2^{T/H}}$). The question is, How adequately does this equation describe the actual gas uptake curve? Implicit in this equation is that gas uptake (time to equilibrium) is independent of starting pressure (P_T), and the pressure differential one creates (P_A-P_T). We have completed an animal study in which we have evaluated the effects of both of

$$P_F = P_T + (1-\frac{1}{2^{\frac{T}{H}}})(P_A - P_T)$$

Fig. 5. Equation describing Haldanian gas uptake.

these factors. The study was divided into 2 phases: determination of the time to reach pressure equilibrium during different excursions from 1 ATA (Fig. 6), and determination of the time to reach pressure equilibrium during excursions from 1.3, 10, and 20 ATA (Fig. 7). In Phase I, excursions were made to 10, 20, and 30 ATA for 5, 10, 20, 40, or 80 minutes. In Phase II, the rats were saturated at 1.3, 10, or 20 ATA for 60 minutes; each saturation exposure was followed by a 10-atm excursion of either 1, 5, 10, 20, or 40 min. After each exposure the rats were abruptly decompressed to a lesser pressure for observation. The observation pressure levels were selected to establish an ED_{50} (effective dose to produce signs of decompression sickness in 50% of the animals). Results are shown in Figs. 8 and 9, and suggest that neither the starting saturation pressure nor the differential excursion pressure alters the time required for an animal to reach equilibrium with the surrounding environment. From these experimental exposures it would appear that the equation suggested by Haldane does a fairly adequate job of describing gas uptake during exposures to increased pressures.

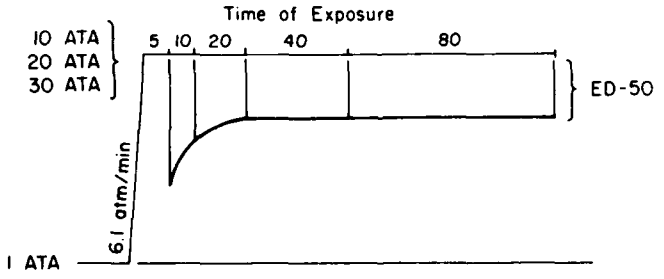

Fig. 6. Time to reach equilibrium during excursions from 1 ATA.

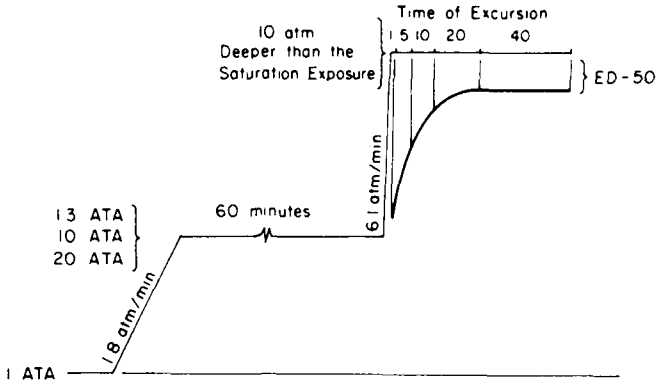

Fig. 7. Time to reach equilibrium during 10-atm excursions from 1.3, 10, and 20 ATA.

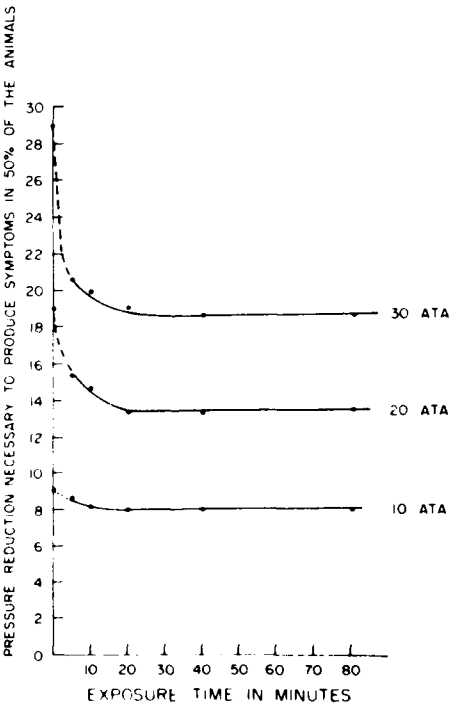

Fig. 8. ED_{50} for decompression sickness for excursions to 10, 20, and 30 ATA from 1 ATA.

Brian D'Aoust agreed with the adequacy of the Haldane equation for calculating saturation states for different compartments; however, he suggested that it might be useful in mixed gas (He and N_2) tables to account for the total amount of dissolved gas (rather than the total inert $He+N_2$ tension) by use of an average solubility for that compartment.

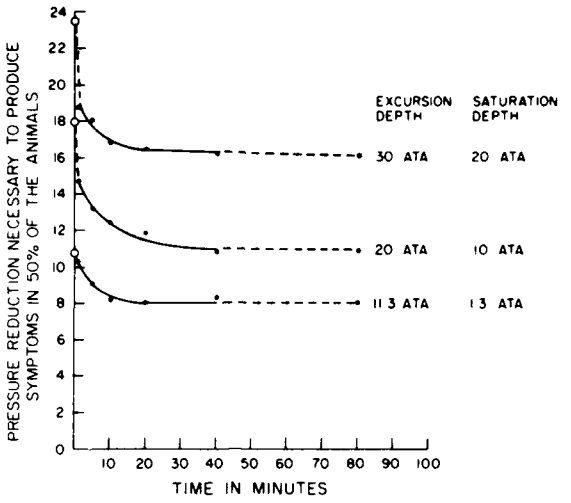

Fig. 9. ED_{50} for decompression sickness for 10-ATA excursions from saturation depths of 1.3, 10, and 20 ATA.

David Yount was also in general agreement with the Haldanian uptake equation, but believed that rather than dealing with tissue compartments we would be better served by thinking in terms of a continuous process described by a series of time constants.

Albert Behnke and Dick Vann both argued that the Haldane equation does not permit one to deal with individual differences in that there are no physiological parameters such as cardiac output, blood perfusion of tissue beds, or size of the body's gas reservoir.

Certainly, if we are ever to individualize decompressions, this type of information is going to be essential. The list of potential factors that might be included in such a model could be expanded to include those in Fig. 10. All of these physiological variables have been mentioned in the decompression literature as possibly contributing to the problem. Before we start individualizing decompressions, however, it is important to get some type of handle on the effects of the various environmental and procedural parameters (Figs. 11 and 12).

SUMMARY

From the information that was presented we can probably conclude that the Haldane equation comes close to describing the effects of the major parameters (time and differential pressure) in the gas uptake process. There are numerous potentially beneficial parameters that are not presently included in the equation; their addition to the model is contingent upon future research. Presently, all of our knowledge concerning pressure physiology is summarized in the tissue half-time concept. One modification that should be seriously considered is the incorporation of the Hennessy three-component equation in place of the present tissue half-time method of handling the changes across time. The resulting equation would be:

$$P_t = P_o + (1 - A_{1e}^{-k_1 t} - A_{2e}^{-k_2 t} - A_{3e}^{-k_3 t})(P_a - P_o)$$

This single equation could be used in place of repeated applications of the Haldane equation. Once the constants are defined this single equation will simplify the calculation of gas uptake.

PHYSIOLOGICAL FACTORS

Species differences

Sex differences

Race and nationality

Age

Body build

Fat content

Diet and fluid intake

Psychological (anxiety)

Fatigue

Previous injury, disease or illness

Cardiovascular differences

Biochemical factors (drugs)

Drinking

Smoking

Blood differences

Respiratory differences

Fig. 10.

ENVIRONMENTAL FACTORS

Inert gas used

Inert gas partial pressure

Duration of exposure

Duration of decompression

Immersion

Time of day

Seasonal variation

Carbon dioxide

Atmosphere contamination

Air ions

Humidity

Temperature

Exercise on the bottom

Fig. 11.

PROCEDURAL FACTORS

Rate of ascent

Distance traveled (step size)

Exercise during decompression

Repeated exposures (adaptation)

Oxygen partial pressure

Stage versus uniform ascent

Posture during decompression

Fig. 12.

ASSUMPTION NUMBER 3.

Only the inert gas partial pressure need be considered in the decompression problem.

Although Haldane made his original calculations using the total gas pressure, he alluded to the fact that only the inert gas partial pressure need be considered in the decompression process. The neo-Haldanian theorists that followed have continued to base their calculations solely on the partial pressure of the inert gas. This approach disregards the possible effects of the other gas constituents such as oxygen and carbon dioxide. The experimental evidence that is available indicates that both oxygen and carbon dioxide are potent ingredients in the decompression problem. The effect of the trace elements in the breathing medium is still undetermined.

We completed three animal studies that relate to this assumption. The first was a mouse study in which we varied exposure pressure and oxygen partial pressure to determine their effects on the incidence of decompression sickness. Figure 13 presents these results. To look at these results in terms of Assumption No. 3, we redrafted them as a function of the inert gas concentration. In Fig. 14, the data points within each box are equivalent inert gas partial pressures. It is obvious that the incidence of bends is not the same for all the dives within a given box—something other than the inert gas partial pressure is having an impact on the decompression outcome. It appears that the differences in the incidence of decompression sickness are caused, in some part, by the oxygen content of the breathing medium. Another interesting result seen in Fig. 14 is that for the low oxygen partial pressures of 0.5 and 1.0 ATA, the incidence of bends increases as the partial pressure of the inert gas increases; however, for the two high oxygen partial pressures of 1.5 and 2.0 ATA, there is a reduction in the incidence of bends as the inert gas partial pressure increases. This finding is completely contrary to the neo-Haldanian model.

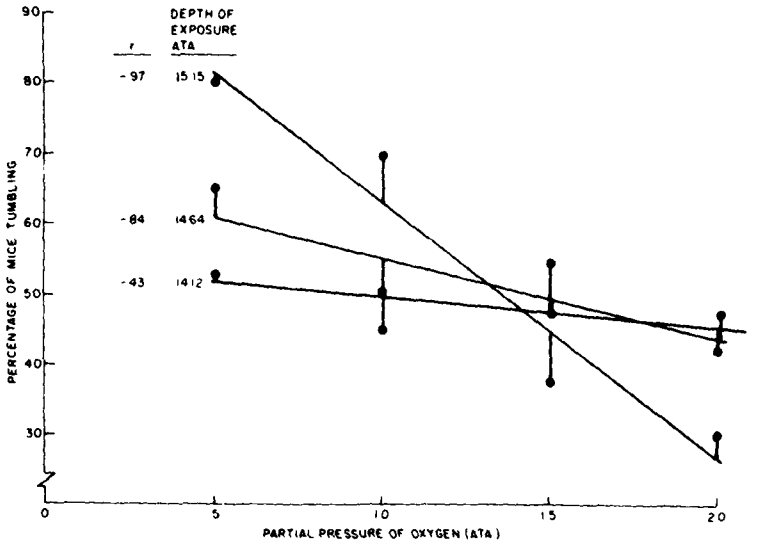

Fig. 13. Relationship between oxygen partial pressure and percentage of mice with serious decompression sickness for various pressure exposures.

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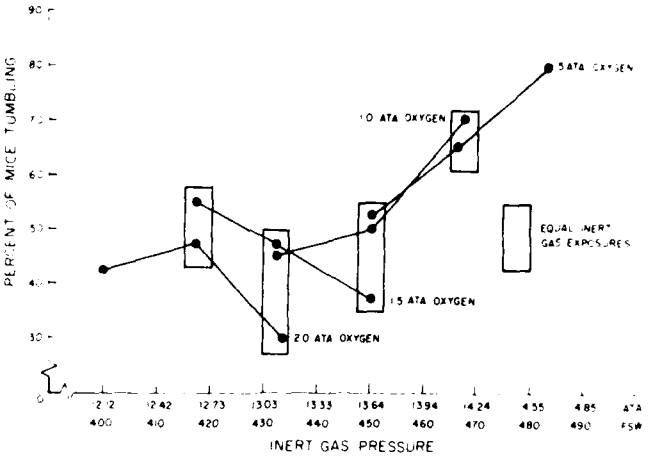

Fig. 14. Incidence of decompression sickness in mice as a function of inert gas concentration.

Assumption No. 3 is the basis of the Equivalent Air Depth (EAD) concept used in the U.S. Navy for calculating decompression schedules for nitrogen-oxygen mixed gas dives. The EAD concept suggests that oxygen does not contribute to the total tissue gas tension and can be disregarded in the decompression process. The validity of this assumption has been experimentally tested by exposing 365 rats to various partial pressures of oxygen at 15 ATA for various lengths of time. If the EAD concept (and the underlying Assumption No. 3) is correct, under constant exposure pressure each incremental change in the oxygen partial pressure should have a corresponding incremental change in pressure-reduction tolerance. Results of the study, shown in Fig. 15, suggest that the concept of EAD does not adequately describe the decompression advantage gained from breathing elevated oxygen partial pressures.

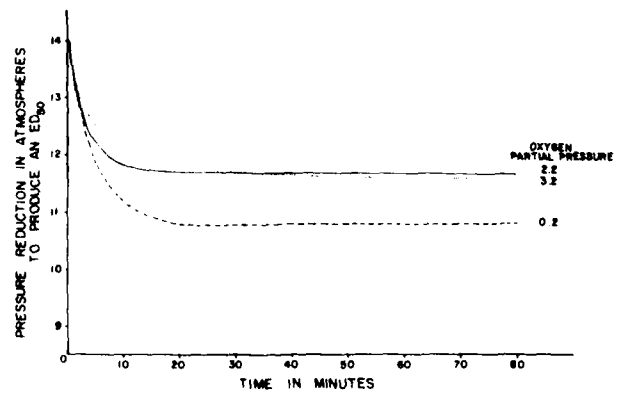

Fig. 15. Relationship between ED_{50} and time.

A second breathing gas factor that may affect the decompression process is the level of carbon dioxide in the inspired gas mixture. The one study we have conducted in this area indicates that carbon dioxide, even in low concentrations, can have a dramatic effect on the incidence of decompression sickness. Our results (Fig. 16) are based upon 680 mice explosively decompressed to the surface after a 15-min nitrogen-oxygen-carbon dioxide exposure to 12, 14, or 16 ATA. Despite the statistical significance of our experimental results and the logical physical rationale based upon the high solubility of carbon dioxide, the literature on carbon dioxide is far from consistent. It is safe to say that carbon dioxide has an adverse effect on the decompression process, but how it should be incorporated in the model is still far from clear.

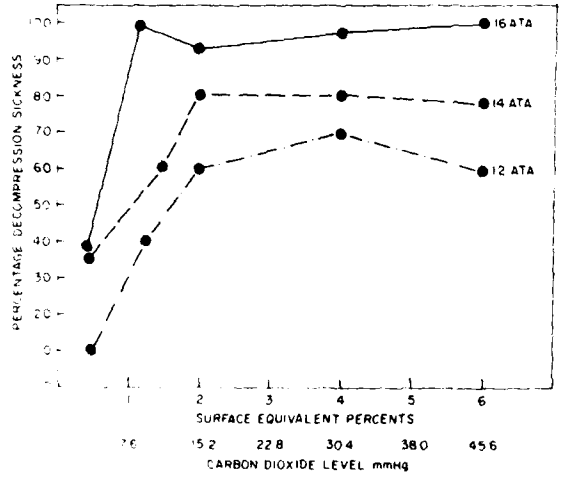

Fig. 16. Incidence of decompression sickness in mice associated with exposure to various levels of carbon dioxide.

Albert Bühlmann indicated during the Workshop that his calculations are based upon the total inert gas pressure in each of the 16 compartments; he uses the sum of all of the inert gas partial pressures. It appears that the oxygen content in the breathing mixture is merely subtracted from the total gas tension to arrive at the inert gas partial pressure for calculation of the decompression. The presence of carbon dioxide in the breathing gas does not appear to be incorporated in Bühlmann's model, either.

In his presentation, Behnke is rather emphatic about the need to include more than just the inert gas partial pressure. He states that all gases, inert and metabolic as well as water vapor, contribute to overpressure and the size-composition of bubbles formed in the process of phase separation.

Yount supports the Behnke position and suggests that to prevent gas nuclei from growing into macroscopic bubbles, it is necessary that the supersaturation pressure P_{ss} remain always less than some critical value, P_{ss}^{*}. The supersaturation pressure, in turn, is defined as the difference between the total dissolved gas tension $\tau = \Sigma\tau_i$ and the ambient pressure P_{amb}

$$P_{ss} \equiv \Sigma\tau_i - P_{amb} \leq P_{ss}^{*}$$

where τ_i are the dissolved tensions of all the gases present, including not only the inert gases, but also the oxygen, carbon dioxide and water vapor. Yount further suggests that oxygen and carbon dioxide –because of their activity and the high solubility of the latter –may play a more significant role than would inert gases at the same tensions.

SUMMARY

It appears that theorists have oversimplified the decompression problem by considering only the inert gas in their calculations. The presence of various levels of oxygen and carbon dioxide has a direct impact on the decompression process and should be incorporated into future models. The problem, of course, is how to incorporate these factors in the model. The basic data are not presently available for estimating the effect of various levels of these variables on man. The best we can do at the present time is acknowledge that decompression is not solely a function of the inert gas partial pressure and that other variables do affect the process.

ASSUMPTION NUMBER 4.

Breathing multiple inert gases appears to provide some decompression advantage.

As shown in Fig. 17, multiple inert gases can be breathed during decompression in either a sequential or simultaneous fashion. Both methods can probably be used effectively to reduce the decompression obligation; however, our research has only dealt with the latter. The advantage in breathing more than one inert

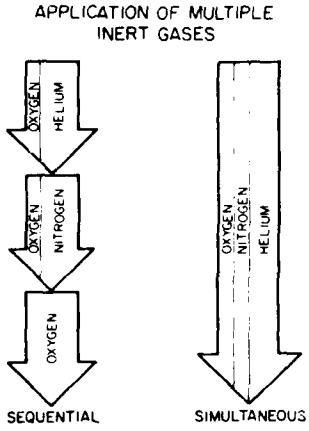

Fig. 17. Schematic of methods of inert gas breathing.

gas is probably due to the different exchange characteristics of each gas. Figure 3 shows that for our rats the rate of gas uptake for helium is about 2.4 times faster than for nitrogen. To determine if one could make use of these different gas exchange characteristics, we designed a study in which rats breathed various combinations of inert gases and oxygen (0.5 ATA O_2) at 7 ATA. After an exposure for a specified time, the rats were abruptly decompressed to the surface. The incidence of decompression sickness for the various experimental conditions is shown in Fig. 18. The results indicate that both time and the inert gas combination have an effect on decompression outcome. The optimum combination of inert gases appears

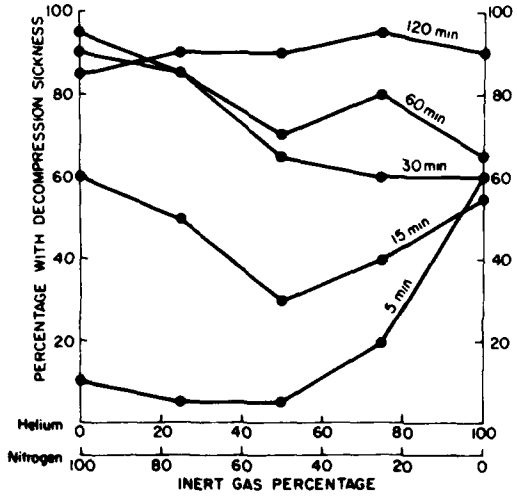

Fig. 18. Decompression sickness incidence for rats under various experimental conditions.

to be a function of exposure time. Figure 19 presents the general shape of this relationship. To optimize the decompression we must increase the relative concentration of helium as exposure time is lengthened

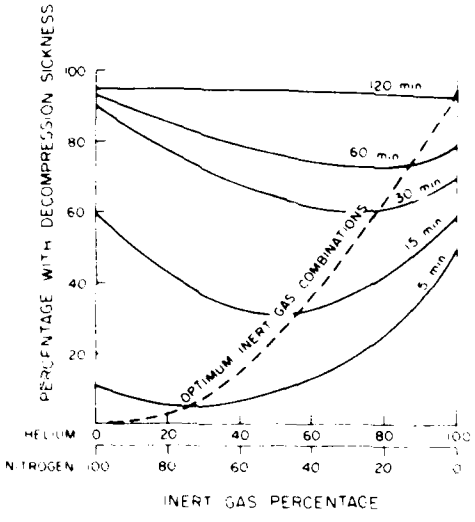

Fig. 19. Relationship between decompression sickness incidence and inert gas combination.

All of the practical diving experience reported by Workshop participants has been in the sequential use of different inert gases. Hamilton reported on a series of deep helium-oxygen dives conducted by Ocean Systems in 1971. During these dives the diver's breathing medium was shifted from helium-oxygen to air at 100 fsw (30 meters). Decompression sickness was experienced after the shift in breathing gas and Hamilton and his colleagues concluded that the Haldanian model, or calculation method, is not valid for describing the physiological response to inert gas shifts.

Professor Bühlmann stated that in Switzerland they also shift to air at 30 meters during the decompression from deep saturation dives. He also reports that they have experienced decompression sickness at shallow depths after the shift to air. An article in *Undersea Biomedical Research* published by Berghage in 1976 provides statistics on the depth of onset of decompression sickness during saturation dives. These statistics indicate that all of the cases of decompression sickness occurring on the U.S. Navy's saturation decompression schedule have occurred at depths less than 20 meters. The U.S. Navy's schedule does not call for the shifting of inert gases during the decompression, so it is difficult to say whether or not the shifting of inert gases has anything to do with the onset of decompression sickness symptoms. We need a well-controlled study to resolve the questions about gas switching during decompression.

David Yount presented data from a University of Hawaii study that indicated that the supersaturation pressure (pressure differential) necessary to produce bubbles does not differ among the inert gases normally associated with diving environments. He went on, however, to suggest that there are other physical properties associated with the various inert gases that could be used to advantage during decompression. He further suggested that the most likely physical property to be utilized in optimizing decompression is the diffusion coefficient. He presents a mathematical example of how the optimization might occur. The example is interesting but what is needed is good, solid experimental evidence.

Brian D'Aoust was more optimistic and suggested that ". . . one can arrange the sequence of gases appropriately using existing models (Keller, Bühlmann, Nishi, etc.) to provide undersaturation and thus a decompression advantage." To the chairman's knowledge this has not been empirically tested. Workman did some initial testing in the late 1960's on the benefits of multiple inert gases. Since then Schreiner, Hempleman, Galerne, Bond, and others have experimented with the idea, but no one has published a comprehensive model and empirically tested it.

SUMMARY

From a theoretical standpoint, it appears that either the sequential or simultaneous breathing of multiple inert gases can improve decompression efficiency. The experimental evidence to support the theory is sadly lacking. The sequential shifting of inert gases during decompression from human saturation exposures has raised some questions concerning this procedure. Several investigators have suggested that the incidence of decompression sickness is higher when the inert gases are changed. The evidence is far from convincing, however, because a control group has never been employed. The two or three animal studies that have evaluated the effects of breathing simultaneously two or more inert gases have provided positive results. The advantages associated with multiple inert gases appear to be related to time. The longer the exposure, the more helium that is needed in the diluent gas.

ASSUMPTION NUMBER 5.

Compression procedures have no effect on decompression.

Recent laboratory experiments by Evans and Walder (1969), Yount and Strauss (1976), and Vann, Grimstad, Neilsen, and Carey (1978) have demonstrated that compression procedures can have an impact on decompression outcome. Dr. David Yount has suggested that the reason for this relationship is the existence in the body of gaseous micronuclei. These pre-existing micronuclei are the basis of the bubbles that form during decompression. The laboratory experiments that have demonstrated the relationship between compression and decompression have used widely divergent rates of compression to very deep depths (high pressures). The question of whether or not these studies have any significance for operational diving remains open for discussion. We have completed one study in which rats were compressed to several different depths using two different rates of compression (60 fpm and 200 fpm). After saturation the animals were abruptly decompressed to determine the pressure reduction that will produce decompression sickness in 50% of the animals (Fig. 20). Regardless of which compression rate was used, the decompression tolerance was the same, which indicates no relationship between compression and decompression.

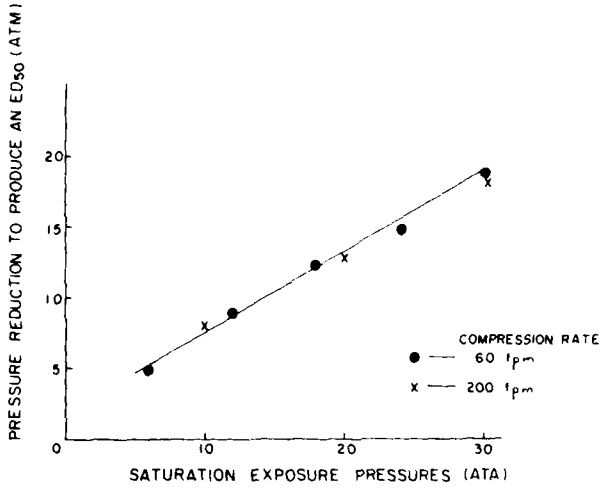

Fig. 20. Decompression sickness ED_{50} for rats compressed at 200 fpm (x) and 60 fpm (•).

SUMMARY

It appears that for normal operational diving the rate of compression can be disregarded in the decompression calculations. The laboratory experiments that have demonstrated a relationship between the two are important from a theoretical point of view. These experiments may have a tremendous impact on the way we think about the decompression problem, but unless we drastically change compression procedures they will continue to have little or no effect on decompression.

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PRESSURE REDUCTION ASSUMPTIONS

ASSUMPTION NUMBER 6.

Tissue supersaturation limits are best described by a series of pressure reduction ratios.

An essential concept in the discussion of the effects of pressure reduction is its mathematical description. The effects of a pressure reduction are probabilistic in nature and therefore must be described using statistics. Several investigators have demonstrated that the incidence of decompression sickness can be described by an S-shaped function of the magnitude of the pressure reduction. Although the magnitude of the pressure reduction that can be tolerated is different for different species, the same general S-shaped curve prevails (Fig. 21).

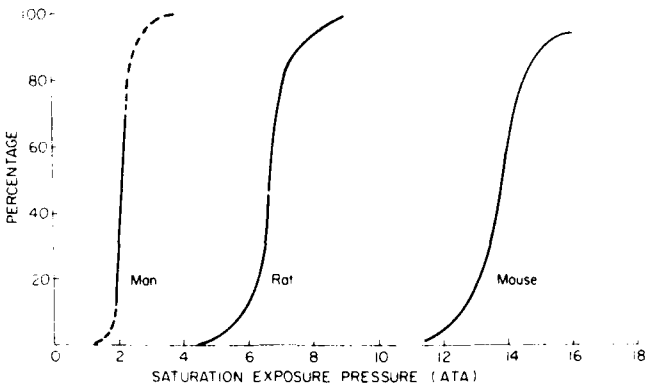

Fig. 21. Tolerance of different species to pressure change after a saturation exposure.

In an initial effort designed to help define the pressure reduction (ΔP) problem, we conducted some demonstration dives. We first exposed mice (40 mice per exposure) to 16 ATA breathing an oxygen-nitrogen mixture (O_2 = 0.5 ATA). After 30 minutes, the mice were abruptly decompressed 15 atm to 1 ATA. One hundred percent of the animals were stricken with decompression sickness. On the second exposure the mice were saturated at 17 ATA and abruptly decompressed to 2 ATA. This is also a 15-atm change in pressure, but it resulted in an incidence of decompression sickness of only 40%. A third saturation exposure to 18 ATA with a subsequent 15-atm decompression to 3 ATA resulted in no decompression sickness (Fig. 22). The message in this demonstration is that we are not dealing with an absolute amount of pressure change, but rather with one relative to the starting and terminating pressures. This is not a revolutionary finding. Haldane had informed us back in 1908 that we were dealing with a constant ratio. To evaluate this Haldanian idea we saturated mice at 13.8 ATA (P_1) on a nitrogen-oxygen mixture. We then decompressed them to 1 ATA (P_2). According to one of our previous studies, this ratio of 13.8 to 1 should produce a 50 percent incidence of decompression sickness. In actuality, we got a 57 percent incidence of decompression sickness (Figure 23). This value is within the acceptance limits of the expected result. We then saturated our mice at 20.7 ATA and subsequently decompressed them to 1.5 ATA, again a ratio of 13.8 to 1. This time, however, we obtained an incidence of 95 percent. It appears obvious that we are not dealing with a constant ratio. This finding is not particularly surprising. Researchers such as Workman, Hempleman, and others have suggested that the relationship between P_1 and P_2 is linear and less than a constant ratio. Regardless of whether one expresses the pressure reduction relationship as $P_2 = f(P_1)$ or $\Delta P = f(P_1)$ (the allowable change in pressure), the mathematical function appears to be linear for pressures (P_1) less than

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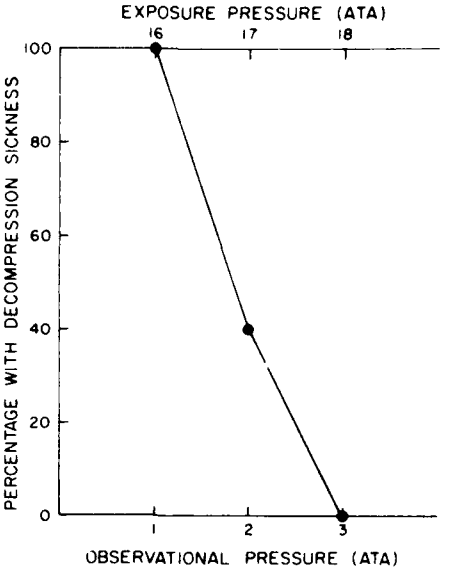

Fig. 22. Incidence of decompression sickness in mice saturated at 18 ATA and then decompressed to 3 ATA.

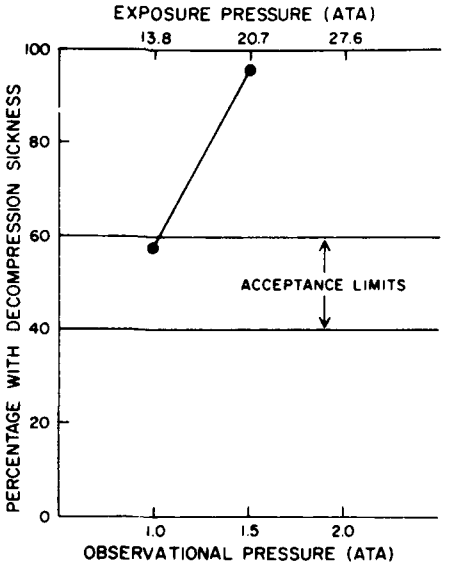

Fig. 23. Incidence of decompression sickness in mice saturated at 13.8 ATA on N_2-O_2 and then decompressed to 1 ATA.

10 ATA and perhaps for pressures as great as 30 ATA. These results, however, do not address the basic question in the above assumption: do we need a separate ratio or linear function for each theoretical tissue or will one single function suffice?

Bühlmann (see section 3 of this report) uses a separate surfacing ratio for each of his theoretical tissues and suggests that this is essential because of their gas exchange characteristics. Hennessy and Hempleman agree that more than one function is necessary to describe pressure reduction tolerance. They suggest that due to the differences in gas solubility between fatty tissue and the fluid of the vestibular and auditory organs, two linear equations are going to be necessary. The results of our small-animal studies suggest that only one pressure reduction equation is necessary, but because of the size and the high metabolic rates of the animals we are using (rats) we may not be confronting the diversity of gas solubilities found in man. D'Aoust, Vann and Yount all use a single equation for describing pressure reduction tolerance. There are differences among their equations but these will be dealt with under the next assumption (No. 7) because they concern the range of pressures over which the equations can be applied.

The pressure reduction question and its description are vital in decompression theory. They are the points of departure for two of the current theories. Vann points out that there have been two principal hypotheses offered to explain bubble formation. These are:

1) Critical supersaturation (or random nucleation)

2) Pre-existing gaseous micronuclei

Which of these hypotheses is adopted will govern how one thinks about the pressure reduction problem. Regardless of which concept is used, however, the empirical results obtained by reducing pressure rapidly are the same, and therefore should lend themselves to a single mathematical description. It should be relatively easy to determine if we need multiple pressure reduction equations by experimenting with animals larger than the rodents presently being used. The chairman personally feels if P_1 is the pressure level to which the body is equilibrated and it is allowed to change with time, a single pressure reduction equation should be sufficient. This concept works with small rodents and needs to be verified on large animals.

SUMMARY

The consensus of opinion of those attending the Workshop seemed to be that a series of ratios is not the best way to describe pressure reduction tolerance limits. The linear model presented by Hennessy and Hempleman appears to describe adequately the available data for exposure pressures (P_1) less than 10 ATA and it may be good to pressures as great as 30 ATA. The question that remains unanswered is how many pressure reduction equations are necessary. The estimates vary between one and one for every theoretical tissue.

ASSUMPTION NUMBER 7.

The pressure reduction ratio for nitrogen-saturated tissue increases with increasing depth, but for helium-saturated tissues the ratio remains constant.

ASSUMPTION NUMBER 8.

Tissue supersaturation limits increase with increasing depth.

Assumptions 7 and 8 will be dealt with together because they are so similar. Assumption No. 7 addresses the pressure reduction differences between helium-oxygen and nitrogen-oxygen gas mixtures, while Assumption No. 8 deals with the relationship between exposure pressure (depth) and pressure reduction tolerance.

When the Haldanian model was extended to make it applicable to helium-oxygen gas mixtures, an interesting change took place. Workman (1965) continues the Haldanian idea that for nitrogen-saturated tissues there is a positive linear relationship between saturation pressure and pressure reduction tolerance; he further suggests, however, that for helium-saturated tissues, regardless of the saturation pressure, the tolerable pressure reduction remains constant (Fig. 24). With very little data available at that time, this was the conservative position to take. Treating the two inert gases differently is intuitively hard to justify, however. The physics of the situation should be very similar. We undertook a small animal study to evaluate the adequacy of this conservative approach. Mice breathing either $He\text{-}O_2$ or $N_2\text{-}O_2$ mixtures were decompressed from selected exposure pressures (P_1) to each of three observational pressures (P_2), 1.0, 1.5, and 2.0 ATA. The ED_{50}'s were determined by linear fits of the decompression results.

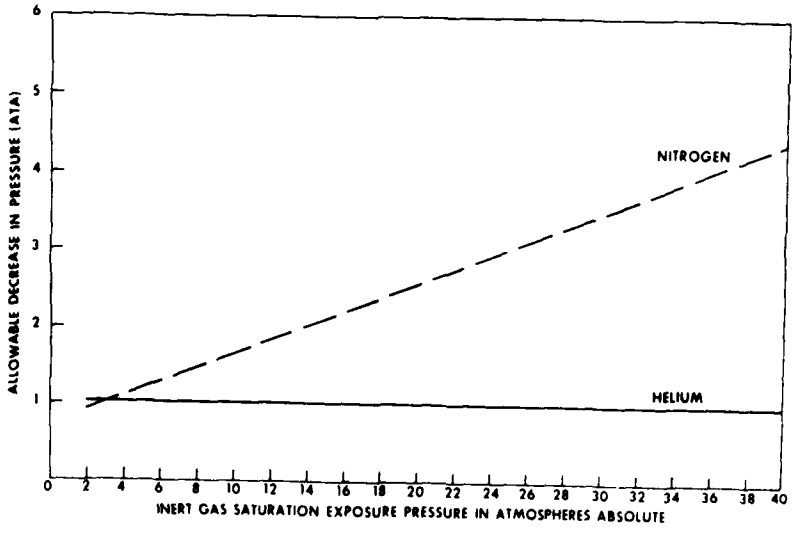

Fig. 24. Allowable decrease in pressure for various nitrogen and helium saturation levels (Workman model).

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A plot of the derived ED_{50}'s for $He\text{-}O_2$ and $N_2\text{-}O_2$ mixtures for three observational pressures (1.0, 1.5, and 2.0 ATA) is shown in Fig. 25. There is a linear relationship between P_1 and P_2, but it is somewhat less linear than the constant ratio proposed by Haldane. The important aspect of these findings is that the differences between $He\text{-}O_2$ and $N_2\text{-}O_2$ are not significant. Both inert gases act in essentially the same way. This is not a startling result, but merely a demonstration of the fact that assumption No. 7 does not hold.

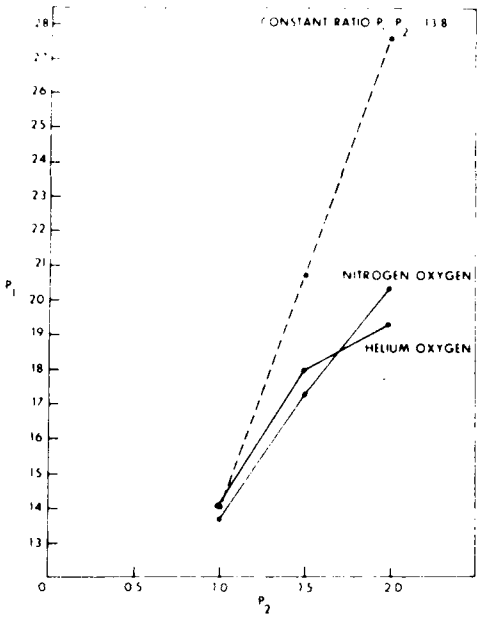

Fig. 25. Plot of derived ED_{50}'s for $He\text{-}O_2$ and $N_2\text{-}O_2$ mixtures at pressures of 1.0, 1.5, and 2.0 ATA.

The question of the linearity of the relationship between P_1 and P_2 or between P_1 and ΔP (tolerable pressure change) is still open to question. The debate is primarily over the range of exposure pressures (P_1's) for which a linear relationship is appropriate. Flynn has suggested that the relationship curves for low P_1's (Fig. 26). Hempleman and Hennessy have suggested that at some pressure, perhaps around 400 fsw, the relationship curves or a second linear function becomes critical. They suggest that this second function is related to gas exchange in the vestibular system. To check on the range over which a single linear relationship holds, a major study was undertaken using rats. ED_{50} pressure reduction values were experimentally derived for 12 different helium-oxygen exposure pressures, ranging from 6 to 60 ATA (Fig. 27). Between the exposure pressures of 6 and 43 ATA, there is a very strong positive linear relationship ($r = 0.99$); from 43 to 60 ATA, the relationship deteriorates and becomes much less precise ($r = 0.44$). There appears to be a qualitative change in the decompression symptoms manifested after an abrupt pressure reduction following a saturation exposure at deep depths. For rats the transition from a precise to a nebulous relationship occurs around 43 ATA, and the Spaur et al. (1976) report indicates that it may be around 35-37 ATA for humans. The best estimates of the pressure reduction tolerance for man after a $He\text{-}O_2$ saturation exposure are shown in Fig. 28.

At the one extreme we have the Haldanian idea of being able to halve the pressure; at the other extreme we have the Workman idea of limiting pressure reduction to 30 fsw regardless of the exposure pressure. The actual pressure reduction tolerances for man probably lie somewhere between these two extremes. The

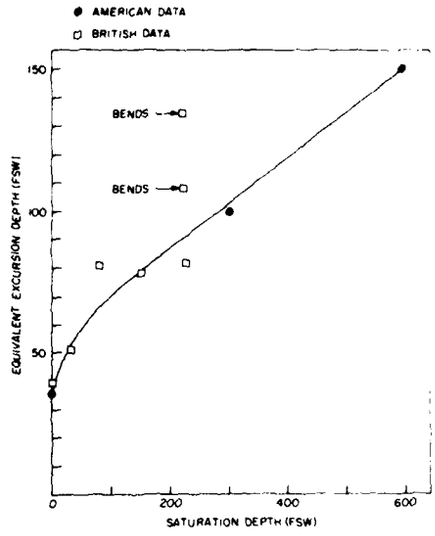

Fig. 26. Plots of British and American P_1 :P_2 ratios.

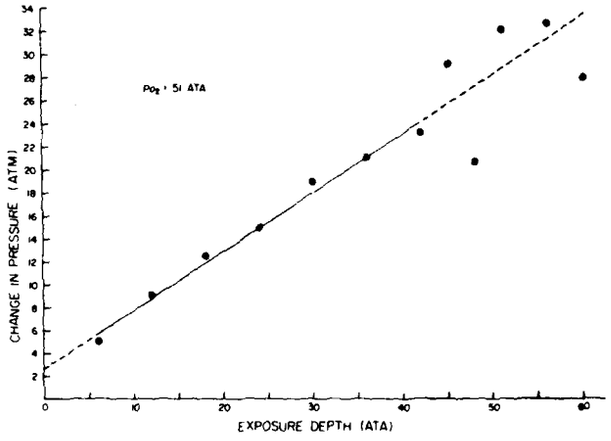

Fig. 27. Reduction in pressure necessary to produce decompression sickness in 50% of rats after an He-O_2 saturation exposure.

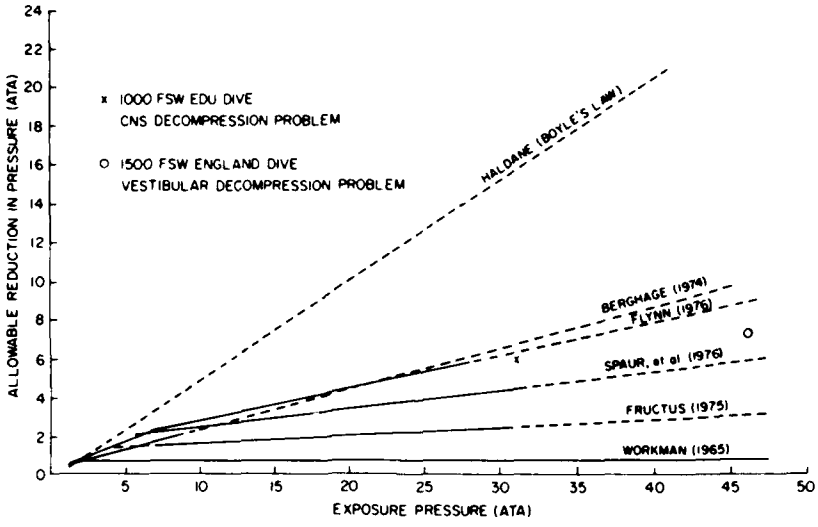

Fig. 28. Estimates of pressure reduction limits for man.

circle and the "x" on Fig. 28 are cases of decompression sickness that occurred on deep helium-oxygen saturation dives. The limits presently published in the U.S. Navy Diving Manual (derived from the Spaur group's report) are probably the best pressure reduction estimate for man now available.

One of the more interesting findings in the Berghage et al. (1976) rat study was a changing confidence interval associated with increased exposure pressure (Fig. 29). This idea has now been challenged by Vann

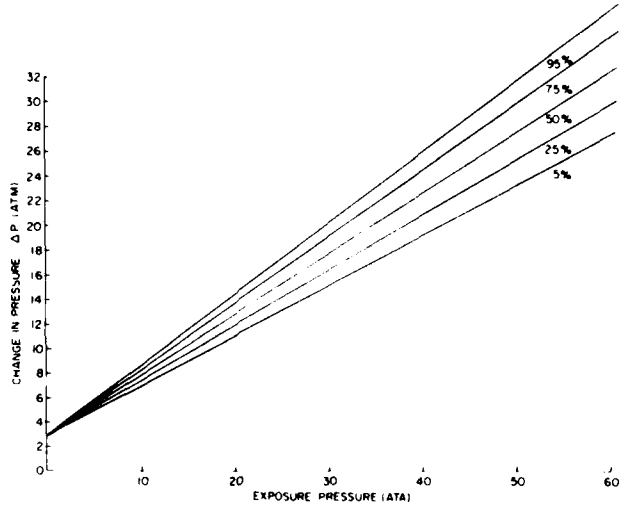

Fig. 29. Pressure reduction required to increase decompression sickness incidence from 5 to 95% for various saturation exposure pressures.

and co-workers. Through a subsequent statistical analysis of the rat data, Vann and his associates have concluded that the confidence interval lines should be parallel rather than diverging as shown in Fig. 29. An additional study or two will be needed to resolve this question.

A follow-up study designed to evaluate pressure reduction tolerance after subsaturation exposures was undertaken next. Results (Fig. 30) indicate that pressure reduction limits after a short 10-atm excursion are shifted upward an equal amount for all saturation pressures. The results of this study have allowed us to develop a model for predicting pressure reduction tolerance limits for rats after any time/pressure combination. Although we have achieved this capability with rats, we are still a long way from being able to do it for humans. Until we can do this for man we can not pretend we understand the decompression phenomenon.

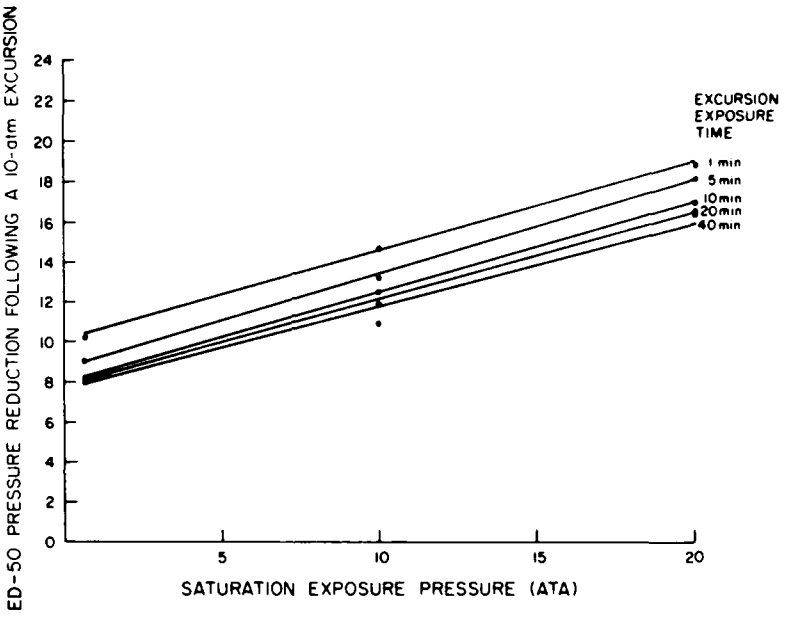

Fig. 30. Pressure reduction tolerance after 10-atm excursions from various saturation pressures.

When I started the discussion of pressure reduction, I suggested that dealing with pressure differentials rather than pressure ratios would be a more parsimonious way of describing pressure reduction. Now that we have demonstrated with experimental data the existence of a linear relationship between pressure reduction and at least part of the exposure pressure range, it is important to point out that had we used pressure ratio in these studies we would have had a quadratic function (Fig. 31). Investigators in the field are encouraged to abandon the idea of supersaturation ratios and pressure reduction ratios in favor of pressure differentials. It makes it much easier to quantify the relationship between P_1 and P_2 and/or ΔP.

David Yount agrees (see his paper in Section III) that there is a positive relationship between exposure pressure and pressure reduction tolerance. He suggests, however, that it is slightly curved rather than linear and that for extremely long exposures the pressure reduction tolerance approaches the values normally found at 1 ATA. In other words, for very long exposures Dr. Yount is saying that pressure reduction tolerance becomes independent of exposure pressure. This concept is a rather basic one for the bubble nucleation theory and it should be rather easy to evaluate. The validity of this concept should be tested by someone before we go much further: the limits of pressure reduction are an essential concept for decompression theory.

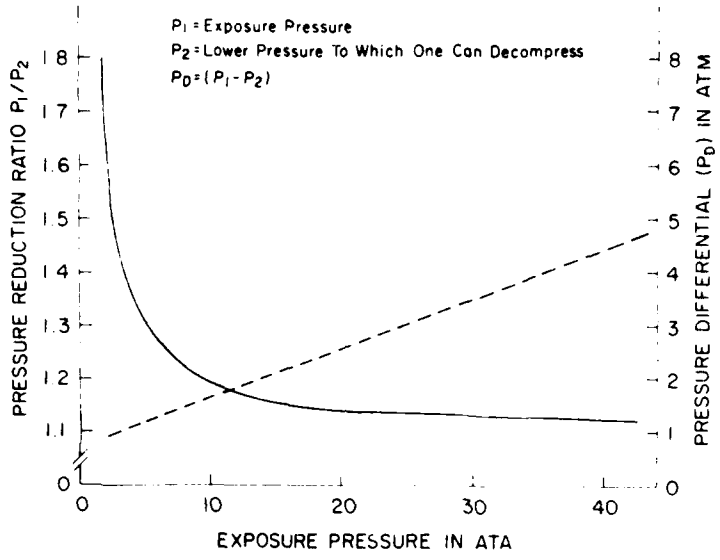

Fig. 31. Shape of quadratic function pressure reduction ratio and pressure differential.

Brian D'Aoust states that virtually all empirical experience except that for very long dives supports the idea of an increasing pressure reduction tolerance with increasing exposure pressure. No comment was made, however, concerning the shape of the relationship. Dr. D'Aoust suggests that the Hennessy and Hempleman critical volume model explains this relationship quite well. He also feels that the Yount nucleation theory appears sound in its interpretation of the facts (see Dr. D'Aoust's paper in Section III). The two models, however, come to different conclusions concerning the shape of the relationship between exposure pressure and pressure reduction tolerance. The Hennessy-Hempleman model is based upon a linear relationship, while the Yount model produces a curved relationship. The two models become compatible when Hennessy and Hempleman add a second linear relationship to their model for deep dives. It is suggested that this second relationship is necessary because of a shift in the anatomical site affected by decompression. The consensus seems to be that for shallow dives (exposures to pressures of less than about 20-30 ATA), a single linear function accurately describes pressure reduction tolerance. For deep dives the picture becomes less clear. Some additional research is needed in this area.

ASSUMPTION NUMBER 9.

Pressure differentials can be maintained indefinitely.

Although this question is becoming more and more important as decompressions become longer and longer, very little research information is available on the topic. We have done a single study on the question and it suffers from the tenuous nature of the underlying assumptions. But because it is the only study I know of that deals with the question, I will present the results here. Male guinea pigs were compressed to one of three exposure pressures, 23.7, 46.4, or 61.6 ATA. After a 1-hour saturation exposure a P_D (pressure differential) was established by an abrupt decompression. The established P_D was maintained during the remainder of the ascent, or until all three animals died. The constant P_D was maintained by reducing the ambient pressure at the same rate at which gas is eliminated from the body. Body gas elimination was calculated on the basis of a tissue half time of 10 minutes. The 10-minute half time was selected because of previously reported data on gas uptake and it was assumed that gas uptake and elimination curves were symmetrical in shape. This assumption, as will be pointed out in the next section, is probably not correct. With this short-coming in mind, we can present the results shown in Fig. 32. The mean survival time (MST) for the animals is related to both exposure pressure and induced P_D. The greater the exposure pressure the longer the MST. The open circles in Fig. 32 are for the 61.6-ATA exposure. The solid circles and open triangles are for the exposures at 46.4 and 23.7 ATA, respectively. The highly significant relationship between the P_D's and mean survival time is an indication that there are finite time limits for any given P_D. The time limits over which one can maintain a given pressure differential need to be evaluated for humans because of the implications for saturation decompressions.

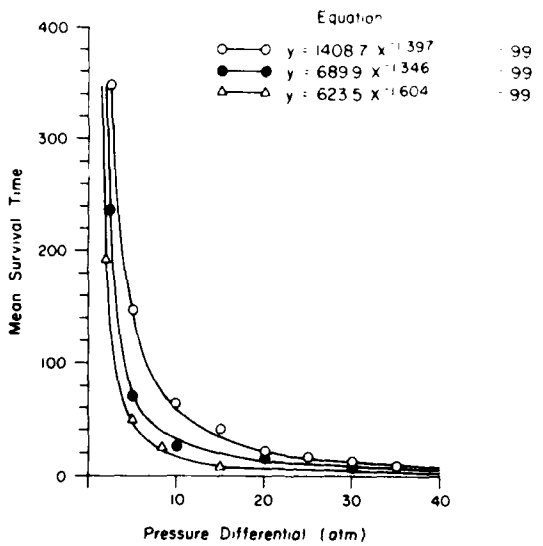

Fig. 32. Relationship between mean survival time and pressure differential.

Bill Hamilton suggested that pressure differentials have finite exposure periods by pointing out that the computation procedures proven on shallow, short dives rarely work when extrapolated to deeper depths or longer times. Existing data clearly indicate that the neo-Haldanian model does not work without modification across the entire range of pressure exposures now known to be tolerable for man.

SUMMARY

Very little is actually known about the time limits for differential pressure exposures. The body rapidly equilibrates after any pressure change and therefore the time limits associated with the differential pressure are rarely confronted. It has only been with the advent of the saturation dive and its associated extended decompression that the question has been raised. What evidence is available indicates that extended decompressions do produce problems. Some well-designed and controlled studies are needed to improve our understanding of the problems of long decompressions.

GAS ELIMINATION ASSUMPTIONS

ASSUMPTION NUMBER 10.

Gas uptake and elimination are symmetrical relationships.

This assumption is a basic element in Haldane's original model. However, experimental evidence has been presented by both Hempleman and D'Aoust that casts some doubts on the validity of this postulate. It has been suggested by these authors that gas elimination might be affected by what was termed "silent bubbles." It was intimated that the dynamics of bubble formation and growth will alter the gas elimination process. In a recent study in our laboratory, we found that for rats saturated at 30 ATA, the time required to re-establish equilibrium between the body and the ambient pressure environment after a pressure reduction was 10 times longer than it was after a pressure increase. In a subsequent study it was discovered that the time required to re-establish equilibrium after a pressure decrease is related to the ambient pressure (Fig. 33). The lower the hydrostatic pressure, the greater the time required to re-establish equilibrium between the body and the ambient environment. It is apparent that time spent at greater pressures is more efficient in removing gas than time spent at shallow decompression stops.

A secondary observation made during the gas elimination studies in our laboratory indicates that there may be an optimum time for each decompression stop (Fig. 34). After a pressure reduction there is an initial period in which the organism is at a disadvantage for any subsequent decompression. The test subject must stay at the decompression stop some fixed amount of time before his condition is sufficiently improved to continue decompression. It appears there is an optimum stop time during which the test subjects experience their greatest rate of improvement. Beyond this optimum stop time, their condition continues to improve, but at a slower rate. If this observation is true, then the efficiency of a stage decompression could be improved by utilizing the optimum times associated with the maximum rate of gas exchange.

In his comments in Section III of these proceedings, Behnke suggests that the saturation uptake curve is symmetrical with the desaturation curve. Weathersby presents data in Section III that support Behnke's position; he has found that inert gas uptake and elimination can be characterized by the same response function.

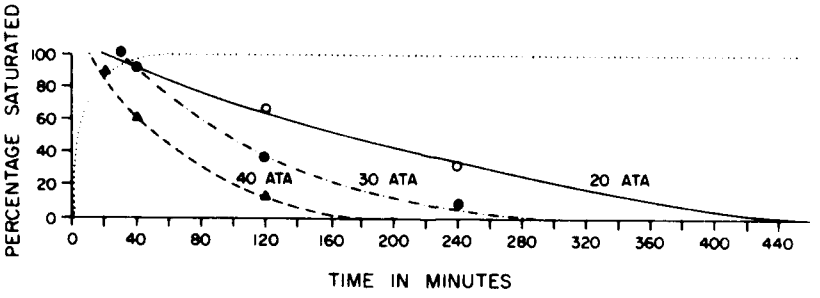

Fig. 33. Relationship between time to equilibrium after a pressure decrease and ambient pressure.

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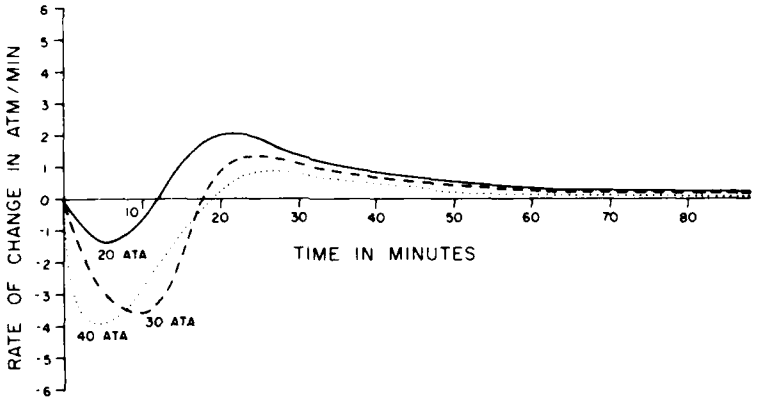

Fig. 34. Relationship between rate of gas exchange, pressure, and time.

D'Aoust suggests that the symmetry between gas uptake and elimination only holds for isobaric conditions. He has experimentally demonstrated that the rate of gas elimination can be drastically reduced by decompression. Both Hills and Vann support this idea and point out that as soon as a stable gas phase is formed the rate of elimination is greatly diminished. Hills suggests that only gas in true physical solution can contribute to the driving force for gas elimination. Vann feels that the drive force for removing inert gas from tissue is the inherent unsaturation (partial pressure vacancy) plus the inflation pressure in the gas nucleus due to tissue elasticity. The rate at which dissolved gas is removed from tissue depends upon the inspired oxygen partial pressure (PI_{O_2}) and the blood flow.

SUMMARY

The laboratory evidence presently available indicates that the normal isobaric symmetry between gas uptake and elimination is altered during the decompression process. The differences between gas uptake and elimination time are probably due to the presence of the free gas that is routinely produced during decompression and the body's hematological response to this foreign substance. The rheological syndrome

(reported by D'Aoust et al. and Wells et al.) may be the single most cogent variable for future decompression research. Unless we can maintain circulation intact, it will be impossible to decompress living organisms safely and expeditiously.

The existence of an optimum decompression time needs to be verified on something other than saturated rats. If this idea is confirmed, it could be coupled with an optimum pressure reduction scenario that maximizes gas exchange and minimizes hematological changes and the time/pressure combination utilized to produce an idealized decompression schedule.

ASSUMPTION NUMBER 11.

Oxygen breathing enhances gas elimination by increasing the pressure differential between the tissues and alveolar air.

For over 70 years decompression has been facilitated by the use of elevated oxygen partial pressures. Oxygen has been administered even though little is known about the proper dosage or the way in which the benefit occurs. Two general concepts have developed out of this historical use: 1) The decompression advantage available by using oxygen seems to be proportional to the amount of inert gas replaced by oxygen (which is the basis of the equivalent air-depth decompression procedure); and 2) Oxygen breathing during decompression increases the gradient or partial pressure difference between the tissues and the alveolar air, and therefore improves the inert gas elimination process. Behnke called this latter concept the "oxygen window" method of decompression (1967). Both of these ideas are based upon the chemical and physical properties of the gases (Fick's, Graham's, Dalton's and Henry's Laws). No account is taken of the physiological dynamics of the situation. Few would deny, however, that the body's response to elevated oxygen partial pressures can alter gas exchange and can affect the effective use of oxygen.

We have attempted experimentally to test these evolved concepts. To test the equivalent air depth (EAD) theory, we replaced the inspired inert gas with oxygen in quantifiable increments to determine if the pressure-reduction bends threshold was increased in a corresponding fashion. Several different exposure times were used in the study to evaluate the time course for gas uptake. We were interested in knowing if the level of inspired oxygen would alter the time needed to reach a steady-state condition (saturation). The results shown in Fig. 35 demonstrate that the time course for gas uptake is unaltered in any systematic way by the inspired oxygen partial pressures used. The incremental change in pressure-reduction tolerance does not correspond to changes in oxygen partial pressure either.

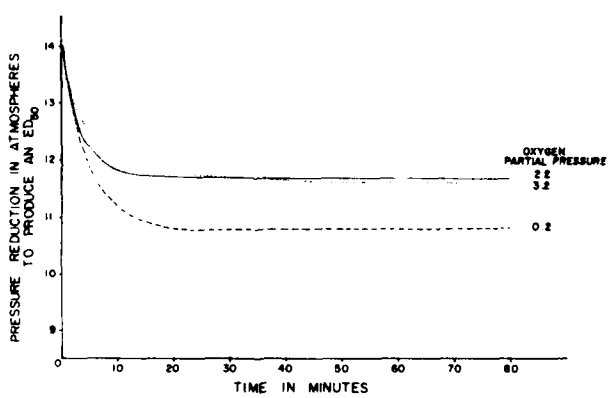

Fig. 35. Relationship between PO_2 and time to reach steady state.

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It would appear that the theoretical assumption that underlies the EAD concept lacks experimental support. With the total pressure held constant at 15 ATA, the trade-off between oxygen and the inert gas does not appear to produce a corresponding linear change in pressure-reduction tolerance. Admittedly, these results are based upon research using rats as subjects, and we do not fully understand the decompression relationship between rats and man. Also, the oxygen partial pressures in the study are higher than those normally used. Small increases in the oxygen partial pressure breathed by large animals do initially result in increases in pressure-reduction tolerance. There is obviously some decompression advantage gained by breathing elevated oxygen partial pressures. It does not appear, however, that subtracting it from the total gas present is an accurate way of describing its contribution. Oxygen is physically and biologically active, and its handling in the decompression problem is probably much more complex than present theories would have us believe.

The historical literature indicates that there is an envelope or narrow range of oxygen partial pressures that can be used. If the oxygen is too low, the incidence of decompression sickness increases; if the oxygen is too high, oxygen poisoning becomes a problem. Our second study was designed to explore this oxygen envelope and to define the relationships between oxygen partial pressure, exposure time, and pressure, and to delineate their effects on pressure-reduction limits.

Figure 36 graphically displays the relationship between these three independent variables. The relationships shown in Fig. 36 are least-squares best-fit polynomials between oxygen partial pressure and ED_{50} pressure-reduction values. The results support the idea that there is an optimal level of oxygen to breathe during a dive to facilitate decompression.

The optimum oxygen level appears to be affected by the exposure pressure, the exposure time, and an interaction between the two. As shown in Fig. 37, the greater the exposure pressure the lower the optimum oxygen level; the longer the exposure time, the lower the ideal oxygen level.

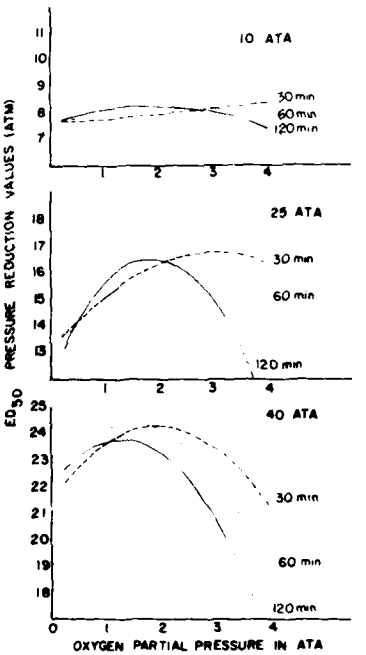

Fig. 36. Relationships among PO_2, exposure time, and pressure.

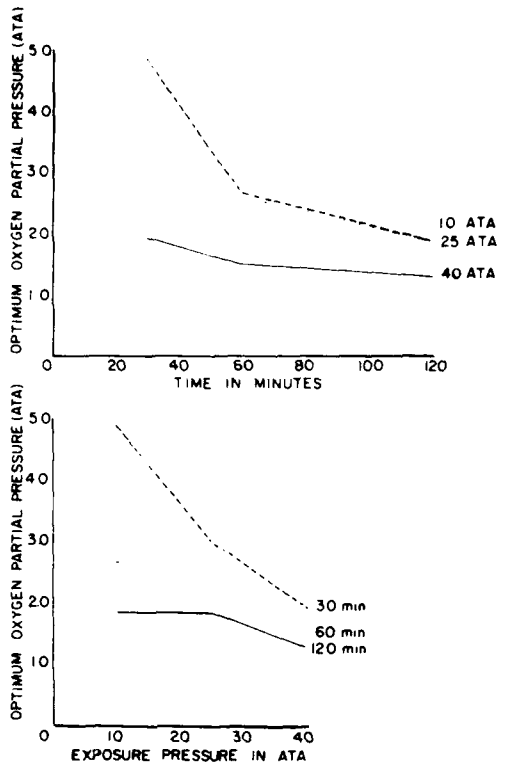

Fig. 37. Relationship between optimal O_2 level and exposure pressure and duration.

Between the extremes of anoxia and CNS oxygen toxicity, there is an envelope of usable oxygen partial pressures. The size of the oxygen envelope depends on the ambient hydrostatic pressure and the exposure time. For short "shallow" exposures, the envelope is quite large; for long "deep" exposures, the envelope is rather restricted. Within each oxygen envelope there is an optimum level that can facilitate decompression.

In commenting on Assumption No. 11, Brian D'Aoust agrees that this explanation for oxygen's effectiveness is logical, but he notes that it seems oversimplified. It does not take into consideration the vasoactive effects of oxygen. David Yount also has serious reservations about the mechanisms involved. All agree, however, that oxygen can be used beneficially in the decompression process.

SUMMARY

The Workshop exchange on this topic did little to elucidate the mechanisms involved in oxygen's initial beneficial and subsequent detrimental effects. It is assumed that the benefits derived from higher oxygen levels is the result of the action of the two principles previously mentioned, i.e., replacement of the inert gas, and gas-exchange facilitation; however, neither of these concepts can fully explain the existing experimental results. It is obvious that oxygen can be used beneficially during decompression, but at what dose is unknown. It now appears that there is an optimum level of oxygen, one that is affected by both ambient pressure and exposure time. By keeping the oxygen level close to optimal, it may be possible to expedite future decompressions.

ASSUMPTION NUMBER 12.

The concept of residual nitrogen time is sufficient to explain repeated exposures.

The research outlined under Assumption No. 10 indicates that gas elimination after decompression is a *much slower process* than gas uptake. If these research findings can be extrapolated from small rodents to man, the present concepts concerning the surface interval between dives are no longer valid. Calculation of residual nitrogen time is based upon the idea that gas uptake and elimination are symmetrical relationships, and this does not appear to be true.

Another aspect of this problem is what has loosely been called "adaptation"; this idea states that an organism changes during exposure to high pressures and is in some way less sensitive to the stresses of decompression. There is some statistical evidence to indicate that this process actually takes place. We have conducted one small pilot study to investigate this phenomenon. The study involved exposing mice on air for 30 minutes to either 6 or 14 ATA. After the adaptation exposure, the animals were slowly decompressed to the surface. The number of adaptation exposures varied, depending on the experimental condition being investigated. After remaining on the surface for a fixed period of time, the animals were exposed to a test dive to 14 ATA for 30 minutes. After the test exposure the animals were abruptly decompressed to the surface (1 atm/sec), where they were evaluated for decompression sickness. As shown in Fig. 38, the incidence of decompression sickness decreased as the number of adaptation exposures increased.

David Yount suggested that the idea of *residual nitrogen time* is inadequate for explaining repetitive dives, for many reasons (see his comments in Section III). He feels the subject's previous pressure exposure

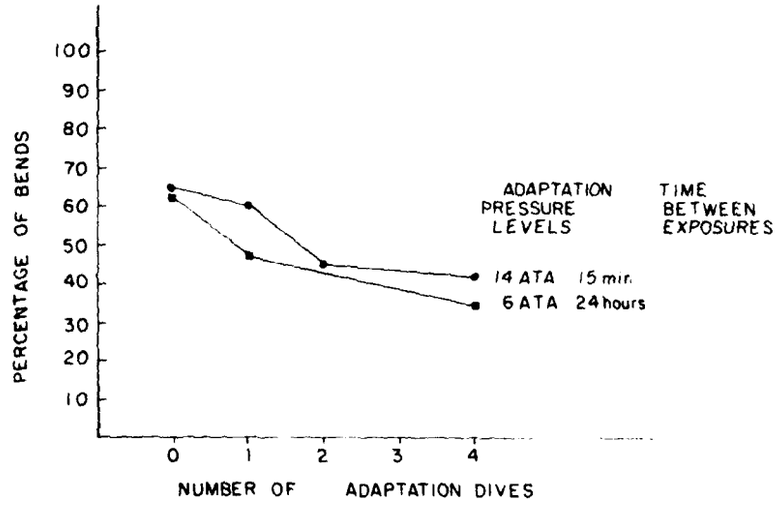

Fig. 38. Effect of repeated exposure on incidence of decompression sickness.

history must be taken into consideration. Brian D'Aoust suggested that the residual nitrogen time concept could be useful if it were modified to include previous pressure history, nuclei regeneration, and other inert gases.

SUMMARY

Statistical and experimental data presently available support the idea that an organism changes physiologically with repeated exposures to high pressures and decompression. This adaptation process complicates the calculation of decompression schedules for repetitive dives. The historical concept of residual nitrogen time is an oversimplification of the problem and does not adequately describe the process. Additional experimental work is needed on this topic.

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SECTION III

Workshop Contributions

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WORKSHOP COMMENTS

Albert R. Behnke

PART I. RESPONSES TO THE ASSUMPTIONS

The Decompression Theory Workshop was structured to discuss specific propositions and problems, and the following statements comprise concise responses to these assumptions. Part II of this paper will consist of a systematic compilation of substantive material pertinent to the improvement of Navy decompression practice.

GAS UPTAKE

Assumption 1. Multiple tissues or tissue compartments must be used to describe whole body gas exchange.

This is true, unfortunately. In the absence of quantitative data to describe whole body gas exchange, and failure to implement Brian Hills' recommendations in the section of the *National Plan* dealing with quantitative gas transport, our only recourse is a mathematical model of tissues and half times that does not require a knowledge of the quantity of gas in different tissues and compartments. "Proponents of mathematical models with some justification state that when we know more about the mechanism of inert gas transport in the human body, of gas phase separation and behavior, we may be able to quantify decompression with confidence in the consequences." Whatever the sophistication of the mathematical system, the results are obtained by trial and error. The present goal is a composite one, a compromise between safety and efficiency.

Assumption 2. Tissue gas uptake is described by the following items: exposure time, pressure differential, and tissue half times.

This is true. One problem is variation in gas uptake due to wide differences in cardiac output, in blood perfusion of tissue beds, and in the size of the body's gas reservoir.

Assumption 3. Only inert gas partial pressures need be considered in the decompression problem.

This is not true. All gases, both inert and metabolic, as well as water vapor, contribute to overpressure and the size-composition of bubbles formed in the process of phase separation. In altitude ascent, for example, exercise attended by increased CO_2 production greatly augments the incidence and severity of decompression sickness.

Assumption 4. Breathing multiple inert gases . . . .

I have no comment on this assumption.

Assumption 5. Compression procedures . . . .

To depths of 500 feet, a descent rate of 100 ft/min was routine in an earlier era.

PRESSURE REDUCTION

Assumption 6. Tissue supersaturation limits are best described by a series of pressure reduction ratios.

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The pressure reduction ratios for various tissues are part of the mathematical strategem for computerization of data in the absence of systematic quantitative knowledge of gas transport.

Assumption 8. Tissue supersaturation limits (pressure reduction ratios) increase with increasing depth.

The opposite is true: ratios decrease with increasing depth but ΔP values increase. At a 1000-ft depth, for example, the decompression ratio may be 1.1, a reduction of 1.58 to 1 at 2 ATA, while ΔP increases from 23 ft (at 2 ATA) to 92 ft at 31 ATA (1000 ft).

Assumption 9. Pressure differentials during decompression can be maintained indefinitely.

This is only true if tissue inert gas pressure does not exceed ambient pressure, i.e., if decompression is isobaric in accord with the 'oxygen window' principle. There is no proof, by contrast, that an overpressure of inert gas can be sustained during the course of decompression without cumulative accretion of bubbles.

The U.S. Navy decompression table for saturation diving is an empirical example of the fact that even an apparently isobaric rate of decompression cannot be sustained uniformly for extended periods. In the section in Part II dealing with percutaneous diffusion of helium into the body, I have outlined a glaring complication in the decompression of divers.

GAS ELIMINATION

Assumption 10. Gas uptake and elimination are symmetrical relationships only when the saturation uptake curve is compared with the desaturation curve. Remarkably, the body appears to behave as a unit such that the time for gas elimination after partial saturation is equal to the time required for gas elimination after complete saturation. (This condition is described more fully in Part II.)

Assumption 11. Oxygen breathing enhances gas elimination by increasing the pressure differential between the tissues and alveolar air.

This is emphatically true, and it does so most effectively between pressure equivalent levels of 33 and 60 feet. Benefit from oxygen, however, is restricted to about one-half of its theoretical effectiveness because of peripheral vasoconstriction and a decrease in tissue perfusion. An Oxygen Table for decompression of workers in compressed air and for long exposures of divers in air at depths of 90 ft or less is presented in Part II (see Oxygen Decompression Tests section and Table 7).

There is some quantitative data (Aeromedical Laboratory, Brooks Air Force Base) that periodic interruption of oxygen inhalation by 'air breaks' as short as five minutes appreciably augments nitrogen storage. The technique of oxygen inhalation is of major importance and will be dealt with in Part II.

Assumption 12. The concept of residual nitrogen time is sufficient to handle repeated exposures.

Thousands of dives in connection with the University of California-Scripps Institution unequivocally support this concept. The dives, however, have either been no-decompression or of relatively short duration. Under conditions of diving in which high overpressures of inert gas are maintained, e.g., ratios of 1.6 to 1, as in the tests of Bühlmann, Frei, and Keller, the half time for 'slow' N_2 elimination was not 120 minutes, which is the period implicit in USN residual nitrogen time, but 480 minutes. The concept of residual N_2 time may be vitiated in part if appreciable quantities of gas evolve from tissues as bubbles in the circulation.

PART II. SOME QUANTITATIVE ASPECTS OF DECOMPRESSION PROCEDURE

Although investigators generally concede the need for quantitative studies of gas transport in accord with the outline in Brian Hills' section of the *National Plan* dealing with the exchange of inert gases, oxygen, and carbon dioxide, it is in the striking absence of such knowledge that empiricism alone has produced our decompression schedules.

In the mathematical formulation of inert gas transport, the number of compartments or body tissues in some models is projected to be as high as 15 (range of half times from 5 to 1280 minutes) for nitrogen. Since the longest empirical estimate of an N_2 half time is 480 min (from the chamber tests of Bühlmann, Frei, and Keller 1967), a value of 1280 min is well out of the range of probability.

It is worthwhile, therefore, to present in Tables 1, 2, and 3 an estimate for the slowest saturating tissue (in our concept, fat) in relation to the quantified aqueous and fat solvents of the body for inert gas, in the light of regional distribution of blood flow.

The following material and tables emphasize quantitative data and elementary formulations (in the tables) in the analysis of decompression data, and are arranged as follows:

1. Summary of guidelines from earlier studies.
2. Experimental determination of desaturation time for slowest tissue, depicted in part by four figures.
3. Requirement for inert gas recovery breathing oxygen.
4. The importance of percutaneous diffusion of inert gases.
5. Tables pertinent to this Workshop.
   a. Formula for the calculation of unlimited duration heliox depth excursions from saturation depths (Tables 4, 4A).
   b. Calculation of the duration of limited excursions from saturation depths (Tables 5 and 6).
   c. Calculation of excursions in compressed air (Table 7).
   d. A table of isobaric decompression from saturated air depths (Table 8).
   e. A prototype oxygen table and outline of effective techniques of administration.

GUIDELINES EVOLVED FROM EARLIER STUDIES

The Question of Supersaturation. "Although for the diving depths and times of exposure with which Haldane was concerned the 2 to 1 ratio appeared to be satisfactory, subsequent caisson and diving experience, particularly following saturation exposures or following dives to deep depths, has indicated with certainty that no one ratio is applicable for all tissues, and that probably no degree of supersaturation is maintained in the body during decompression. It may well be that bubbles form as soon as a state of supersaturation is initiated and what appears to be a ratio of saturation tolerance is in reality an index of the degree of embolism that the body can tolerate. . . . Even under ideal conditions the supersaturated state of gases in liquids is extremely unstable and bubble formation consequently unpredictable (Behnke 1951)."

"The proof that the absolute pressure can be halved is not demonstrable by short exposures up to 20 or 30 minutes since during so short an exposure the body is only partially saturated with fat containing less nitrogen than body fluids. . . . Tests for the validity of the assumption that the absolute pressure can be safely halved must therefore be made after the nitrogen tension in the body has come into equilibrium with the nitrogen tension in the lungs (Behnke 1937)."

Table 1. Body composition data relative to inert gas content

Composition Analysis (kg)		Solubility Data*			
		P_{N_2}, P_{He} = 570 mmHg, 37°C			
Fat in adipose tissue	9.0	N_2	in body fluids	9	ml/kg
Lean body weight (LBW)	63.5	N_2	in fat	54	ml/kg
Essential organ, marrow fat	2.0	He	in body fluids	5.9	ml/kg
Fat-free weight	61.5	He	in fat	12.2	ml/kg
Total body water (TBW)	45	Inert Gas Content, 1 ATA			
Blood and interstitial	20	N_2	in fluids	405	ml
Intracellular	25		in fat	595	ml
				1000	
		He	in fluids	266	ml
			in fat	134	ml
				400	

Reference diver's characteristics: age, 24-29; height 174 cm; weight 72.5 kg: *Bunsen solubility coefficients at 37°C

	Helium	Nitrogen
Water	0.0087	0.0125
Oil	0.0148	0.0609

The tabular data incorporate corrections for the decreased solubility of inert gases in body fluids and for the density of fat. It is hoped that the data represent good approximations (dfluids 1.010 - 1.020; dfat 0.90 (lipid)).

Table 2. Groups of Tissues Relative to Their Weight, Nitrogen Content and Blood Perfusion Rate

	Tissue Groups				
	I	II	III	IV	V
	Brain Lungs Heart Kidneys	Muscle Skin Spinal cord Nerves	Bone* matrix	Bone** marrow	Fat in adipose tissue
Weight, kg	15.0	37.0	3.5	1.5	9.0
Blood perfusion rate, ml/min	4000	1200	80	50	400
N_2 content, ml	405 (fluids) 109 (organ fat)				486 (fat)

Reference diver's characteristics: age, 24-29; height, 174 cm; weight 72.5 kg. *Fat-free, mineral-free; blood supply to bone, speculative. **Fat in bone marrow and other organs, estimated at 2 kg.

Table 3. Representative Values for N_2 Elimination from a Reference Diver*

Time, min	Component (half time): 5 k:*** 0.14 N_2 in component, ml: 180	14 0.05 225	120 0.0058 595	Total 1000	% Desat. 0	RNPL** % Desat. 0
5	90	50	17	157	16	14.3
10	135	88	33	256	26	20.2
20	169	142	64	375	38	28.6
30	177	174	95	446	45	35.0
40	179	194	123	496	50	40.5
50	180	206	149	535	54	45.2
60	–	214	174	568	57	49.5
90	–	222	241	643	64	60.3
120	–	225	298	703	70	68.7
240	–	–	446	851	85	87.9
360	–	–	520	925	93	95.3
480	–	–	558	963	96	98.2
540	–	–	569	974	97	98.9
600	–	–	577	982	98	99.3
660	–	–	582	987	98.7	99.5
720	–	–	585	990	99	99.7

Reference diver's characteristics: age, 24-29; height 174 cm; weight, 72.5 kg. *During the course of continuous oxygen inhalation; after 6 hr, values are projected. **RNPL table: Empirical values based on diving data with an interweave of No-Stop decompression data. ***Rounded values for the rate constant k = 0.693/T/2. (RNPL data from the report of Crocker and Hempleman, March 1957, The Decompression Problems in Diving to 600 Feet.)

Table 4. Genesis of a Formula for the Calculation of Unlimited Duration Heliox Depth Excursions from Saturation Depths

The unlimited duration excursions from saturation depths of 150 to 820 feet represent a notable recent advance in extension of diving depths (Spaur, Thalmann, Flynn, Zumrick, Reedy, and Ringelberg 1978). The following calculations are presented for depth excursions of unlimited duration for comparison with experimental values:

Saturation Depth, fsw	Excursion Depths of Unlimited Duration, (fsw) Calculated*		Experimental
	(1)	(2)	
150	75	83	75
200	85	94	83
300	101	112	99
400	115	128	114
500	128	142	130
600	136	151	146
700	150	167	161
820	162	180	180
900	169	188	--
1000	178	198	--
1200	195	216	--
1400	210	233	--

*Calculated excursion depths for P_1 saturation depth: $\sqrt{P_1/P_2} \times K$ = excursion depth, fsw; P_1 and P_2 fsw + 33; P_2 = saturation depth, 1400 fsw + 33; (1) K = 210 fsw (unlimited excursion depth from 1400 fsw), (2) K = 233 fsw (unlimited excursion depth from 1400 fsw).

Below 500 fsw, (2) gives a better approximation of experimental unlimited excursion depth than (1), which underestimates excursions from deeper saturation depths. Hence, the elementary formula may require, for estimates of excursions from deep depths, the inclusion of Van der Waal's (a and b) constants or related modification.

Note: Formula (1) above can be restated as: Descending Depth = $5.55 \sqrt{P \text{ abs, ft}}$

Table 4A. Heliox Excursions of Unlimited Duration Deeper (E_d) and Shallower (E_s) than Habitat Depth (P) Followed by 'No-Stop' (One-Stage) Decompression to Habitat Steady-State Depth. Comparison of Experimental Data with Calculated Values for E_d and E_s Computed from a Square Root Formula and Two Linear Equations.

P, fsw	Excursions deeper than P depth			Excursions shallower than P depth	
	Experimental, fsw	Calc. (1)[a] fsw	Calc. (2)[b] fsw	Experimental, fsw	Calc. (3)[c] fsw
0	–	32	32	–	–
32	–	45	38	–	0
50	–	51	42	–	35
100	–	64	52	–	43
150	75	75	62	–	52
200	83	85	72	50	60
250	91	93	82	78	68
300	99	101	92	85	77
350	106	109	102	92	85
400	114	115	112	99	93
450	122	122	122	105	102
500	130	128	132	112	110
550	138	134	142	119	118
600	146	140	152	126	127
700	161	150	172	139	144
750	169	155	182	146	152
820	180	162	196	156	164

*Experimental data from Spaur et al. (1978). (1) a. $E_d = 5.55 \sqrt{P \text{ abs. ft}}$; (2) b. $P_d = 1.2\ P + 32$, $E_d = P_d - P$; (3) c. $P_s = 0.833\ (P - 32)$, $E_s = P - P_s$. (Data in this table were calculated and tabulated after the workshop at which this paper was presented.)

Table 5. Calculation of Depth Excursions (ΔP) Followed by No-Stop Decompression from the Surface and Habitat Saturation Depths for Periods of 25 to 60 Minutes

Conditions Divers at rest, oxygen 0.4 ATA
Rate of return to habitat depth to be determined

Formula Adjusted *Hempleman equation*, with application of the 'square root' principle.

$$\sqrt{\text{Time, min}} = \frac{K}{\Delta P, \text{ fsw}}$$

Surface K taken as 493

K (depth) = $\sqrt{P_s + 33}$ x K (surface) (P_s = saturation depth)

Depth (P_s), fsw	$\sqrt{P_s + 33}$	K (adjusted)	ΔP Excursions, fsw 25′	40′	60′
(Surface)	1.0	(493)	99	78	64
150	2.357	1162	232	184	150
200	2.657	1310	262	207	169
300	3.128	1542	308	244	199
400	3.622	1786	357	282	231
500	4.019	1981	396	313	256
600	4.379	2159	432	341	279
700	4.713	2323	465	367	300
820	5.084	2506	501	396	324
900	5.386	2655	531	420	343
1000	5.594	2758	552	436	356
1100	5.859	2888	578	457	373
1200	6.111	3013	603	476	389
1300	6.336	3124	625	494	403
1400	6.590	3249	650	514	419

Table 6. Calculation of Depth (ΔP) Excursions from Saturation Depths Followed by No-Stop Decompression for Periods of 60 to 360 Minutes

ΔP excursions for exposures of 60 to 360 min are computed from multiples of the ΔP for unlimited duration exposures. The multiples are derived from the projected desaturation time of a 60 min half-time 'slowest tissue' (for helium) as follows:

	Desaturation Time of 'Slowest' (Heliox) Tissue						
Time, min	360	300	240	180	120	90	60
% Desat.	98.4	96.9	93.8	87.5	75	64.6	50
Multiple	1.016	1.032	1.066	1.14	1.33	1.55	2.0

TABULAR DATA

Depth (P_s),		ΔP, fsw Excursions for the Following Periods, min						
fsw	Unlimited	360	300	240	180	120	90	60
(Surface)	32	32.5	33	34	36	43	50	64
150	75	76	77	80	86	100	116	150
500	128	130	132	136	146	170	198	256
820	162	165	167	173	185	215	251	324
1400	210	213	217	224	239	279	326	420

SUMMARY Syncopated Tabular Data Relative to ΔP Excursions from Saturation Habitats over the Whole Time-Depth Spectrum

		From Multiples of 60-min half time:					:From Hempleman's Adjusted Formula		
Depth (P_s),	Time, Unlimited	240	180	120	90	60:	: 60	40	25
fsw	min								
(Surface)	32	34	36	43	50	64:	: 64	78	99
150	75	80	86	100	116	150:	:150	184	232
500	128	136	146	170	198	256:	:256	313	396
820	162	173	185	215	251	324:	:324	396	501
1400	210	224	239	279	326	420:	:419	514	650

The unlimited excursion from the surface of 32 ft relates to an oxygen partial pressure of 0.4 ATA (equivalent to 13.2 ft). For a normoxic helium mixture (0.2 ATA), unlimited excursion depth would be reduced from 32 ft to about 25 ft, somewhat higher than the unlimited excursion depth from the surface for air.

Table 7. Calculated Duration of Excursions in Compressed Air from Relatively Low Saturation Depths

Saturation Depth, ft	K	No-Stop Decompression Excursion Time, min									
		25*	45*	60*	85*	100*:	:120	240	360	480	Unlimited
Surface	475	95	79	61	51	48 :	: 45	30	26	24	22.5***
20	602**	140	110	98	86	80 :	: 77	58	53	51	48.5
30	656**	161	128	115	101	96 :	: 92	71	65	61	61.0
40	706**	181	145	131	117	111 :	:107	85	78	75	73.5
50	753**	201	162	147	132	125 :	:122	98	91	88	86.0

*From Hempleman's formula for the first 100 minutes: excursion depth, ft = 475 $\sqrt{\text{T, min}}$; ** = author's adjustment of the surface (K) constant for habitat depths: Adjusted K = $\sqrt{P_2/P_1}$ x surface K; P_1 = 33 ft, P_2 is habitat depth + 33. *** Unlimited saturation excursion is 22.5 ft (10 psig) from surface (1 ATA) and is the bends-free pressure level for prolonged exposures in compressed air followed by no-stop decompression to surface (author's experience). $\sqrt{P_2/33}$ x 22.5 + habitat depth = depth for unlimited exposure at the respective P_2 habitat increment levels, where P_2 is footage absolute, or $\Delta P = 3.92 \sqrt{\text{P abs.}}$. Sub-saturation excursions (120 to 480 minutes) are computed from the percentage saturation of the 120-min (slowest tissue): 50% (120 min), 75% (240 min), 87.5% (360 min) and 93.8% (480 min). For example, at surface an excursion of 45 ft (22.5/0.50) may be made for 120 min. At habitat depth of 20 ft, an excursion to 77 ft (28.5/0.50 + 20) may be made for 120 min. Values are excursion depths (ΔP).

Table 8. Calculated Continuous Isobaric Decompression from Saturated Depths Under Conditions of Air Atmosphere, 20% Oxygen

Depth, fsw	Min	Depth, fsw	Min	Depth, fsw	Min
50	5.5	40		30	
33.5					
31.5	40.8		40.8		
29.5	46.2				
27.5	48.0				42.
25.5	49.4	25.5			
23.5	51.2	23.5	47.2		
21.5	53.0	21.5	53.0		
19.5	55.2	19.5	55.2		
17.5	57.2	17.5	57.2	17.5	
16.0	44.6	16.0	44.6	16.0	40.4
14.0	61.6	14.0	61.6	14.0	61.6
12.0	64.2	12.0	64.2	12.0	64.2
10.0	67.4	10.0	67.4	10.0	67.4
8.0	70.6	8.0	70.6	8.0	70.6
6.0	74.2	6.0	74.2	6.0	74.2
4.0	78.2	4.0	78.2	4.0	78.2
2.0	82.6	2.0	82.6	2.0	82.6
Surface	87.8		87.8		87.8
Total Time, hr	17.3		14.14		10.52

Data are computerized, and were requested by the Author and programmed through the courtesy of Dr. Hester at the Medical Research Laboratory in New London; controlling half-time tissues range from 5 to 200 min.

The Concept of "Silent" Bubbles. This concept, which postulates bubbles not associated with symptoms of decompression sickness, was based initially on the observation that after every decompression, minute intravascular bubbles were present. In 1939-1940, this impression was strengthened by altitude decompression tests in which oxygenation to promote nitrogen elimination was less effective at altitude than at ground level. In tests of Tobias et al. (1949), one subject, susceptible to bends on previous exposures to altitude, showed a definite slowing of krypton elimination during an altitude test in which he experienced no pain. This retardation could be attributed to the presence of "silent" bubbles (Behnke 1942b).

Isobaric "Oxygen Window" Principle of Decompression and Brian Hills' Zero-Supersaturation Concept. During the course of blood perfusion of the tissues, oxygen is unloaded in different quantities in the various tissues. The result of this transfer of oxygen is a space available for transport of inert gas in solution, designated by Momsen and medical officers of the Experimental Diving Unit (Behnke, Yarbrough, and Willmon) as the "partial pressure vacancy."

The Body Can Be Regarded as a Unit. "With the exception of organ tissues of high fat content (in bone marrow, spinal cord, myelin sheaths), the division of the body into tissues that saturate and desaturate at different rates is largely arbitrary, and the body can be regarded essentially as a unit (Behnke 1937)."

Short exposures in air at high pressures were equated with long exposures at low pressures. After high exposures for short periods, tissue pressures tended toward equality such that the duration of time required for inert gas elimination was approximately the same as decompression time after prolonged exposure.

In the experiments of Tobias et al. (1949), it is noteworthy that irrespective of the duration of the preliminary period of krypton inhalation, the half-time values of each of the three exponential components were nearly identical. Thus, after a 20-min period of krypton uptake, the half-time values recorded on desaturation were 6, 42, and 320 minutes. After saturation uptake, the half times recorded were 6, 41, and 310 minutes for the respective components.

These findings are in accord with nitrogen elimination in the dog (Fig. 3) and limited helium recovery data in man, namely that the tension of inert gas in various parts of the body (excluding bone-encased osseous and nervous tissue) tends toward equality during desaturation, irrespective of the degree of partial saturation.

Bone Considered a Separate Category. The high absorption coefficient of marrow for nitrogen and the sluggish, sinusoidal type of circulation with rigid obstruction to exit of bubbles in the dichotomous branches of the vein traversing the cortex "serve to make bones a trap for gas bubbles disseminated from the general circulation or forming in situ (autochthonous bubbles) in the marrow (Behnke 1942a)."

Danger of Too Rapid Ascent to the First Stop (1938-1940). "Symptoms indicative of embolism have appeared during helium-oxygen diving at depths of 180 and 90 feet. Ascent rate was then limited to 25 ft/min and an arbitrary period of 7 min was taken at the first stop in order to facilitate blood transport to the lungs of the large amounts of helium diffusing into the blood stream."

Experimental Determination of Desaturation Time for the Slowest Tissue

Campbell and Hill (1931) measured the nitrogen eliminated by man when oxygen was breathed for short periods of time, and contributed quantitative data with reference to nitrogen absorption in the brain, liver, and bone marrow of the goat. What prompted these studies at the time was the occurrence of accidents, chiefly after long exposures to high pressure. Subsequently, only a few tests have been conducted with human subjects for the purpose of nitrogen recovery for longer than 6 hr of oxygen breathing.

In lean men, nitrogen recovery during the course of oxygen breathing after nine hr was chiefly extraneous, from the inward diffusion of air-nitrogen into the recovery system and through the subject's skin from the atmosphere. Surrounding the body with oxygen eliminated percutaneous diffusion of atmospheric nitrogen, and nitrogen recovery from the lungs approached the limits of analytical error after 9 to 12 hours of oxygen inhalation. Figure 1 represents N_2 recovery from lean men. In current tests, the lung rinsing period in the early tests (5 min) could be reduced to one minute or less, but an analytical accuracy (for nitrogen in contrast to helium) of ± 1 part in 100,000 remains to be emulated.

In anesthetized lean dogs placed in an oxygen chamber, nitrogen elimination was apparently complete in 3 to 4 hr, with an end point of ± 3 ml per hr (Harvard tests of Shaw et al., 1932-1935). Figures 2 and 3 show typical results obtained, but this type of meticulous quantitative assessment, essential for an understanding of gas transport, has not been replicated.

In experiments done by Groom, Morin, and Farhi (1967), the half time of the slow compartment in awake dogs was 80 min, in contrast with 102 min in anesthetized animals. The determination of the end point of N_2 recovery and estimation of body nitrogen stores from the arterial-venous N_2 difference (and without determination of cardiac output) is subject to re-evaluation. The A-V N_2 difference is not defined precisely in such tests and the difficulty of obtaining a "blood-end-point" is evident from the following calculation. If ± 3 ml/hr is the end point for cumulative N_2 recovery from the lungs, it is apparent that 3 ml of N_2 in 132,000 ml of blood (60 x cardiac output of 2.2 liters estimated for the dog) is beyond the limit of technical accuracy in an analysis of a 10-ml sample of blood.

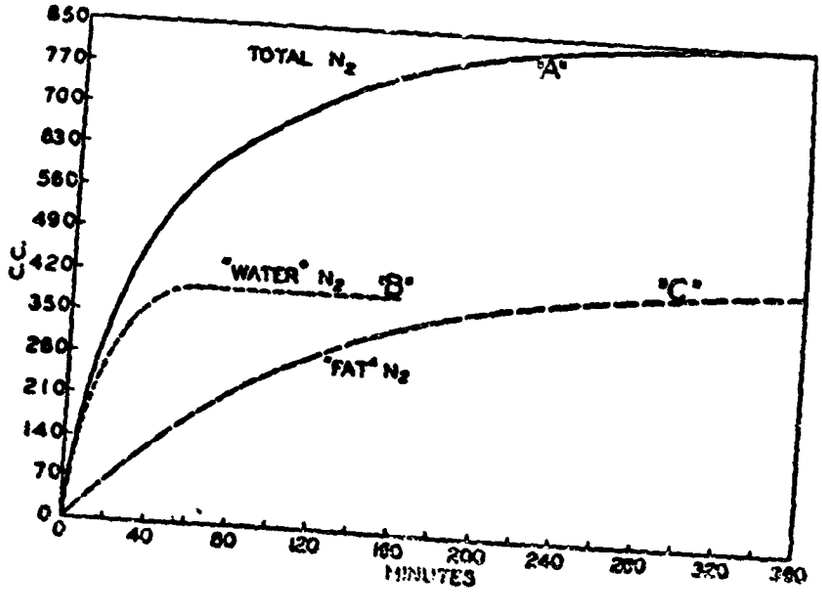

Fig. 1. Nitrogen elimination in man. Curve A represents average values for cumulative nitrogen from three lean men (average weight 64 kg) who breathed oxygen at atmospheric pressure in a helmet system. Water-N_2 (B) and fat-N_2 (C) are hypothetical curves that represent N_2 elimination from the chief body solvents. Nitrogen recovered during first 5 min (rinsing period) would add a third (faster) half-time component, while increased fat would add a fourth or even a fifth half-time component (Behnke 1937).

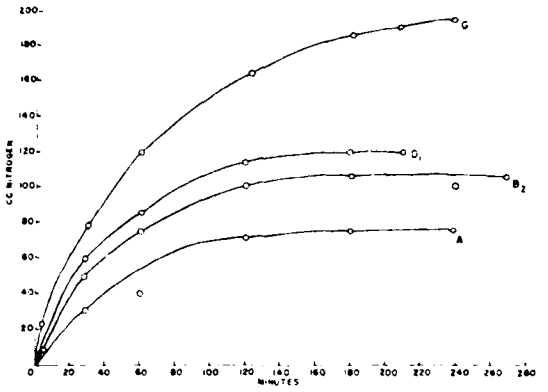

Fig. 2. Nitrogen recovery from four anesthetized dogs placed in a closed oxygen (99% system for periods up to 280 min at 1 ATA. At end of this period N_2 *elimination is not complete* in dog (G), which was old and fat (from Shaw et al. 1935).

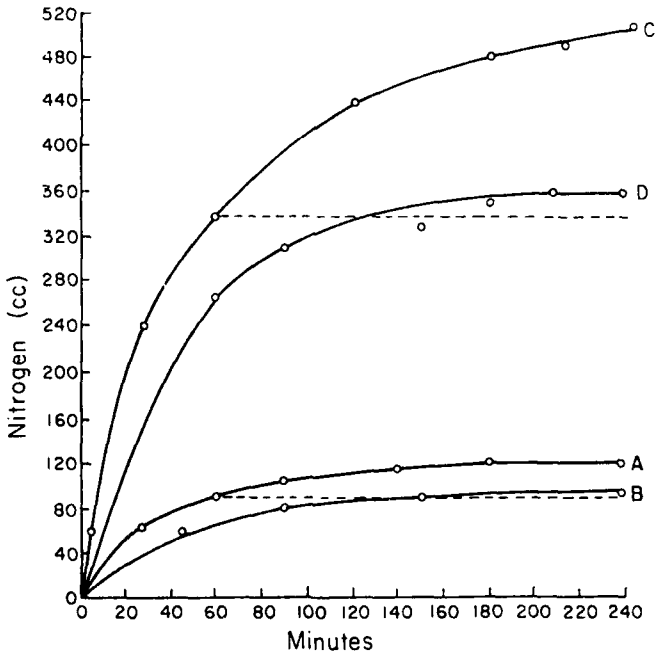

Fig. 3. Saturation time compared with desaturation time for anesthetized dog (D). Nitrogen eliminated during first 7 min (lung rinsing) was not measured. Curve A follows equilibration at 1 ATA. Dog then breathed air for 67 min and curve B represents N_2 elimination in the oxygen atmosphere after the air exposure. Curve C represents N_2 elimination on a different day in the same anesthetized dog after exposure in compressed air (4 ATA) for 4 hr. After desaturation, the dog breathed air for 67 min at 4 ATA, and curve D represents subsequent N_2 recovery in an atmosphere of pure oxygen (from Shaw et al. 1935).

An objective in the early nitrogen recovery tests (Behnke 1937, 1969) was to obtain from the cumulative N_2 curve successive rate (k) values applicable to the individual diver. One derivation of interest to decompression theory may be exhumed in the form of the following quotation, "The curve representing cumulative nitrogen recovery from the body has been analyzed in terms of exponential components, each represented by

$$Y = A(1 - e^{-kt})$$

$$A = \frac{(Y_1)^2}{2Y_1 - Y_2}$$

and

$$k = \log_e \frac{A}{A - Y} \cdot \frac{1}{t}$$

where Y is the quantity of nitrogen recovered at time (t), and A is the total nitrogen for the segment of the whole body curve represented by the single exponential member in the first equation; A can be calculated from Y_1 at time t_1 and from Y_2 at time t_2 provided that the time interval of t_2 is in each case twice that of t_1" (Behnke 1937).

An Essential Requirement for Inert Gas Recovery Breathing Oxygen

An early limitation to the prolonged recovery of inert gas dissolved in tissues was the adverse effect of oxygen inhalation after four to six hours. This limitation can be circumvented by a simple arrangement that converts the hyperbaric chamber into an altitude chamber so that at 34,000 ft (0.25 ATA), inert gas recovery tests can be prolonged for 24 hr or longer. In one EDU test (circa 1940), the author, breathing oxygen in a helmet-bag system, developed pulmonary symptoms during the 4th hr at 1 ATA. The ambient pressure was then reduced to 0.25 ATA and the experiment continued for an additional 24 hr; during this period there was remission of all pulmonary symptoms in the normoxic atmosphere, in accord with a principle clearly expounded by Paul Bert.

Switching of Gases—an Unresolved Problem

The practice of substituting air for a heliox mixture was routine at the Experimental Diving Unit during the 1938-1941 test period. Pruritus, macular skin lesions, and delayed onset of fatigue occurred in some divers after a switch from heliox to air; these were attributed to bubbles in subcutaneous vessels caused by abridged decompression, and were especially likely to occur after chilling of the skin in the abrupt first drop of pressure. Hypothermic circulatory stasis served to explain this adverse phenomenon.

In the changeover from heliox to air, often as deep as 200 ft, it is not likely that counterdiffusion compounded the decompression problem since the changeover to air was gradual, the body was surrounded by air when air was breathed, and the earlier helium exposures were of relatively short duration. The following tabular data are representative of a dive during the 1938-1941 period.

Feb. 17, 1938, dive to 425 ft for 20 minutes (87% He, 6% N_2, 7% O_2); decompression, 425 to 210 ft in 20 minutes. Recirculate air for 12 minutes from 210 to 180 ft; at 180 ft, proceed on open-circuit air to the surface as follows:

Depth:	180	170	160	150	140	130	120	110	100	90	80
Time, min:	2	1	1	2	2	2	2	2	3	3	6
Depth:	70	60	50	40	30	20	10				
Time, min:	6	8	10	15	36	45	54	Total: 220 min			

After decompression, the diver had a decrease in vital capacity from 4.25 to 4.10 liters; diver also developed a rash over the abdomen during the first half-hour and was given oxygen to breathe but was not recompressed. In none of the many test dives in which a shift was made from helium to air at deep depths (180 - 150 ft) were there any symptoms or signs pointing to vestibular or auditory involvement.

Importance of Percutaneous Diffusion of Inert Gases

Cutaneous Diffusion of Nitrogen. Tests of Groom and Farhi (1967) confirmed earlier studies of percutaneous gas transfer (Behnke and Willmon 1941). The practical importance of their finding, that the cutaneous transfer of nitrogen from the atmosphere will increase the half time of the slowest tissue by a factor of more than 2.5 in the dog compared with tests in which the ambient gas is oxygen, should be emphasized. However, it is pointed out that any incision of the skin left open after insertion (say of a tracheal cannula) greatly augments inward uptake of ambient nitrogen. Additional tests are required on intact animals to provide unequivocal data.

Percutaneous Diffusion of Helium. In a euthermic environment (skin temperature 31-32°C), approximately 22 ml of helium diffuses through the skin/square meter of body surface per hour (P_{He} 700 mmHg). If the ambient temperature is elevated to induce sweating with concomitant increase of blood flow to the skin, there will be a threefold increase inward of helium transfer (Fig. 4) (Behnke and Willmon 1941).

Consider the following example of percutaneous helium transfer (PHT) in a euthermic environment:

If a diver has a height of 174 cm (68.6 in), weight 72.5 kg (160 lb), and surface area 1.87 m^2, then PHT = 1.87 x 22 = 41 ml He per atmosphere/hr: at 31 ATA (1000 ft), the PHT (potential) = 1271 ml/hr.

Isobaric pulmonary elimination of helium during the course of decompression will be approximately 170 ml/hr (P_{O_2} 0.3 ATA to provide an "oxygen window"). For example, at 50 ATA (+ 1600 ft), body helium

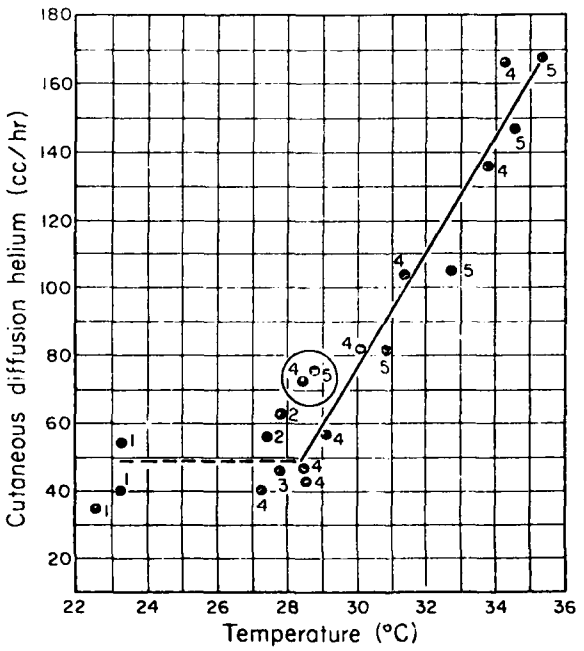

Fig. 4. Cutaneous diffusion of helium in relation to ambient temperature, measured as ml (cc/hr) when the body, head out, is immersed in helium (P_{He} = 700 mmHg). Numbers 1 to 5 refer to different subjects. Encircled values were obtained after previously heated ambient helium had been cooled to 29°C (from Behnke and Willmon 1941).

content is 25,000 ml. The time required for isobaric elimination of this quantity of helium (in the absence of inward cutaneous passage of He) can be computed as:

25,000/170 = 147 hr (+ 6 days), or 9 days if daily 'stop' periods are eight hr.

In the USN 1600 ft wet-pot dive (1973), decompression extended over a period of 17 days, in accordance with the U.S. Navy's conservative (quasi-isobaric) schedule. Yet this prolonged decompression was not free from Type I symptoms.

A prime factor complicating this type of decompression is evident from a comparison of inward percutaneous He diffusion and He pulmonary loss:

Pressure (ATA)	50	40	30	20	15	10	2
PDH (mm/hr):	2000	1600	1200	800	600	400	80
*P_a He (mm/hr):	170	170	170	170	170	170	170

*Pulmonary isobaric He elimination: O_2 window 0.3 ATA

Oxygen Decompression Tests, Bay Area Rapid Transit Project (1967-1969)

Since at pressures above 26 psig, the Washington State tables were not satisfactory in providing a low bends incidence, tests were made (one subject, overage, overweight) to ascertain the value of oxygen decompression. The apparatus was a closed oxygen breathing system that could be replenished periodically and provided moist 95 + percent oxygen, with no resistance to inhalation and about 5 mmHg resistance to exhalation. Occasional deep breaths were taken, and oxygen was breathed without interruption for periods of 2 hours at pressures higher than 30 psig and usually between 25 and 15 psig (+ 5 min from 15 psig to normal pressure).

Dry chamber runs were made, with moderate exercise, for 45 minutes out of each hour. Results were:

		Decompression
20 psig	6-hr shift	60 min O_2
30 psig	6-hr shift	120 min O_2
40 psig	4-hr shift	120 min O_2

For each hour of a fractional shift, oxygen decompression was performed as follows: 10 min at 20 psig; 20 min at 30 psig; and 30 min at 40 psig. In tests which extended over 3 months, there were no symptoms of oxygen toxicity in a relatively sensitive subject, e.g., pulmonary irritation had occurred in the past in Experimental Diving Unit tests after pure oxygen inhalation for 4 hours at 1 ATA. The subject remained in good condition throughout the test period. There were no prodromal symptoms of decompression sickness, such as fatigue, itch, rash.

Table A1. Duration of Excursions in Compressed Air from Surface and Habitat Pressure Levels

Formulas

$D_{max} = 1.443\ D_H + 12$ for a 4-hr dive
$D_{max} = 1.390\ D_H + 9$ for a 6-hr dive
$D_{max} = 1.361\ D_H + 7$ for a dive of indefinite duration

where D_H is habitat depth in meters; converting meters to feet, the following table is derived from the above formulas (from Hennessy).

		No-Stop Decompression Excursions		
		4 hr	6 hr	Indefinite
Surface				
	ft	39	30	23
Habitat				
	20 ft	68	57	50
	30 ft	83	71	64
	40 ft	97	85	77
	50 ft	112	99	91

These values are in close agreement with the uncorrected tabular data (see above) for 240, 360, and 480 min duration exposures where the duration of excursion times was calculated from saturation habitat + ΔP increment and not from ΔP increment alone. For example, at 50-ft saturation depth, total excursion depth is 86 ft (50 + ΔP of 36 ft). For an excursion time of 6 hr in the uncorrected data, the unlimited depth of 86 ft was divided by 0.87.5 (fractional saturation at 6 hr of the 120-min (slowest) tissue to give a depth of 98 ft. Since the body is in equilibrium with nitrogen at 50 ft, it is the incremental depth (ΔP) of 36 ft/0.87.5 = 41 ft which represents the net excursion from 50 ft. If this type of correction is made (see above) there is then continuity between the calculations from *Hempleman's formula (25' to 100' exposures)* and the longer exposures computed in accord with the 'square root' principle.

Table A2. Calculated No-Stop Decompression Schedule for Tunnel Workers from a Pressurized Habitat

Habitat Pressure,		Excess Nitrogen*,		Air Equivalent,	
Psig	ft	psig	ft	psig	ft
0	0	8	18	10	22.5
4.45	10	13.4	30	16.7	37.5
8.9	20	18.7	42	23.4	52.5
13.4	30	24.1	54	30.1	67.5
17.8	40	29.4	66	36.7	82.5
22.3	50	34.7	78	43.4	97.5

(Tunnel worker's schedule: excursions to shift pressures and no-stop return to habitat)

Habitat,		8-hr Shift,		6-hr Shift,		4-hr Shift,	
psig	ft	psig	ft	psig	ft	psig	ft
0	0	10.7	24	11.6	26	13.4	30
4.45	10	17.4	39	18.3	41	20.9	47
8.9	20	24.5	55	25.4	57	28.1	63
13.4	30	31.2	70	32.5	73	35.6	80
17.8	40	37.9	85	39.6	89	43.2	97
22.3	50	44.5	100	46.3	104	50.3	113

Calculations were as follows: Δ Air equivalent (saturation values) /divided by 0.93.8 (8-hr shift), -/0.87.5 (6-hr shift), or -/0.75 (4-hr shift). Divisor fractions represent percentage saturation of the 120-min 'slowest' tissue. Example: Δ Air equiv. (50 ft) = 47.5: 47.5/75 = 63 + 50 = 113. *Tolerated excess N_2 in the 120-min (slowest tissue) that allows a no-stop decompression to habitat – an adaptation of Workman's M matrix concept (Behnke, unpublished data, 1974).

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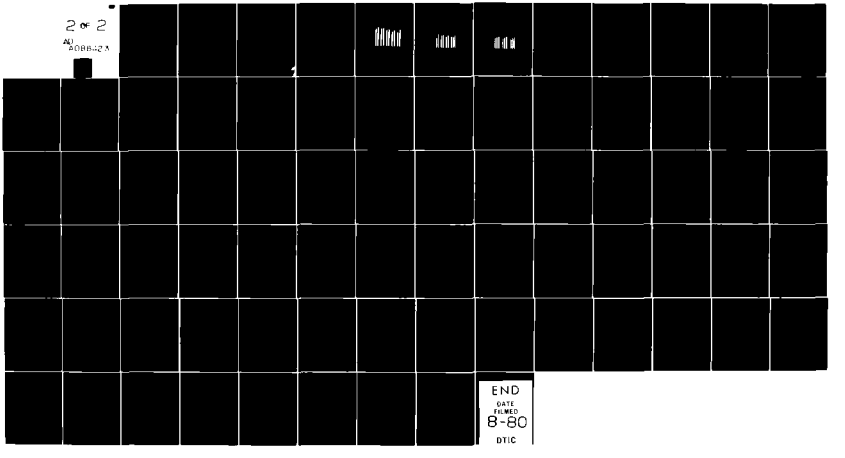

Table A3. Comparison of Depth Excursions Followed by No-Stop Decompression Calculated by Several Methods

	1. Depth of Excursions of Unlimited Duration		
	Hennessy	Author's M adaptation	Author's $\sqrt{\ }$ principle
Surface, ft	23	22.5	22.5
Habitat, ft			
20	50	52.5	48.5
30	64	67.5	61.0
40	77	82.5	73.5
50	91	97.5	86.0
	2. 6-hr Workshift		
Surface, ft	30	26	26
Habitat, ft			
20	57	57	53
30	71	73	65
40	85	89	78
50	99	104	91
	3. 4-hr Workshift		
Surface, ft	39	30	30
Habitat, ft			
20	68	63	58
30	83	80	71
40	97	97	85
50	112	113	98

Table A4. Comparison of Bornmann's Data on Duration of No-Stop Decompression Excursions from Saturated Heliox Atmospheres with Current Experimental and Calculated Values

Bornmann*		Current	
Sat. depths between 150 and 300 feet		150 ft	300 ft
Exposure Time	Excur. Depth	Excur. Depth	
Unlimited	+25	+75**	99**
270 min	+50	+78***	+104***
150 min	+75	+91***	+120***
60 min	+100	+150***	+198***

*From Research Report 1-70, Decompression Schedule Development for Repetitive Saturation-Excursion He-O_2 Diving. Deep Submergence Systems Project Office, 1 June 1970. **Experimental data from Spaur et al. (1978); ***calculated in accord with the square root principle.

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GAS UPTAKE, GAS ELIMINATION
Decompression Theory: Swiss Practice

A. A. Bühlmann

No factors other than inert gas pressure gradients, exposure times, and half times for 16 compartments are considered. The half-time ratio N_2/He is 2.6455 for each compartment (Table 1). The total inert gas pressure (P_{igt}) in each of the 16 compartments is the sum of the computed N_2 and He pressures, respectively, and the pressures of other inert gases like H_2, Ar, and Ne.

The compartments can be associated with specific tissues by typical symptoms of decompression sickness like skin bends, inner ear disorders (vertigo bends), or pains in muscles and joints (Table 1).

PRESSURE REDUCTION

The tolerated ambient pressure is calculated by a simple formula (Tables 2 and 3). The surfacing ratios decrease with increasing half times (Table 3).

Table 1.

Compartment No.	Helium Half Time, min
Blood, CNS	
1	1.15
2	3.8
3	9
Skin	
4	14
5	20
6	30
Inner Ear	
7	45
8	60
9	75
10	90
Joints, Bones	
11	110
12	130
13	150
14	175
15	205
16	240

N_2/He ratio = 2.6455; H_2/He ratio = 0.7097.

Table 2. (Equation)

P_{igt} = inert gas tissue pressure, atm, calculated on the basis of half time and ΔP.
$P_{amb\ tol}$ = tolerated ambient pressure, ATA.

$$P_{amb\ tol} = (P_{igt} - a) \cdot b$$

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Table 3. Surfacing Ratios, Factors a and b

Factor	Compartment No.					
	1	2	3	4	5	(6 + 7)
a	1.80	1.07	0.67	0.54	0.48	0.42
b	0.830	0.855	0.855	0.880	0.885	0.90
Surfacing Ratio	3.0	2.24	1.84	1.68	1.61	1.53

Factor	Compartment No.		
	(8 + 9 + 10)	(11 + 12 + 13)	(14 + 15 + 16)
a	0.40	0.36	0.30
b	0.905	0.910	0.947
Surfacing Ratio	1.50	1.46	1.36

Ascent rate, ATA/min = (P_{amb} • 0.2) + 0.6; maximal rates are limited by the leading tissue, e.g., compartment 6 = 1 ATA/min.

A series of new experiments at 30 m (4 ATA) is appropriate for comparing N_2 and He with respect to half times and surfacing ratios. Table 4 demonstrates the differences in bottom time and decompression time required to produce or avoid identical symptoms, like itching of the skin and red spots. Tissue compartments 4, 5, and 6 appeared to be the most critical ones in these experiments. There was no significant difference in the surfacing ratio for N_2 and He (Table 4).

Table 4. Decompression Times and Bends Incidence in 4-ATA Exposures Involving Physical Work

Breathing Time, min	Breathing Mixture	Decompression Time, min	Bends Incidence
73	Air	a. 58 - 63	1/16
		b. 38 - 43	5/12
23	59% He, 20% N_2*	a. 27	0/12
		b. 18	6/12

*PN_2 = 0.79 atm.

CONSTANT BOTTOM TIME OF 120 MIN (TABLE 5)

If the oxy-helium mixture is replaced after 45 min by normal air for 75 min, a decompression time of 35 min breathing 100% O_2 is sufficient. Shorter decompression times (27 - 30 min) produce mild skin symptoms. Particularly involved is Compartment 6, as is also true after breathing air for 120 min at 4.0 ATA. After a 120-min exposure breathing oxy-helium, a decompression time of 90 min is needed. Insufficient decompression after such an exposure produces mild pains in the arm. The involved compartments are compartments 11 through 15.

These experiments illustrate well our old hypothesis that decompression time can be shortened by changing from a light to a heavy inert gas at the right moment.

Figures 1, 2, and 3 demonstrate the changing composition of the total inert gas pressure with N_2 and He.

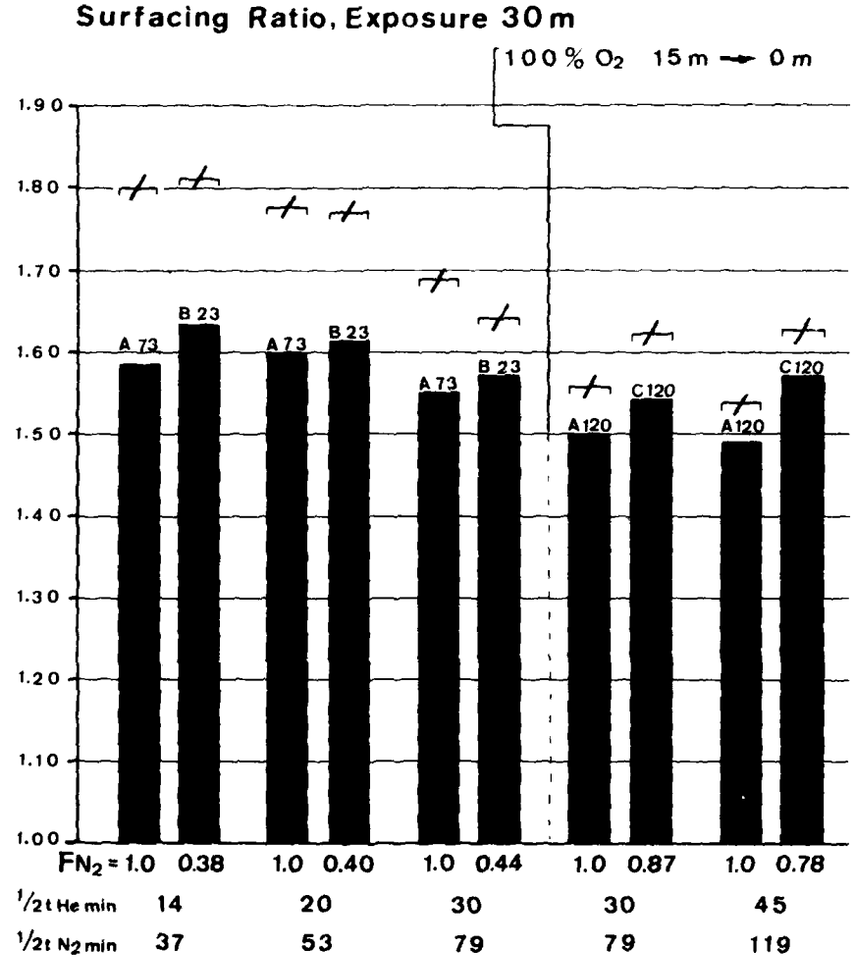

Fig. 1. Calculated inert gas pressures at end of decompression for compartments with different half times. $FN_2 = N_2$ fraction of computed inert gas pressure; ⊓⊓ = inert gas pressure and DCS symptoms; A73 = 73-min bottom time at 4 ATA breathing air (and decompression breathing air) (see Table 4); B23 = 23-min bottom time at 4 ATA breathing 59% He, 20% N_2 (and decompression breathing same mixture) (see Table 4); A120 = 120-min bottom time at 4 ATA breathing air (decompression breathing 100% O_2 by mask) (see Table 5); B120 = 120-min bottom time at 4 ATA breathing 79% He (including 1.5% N_2) (decompression with 100% O_2 by mask) (see Table 5); C120 = 45 min bottom time at 4 ATA breathing 79% He (including 1.5% N_2) and an additional 75 min breathing air (decompression breathing 100% O_2 by mask) (see Table 5); A320 = 320-min bottom time at 4 ATA breathing air (decompression breathing 100% O_2 by mask); C240 = 90-min bottom time breathing 79% He (including 1.5% N_2) and an additional 150 min breathing air.

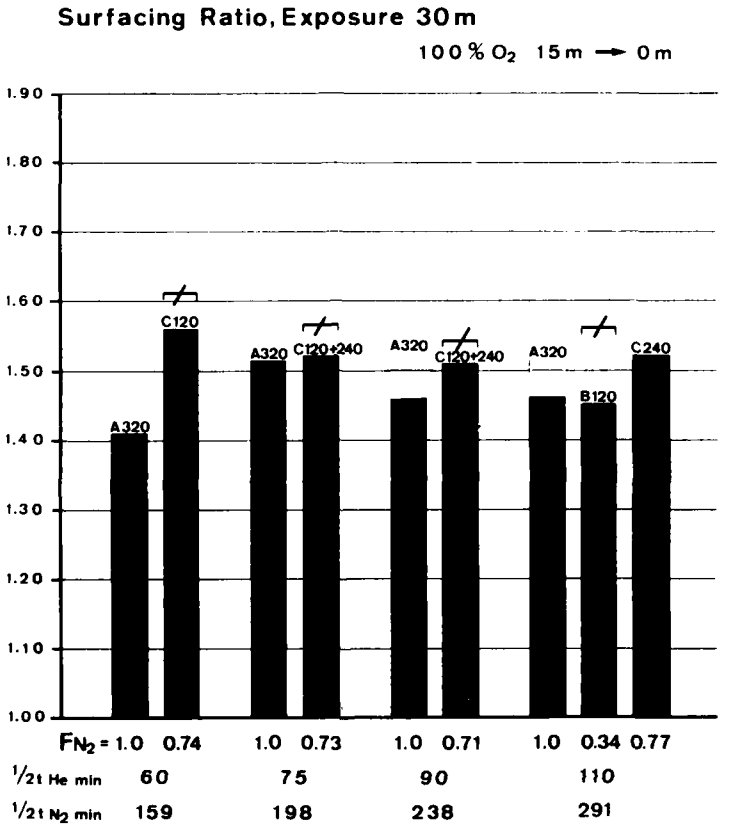

Fig. 2. See Figure 1 legend for details.

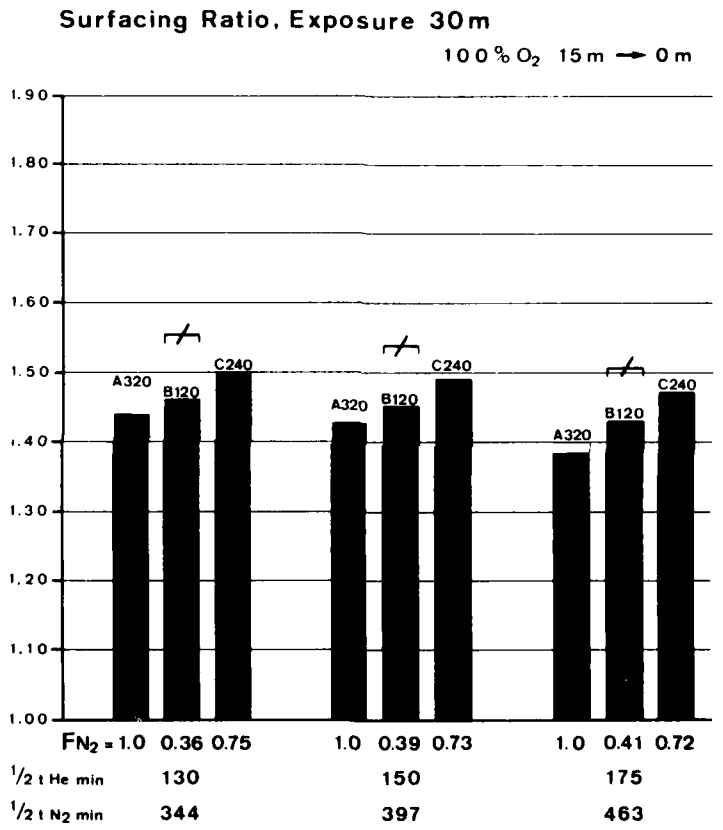

Fig. 3. See Figure 1 legend for details.

Table 5. Decompression Times and Decompression Sickness Symptoms in 4-ATA Exposures Involving Physical Work

Breathing Time, Min	Breathing Mixture	Decompression Time, Min	Symptoms	
120	Air	a. 52		3/16
		b. 45 - 48	Skin-	6/17
120	79% He, 1.5% N_2	a. 90		0/12
		b. 73	Bends-	3/8
45	79% He, 1.5% N_2	a. 35		0/12
+75	Air (1.5% He)	b. 27 - 30	Skin-	5/6
120				

Decompression from 4 ATA to 2.5 ATA in 1 min; 100% O_2 breathing from 2.5 to 1 ATA.

In diving with oxy-helium, it is important to calculate the N_2 pressure for all compartments and to know the extent of N_2 contamination of the breathing mixture.

After saturation with oxy-helium, we normally change to air at 30 m. Table 6 gives the decompression times related to two different N_2 pressures in Compartment 16 at the beginning of air breathing. In our saturation dives with oxy-helium, we had occasional bends during or after the final phase beginning at 30 m. In the experiments, the decompression time from 30 m to the surface breathing air lasted only 16 to 18 hours. We never had bends at the beginning of the decompression. Figure 4 shows the first step of our saturation dives, with depths between 30 m and 300 m. The points lie on a straight line. There are exactly three lines: P_{igt} = 1.36 - 4.0, 4.1 - 7.0, and 7.1 - 50 atm. The line on top represents Compartment 2, with an *He half time of 3.8 min. It was derived from a series of bounce dives with short exposures from 150 m* up to 500 m. The maximal ascent rate of Compartment 2 is 5 ATA per minute.

Table 6. Decompression Times After Saturation

	Surfacing Ratio	P_{igt} 1.37 atm	
P_{He} half time = 240 min	–	4.30	3.91
P_{N_2} half time = 635 min	4.70	0.40	0.79
P_{igt} atm	4.70	4.70	4.70
First Step 4 ATA			
Breathing Air	62 hours	20 hours	22 hours
Breathing He 79%, O_2 21%	–	30 hours	32 hours

Decompression from 4 to 1 ATA.

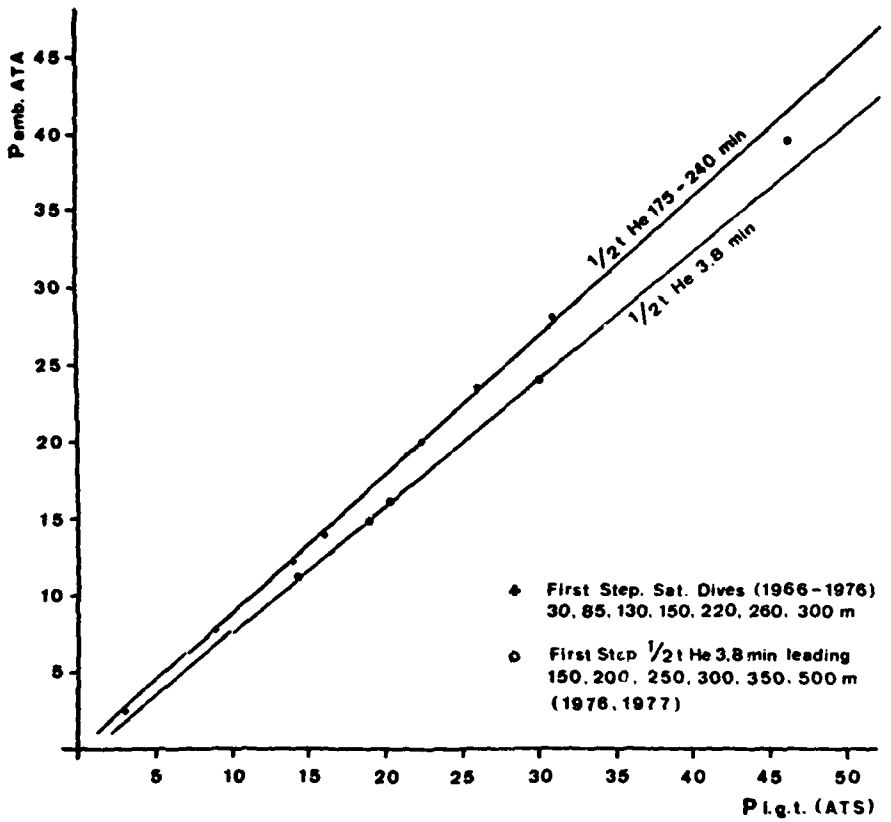

Fig. 4. Pamb tol/computed Pigt; + = first step of saturation dives breathing O_2-He, ascent rate 1 ATA/min; O = first step after short exposure breathing O_2-He between 150 and 500 m. Compartment has He half time of 3.8 min; maximal ascent rate 6 ATA/min.

CONCLUSION

The method employed to calculate inert gas uptake and decompression is simple. It produces useful results in air diving and oxy-helium diving, and also in cases involving multiple inert gases at the same time or in succession. Supersaturation limits of the different compartments or tissues are empirical, and there are some gaps in our knowledge.

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VMRC'S DATA RELEVANT TO CURRENT ASSUMPTIONS USED IN USN DECOMPRESSION CALCULATION

Brian G. D'Aoust

The program in the Department of Hyperbaric Physiology at Virginia Mason Research Center (VMRC) is ultimately aimed at elucidating the etiology and pathogenesis of decompression sickness. Accordingly, we are presently engaged in studies of gas uptake and elimination during the initial phases of saturation and desaturation, studies of the biophysical bases of ascent criteria, and investigations on the temporal relationship of gas elimination to the detection of vascular bubbles, and tissue and hematologic effects as a result of decompression profiles after both saturation and non-saturation dives. In the course of these studies, we have obtained results that bear on the implied assumptions currently held by the USN regarding decompression theory.

We wish to emphasize that the fact that the USN tables are calculated in a particular fashion does not necessarily mean that all USN personnel agree with such views and assumptions. In fact, it has been made abundantly clear by this time (see Hennessy, this Workshop) that many mathematically different functions that are constrained by a unique physical situation, process, or model are quantitatively very similar and thus often indistinguishable on the basis of physiological data. This being the case, the problem of "optimal" decompression calculation can quite justifiably be simplified to very simple procedures based chiefly on empirical experience (see Hennessy and Hempleman 1977; Bühlmann and Keller, this Workshop). Figure 1 summarizes this situation graphically.

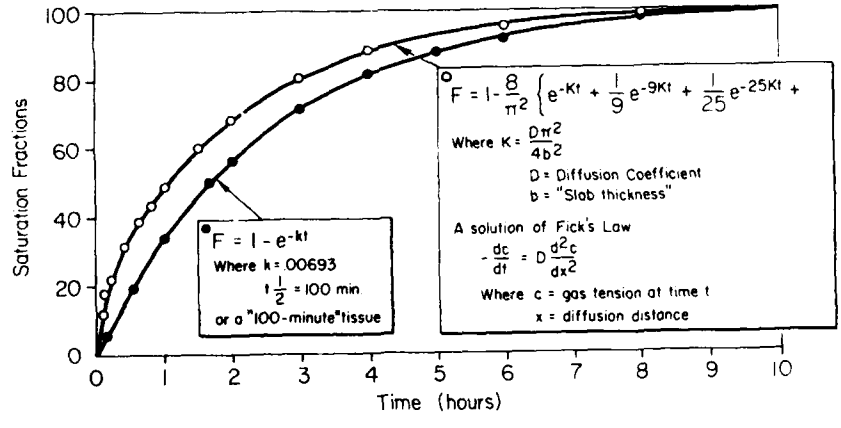


Fig. 1. Comparison of a single-tissue diffusion slab model (o–o) with an "average" 100-min half-time tissue model (•–•). Although basically different, it would challenge physiological measurements to distinguish between the perfusion-based model and the single-tissue diffusion-based model.

It is, we believe, the need to understand the etiology and course of decompression sickness more completely that provides the added stimulus to search for a physiologically predictive model. Though unnecessary to predict optimal decompressions, such a model's chief value will be the understanding of what happens when the model fails. With this introduction, we move to considering the first assumption on the agenda.

ASSUMPTION NO. 1. Multiple tissues or tissue compartments must be used to describe whole body gas exchange.

The critical word here is "must." For the reasons given above, a completely empirical single "tissue" approach would work, but we would suggest and are investigating the use of a multiple tissue model simply because the human body cannot be considered homogeneous in regard to gas saturation, and in fact since non-saturation dives present the real difficulty in estimating gas uptake, the concept of multiple tissues is logical.

However, I do not agree with the continuum approach, i.e., using 10 tissues or more, because such models do not lend themselves to interpreting the reasons for their failure. We know enough now that we may assume a fair correlation between CNS symptoms and the appearance of vascular bubbles even though the correlation for prediction of pain is poor.

Since the blood carries vascular bubbles centrally, it is "sampling" the tissues where the bubbles almost certainly formed first. Thus, the blood is sampling a number of different tissues or half times, and Doppler bubble detection can only provide some information on both the half time of the stressed tissue and the degree of stress. Accordingly, it seems logical to use a smaller number of tissues with a heavier emphasis on physiological relevance. This has long been a practice in anesthesiology, where much more soluble gases, such as ether, N_2O, halothane, and cyclopropane, have been considered. Accordingly, one speaks of a vessel-rich group, a vessel-poor group, a fatty group, and so on (Papper and Kitz 1963).

Behnke's original suggestion, put forth in the first edition of Bennett and Elliott (1969, p.241), which involves six tissues, still seems useful. Although the constraints would undoubtedly require some change from the originals, they would have the advantage of being based on actual tissue capacities and exchange rates.

The studies we have done on gas uptake and elimination in both awake dogs and goats are best fitted with a sum of two exponentials according to the equation

$$Y = Ae^{-k_1 t} + Be^{-k_2 t}$$

This fit is shown in Fig. 2a for dogs and Fig. 2b for goats. Though our data do not lend themselves to fitting more than one exponential, they do strongly support the use of several half times, tissue compartments, or "situations," as the most recent workshop jargon has appropriately called them.

In summary, though not strictly necessary for predictive use (one time constant is sufficient, as indicated in this Workshop; see Fig. 1), we support the use of several parallel exponential compartments. Such a model lends itself better to interpretations. It will be evident that this will only be so if the time constants are initially based on known and experimentally supported physiological quantities and constraints.

ASSUMPTION NO. 2. Tissue gas uptake is described by exposure time, pressure differential, and tissue half time according to the equation

$$P_t = P_o + (1-2^{-T/H})(P_o-P_a)$$

This equation is inadequate for calculating the saturation state for different compartments; however, it may be useful in mixed gas (He and N_2) tables to account for the total amount of dissolved gas (rather than the total inert He + N_2 tension) by use of an average solubility for that compartment, e.g.,

$$P_t = P_o + (1 - 2^{-T/H})(P_o - P_a) \cdot (\bar{\alpha}N_2) \text{ or } (\bar{\alpha}He) \quad \text{(see Fig. 2c)}$$

where $\bar{\alpha}N_2$ denotes the "average" solubility of N_2 in the compartment. This will of course necessitate the use of such a parameter in the ascent criteria. This discussion becomes pertinent when considering Assumptions 3 and 4.

ASSUMPTION NO. 3. Only the inert gas partial pressure need be considered in the decompression problem.

ASSUMPTION NO. 4. Breathing multiple inert gases appears to produce some decompression advantage. Methods of incorporating these advantages into the decompression model are not yet understood.

The first part of this assumption is true, provided one is careful to specify the appropriate sequence of gases. We have presented data (D'Aoust et al. 1977) indicating the transient potential hazards of gas switching. These data are shown in Fig. 2c.

By the same reasoning (first introduced by Keller in 1962), one can arrange the sequence of gases appropriately, using existing models (Keller, Bühlmann, Nishi) to provide undersaturation and thus a decompression advantage. I believe the same methods and assumptions used to compute tables can be used both to exploit the potential advantages of gas sequencing and to avoid its hazards.

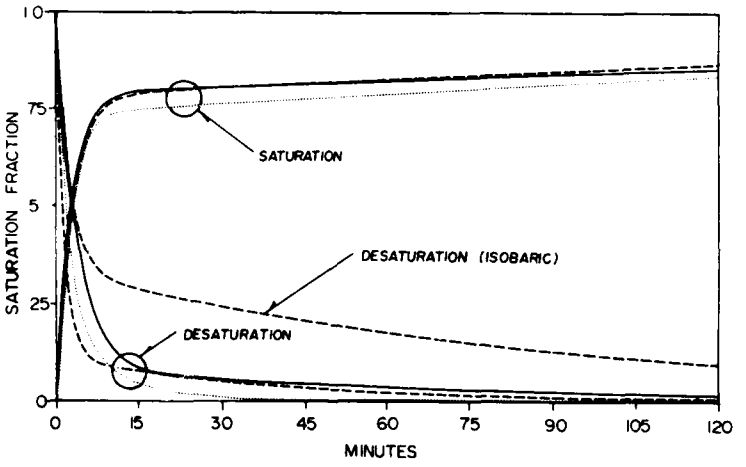
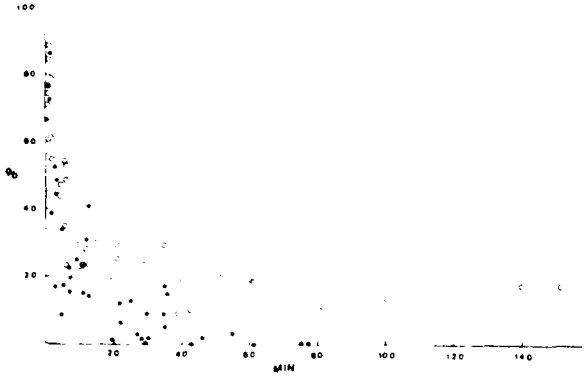
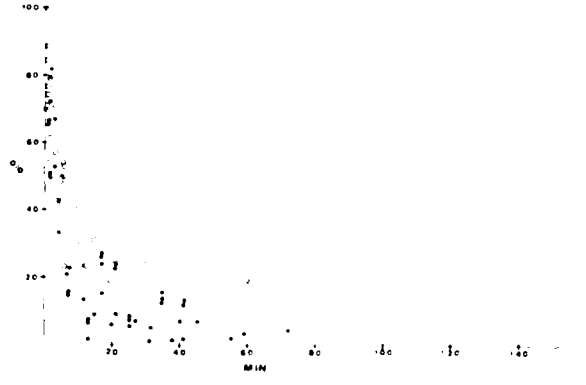


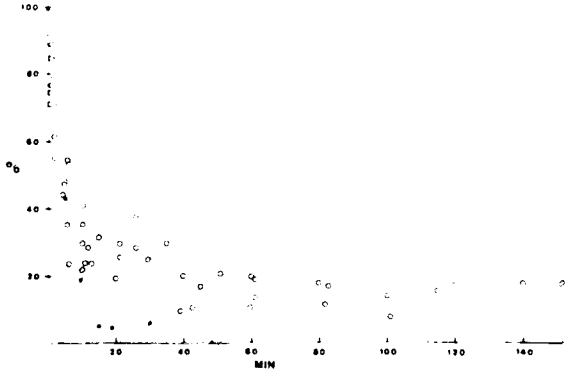
Fig. 2a. Comparison of average desaturation and saturation rates in awake dogs under isobaric and decompression situations. Notice the significant difference in desaturation curves after decompression compared to desaturation after a switch to oxygen breathing. The three curves for both saturation and desaturation have been taken at 33, 66, and 99 fsw.



$[\bar{v}N_2]$ (black dots) plotted against time after leaving bottom (ascent is in all cases less than 3 minutes) from a 33' saturation superimposed on this data is the isobaric elimination (○).



$[\bar{v}N_2]$ (black dots) plotted against time following decompression from a 66' saturation exposure; same superposition of $[\bar{v}N_2]$ from isobaric desaturation. 6 experiments shown.



$[\bar{v}N_2]$ (black dots) plotted against time following decompression from saturation at 100 fsw. Only one experiment is shown, since a 100 fsw decompression from saturation requires recompression.

Fig. 2b. Desaturation of mixed venous blood nitrogen content in awake goats after decompression from 33, 66, and 99 fsw. In each figure, the isobaric desaturation curve (open circles) has been superimposed on a fractional basis. It is clear that, like the curves for the dog in Fig. 2a, the effect of decompression is to retard the rate of nitrogen elimination. This is manifested by a more rapid drop in mixed venous blood nitrogen concentration after decompression, appearing as a more rapid elimination rate. However, because we have documented that cardiac output does not increase measurably during this time, it must be assumed that considerable nitrogen is left in the animal.

If multi-compartment "real" models are used in this endeavor, there may be theoretical reason to expect different rates of equilibration in different tissues between He, H_2, Ne, and N_2; however, whether these would be markedly different from the ratio of 2.65 (Bühlmann, this Workshop), which approximates the Graham's law relationship, or 2.80, which approximates the ratio of their aqueous diffusion coefficients, is uncertain and not likely to be easily resolved experimentally. Current understanding of the decompression problem is at least adequate to exploit the obvious decompression advantages of this approach. More important, such attempts cannot help but provide further needed insight into the entire decompression problem, including that of the effects of vascular bubbles.

ASSUMPTION NO. 5. Compression procedures have no effect on decompression.

This assumption has been ignored too long. It is theoretically wrong, according to nucleation theory (Yount, this Workshop), and physiologically wrong in that HPNS is very much related to compression rate, albeit at considerable depth. The influence of nuclei regeneration has not been well documented, but probably has influenced the outcome of many decompressions. This probably should be taken into account in future models. Since we have isolated very *low* critical ratios in our fish, we will be able to investigate this effect on rather large nuclei.

ASSUMPTION NO. 6. Tissue supersaturation limits are best described by a series of decreasing pressure ratios ranging from 1.8 to 4.1 to 1 for N_2 and 1.5 to 1.0 to 1 for He.

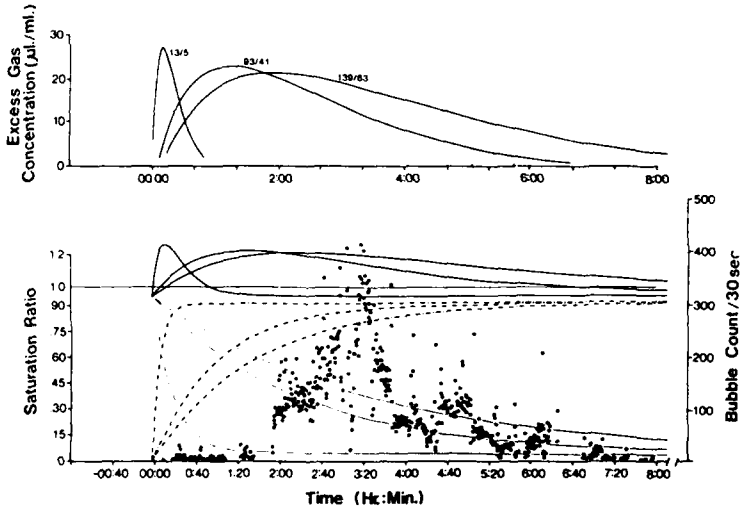


Fig. 2c. Computed helium-nitrogen tissue half-time pairs superimposed upon bubble counts with time after an isobaric gas switch at 7 ATA. The top figure shows the supersaturation expressed as an excess gas concentration (microliters/milliliter). Assuming that the maximum numbers of bubbles per minute reflects collection of bubbles from the compartment with the greatest gas capacity, it should be possible to check this information against largest compartment's half time.

I feel these are the *least* satisfactory of the assumptions currently used in models. First of all, they are illogical. If one assumes that separated gas is the primary etiologic agent in decompression sickness, there must be a certain volume and mass involved. Because so much empirical work correlates well with relative volume ratio (and we have also demonstrated this in our decompression studies with fish; see Fig. 3), the impression is gained that computed partial pressure is adequate as our ascent criteria. However, it can be shown that a very small proportion of the total gas dissolved is ever involved in decompression sickness. This small proportion is eliminated according to different physical (and mechanical) processes, or, in fact, may not be eliminated at all! However, it has a profound physiological effect in that it can change the rate of elimination of the remaining gas that may later contribute to symptoms.

Further, once gas is separated in tissues, its volume depends chiefly on ambient pressure, ΔP, and tissue distensibility, as pointed out earlier (D'Aoust 1977; Yount, this Workshop). In addition, the time required for elimination of a given degree of supersaturation of one gas vs. another depends on the relationship of their diffusion coefficients as well as solubility. In our studies of fish decompression, we have incorporated these ideas into a critical volume concept based on the assumptions that within certain limits of time, the response of fish to a given decompression is related directly to separated gas volume. This implies that a given degree of supersaturation of a less soluble gas will be less damaging than the same degree of supersaturation of a more soluble gas. This is illustrated in Fig. 3a, b, and c, where Ar, N_2, He, and O_2 are compared on the basis of response to similar decompressions.

These data show that when one attempts to account for different volumes of gas present by assuming that this separated volume is proportional to D, ΔP, and α and inversely proportional to absolute pressure (P_f), one can show little difference between He and N_2. *However, this does not permit one to say that "N_2 and helium are the same"* in decompression; rather, one can say only that there are circumstances where the differences (the more rapid diffusion of He vs. the greater solubility of N_2) are self cancelling. It appears that these circumstances occur frequently in current decompression procedures.

Other studies also support this concept, as shown in Fig. 4a and b, which shows a comparison between two different "stress criteria," which—with some caution—we may interpret as tentative "ascent criteria." In Fig. 4a, the stress is assumed to be proportional only to the ratio of initial pressure (P_i) to final pressure (P_f), (P_i/P_f).

This shows an obvious difference in He, N_2, and argon. On the other hand, Fig. 4b indicates the same data plotted as a response to a different ascent criterion. This criterion is based on the assumption that the damage of decompression is related to the volume of separated gas and that the latter is proportional to the original gas concentration, to diffusion (assuming resolution of bubbles is diffusion limited), and inversely proportional to the final pressure. Clearly there is statistically less difference in the gases when their "dose" is expressed in this way, as $\gamma = (\frac{Pi}{Pf} - 1) \cdot \bar{\alpha} \cdot D \cdot 10^2$.

The valueof these data is chiefly conceptual; there is no direct means of adjusting such an ascent criteria to man because far less stressful effects are considered; however, Fig. 4 does demonstrate the importance of solubility and diffusivity in critical volume concepts. It is also of interest because in the extreme it might seem to contradict the conclusion of Yount (this Workshop) that the supersaturation pressure ΔP is the parameter that correlates best with the initial numbers of bubbles. *However, total bubble numbers will only correlate with total separated volume at the same pressure.* For a given distribution of nuclei, the ability of an original nuclei or "bubble site" to continue to produce vascular bubbles depends also on the total gas dissolved in the vicinity of the nuclei, i.e., solubility as well as ΔP. Thus our data provide no direct disagreement with the Yount model, and can be interpreted on the basis of that model's assumptions.

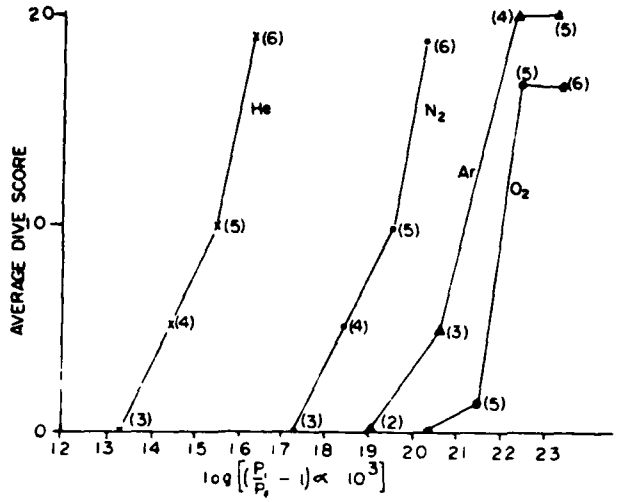


Fig. 3a. Response of salmonids to decompression from various depths of saturation with helium, nitrogen, argon and oxygen. Stress is expressed as ΔP/hydrostatic pressure x solubility.

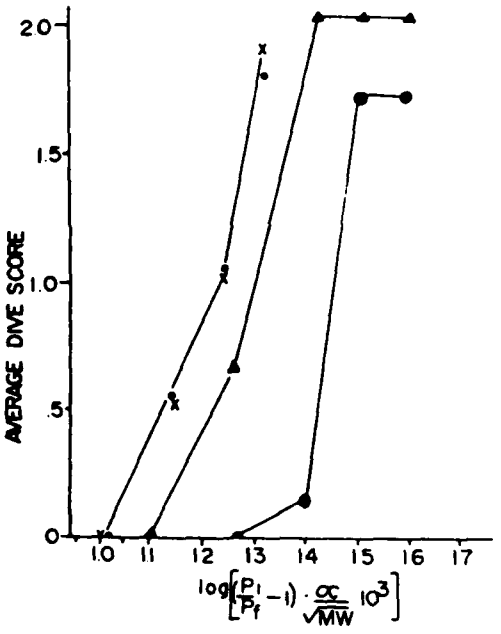


Fig. 3b. Stress is defined in same way, except correction is made for diffusion by dividing by square root of molecular weight. When same data as in a are plotted against this stress parameter, there appears to be no difference between helium and nitrogen in response, indicating that under some circumstances, He and N_2 can behave similarly, probably because more rapid diffusion of He makes up for extra solubility of N_2.

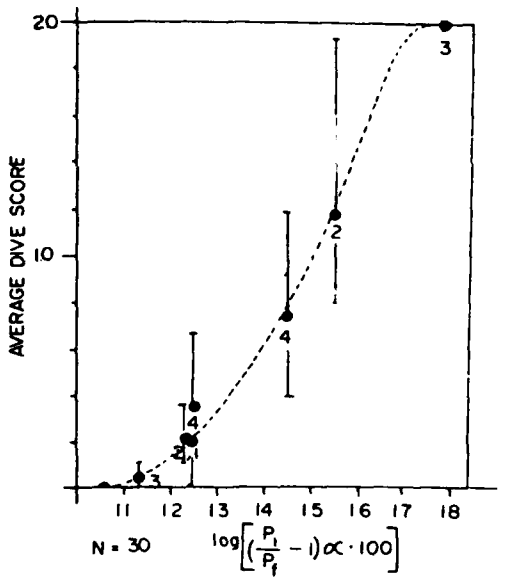


Fig. 3c. Response of salmonids to decompression from various depths of saturation with air. Numbers to right of dots indicate ΔP values imposed. A ΔP of 3 ATA (top right) can provide 100% mortality if final pressure is 1 ATA, whereas a ΔP of 3 (lower left) can produce almost no response when final pressure is 3 ATA.

With respect to Assumptions Nos. 3 and 4, our experience points to the Yount model as the one most useful functionally. The fact that all nuclei have critical supersaturation pressures pretty well explains why concepts of stable supersaturation have worked at all.

If one were to add any constraint to that of pressure ratios in designing ascent criteria, it would be that of the integral ΔP x time. As shown in Table 1 (from D'Aoust 1977), this correlated best with a simple concept of decompression insult based on 1) bubbles, and 2) bubbles and bends. Thus, the concept of "compartment history" is introduced.

Table 1.

		0	10	20	40	80	120
180 ft/45 min	SSt	410	1016	1319	1347	1485	1510
	ΔP	56(50)	65(50)	55(40)	53	37	27
220 ft/35min	SSt	606	1274	1535	1551	1654	1637
	ΔP	69(50)	81(50)	63(30)	56(30)	38	27
240 ft/25 min	SSt	803	1400	1825	2050	3000	2025
	ΔP	73(50)	81(50)	59(40)	55	34	23

Maximum values of ΔP (= tissue N_2 tension - hydrostatic pressure, P) or supersaturation for different tissue half times in dives to 180 ft/45 min (no bubbles, no bends); 220 ft/35 min (bubbles but no bends); and 240 ft/25 min (bubbles and bends). Depths of limiting ΔP are shown in brackets; otherwise, ΔP values are those on arrival at surface prior to recompression. "SSt" values are calculated, during and after dive ΔP, in feet of seawater (fsw), SSt in (fsw) (min).

The 240 fsw/25 min profile shown in Table 1 was the most stressful, judged on the basis of the most divers showing both bubbles and bends. It is therefore interesting that though there is little difference in the surfacing values of ΔP between each profile, there was a great deal of difference between the integral of ΔP · t in the same compartment. This is true for the 10, 20, 40, 80, and 120-min tissues. This illustrates the utility of this parameter in decompression calculation, and it is now in use (Lambertsen, personal communication). Further, if a given decompression produces a certain number of "sites" for bubbles to grow, the time-dependent nature of the response can be handled computationally in this manner by a simple reprogramming of current models. A simple explanation of the better correlation shown in Table 1 would include the fact that, as shown by the Yount model, the amount of gas as "supercritical nuclei" is small relative to the amount dissolved between adjacent nuclei. Thus both ΔP (P_{ss}), the nuclei distribution, and time constrain the amount of gas entering the vascular system (as bubbles) in an unphysiologic manner. Taking the explanation further, the reason that high supersaturations can "exist" for a relatively short time is that the distribution of nuclei is such that there is neither time nor sufficient nuclei to create too many bubbles.

ASSUMPTION NO. 7. Pressure reduction ratios for nitrogen-saturated tissues increase with increasing depth while those for helium-saturated tissues remain constant.

Relative to the last assumption (No. 6), this is a contradiction. Based on human experience, the permissible pressure ratios *decrease* with depth, whereas permissible pressure supersaturation increases. Further, permissible supersaturation should not be expressed as a pressure ratio. It is much more likely that differences between helium and nitrogen are related to gas uptake and elimination than to any inherent response

to supersaturation, although obviously a given "sheet" of helium will fill a nucleus faster. As shown in Fig. 3a and b, experience with nitrogen and He can, under certain situations, be identical.

Once again, the old dilemma is introduced. If a profile produces too many bends, is it better to assume that more gas was taken up than originally calculated and the permissible supersaturation is correct, or that the amount of gas taken up was as calculated and the permissible supersaturation is too high?

It is this dilemma which I feel supports the development of a physiologically relevant model; with the theoretical and experimental tools at our disposal, such a model should help us to solve this problem, at least of calculation, with a minimum of further research, and still provide a model that can be useful in understanding the etiology and pathogenesis of decompression sickness. This goal would seem to provide the best compromise between too empirical vs. too theoretical a model.

ASSUMPTION NO. 8. Tissue supersaturation limits (pressure reduction ratios) increase with increasing depth.

This assumption is in fact summarized by the data in Fig. 3c, in which a ΔP of 3 ATA applied at an original P_i of 7 ATA had virtually no effect, whereas the same ΔP with a final P_f of 1 ATA had 100% mortality. The permissible pressure reduction ratios must *decrease* with depth because the permissible ΔP increases. This fact is consistent with a critical volume model (Hennessy and Hempleman 1977), which is supported by the data in Figs. 3 and 4 and virtually all empirical experience, except that for very long dives. Yount's interpretation of this fact appears both sound and testable, and in part guides our current approach.

We now have evidence that there may be something empirically much more fundamental in the Haldane ratio principle than the accidental physiological situations described above (and by others in this Workshop). This has come to notice in our fish experiments. The dose-response curves shown in Fig. 4a and b were obtained by assessing the response to decompression 30 min after surfacing. In doing this we were aware from previous experience that we were also observing the effects of both internal and external supersaturation on mortality (see the scheme in Fig. 5, from Beyer 1977).

By assessing the response to the same decompression 15 min after surfacing, we would obviously be examining more closely the effects of internal supersaturation, that is, the damage done only by the gas dissolved in the fish. It is therefore surprising that when this was done it revealed that the ED_{50} pressure ratios (P_i/P_f) for He, Ne, N_2, and Ar were similar *at several depths*, and ranged between 2.4 and 2.8! This is shown in Fig. 5a, b, c, and d. These ratios are very much closer to the original Haldane ratio of approximately 2.4. In other words, the importance of solubility or diffusivity was no longer obvious. Of even greater interest is the fact that if one compares these ratios to those reported for rats (Berghage 1976), they are very much smaller and similar for each gas! On the other hand, if plotted according to Yount's data in Fig. 7, the slope is similar to that of Berghage's 1976 data.

It appears that we may be observing a critical pressure ratio that is consistent with a critical volume hypothesis. Further, it is similar to the magnitudes postulated by Yount et al. (1978) for "critical tissue deformation pressure" or "tissue distensibility," as we recently described it (D'Aoust 1977). On the other hand, these results seem to contradict the conclusions that the critical pressure ratio decreases with depth. Since we have not used pressures greater than 10 ATA, we cannot yet check this. Our pressure chamber hatches would have to be modified for this purpose, and we hope to accomplish this in the next year.

If one assumes that separated gas volume is the main stress, these results indicate that a constant pressure ratio *of any gas* can provide the same stress under certain circumstances. This is consistent with the assumptions:

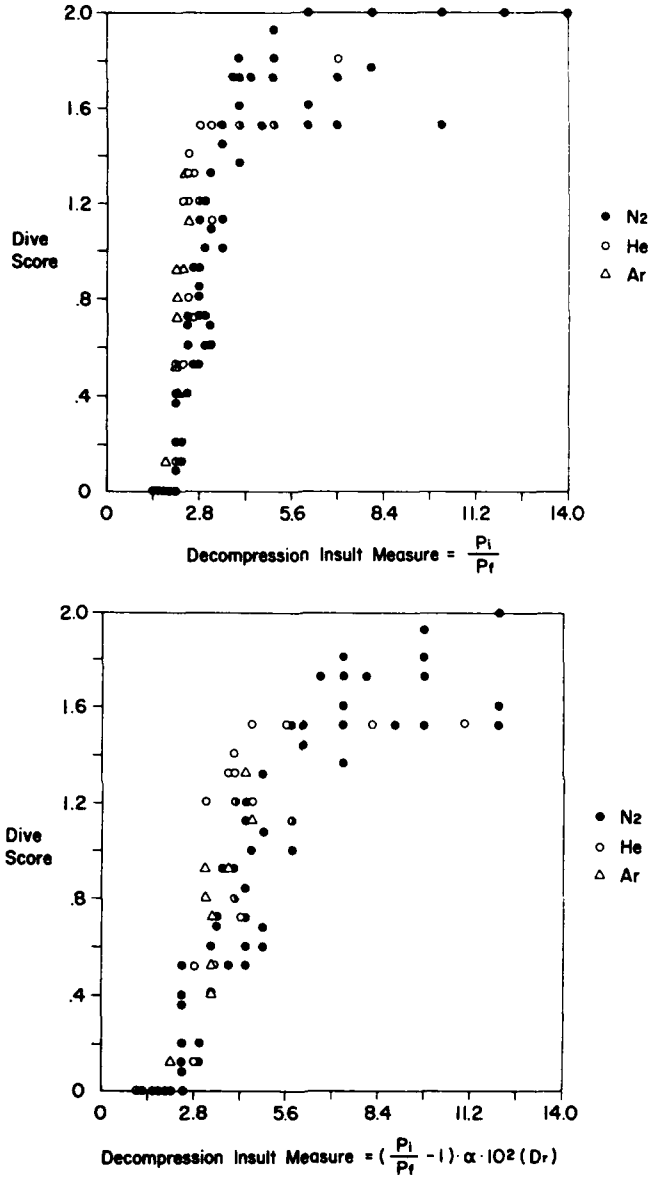


Fig. 4. a, Response of fingerling salmonids saturated with nitrogen, helium, and argon to decompression from a range of pressures. Data are plotted according to decompression ratio only. In this situation, it is clear that the points for argon, the most soluble gas, appear along the left; helium, more toward the middle; and nitrogen more to the right, although differences are slight; b, same data in Fig. 4a plotted according to stress parameter $\gamma = (\frac{P_i}{P_f} - 1) \circ \bar{\alpha} \bullet D \times 10^2$. When plotted against this parameter, there is less separation between each gas. This encourages further work on multi-parametric approaches to ascent criteria.

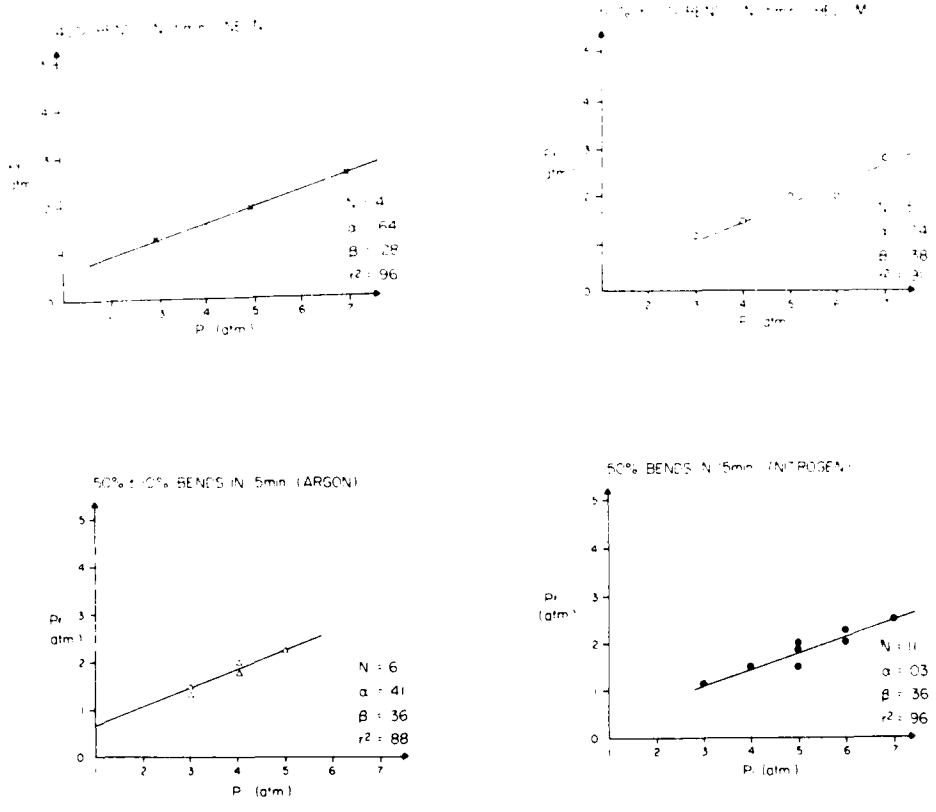


Fig. 5. Data show same experiments providing data plotted in Fig. 4a and b, analyzed at 15 rather than 30 min. The LD_{50} are taken at 15 min after decompression, and the differences between gases disappear. All are apparently described by a similar decompression ratio. If these data are plotted in the same way as the Berghage data (this Workshop), the slope of the line is similar (expressed in the very lower left corner of his plot (see Fig. 7 of Yount, this Workshop)). Thus, decompression of fish provides a means of examining these factors at pressures lower than can be used with rodents.

1) That we are observing the "recruitment" of a number of nuclei

2) That this critical number of nuclei provides either *collectively* or *sequentially* enough separated gas volume to produce the response, regardless of the amount of gas dissolved

3) Because of the apparently small critical ratio, we are observing either the effects of a) larger nuclei, or b) weaker tissue (Berghage could not reproduce these ratios except at higher ΔP!).

4) The critical *volume* of separated gas associated with the observed ED_{50} is so small relative to the amount dissolved as to be initially independent of solubility.

ASSUMPTION NO. 9. Pressure differentials during decompression can be maintained indefinitely.

Assuming "pressure differentials" means "supersaturation," this assumption is theoretically wrong. However, I assume the statement has something to do with the pros and cons of supersaturation vs. no-supersaturation models, and should be qualified by the phrase "for all practical purposes." There is no black-and-white answer to this question.

There can be no doubt that some degree of supersaturation is "stable," that is, *insufficient to trigger the growth of existing* nuclei against their size and tissue regime or to allow bubble formation by tribonucleation. Under these conditions, gas elimination can be treated by physiologic models. Our comparative studies on fish and our environmental studies emphasize the quasi-stability of supersaturation and the unreality of no-supersaturation models.

ASSUMPTION NO. 10. Gas uptake and elimination are symmetrical relationships. They are mirror images of each other.

Under isobaric conditions, this has been experimentally verified and is theoretically sound. However, we have shown experimentally that this assumption does not hold after decompression from saturation (D'Aoust et al. 1976). The rate of gas elimination can be drastically reduced by decompression. The remaining question is how severely affected by decompression is gas elimination? From the point of view of computation one can "correct" for this effect either by adjusting the gas transport time constant or by reducing ascent criteria; the choice is probably unimportant unless one is using a "physiological" model.

However, the question bears heavily on residual nitrogen time, repetitive diving, nuclei regeneration, and altitude diving, and an attempt might be made to adjust desaturation time constants in any model by an amount proportional to the rate of ascent.

ASSUMPTION NO. 11. Oxygen breathing enhances gas elimination by increasing the pressure differential between the tissues and alveolar air.

This is demonstrated beyond doubt; however, the actual vasoactive effects of oxygen are not taken into account, and would seem to be capable of diminishing its effectiveness. The explanation for its effectiveness, although logical, seems oversimplified.

ASSUMPTION NO. 12. The concept of residual nitrogen time is sufficient to handle repeated exposures.

Since a certain degree of supersaturation can be viewed as stable because of nuclei size and tissue distensibility, this concept can still be useful if it takes into account previous history, nuclei regeneration, and all gases.

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COMMENT ON USE OF DIFFERENT GASES

R. W. Hamilton, Jr. and D. J. Kenyon

There are two aspects to the use of different gases in decompression: gas switching, and the use of multiple gas mixtures. Regarding the former, laboratory experience with the use of a switch to air at 100 fsw has caused us to question the premise that this can be managed with straightforward Haldanian computation. Figure 1 shows profiles of a series of 4 dives performed in Ocean System's laboratory in Tarrytown in the fall of 1971. Characteristics of these dives were as follows:

Depth: 680 fsw

Bottom gas: Helium 20%
Neon 70%
Nitrogen 5%
Oxygen 5-6%

Gas Switches: O_2 raised to 16% at 300 fsw;
air at 100 fsw

Oxygen breathing: None in first dive (71-28);
Last 70 min of 10-ft stop in others

Rates: Compression 100 fpm
-60 fpm to first stop
-10 fpm to 50 fsw (100 in later dives)
-1 fpm to surface

Matrix 31/02

In the first dive, bends occurred at 80 fsw. In later dives the switch to air was changed (see profiles, Fig. 1) and was carried out over several stops in both the computation and the dive. This resulted in clean dives except for a bend at 20 fsw in the last dive.

One reason that we suspected that the shift to air might be responsible for the bends was feedback from the field, where there was a feeling that bends were likely to develop after the shift to air.

Our analysis was complicated by the use of neon as a component in the mixture: whether neon, or the use of three inert components, had any specific bearing on the results cannot be determined from the data. In three dives to 640 fsw at sea in Oct-Nov 1972, with 2 divers each, limb bends were encountered at 70 fsw in one diver and after surfacing in another.

Results of another dive with characteristics similar to these, but uncomplicated by neon, are shown in Fig. 2. This dive was conducted at Duke University in September 1976, and resulted in two cases of bends, one of them difficult to treat. The computations were based on a "diffusion model," but the important thing is that a sharp change in ascent rate followed the shift to air—pure Haldanian computations could yield a similar profile. Another factor that contributes to this acceleration is the increase in oxygen at the same time.

CONCLUSIONS

We feel these data suggest that direct Haldanian calculations (or other models, for that matter) are not valid when they imply unusually fast gas unloading and call for acceleration of decompression as a result of an abrupt change in the inert gas.

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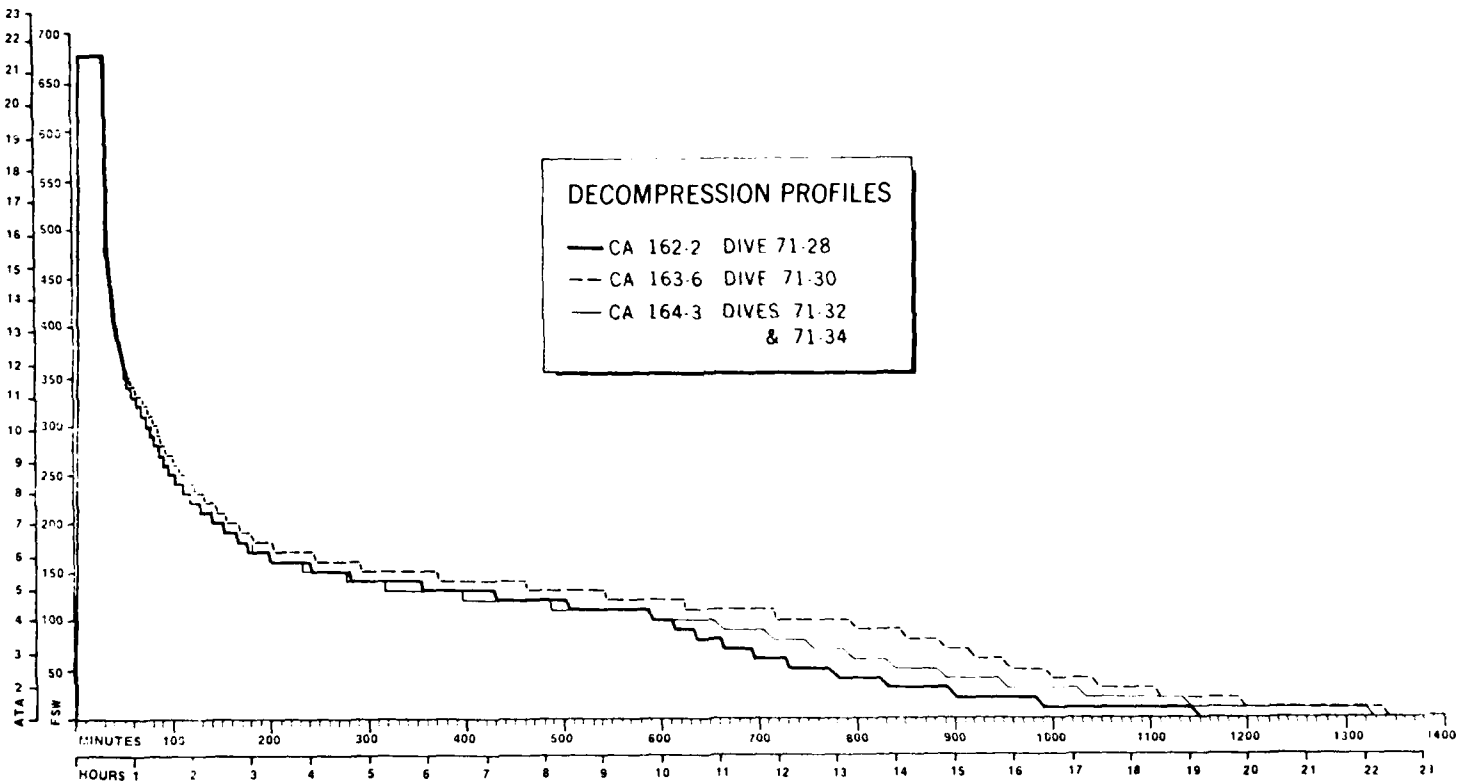

Fig. 1. Laboratory dives using 5% O_2, 70% Ne, 20% He, and 5% N_2. Oxygen breathing was used during the last 70 min of the 10-ft stop. Bends occurred at 70 fsw in dive 71-28, and at 20 fsw in dive 71-34. There were two divers on each dive.

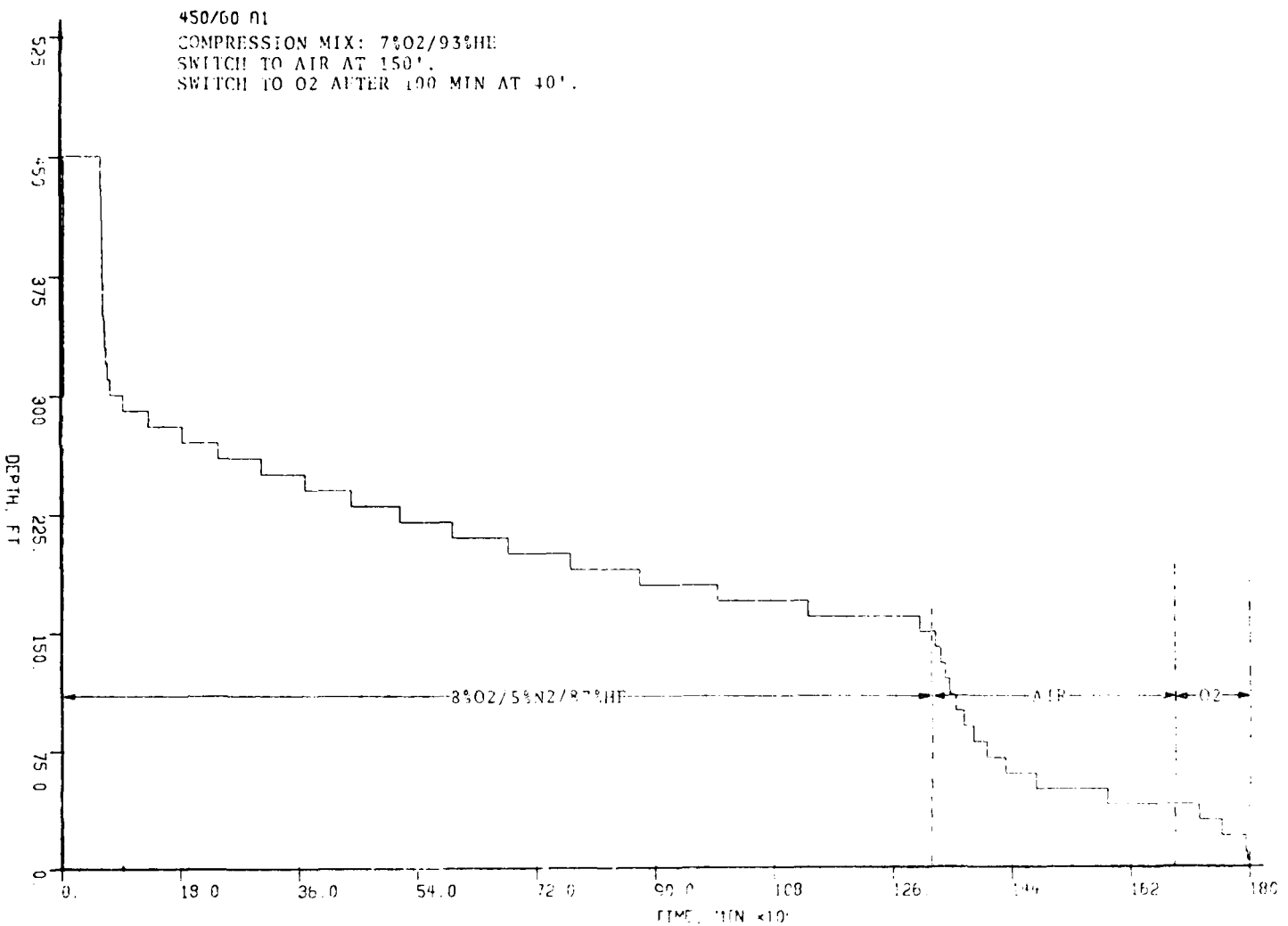

Fig. 2. Laboratory dive conducted at Duke University (Sept. 1976), that caused decompression sickness in two of six divers.

EFFECT OF DURATION OF EXPOSURE TO M VALUES ON THEIR VALIDITY

R. W. Hamilton, Jr., D. J. Kenyon and R. E. Peterson

That duration affects the validity of M values is a principle well appreciated by decompression researchers, but it is difficult to demonstrate in a quantitative way. The observation can be expressed in two ways, by saying either that computation procedures proven on shallow, short dives rarely work when extrapolated deeper and longer, or conversely, that a set of tables tested in dives in the deep range generally are too conservative in the shallow end.

A crude representation of this observation is shown in Table 1. This table compares five commercial decompression tables with the U.S. Navy helium-oxygen tables. The Navy tables, and Ocean System's Mark VI, have reasonable records in the short shallow range (200/30), but note that the commercial tables all have much longer decompression times in this range. At the other end of the table (300/60), where the commercial tables have a better record than the Navy tables, the times for the commercial tables are again longer.

We feel that this trend indicates clearly that the unmodified neo-Haldanian model does not work across the entire range, but that it can be made to work. A term or modification is needed in the computation scheme to account for this empirical observation. Theoretically, it could be stated that the supersaturation or bubble count or whatever is being monitored by the computations is time-related, and that exposure to these conditions, e.g., gas loadings, for longer times increases the probability that the limitations will not be adequate and that decompression sickness will occur.

Table 1. Comparison of several mixed-gas decompression tables

fsw/min	USN Partial Pressure	OSI Mk VI	3X Hybrid	TM-1		HKS-2* (surface-supplied)	HKS-3* (for bell use)	
200/30	14	10	14	14		14		% Oxygen
(60 msw)	80	90	40	30		147		First stop, fsw
	97	67	230	207		195		decompression time
200/60	14	10	14	14		14		% Oxygen
	80	120	120	120		147		First stop, fsw
	138	175	410	357		375		decompression time
300/30	14	10	10	14	10	14	10	% Oxygen
(90 msw)	130	150	170	160	180	147	179	First stop, fsw
	158	168	495	356	392	378	404	decompression time
300/60	14	10	10	14	10	14	10	% Oxygen
	130	200	220	210	220	195	212	First stop, fsw
	212	799	815	660	705	731	734	decompression time

*Nearest metric tables used: 45 msw = 147 fsw
55 msw = 179 fsw
60 msw = 195 fsw
65 msw = 212 fsw
90 msw = 293 fsw

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Not shown in the chart are several important factors, such as use of oxygen breathing, environmental conditions like work rate and cold, and the bends incidence. All these tables call for some oxygen breathing.

To assess properly results of the computations, one needs to look at results. These data are difficult to obtain, and possibly even more difficult to use once in hand. A small number of results of Navy dives might be available through the Safety Center, but most of the relevant dives on these tables have been commercial.

In a sample of 434 commercial dives on the Ocean Systems Mark VI tables, the following results were obtained. (Note: There was no statistical basis to the selection of this sample, and its relevance to company operations cannot be determined.) These showed dive/hit scores as follows:

	Duration		
Depth, fsw	0-20 min	20-35 min	35-50 min
190-310	84/0	27/2	53/4
310/430	164/14	83/8	23/0

Looking at this Mark VI data, we considered whether the dives were done at "face value" or had conservatism factors. Surprisingly, most were close to the nominal values, that is, the dives were carried to 80-85% of the table time and depth. However, almost all logs showed a longer total decompression time than the table called for (and we were not usually able to tell where the extra time was added), and generally more oxygen breathing.

Scores on the other deep tables shown in Table 1 are essentially all quite good. All have had limited to extensive field experience (a few to dozens of dives), including many to depths deeper than the depths shown in Table 1. We know of only one or possibly two cases of bends in dives on which the tables have been used properly.

IMPORTANCE OF OXYGEN IN MIXED-GAS DECOMPRESSION

R. W. Hamilton, Jr., D. J. Kenyon, M. R. Powell, and M. Freitag

This is an observation based on a series of previously unreported human laboratory dives conducted for the purpose of comparing the decompression properties of neon and helium. The dives were performed in 1974 at the Union Carbide Laboratory in Tarrytown. A total of 34 exposures were made to depths of between 150-400 fsw and for bottom times ranging from 30-120 minutes.

Decompression profiles were computed based on the Haldane-Workman-Schreiner approach. A 13-compartment model was used, with gas loadings compared to a matrix of ascent-limiting M values. All inert gases were added up in each compartment. The Schreiner half times reported at the Fourth Symposium (Schreiner and Kelley 1971) were modified using information from animal experiments and mass spectrometer tissue measurements. We assumed that all gases behaved in the same manner in the fastest compartments, i.e., were totally perfusion limited, and that they were proportional to diffusion constants in the longer half-time compartments; they were interpolated in between. Classical helium half times were used as the basis. Half times chosen were:

Compartment No.	1	2	3	4	5	6	7	8	9	10	11	12	13
Nitrogen, min	5	15	30	50	72	109	140	180	240	272	324	380	480
Helium, min	5	15	30	45	60	80	100	120	140	160	180	200	240
Neon, min	5	15	30	45	61	88	119	153	190	230	276	323	410

When this decompression computational model is used, previous experience can be injected by making adjustments in the ascent matrix. Two matrixes were used (Table 1), both based on the matrix developed in the NOAA OPS experiment (Hamilton et al. 1973). The reasons we liked the NOAA OPS matrix was that it was based directly on empirical data, and it had worked well. The NOAA OPS procedures have been called "conservative," e.g., by Hennessy (1977) and Keller (this symposium). In retrospect, it might have been more reasonable to start with an established helium matrix.

The dive series was in two parts. We started out to test representative parts of the time-depth range, but had to give this idea up because of an unusual response to the initial dives—nearly half the divers had decompression sickness, and some of these cases were troublesome and did not respond well to treatment. One of the original premises was that we could get a better comparison between the gases if we carried the divers all the way to the surface on the inert gas, without oxygen breathing. Attaining this proportion of decompression sickness (8 out of 20 exposures) was perhaps useful for comparing gases, but it was a disaster in every other respect. After making enough dives to determine we were getting nowhere (20), we revised the matrix, added oxygen breathing, and concentrated on a single table, 250 fsw/60 min. Results are shown in Table 2. This did a fair job of cleaning up the dive tables (box score: 1 limb bend in 14 dives).

Although both of the revised tables (the two on the right in Table 2) are longer, this amount of change in bends incidence is not consonant with this time change; we are convinced that the big factor in reducing the bends incidence was the addition of four 20-minute cycles of oxygen breathing. The oxygen breathing in the second series was not considered in the inert gas calculations. This is essentially the position we have subsequently arrived at in commercial computations; our tables require a specified session of oxygen breathing, which is not considered in gas loading computations. It is not optional, and in fact the matrixes we have developed are appropriate only if oxygen breathing is included.

Table 1. Matrix TL-01-M1

Depth, fsw	Compartment Number 1	2	3	4	5	6	7	8	9	10	11	12	13
100	230	193	173	170	167	162	157						
90	220	180	159	158	156	149	142						
80	210	170	149	141	138	132	119	118	118	118	118	118	118
70	200	161	140	130	127	123	114	113	113	113	113	112	108
60	190	151	131	119	117	110	108	106	105	105	104	103	98
50	172	140	120	108	106	99	98	97	94	94	93	91	87
40	154	126	108	97	93	88	87	85	83	82	81	80	77
30	136	111	93	83	80	76	76	74	72	72	71	70	69
20	118	96	78	69	67	64	64	64	61	61	60	59	57
10	100	80	64	56	54	53	52	51	50	50	59	48	46

Values are M values, maximum total inert gas for safe ascent (10 fsw); these values were used to prepare tables for the first 20 dives of the neon Spring series.

Matrix TL-01-M2

100	230	192	176	166	160	157	152	151	147	146	143	142	140
90	220	181	163	153	148	145	141	140	136	135	133	132	130
80	210	171	150	141	136	132	129	128	125	124	122	121	119
70	200	161	140	130	124	120	118	117	114	113	112	111	109
60	190	151	130	120	113	110	107	106	104	103	101	100	98
50	172	139	120	108	102	100	96	95	93	92	91	90	88
40	154	125	106	95	91	89	85	84	82	81	80	79	77
30	136	110	92	82	79	77	74	73	71	70	69	68	66
20	118	95	78	69	67	65	63	62	60	59	58	57	55
10	100	80	64	56	54	53	52	51	50	49	48	46	44

Used in later exposures in the neon Spring series.

Table 2. Comparison of 250/60 dives with and without oxygen

Schedule	250-H-1	250-H-2	250-H-3	250-N-2
Matrix	TL-01-M1	TL-01-M1	TL-01-M2	TL-01-M2
Gas	Helium	Helium	Helium	Neon
Oxygen used	No	No	Yes	Yes
First stop, fsw	130	130	130	130
Depart 20 fsw, min	273	279	281	223
Linear ascent used	No	No	20 to 10 fsw, 10 fsw to sfc	10 fsw to sfc
Linear ascent rates	–	–	-15 and -25 mpf	-20 mpf
Total time, min	425	449	681	523
Divers/bends	2/1	6/3	6/0	8/1

All bottom mixtures are 10% oxygen, balance inert. Neon is 75% neon, 25% helium. Oxygen breathing: 4 cycles of 20 min on, 5 min off.

The experiment revealed the following:

1. An unusual degree of non-pain-only decompression (4 of 8), regardless of inert gas, and these cases were hard to treat. They included numbness, blurred vision, nausea, and mild shock.
2. In both series, no difference in incidence of decompression sickness for neon and helium. The neon table was somewhat shorter for an equivalent dive, but this was the only one-to-one comparison made.
3. Oxygen breathing greatly improved the probability of achieving a clean decompression with this model.

This series provides another point relevant to the theme of this Workshop. Using a model with several new parameters, e.g., half times, M values, it was possible in just one iteration to produce reasonably workable decompression profiles. This supports our premise that "Haldane works if you use it properly." This has been stated by others in this Workshop, e.g., Keller, Bühlmann, Barnard . We long ago took an attitude different from the Navy's philosophy, which tends to cast diving procedures "in concrete," treating them (necessarily) with a high degree of dogma. Our policy has been that a set of decompression tables is a living thing, that it grows and conforms to its usage more or less continuously. We feel the Haldane technique, with all of its limitations, is a usable tool.

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THE ALTERNATING BUBBLE THEORY OF DECOMPRESSION

Brian A. Hills

VITAL QUESTIONS

Most commercial and naval decompression procedures have been formulated by trial and error or by empirical calculation methods; very few have been designed according to a model involving any physics or physiology. In calculation methods, it does not matter how many equations or coefficients are used, since these are determined empirically and amount to the art of curve-fitting. Such approaches have therefore provided useful means of interpolating between dive parameters; however, they leave much to be desired when extrapolating—for instance, a decompression method that is successful to 400 ft may fail completely when used to formulate a table for 500 ft.

It is therefore desirable to synthesize a mathematical model from fundamentals, but this requires answers to several vital questions for which answers must be found or assumed. These may be listed as follows:

1. IS CALCULATION WORTHWHILE?

Perhaps the best evidence on which to try to answer this difficult question is provided by the "bounce dive" curve, otherwise known as the no-stop decompression limits for air and heliox diving. These are purely experimental and yet, for the same individual, they show a well-defined relationship between depth and time. In fact these curves are so well defined that they offer strong evidence to support an underlying rationale and, hence, one well worth attempting to describe mathematically. These curves, however, are based upon limb bends only, and no such clearly defined relationship has been shown for neurologic decompression sickness or for any one of its sub-categories.

For a single decompression from steady state at a pressure P_1 to another P_2, the P_1 vs. P_2 relationship also indicates a characteristic relationship and hence an underlying rationale favoring the application of mathematical methods. This again is based upon limb bends only, although there is some indication that vestibular problems might show a similar tendency.

Unless there has been an accident, such as a blow-up, there is very little, if any, correlation between the incidence of limb bends and other symptoms. It is therefore difficult to justify invoking calculation for preventing anything other than limb bends.

2. HOW MANY TISSUES ARE INVOLVED?

Each "tissue" represents another constraint in the formulation of a decompression table and, hence, another equation to apply. A spectrum of half times for these represents a range of perfusion rates, as Haldane himself indicated. However, allotting different coefficients to these "hypothetical" tissues, e.g., M values, implies a different identity for each. The same symptoms, however, seem to occur whether we violate the 10-min tissue or the 40-min tissue, and none of the advocates of the Haldane calculation method have even shown any correlation between the hypothetical "tissue" violated and the nature of the symptoms.

It is therefore difficult to justify the involvement of more than one anatomical tissue. This is emphasized if we accept the answer offered to the previous question—that it is only worthwhile to calculate the imminence of limb bends. Reasoning such as this led Hempleman (1952, 1957) and others at R.N.P.L. to pursue a single-tissue model.

3. WHAT CAUSES LIMB BENDS?

It has long been known that the coefficients (M values) derived for the onset of bends are not the best values to use in formulating a decompression table. This is one example indicating that it is more economical in total decompression time to keep appreciably deeper than the bends-provoking depth even though the lowest pressure should give the fastest rate of 'outgassing' of tissues if critical supersaturation were the criterion for bends.

This indicates that there is not one critical point but two:

(a) a point where the primary insult is initiated—probably the formation of a stable gas phase; and

(b) a second point where that gas has grown to a size where the bubble can exceed a pain threshold. This pain threshold is probably mechanical in nature since limb pain, at least, is usually so easily and rapidly reversed by recompression.

The differentiation between these points is important, since it indicates that any coefficient, M value or otherwise, based upon a bend may be far from optimal for that stage of the calculation. In my opinion, the primary event is the initiation of a stable gas phase, and limb pain is caused only when the bubble has grown in a 'tight' tissue to a stage at which its pressure differential in excess of that of the adjacent tissue bends a nerve ending beyond its pain threshold (as induced experimentally by Inman and Saunders (1944)).

4. WHICH TISSUE IS RESPONSIBLE?

If one follows the above argument—that it is only feasible to calculate for limb bends and only one tissue is involved—it becomes highly desirable to identify that tissue anatomically. With all manner of monitoring devices available to us these days, it is essential to know where to look for bubbles. After all, Doppler meters used in the popular precordial position may be monitoring irrelevant bubbles from a fatty tissue that may be a poor analogue for the relevant tissue, especially after a long, complex decompression.

There is much evidence to implicate tendon, since it is a well-innervated, "tight" tissue located around joints and produces a pain identical to limb bends when its nerves are abused by local pressure differentials.

5. DOES THE AVOIDANCE OF MARGINAL LIMB BENDS AVOID OTHER SYMPTOMS?

In using the single-tissue approach, it is difficult to know whether one can avoid other symptoms. However, this is probably safe providing that one also avoids a number of factors that can potentiate neurologic symptoms and that have no direct bearing on the limb bends calculation. There may be many factors, but there is now evidence to identify two of these:

(a) Avoid unnecessary gradients of 'heavy' gases, e.g., nitrogen, between the middle ear cavity and the breathing mix to minimize the chances of vestibular problems. It is our experience that partial compression on air and the use of graded tri-mixes seem to eliminate vestibular decompression sickness after switching to air in the DDC when returning from 500-600 feet.

(b) Avoid arterial bubbles by keeping the lungs in good condition and not overloading them with venous bubbles. In our laboratory we have recently shown that factors, including recompression, oxygen poisoning, etc., can influence the effectiveness of dogs' lungs as a bubble trap. It is therefore my opinion that the danger of too much oxygen is not so much in injury to lung tissue but in facilitating the escape of bubbles into the arterial system.

Many factors in addition to (a) and (b) may also potentiate neurologic symptoms and may do so by mechanisms other than arterial bubbles. However, it seems that, in common with those factors cited above, they will not be conducive to mathematical formulation but may be avoided by paying heed to some aspect of the dive other than those aspects normally included in the computation.

6. HOW IS GAS TAKEN UP?

Once a tissue is implicated by name, whether correctly or not, it is possible to study the circulation to that tissue and ascertain its diffusion coefficients to determine whether blood perfusion, diffusion, or both processes control gas transfer. In the past the evidence on this issue has been most conflicting. Differences in the helium and oxygen bounce-dive curves do not follow Graham's law (favoring perfusion), and yet both conform to a $\sqrt{t}$ relationship (favoring diffusion). However, direct observation of the capillaries in tendon offered a simple explanation, as will be discussed below in connection with the alternating bubble theory of decompression.

7. WHAT IS THE OPTIMAL DECOMPRESSION?

There are several schools of thought on how to select the optimal depth for a particular tissue gas content, assuming this to be correct. To consider only the extreme views:

(a) By neo-Haldanian reasoning, one decompresses as far as the M value will allow in order to create the greatest degree of supersaturation without forming bubbles, and hence the largest driving force for eliminating more gas.

(b) By "thermodynamic" reasoning, on the other hand, one cannot exceed true saturation without provoking some bubble or other. In this case it should be remembered that the point of true saturation must account for the inherent unsaturation occurring by virtue of metabolic consumption of oxygen and the failure of carbon dioxide to complement it tension-wise. This leads to much deeper initial stops in the decompression table. If a stable gas phase is formed, the rate of elimination is greatly diminished, since only gas in true physical solution can contribute to the driving force.

This conflict of views has produced an interesting controversy over the last decade but, once one names a tissue and actually observes it under a microscope during decompression, the whole issue seems academic. It is now my opinion that although the thermodynamic approach postulated bubbles for any potential degree of supersaturation, it did not go far enough. In other words, it is not feasible to avoid separation of gas from solution, and the real issue is how to control the sizes of the bubbles present almost from the outset of a decompression.

THE ALTERNATING BUBBLE APPROACH

TIME AVERAGING

The thermodynamic approach and all others, neo-Haldanian or otherwise, have all implicitly assumed time averaging. For instance, all tissue gas contents vary continuously according to the same function, whether this is related to blood perfusion, diffusion, or is just derived empirically.

Most studies of blood flow distribution refer to skeletal muscle: hence the fact that blood flow in one small artery fluctuates by a ratio of 10:1 is ignored, since it does so twice per minute. In any case, there is collateral flow at the microvascular level, so there is no reason to question time averaging.

However, the pattern may be much different in a tissue of much lower metabolic rate, and therefore a direct study has been made of the microvascular dynamics of tendon.

MICROVASCULAR DYNAMICS OF TENDON

A study of the Achilles tendons of 40 bullfrogs and 8 guinea pigs has shown that adjacent capillaries open and close as described for muscle by Krogh (1918), but rather more slowly. However, superimposed upon this 'flickering' is a much slower process in which whole bundles of 20-147 capillaries open and close, with little overlap in the tissue areas perfused by each. These zones spanned 1-2 mm., i.e., large diffusion distances by tissue standards. Periods of no flow averaged 39 min in 70 bundles in bullfrogs and 43 min in 34 bundles in guinea pigs. Moreover, a few bundles failed to open in 100 min (Hills 1978).

This immediately raises the question of what would happen if a bundle were to close just before the start of a long decompression.

OBSERVATION WITH DECOMPRESSION

When the tendon is observed in a lightly anesthetized guinea pig under decompression, a mass of small bubbles can be seen, primarily extravascular but a few intravascular. During decompression, these grow in the non-perfused zones and then rapidly decrease in size when the capillary bundle in which they are located is perfused. Thus a pattern of alternating bubbles develops in which most are growing slowly while others are shrinking rapidly.

Unfortunately, the capillary bundles do not open sequentially and there is a certain degree of randomness to their patency. This might add credence to a minority viewpoint—that however much decompression time is allowed, there is always a significant chance of a bend occurring. This "alternating bubble" theory is compatible with the concept that all decompressions are really treatments, and may explain why tables with widely differing profiles can be equally successful or unsuccessful.

This theory can explain the complex mixture of perfusion and diffusion parameters, since the tissue is primarily controlled by perfusion in, however, two zones:

(a) very well-perfused zones in the open bundles where the high flow rate makes diffusion an insignificant factor, or

(b) non-perfused zones where gas can only escape to a neighboring open bundle by diffusion.

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DECOMPRESSION THEORY WORKSHOP: RESPONSES TO THE TWELVE ASSUMPTIONS PRESENTLY USED FOR CALCULATING DECOMPRESSION SCHEDULES

David E. Yount

INTRODUCTORY STATEMENT

Decompression sickness studies at the University of Hawaii have emphasized nucleation—the mechanism by which bubble formation and growth are initiated in aqueous media. In this sense, we have been influenced more by E. Newton Harvey than by J.S. Haldane, and our views frequently differ from the neo-Haldanian model that forms the basis of the U.S. Navy's current decompression calculations. To facilitate a comparison of the two schools of thought, we have elected to respond to all twelve of the assumptions around which this Workshop has been organized. Since our starting point is different and since there is a good chance that some of our responses, taken out of the context of our model, might be misunderstood, we shall begin with a brief outline of our own present assumptions for calculating decompression schedules.

1) Decompression sickness is associated with bubble formation in blood or tissue.
2) Bubble formation in tissue is dominant. Formation also occurs in blood, but the formation threshold is higher and the blood volume much smaller. Most bubbles observed in blood originated in the tissue.
3) As a first approxi- ation, tissue is considered to be homogeneous. For example, differences in body organs, the detailed geometry of capillary beds, and the presence of cell walls are not taken into account.
4) At the onset of symptoms of mild decompression sickness, bubble growth in tissue is limited to a few microns in radius by the tissue deformation pressure δ. Bubble growth in blood is not limited in this way, and bubbles exuded from tissue into blood frequently exceed 50 microns in radius.
5) Bubble formation is initiated by gas nuclei that are stable over periods of hours or days.
6) Gas nuclei are ordinarily present in all aqueous media, including blood or tissue, unless some means has been provided for removing them.
7) While gas-filled crevices can serve as nucleation sites, most bubbles found in tissue originate as spherical gas nuclei surrounded by a stabilizing film or membrane of surface-active molecules.
8) The number of bubbles which form in a given situation is determined by the number of gas nuclei larger than some critical radius r_c. Nuclei smaller than r_c will not grow into bubbles and can therefore be neglected.
9) The critical radius for bubble formation is related to the supersaturation pressure by means of an equation of the form

$$p_{ss} = \xi / r_c \tag{1}$$

where ξ depends upon the characteristics of the nuclear skin and where the supersaturation pressure is defined to be the difference between the dissolved gas tension τ and the ambient pressure p_{amb}:

$$p_{ss} \equiv \tau - p_{amb} \tag{2}$$

If there is no skin, or if the skin is no longer intact, then ξ is equal to twice the surface tension γ, as in the case of ordinary bubbles.
10) It follows from 4) and 8) that the volume of gas released from tissue in the form of bubbles is proportional to the number of nuclei that were present with radii larger than r_c when the supersaturation pressure reached the level given in Eq. 1.
11) At the onset of symptoms of mild decompression sickness, the number of supercritical nuclei per unit tissue volume is small, e.g., the average distance between nuclear centers is at least two orders of

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magnitude larger than the limiting radius for bubble growth determined by the tissue deformation pressure δ. It follows from 4) and 10) that the quantity of gas released into bubbles is a small fraction, e.g., less than 10^{-5}, of that originally dissolved.

12) The volume of released gas is critical in determining the onset of decompression symptoms. From 10) this is equivalent to saying that the number of nuclei larger than r_c is critical since this determines the number of bubbles and hence the released gas volume.

13) From 12) it follows that decompression schedules yielding the same bubble number will also yield, on the average, the same decompression outcome and that the isopleths for constant N will also be isopleths for constant effective dose ED.

14) From 9), 10), and 12), it follows that decompression schedules should be designed so as not to exceed the critical supersaturation pressure p^*_{ss} that is associated via r^*_c and N* with an acceptable effective dose ED*. (The Haldane ratio principle is replaced by a pressure-difference principle.)

15) The number of nuclei N[r(t)] larger than a given radius r(t) at any time t in a particular pressure schedule can be derived from the initial distribution N[r(0)], the pressure history, and the dynamic characteristics of the nuclei involved. Conversely, one can work backwards through an appropriate set of pressure histories and outcomes to infer the initial radial distribution and the behavior of nuclei typical of that group of subjects. In this way, the decompression criteria p^*_{ss} for the group and for an arbitrary exposure can be determined.

Once the allowed supersaturation p^*_{ss} has been found, the calculation of the Hawaii decompression tables parallels that of the U.S. Navy. A range of time constants or tissue half times is used to describe gas uptake. The same set of constants is used to describe gas elimination. The gas tension τ associated with each time constant is then computed as a function of time t. Decompression is carried out by requiring that the difference between the dissolved gas tension and the ambient pressure never exceed p^*_{ss}

$$p_{ss} \equiv \tau - p_{amb} \leq p^*_{ss} \tag{3}$$

Ordinarily, p^*_{ss} is constant for a particular pressure history and has the same value for each tissue compartment throughout decompression. Hence decompression is controlled at any given time by that tissue which has the highest dissolved gas tension τ. In cases where spontaneous regeneration of gas nuclei is significant, p^*_{ss} decays exponentially with time, approaching its usual value at atmospheric pressure, about 0.6 atm. Our views on these and other topics are given in more detail in the discussion which follows of the U.S. Navy's decompression model.

THE U.S. NAVY'S NEO-HALDANIAN MODEL

A. GAS UPTAKE

1) Multiple tissues or tissue compartments must be used to describe whole body gas exchange.

We are in general agreement with this assumption, although we prefer to discuss time constants or half times rather than tissues or tissue compartments. There is ample evidence that a wide range of time constants is involved in decompression since, for example, the susceptibility of a diver exposed to elevated pressure increases with time, not only during the first few minutes or hours, but even during the first few days or weeks. On the other hand, whereas some of these differences may be associated with specific tissues, others may depend upon such poorly understood phenomena as the *in vivo* elimination and regeneration of gas nuclei.

2) Tissue gas uptake is described by the following items: exposure time, pressure differential, and tissue half times (gas characteristics). This relationship can be expressed mathematically by the following

equation

$$Pt = Po + (1 - 2^{(-T/H)})\,(Pa - Po) \tag{4}$$

Again we are in general agreement with the stated assumption, although we reiterate our reservations about tissue half times *per se* and view this proposition merely as a convenient method for introducing a range of time constants. To illustrate this point, we note that Eq. 4 can be obtained by solving the perfusion equation

$$dP\,(T)/dT = [Pa - P\,(T)]\,(c/s) \tag{5}$$

where c is the gas uptake parameter and s is the solubility. It follows that the half time H is related to c and s by the equation

$$H = s\,\ln\,(2)/c \tag{6}$$

Now the ratio c/s can increase by an order of magnitude during exercise: hence, the half time H in Eqs. 4 and 6 is by no means constant and can vary by a similar factor. Furthermore, whereas exercise tends to decrease H and would seem to be helpful in eliminating gas during decompression, the net effect is detrimental. This paradox can be resolved by noting that exercise, while it does speed up gas elimination, also stimulates the growth of pre-existing gas nuclei into macroscopic bubbles. The latter is more decisive than the former in the etiology of this disease syndrome.

3) Only the inert gas partial pressure need be considered in the decompression problem.

Technically, this assumption is certainly wrong. To prevent gas nuclei from growing into macroscopic bubbles, it is necessary that the supersaturation pressure p_{ss} remain always less than some critical value p^*_{ss}. The supersaturation pressure, in turn, is defined to be the difference between the total dissolved gas tension $\tau = \Sigma\tau_i$ and the ambient pressure p_{amb}

$$p_{ss} \equiv \Sigma\tau_i - p_{amb} \leqslant p^*_{ss} \tag{7}$$

where τ_i are the dissolved tensions of all of the gases present, including not only the inert gases, but also O_2, CO_2, and water vapor. In some cases, the tensions of O_2, CO_2, and water vapor may be negligible in comparison with the critical supersaturation p^*_{ss} and with the inert gas tensions, but this is not true in general. Furthermore, O_2 and CO_2—because of their activity and the high solubility of the latter—may play a more significant role than would inert gases at the same tensions.

4) Breathing multiple inert gases appears to produce some decompression advantage. Methods of incorporating these advantages into the decompression model are not yet understood.

As outlined above, safe decompression requires that the supersaturation pressure p_{ss} remain always less than some critical value p^*_{ss}

$$p_{ss} \equiv \Sigma\tau_i - p_{amb} \leqslant p^*_{ss} \tag{8}$$

where p_{amb} is the ambient pressure and $\Sigma\tau_i$ is the sum of the various gas tensions and the vapor pressure of water. At the present level of our approximations, no distinction is made between different gas types, either in summing the gas tensions to evaluate $\Sigma\tau_i$ or in determining the decompression criterion p^*_{ss}. This is not an intrinsic feature of our cavitation model but relates instead to the way in which we have begun to apply

the model to decompression sickness. For example, we have thus far assumed that a particular effective dose ED or level of severity is associated with a definite bubble number N and that N is independent of the type of inert gas being used. It is certainly possible that the critical bubble number N* for an acceptable effective dose ED* differs from one gas to another, in which case p^*_{ss} would have to be adjusted accordingly.

The effect that different inert gases have upon bubble counts in supersaturated gelatin has been investigated at the University of Hawaii by C.M. Yeung (1975). His results for *nitrogen, helium, neon, and argon* are summarized in Fig. 1. The rudimentary pressure schedule shown in the inset in the same figure consisted of a rapid compression from atmospheric pressure p_o to the maximum pressure p_m, saturation of the samples at $p_s = p_m$, and a rapid decompression from p_s to the final pressure $p_f = p_o$. The time allowed for saturation at p_s was 5.25 hours for nitrogen and 8.00 hours for the other gases. This is 4.4 or more time constants for gas uptake in each case. Whereas some variation is observed in the bubble counts for different gases, particularly above 100 psi, the close similarity of the four data sets is readily apparent.

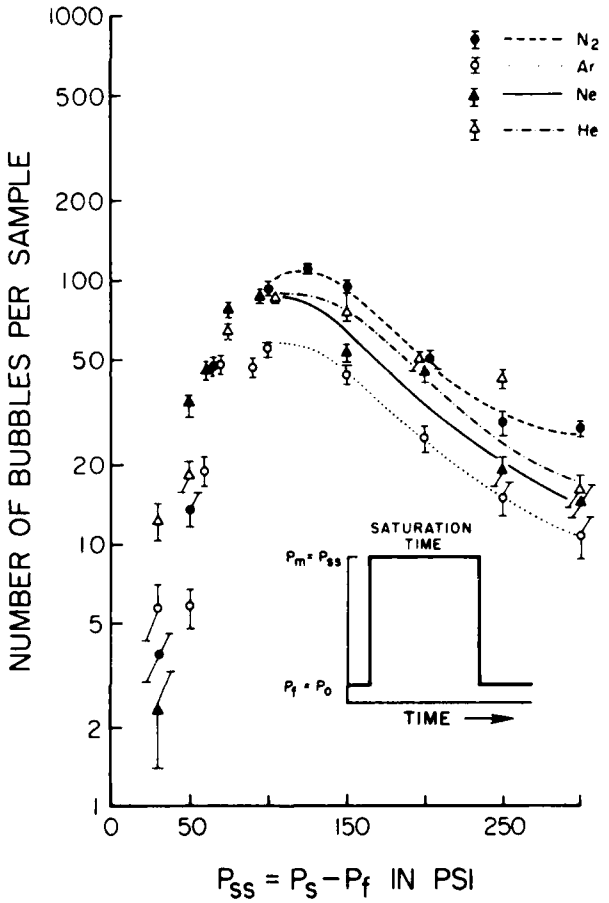

Fig. 1. Bubble counts per 0.4 ml gelatin sample vs. supersaturation pressure p_{ss} in psi. Results plotted are for nitrogen, argon, neon, and helium.

Even when inert gases are indistinguishable as far as the decompression criterion p^*_{ss} is concerned, they have other physical properties that can be exploited to gain a decompression advantage. The most important of these is probably the diffusion coefficient, which implies that different gases have different rates for uptake and elimination. This gives table calculators another parameter to vary—another game to play in which the object is to find a combination of gases, i.e., time constants, that is optimal for a particular exposure.

To illustrate this point, we consider the rudimentary example shown in Fig. 2. Only one tissue compartment is involved, and the tissue half times for two different inert gases in this compartment are 20 minutes and 40 minutes, respectively. No other gases or vapors are present. The compartment is subjected to a pressure of 4 atm abs for a period of 80 minutes. Any combination of the two inert gases is permitted which satisfies the given conditions, and the goal is to find a pressure scenario such that the compartment can be decompressed as rapidly as possible without exceeding the critical supersaturation p^*_{ss}.

The value of p^*_{ss} can be obtained from the data in Table 1, which is a compilation of critical pressure formulas adapted from a recent article by Hennessy and Hempleman (1977). These authors extracted from oxy-helium experiments by Barnard (1976) and from various oxy-nitrogen decompression tables (Bühlmann 1969; Des Granges 1956; Hempleman 1969; Nishi and Kuehn 1973; Workman 1965) combinations of pressure reductions and exposures characteristic of tissues with the longest half times for gas uptake and elimination. The resulting parameterizations are all of the same form

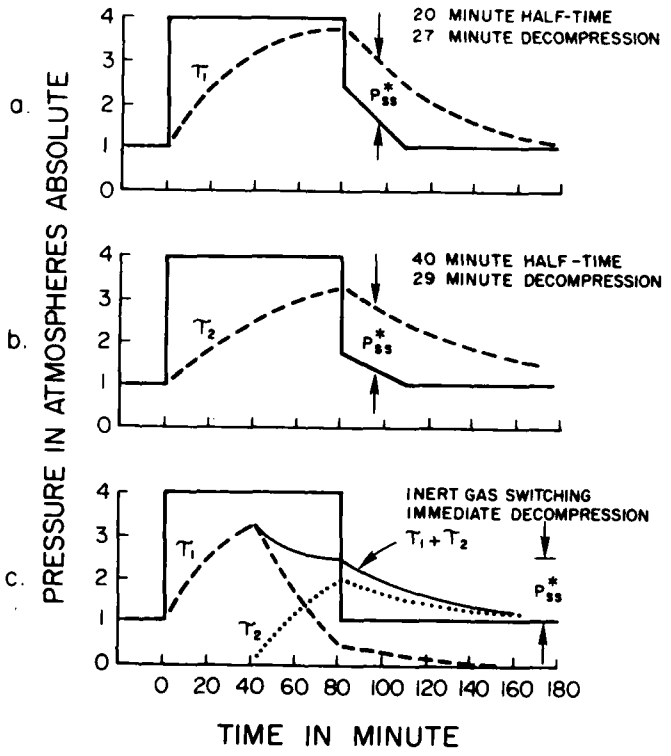

Fig. 2. Illustration of how inert gas switching can be used to gain a decompression advantage.

Table 1. Critical Pressure Formulas

Formula, atm abs	Source	Application
$P_1 = 1.397\ P_2 + 0.57$	Barnard (1976) Experimental data	
$P_1 = 1.518\ P_2 + 0.46$	Des Granges (1956) 120-min Tissue	USN Standard Air Tables
$P_1 = 1.366\ P_2 + 0.56$	Hempleman (1969) $1.9 < P_1 < 7.0$ atm abs	RNPL Tables
$P_1 = 1.375\ P_2 + 0.52$	Workman (1965) M value, 240-min Tissue	USN Mixed Gas Tables
$P_1 = 1.385\ P_2 + 0.42$	Nishi and Kuehn (1973)	Canadian Tables
$P_1 = 1.401\ P_2 + 0.47$	Bühlmann (1969) 240-min Tissue $1.0 < P_1 < 7.0$ atm abs	Swiss Tables

$$P_1 = a\ P_2 + b \tag{9}$$

where P_1 is the absolute pressure breathed by men for a period approaching equilibrium, P_2 is the pressure to which it is just safe to decompress rapidly, and a and b are constants referenced to an equivalent gas mixture of air. The critical supersaturation for $P_1 = 4$ atm abs, averaged over the six entries in Table 1, is $p^*_{ss} = 1.5$ atm.

If only the fast gas is used (Fig. 2a), the time required for decompression is about 27 minutes. If only the slow gas is used (Fig. 2b), 29 minutes are needed. However, if we switch from the fast gas to the slow gas after 40 minutes (Fig. 2c), the sum of the two dissolved gas tensions at the end of 80 minutes is only 2.4 atm abs. Without exceeding $p^*_{ss} = 1.5$ atm, the pressure can immediately be reduced to 1 atm abs, and no decompression stops are required.

5) Compression procedures have no effect on decompression.

This assumption is certainly wrong. It is wrong because bubble formation in aqueous media, including human tissue, is caused by pre-existing gas nuclei and because the status of these nuclei at any point in a dive profile depends in a detailed way upon the pressure history. This introduces into the decompression problem a significant factor that is outside the usual considerations of gas uptake and elimination.

As one example, the rate of compression, if it is sufficiently slow, can be of crucial importance. This is illustrated in Fig. 3, in which the number of bubbles counted by Yount and Strauss (1976) in supersaturated gelatin is plotted as a function of the supersaturation pressure

$$p_{ss} = p_s - p_f \tag{10}$$

where p_s is the pressure at which the samples were saturated and p_f is the final pressure after a single-step

decompression. For our purposes, a "rapid compression" is one that is completed in an interval that is short compared to the relevant sample or tissue half times. A "slow compression" is one that is not. The rapid compressions shown in Fig. 3 were carried out at approximately 3 psi per second, while the slow compressions were performed in 15-psi steps taken every half hour. The half times for that portion of the gelatin volume in which bubbles were counted ranged from 13 to 51 minutes.

The rapid-compression and slow-compression data in Fig. 3 differ because the crushing pressure

$$p_{crush} \equiv (p_{amb} - \Sigma\tau_i)_{max} \tag{11}$$

where p_{amb} is the ambient pressure and $\Sigma\tau_i$ is the total dissolved gas tension are different. For fast compressions, Eq. 11 gives

$$p_{crush} = p_m - p_o \tag{12}$$

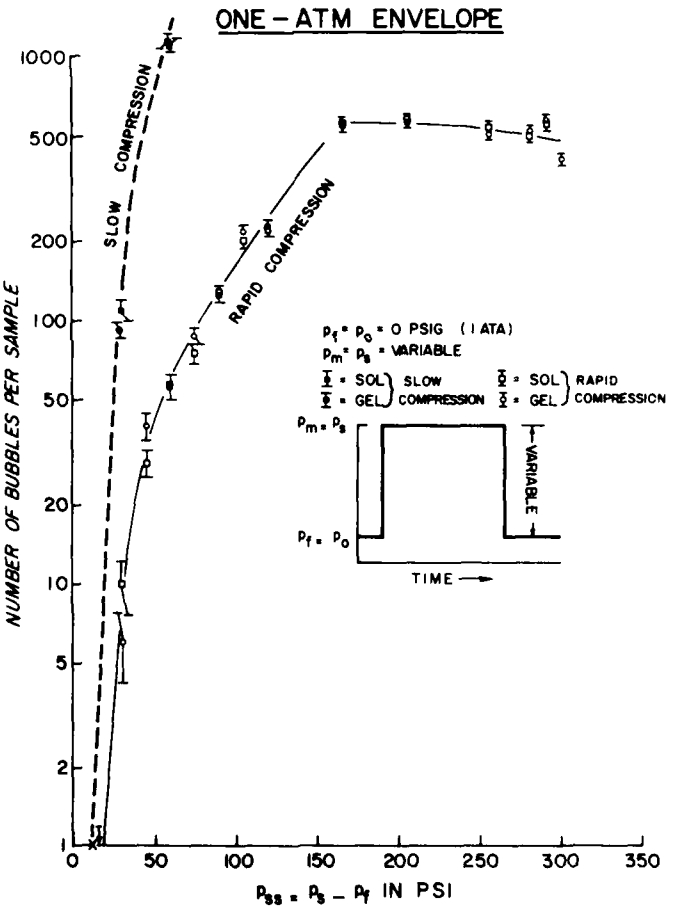

Fig. 3. Number of bubbles per 0.4 ml gelatin sample vs. supersaturation pressure p_{ss} in psi. Slow compression yields are typically an order of magnitude higher than those for fast compression at the same p_{ss}.

where the difference between the maximum pressure p_m and the initial pressure p_o ranges up to 300 psi. For slow compressions, the dissolved gas tension follows the ambient pressure, limiting p_{crush} to values less than or equal to 50 psi.

A comprehensive study of the effect of crushing (Yount and Strauss 1976) is summarized in Fig. 4. In this case, all compressions are rapid, and the strong dependence of the bubble number on both p_{crush} and p_{ss} is readily apparent.

From the data in Fig. 4, combinations p_{crush} and p_{ss} can be found that yield a fixed bubble number: N = 1, 3, 10, etc. In this way, one can obtain a plot of p_{ss} versus p_{crush} for each value of N selected, as in Fig. 5. The slow compression data in Fig. 3 can also be plotted in Fig. 5 once the effective values of

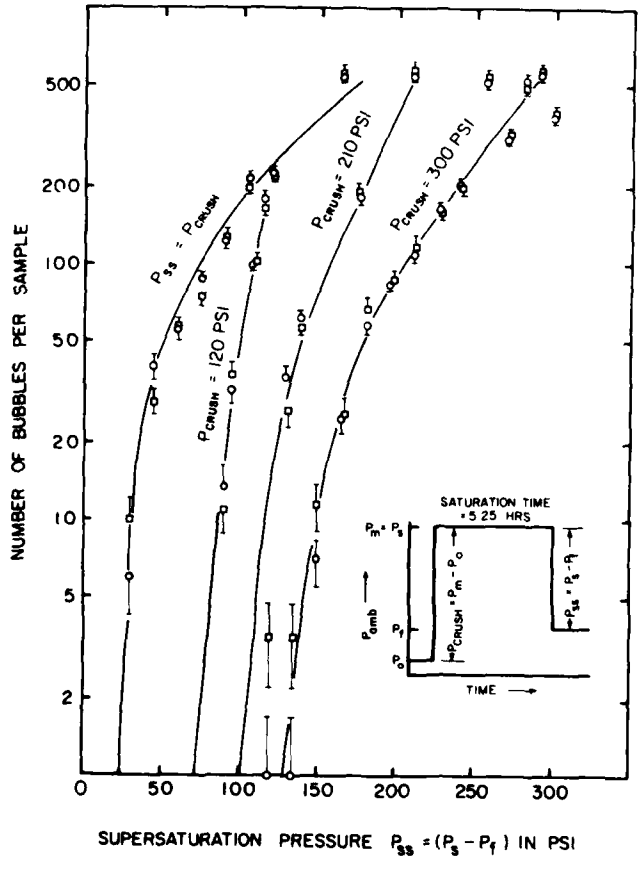

Fig. 4. Comprehensive study of effect of initial compression p_{crush} on bubble yield in supersaturated gelatin. Solid curves are predictions of a cavitation model in which spherical gas nuclei are stabilized by surface-active skins of varying gas permeability.

p_{crush} have been determined. When this is done, it is found that the slow-compression data, confined to values of p_{crush} less than or equal to 50 psi, lie on the appropriate lines of constant N.

As a second example of the importance of compression procedures, another test carried out by Yount and Strauss (1976) is illustrated in Fig. 6. The pressure schedules labeled A and C had the same supersaturation, p_{ss} = 150 psi, but the crushing pressures, p_{crush} = 300 psi and p_{crush} = 150 psi, were different. Schedule A yielded (18 ± 1) bubbles per sample, and Schedule C yielded (519 ± 8) bubbles per sample.

B. PRESSURE REDUCTION

6) Tissue supersaturation limits are best described by a series of decreasing pressure reduction ratios ranging from 1.8 to 1 to 1.1 to 1 for nitrogen and 1.5 to 1 to 1.0 to 1 for helium.

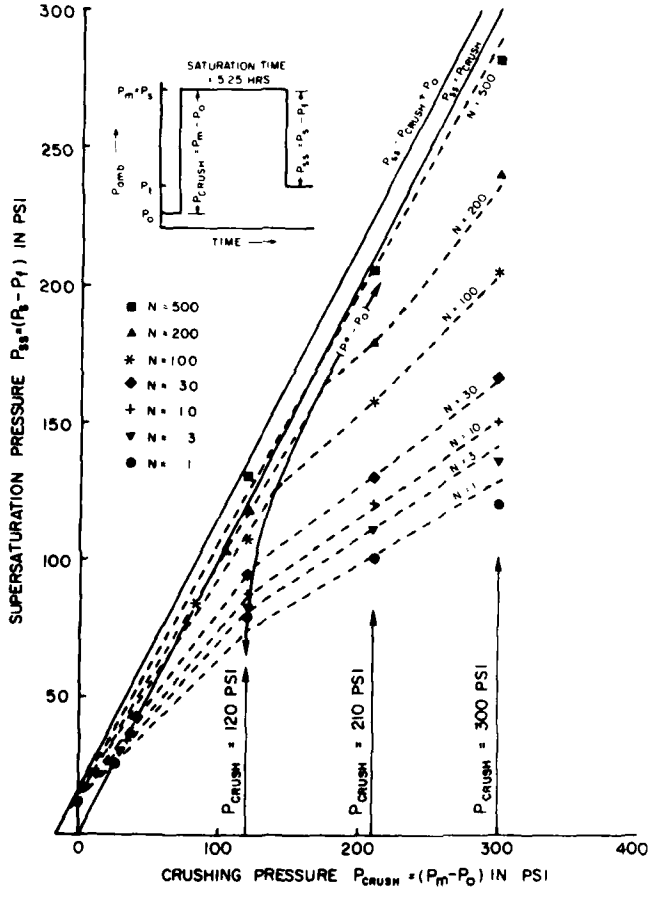

Fig. 5. Supersaturation pressure p_{ss} vs. crushing pressure p_{crush} for isopleths of constant bubble number N = 1, 3, 10, 30, 100, 200, and 500. Dashed curves are predictions of the varying-permeability nucleation model.

This assumption is certainly wrong. A specific test of the Haldane ratio principle in gelatin (Yount and Strauss 1976) is outlined in Fig. 6. For Schedule A the pressure reduction ratio after supersaturation was 11.2 to 1, while for Schedule B it was 1.91 to 1. The difference in these ratios is enormous, yet the bubble counts were nearly the same: (18 ± 1) bubbles per sample in the first case and (14 ± 1) bubbles per sample in the second. This similarity is expected from nucleation theory since samples subjected to the respective pressure schedules have equivalent pressure histories (p_{crush} is the same) and identical supersaturation p_{ss}. The importance of the pressure history was tested with Schedule C, which has the same supersaturation (p_{ss} = 150 psi) as Schedules A and B, but a smaller compression (p_{crush} = 150 psi instead of 300 psi). Schedule C yielded (519 ± 8) bubbles per sample.

Although the basic premise is wrong, values of the pressure reduction ratio P_1/P_2 can still be calculated for various decompression data, where P_1 is the absolute pressure breathed by subjects for a period approaching equilibrium and P_2 is the pressure to which it is just barely safe to decompress rapidly. We consider first the compilation of Hennessy and Hempleman (1977), shown in Table 1. The average of the six entries is

$$P_1 = 1.407\, P_2 + 0.50 \text{ atm abs} \tag{13}$$

For the interval over which Eq. 13 is valid, e.g., for 1 atm abs $\leqslant P_1 \leqslant$ 10 atm abs, we find a range of pressure reduction ratios of

$$2.81 \geqslant P_1/P_2 \geqslant 1.48 \quad \text{(Table 1)} \tag{14}$$

Any resemblance between these values and the pressure reduction ratios in the stated assumption is probably coincidental.

Equation 13 can be extrapolated to pressures above 10 atm abs by using nucleation theory. Two examples from a recent preprint (Yount 1978) are plotted in Fig. 7. The curves labeled VP1 and VP2 coincide exactly with Eq. 13 at exposures P_1 up to 9.2 atm abs and 7.5 atm abs, respectively. A more conservative calculation, VP3, is also shown. Among these, VP2 appears to agree best with the high-pressure data of Flynn and Spaur (1976) and of Spaur et al. (1976), and it passes just below the single-incidence points at 1000 fsw and at 1500 fsw. Certainly VP2 is much more reliable above 10 atm abs than a linear extrapolation of Eq. 13. The pressure reduction ratio for VP2 at 46 atm abs is 1.33. The range for VP2 at exposures 1 atm abs $\leqslant P_1 \leqslant$ 46 atm abs is thus

$$2.81 \geqslant P_1/P_2 \geqslant 1.33 \quad \text{(VP2)} \tag{15}$$

In Fig. 8, nucleation theory (Yount 1978) is compared with the pressure reduction limits obtained for rats by Berghage, Gomez, Roa, and Everson (1976). The data points correspond to a 50% effective dose ED, and were found by these authors by fitting their raw data for the various ED's plotted in Fig. 9. The model calculation labeled VP5 appears to describe the 50% ED data rather well at pressures up to 42 atm abs. At pressures of 45 atm abs and above, the experimental points are highly variable, and no definite conclusions can be drawn. For exposures in the interval 1 atm abs $\leqslant P_1 \leqslant$ 60 atm abs, the pressure reduction ratios for VP5 are in the range

$$7.97 \geqslant P_1/P_2 \geqslant 2.00 \quad \text{(VP5)} \tag{16}$$

The ratios are significantly higher for VP5 than for VP2, not because rats are different from humans, but mainly because the decompression outcomes for rats are far more severe.

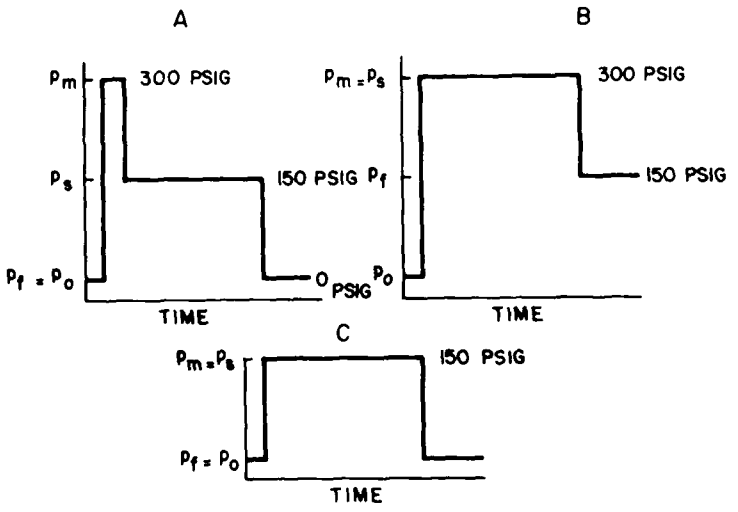

Fig. 6. Test of Haldane ratio principle in supersaturated gelatin. Schedules A and B have pressure reduction ratios (P_1 / P_2 = 11.2 and 1.91) that are very different, yet bubble counts (18 ± 1 and 14 ± 1 bubbles per sample) are nearly the same. Schedules A and C have pressure reduction ratios that are the same (P_1 / P_2 = 11.2), yet bubble counts (18 ± 1 and 519 ± 8 bubbles per sample) are very different.

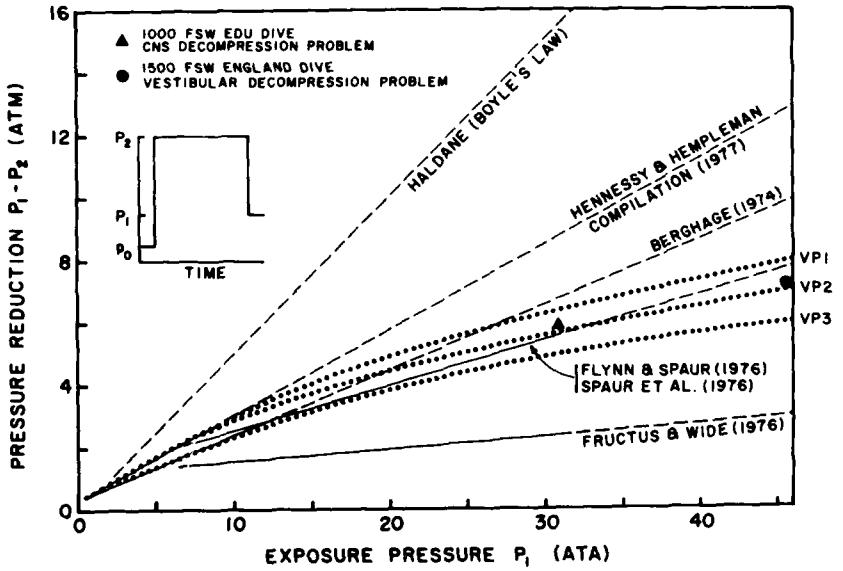

Fig. 7. Pressure reduction limits for humans. Below $P_1 \simeq 10$ ATA, available data can be summarized by straight line $P_1 = 1.407\ P_2 + 0.50$ ATA, derived from compilation of Hennessy and Hempleman (1977) shown in Table 1. At higher pressures, data fall well below this line and are better described by varying-permeability nucleation model calculations, such as VP2.

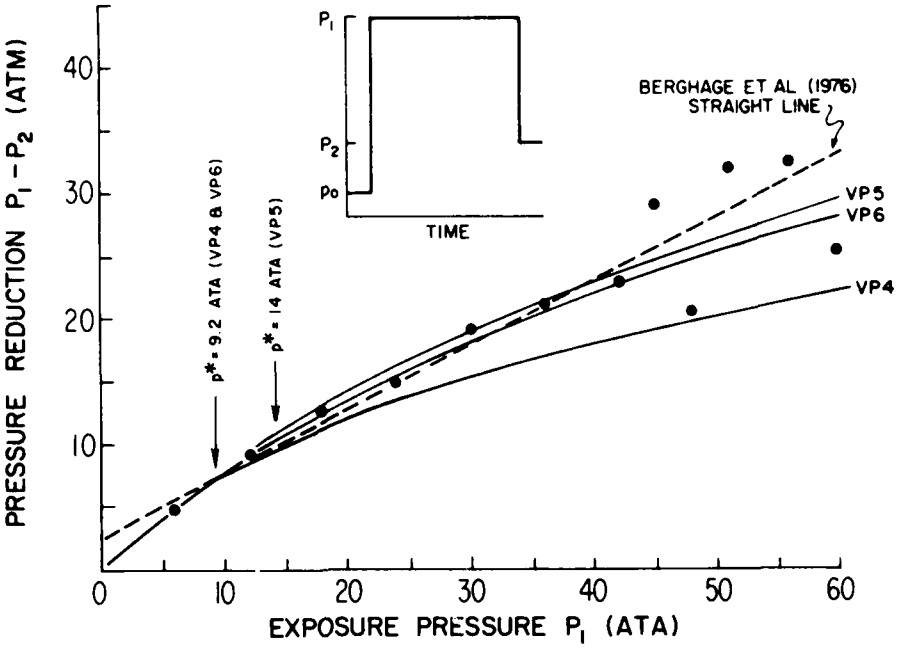

Fig. 8. Pressure reductions required to produce decompression sickness in 50% of rats saturated with helium-oxygen gas, described by Berghage et al. (1976). Curves VP5 and VP6 from varying-permeability nucleation model are in better agreement with data points from 6 to 42 ATA than a straight line. At pressures of 45 ATA and above, data are erratic, and no definite conclusions can be drawn.

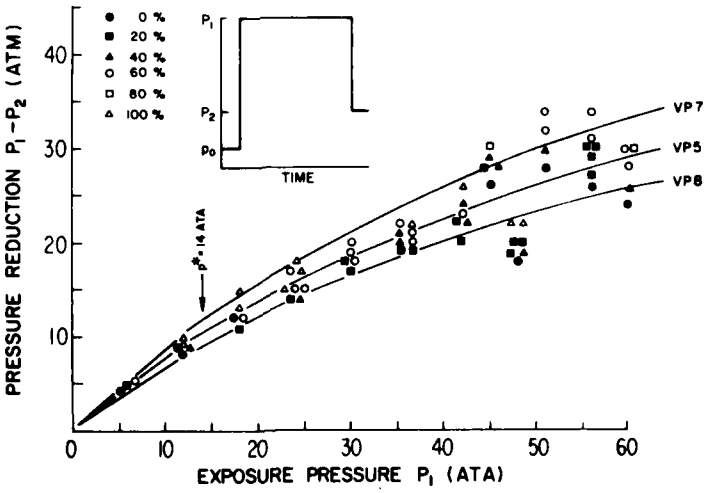

Fig. 9. Pressure reductions for various effective doses of decompression sickness in rats saturated with helium-oxygen gas and described by Berghage et al. (1976). Each dot represents 5 animals. The ED_{50}'s plotted in Fig. 8 were obtained by Berghage et al. (1976) from a least-squares analysis of points shown here at each of the respective pressure settings. Calculation labeled VP5 is in excellent agreement with the ED_{50}'s in Fig. 8, while VP7 and VP8 illustrate how a range of response levels can be accommodated by the varying-permeability nucleation model.

In applying nucleation theory to decompression sickness in rats and humans, we have assumed that lines of constant effective dose ED are also lines of constant bubble number N, i.e., that bubble number is critical in this disease syndrome. This hypothesis is being tested at the University of Hawaii by Watt and Lin (private communication 1978) using a perivascular Doppler probe to detect gas emboli in the rat posterior vena cava caudad to the renal veins. Their preliminary results are plotted in Fig. 10. Also shown are the ED_{50} points of Berghage et al. (1976) at 6, 12, and 18 atm abs, the Berghage et al. (1976) straight line, and three model calculations, VP5, VP7, and VP8 from Fig. 9. There are several important conclusions that can be drawn. First, the two data sets appear to be in excellent agreement in that the bubble formation thresholds (lines of constant N, where $N \sim 1$) measured by Watt and Lin (unpublished observations) lie just below the ED_{50} limits of Berghage et al. (1976), as expected. Second, the straight line of Berghage et al. (1976), which gives an acceptable description of the ED_{50} data in the range 6 atm abs $\leq P_1 \leq 42$ atm abs, is not very useful below 6 atm abs. Finally, the Doppler bubble-detection thresholds determined by Watt and Lin for a second dive performed 24 - 96 hours after the first exposure are higher than those obtained on the first dive. We believe that acclimation of this type results from a depletion of the reservoir of gas nuclei on previous exposures.

7) Tissue supersaturation limits (pressure reduction ratios) increase with increasing depth.

This assumption is improperly stated and should either read: "Tissue supersaturation limits increase with increasing depth" or "Pressure reduction ratios decrease with increasing depth." With this revision, the assumption is generally valid, as can easily be seen from Eq. 13, from the data in Table 1, from the data and model calculations shown in Figs. 5, 7, 8, 9, and 10, and from our discussion of Assumption 6. However, there are exceptions. On dives of very long duration, such as the Tektite series (pressures up to 100 fsw and durations up to 60 days), the limiting supersaturation pressure p^*_{ss} approaches the value normally found at $P_1 = 1$ atm abs, and there is essentially no increase with increasing depth. This can be understood in terms of the *in-vivo* regeneration of gas nuclei, which apparently occurs over periods of days or weeks and which is responsible for the loss of acclimation in divers and caisson workers during vacations.

8) The pressure reduction ratio for nitrogen-saturated tissues increases with increasing depth, while for helium-saturated tissues the ratio remains constant.

In response to this question, we first reiterate our view that pressure reduction ratios *per se* are not germane to the calculation of decompression tables and that the relevant parameter is the supersaturation pressure

$$p_{ss} = \Sigma\tau_i - p_{amb} \tag{17}$$

where $\Sigma\tau_i$ is the sum of the dissolved gas tensions and the vapor pressure of water and p_{amb} is the ambient pressure. The criterion for safe decompression is that the supersaturation remain always less than the critical value p^*_{ss} for which a clinically significant number of bubbles will form

$$p_{ss} \leq p^*_{ss} \tag{18}$$

Although it is of no fundamental importance, the pressure reduction ratio P_1/P_2 (or its reciprocal) can still be calculated, where P_1 is the absolute pressure at which the tissue has been saturated and P_2 is the absolute pressure to which it is just barely safe to decompress rapidly. In terms of P_1 and P_2, the decompression criterion can be written

$$P_1 - P_2 \leq p^*_{ss} \tag{19}$$

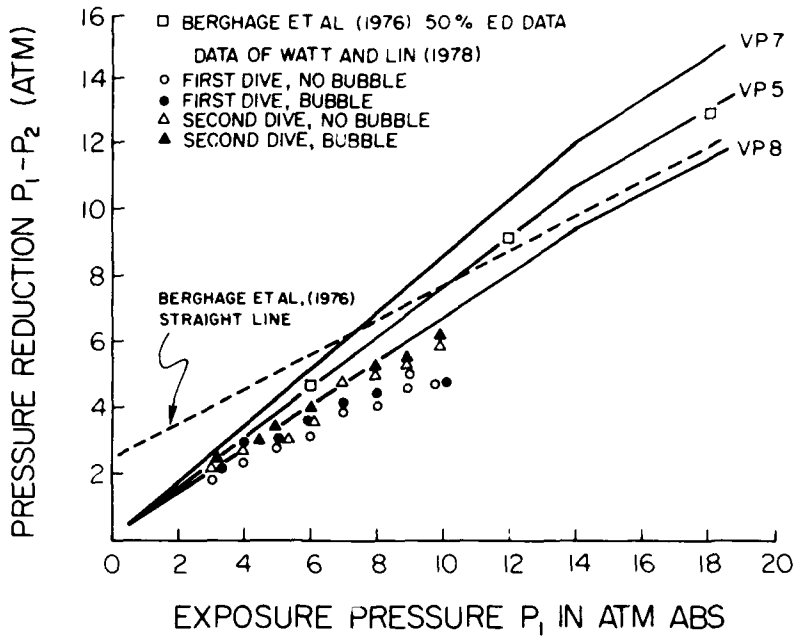

Fig. 10. Pressure reduction thresholds for bubble formation in rats. This comparison of effective dose data of Berghage et al. (1976) with Doppler bubble detection data of Watt and Lin (unpublished observations) suggests that lines of constant effective dose ED are also lines of constant bubble number N, as has been assumed in applying the varying-permeability nucleation model to decompression sickness in rats and humans.

In terms of the pressure reduction ratio P_1/P_2, the criterion is

$$P_1/P_2 \leq P_1/(P_1 - p^*_{ss}) \tag{20}$$

As noted in our responses to Assumptions 6 and 7, the ratio P_1/P_2 generally decreases with increasing depth. This is true for both helium and nitrogen, as can be seen in the data of Barnard (1976) in Table 1 and the data of Flynn and Spaur (1976) and Spaur et al. (1976) in Fig. 7. In the exceptional case of dives of very long duration, for which p^*_{ss} is ultimately independent of P_1, we see from Eq. 20 that the ratio P_1/P_2 decreases to a limiting value of 1 as P_1 becomes arbitrarily large.

9) Pressure differentials during decompression can be maintained indefinitely.

Ordinarily, the maximum safe supersaturation p^*_{ss} does not change during decompression, and the pressure differential

$$p_{ss} - p^*_{ss} \tag{21}$$

can be maintained indefinitely. During decompressions of exceptionally long duration, however, some regeneration of gas nuclei *in vivo* may occur. If it does, the safe supersaturation limit p^*_{ss} at the elevated pressure would gradually decrease, approaching its normal value at sea level, about 0.6 atm. (Eq. 13 gives p^*_{ss} = 0.645 atm for an exposure P_1 = 1 atm abs.)

C. GAS ELIMINATION

10) Gas uptake and elimination are symmetrical relationships. They are mirror images of each other.

This assumption, taken literally, is certainly wrong. It is wrong even if no bubbles have formed and only one tissue having the same time constant for gas uptake and elimination is involved. This can be seen, for example, in Fig. 2a and b. If several time constants contribute, the lack of symmetry is even more apparent, as indicated by the envelope $\tau(t) = \tau_1(t) + \tau_2(t)$ in Fig. 2c. To facilitate discussion, therefore, we shall restate the assumption:

The time constants for gas uptake and gas elimination are the same.

The revised assumption is probably valid in cases where no bubbles have formed. We caution, however, that the time constants for gas uptake and gas elimination are not the only ones that are relevant in calculating decompression tables. For example, the times required to crush and to regenerate gas nuclei may be important. The former are of the order of seconds or minutes, while the latter are probably measured in hours or days.

The important case in which bubbles have formed but in which signs or symptoms of decompression sickness have not appeared or are marginal can be investigated with the aid of Fig. 11, taken from an article by D'Aoust et al. (1977). The scenario, as we view it, begins as follows. The subject goats are first compressed to 198 fsw with a breathing mixture consisting mainly of nitrogen and about 0.3 atm abs of oxygen. At time $t = 0$, the inert gas is switched from nitrogen to helium. Because the uptake rate for helium is faster than the elimination rate for nitrogen, the total dissolved gas tension $\tau = \Sigma\tau_i$ begins to rise. After a few minutes, the inherent unsaturation (oxygen window) is exceeded by the rising tension, and the tissue is supersaturated by an amount

$$p_{ss} \equiv \Sigma\tau_i - p_{amb} \tag{22}$$

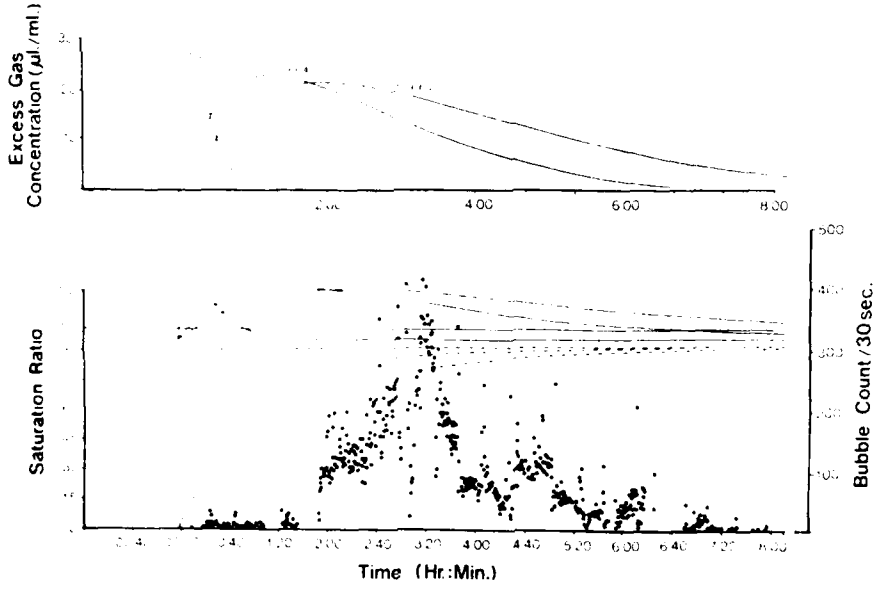

Fig. 11. Doppler bubble counts vs. time after inert gas switching from nitrogen to helium in goats at 7 ATA. This figure, taken from D'Aoust et al. (1977), is discussed in text to illustrate how nucleation theory can be applied under dynamic conditions to obtain information on gas uptake and elimination when bubbles have formed but no signs of decompression sickness have been observed.

Bubble formation in the tissue begins almost immediately after the dissolved gas tension exceeds p_{amb}. This indicates that there are some relatively large nuclei present, e.g., their radii exceed 1 micron. Occasionally, bubbles discharge or are discharged into the bloodstream. Whereas growth in tissue is limited to a few microns in radius by the tissue deformation pressure δ, those gas phases which appear in the blood are unstable and expand whenever the condition

$$p_{ss} \geq 2\gamma/r \tag{23}$$

is satisfied, where γ is the gas-liquid surface tension and r is the bubble radius. Frequently, bubbles in blood grow to a size, e.g., greater than 50 microns, that can be detected by the Doppler apparatus used in this experiment. For mild conditions, such as we have here, the lungs are effective in eliminating bubbles in the blood. Hence, the bubble number in the blood is continuously updated and accurately samples the bubble density existing at any given time in the tissue.

Bubbles continue to be produced in tissue only so long as the supersaturation pressure p_{ss} is rising. After about 3 hours, the bubble number and hence the supersaturation pressure reach their maximum values. At this point, the dissolved tension of nitrogen is decreasing at the same rate that the dissolved tension of helium is increasing. The maximum supersaturation is about 1.4 atm, corresponding to a critical radius or threshold radius for bubble formation of about 0.25 microns. By scaling the calculations of D'Aoust et al. (1977) shown in the same figure, we estimate that the time constant for nitrogen elimination in this experiment is on the order of 200 minutes, whereas that for helium uptake is about 90 minutes—rather longer than is indicated by any of the three curves in the figure or by standard measurements of gas uptake and elimination.

After three hours, therefore, all nuclei originally present in the tissue with radii larger than about 0.25 microns have developed into bubbles, and the remainder of the graph is a measure of the time required to eliminate them. The criterion for a bubble in tissue to diminish in size is that the supersaturation be less than the sum of the surface and tissue deformation pressures

$$p_{ss} < (2\gamma/r) + \delta \tag{24}$$

Evidently, Eqs. 23 and 24 can be satisfied simultaneously even if the surface tension has the same value in blood and tissue. It is plausible, then, that the Doppler apparatus continues to serve as a reliable monitor of the bubble number density in tissue during the period when bubbles are being eliminated or reduced to nuclei. This suggests that the time constant for bubble elimination in this experiment is essentially the same as that for nitrogen elimination. The data in Fig. 11 are consistent with this interpretation if one assumes that the time constants for nitrogen uptake and elimination are the same.

The similarity of the time constants for nitrogen uptake and elimination in this experiment is predictable if one assumes: 1) that the fraction of the gas released into bubbles is much less than 1% of that dissolved; 2) that the tissue deformation pressure is sufficient, e.g., greater than 1.3 atm abs, to maintain a pressure within bubbles comparable with the dissolved gas tension; and 3) that individual bubbles in tissue are small enough to discharge their contents rapidly when the conditions of Eq. 24 are satisfied. This would probably not be the case for severe decompression sickness. Finally, it should be noted that the time constants measured in this experiment refer to those tissues which account for the bubbles actually detected in the blood. They are not necessarily the same as the time constants for whole body uptake and elimination.

11) Oxygen breathing enhances gas elimination by increasing the pressure differential between the tissues and alveolar air.

The first part of this assumption is an experimental fact: oxygen breathing does enhance inert gas elimination. We have serious reservations, however, about the mechanism. Especially, we wonder whether the increase in the pressure differential obtained by oxygen breathing is sufficient to account for the beneficial results. Often the change in the dissolved gas tension $\tau = \Sigma\tau_i$ that is produced by breathing oxygen is modest in comparison either with the original value of τ or with the allowed supersaturation p^*_{ss}, yet it can reduce decompression times by a factor of two. Why is this technique so effective?

12) The concept of residual nitrogen time is sufficient to handle repeated exposures.

This assumption is wrong for several reasons already discussed in response to earlier assumptions. First, the dissolved gas tension $\Sigma\tau_i$ includes not only any inert gases that may be present but also O_2, CO_2, and water vapor. Second, the limiting supersaturation for safe decompression p^*_{ss} depends upon the status of the gas nuclei that initiate bubble formation in humans and hence upon the previous pressure history of the subject. Finally, the presence in the tissue of gross bubbles that have formed on previous exposures can affect the decompression outcome.

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COMMENTS

Richard D. Vann

GAS UPTAKE

There is a large body of literature on the exchange of materials between blood and tissue, from which it may be reasonably concluded that:

1) Blood flow is the primary factor controlling inert gas uptake, and

2) Blood flow is unevenly distributed to the tissues and organs of the body. Two examples from this literature are discussed below.

Häggendal, Nilsson and Norbach (1965) studied the washout of ^{85}Kr from dog brain (Fig. 1). After brief arterial injections of ^{85}Kr solutions, they found multi-exponential washouts that could be resolved by backward projection into two principal components. During subsequent experiments, they performed local injections of ^{85}Kr solutions directly into grey or white matter. The washout curves after these local injections were mono-exponential. The blood flow calculated from the mono-exponential washout of the grey matter was in good agreement ($r = 0.94$) with the blood flow calculated from the fast component of the multi-exponential washouts observed after arterial ^{85}Kr injection. The agreement between the slow-component blood flow and the flow calculated from the mono-exponential washout after local injection in white matter was less good ($r = 0.66$) because, as the authors demonstrate, the slowest component of a multi-exponential curve can be extracted less accurately than the faster components.

Linder (1966) conducted similar ^{85}Kr and ^{133}Xe washout experiments with dog hearts in which one coronary artery branch had been occluded to produce a myocardial zone that was perfused only by collateral flow (Fig. 2). Arterial infusion of ^{85}Kr and ^{133}Xe solutions into these hearts resulted in multi-exponential washout curves with two principal components. Local injections of tracer solutions into normally perfused (N) and ischemic myocardium (I) produced fast and slow mono-exponential washout curves. The blood flow calculated from the normally perfused tissue did not differ significantly ($P > 0.05$) from the blood flow calculated from the fast component of the arterial infusion washout curve. The same agreement existed between the slow component and the mono-exponential washout from the local injection into the ischemic region.

The washout of an ideally perfusion-limited tracer is characterized by a single exponential curve that appears as a straight line in a semi-logarithmic plot. Thus, the experiments of Häggendal et al. (1965) and of Linder (1966) demonstrate, within the limits of experimental error, that multi-component washout curves can be the sum of single component washouts from adjacent regions of tissue that are ideally perfusion limited.

These studies represent only a small part of a large body of literature that leads to the same conclusion. However, buried within the experimental error associated with the backward projection technique are local factors that have a secondary influence on inert gas exchange. These factors include:

1. Countercurrent diffusion shunt (Sejrsen and Tønnesen 1968; Duling and Berne 1970). This mechanism reduces the gas exchange rate by allowing materials dissolved in the blood to diffuse directly between adjacent arterial and venous vessels and so to bypass the capillary beds where the greatest part of gas exchange takes place.

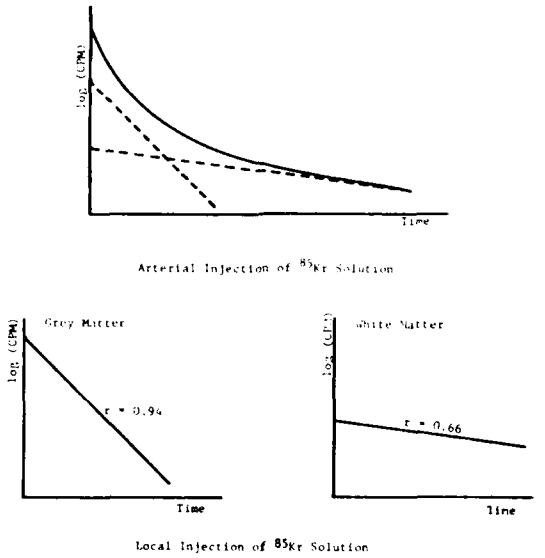

Fig. 1. Cerebral ^{85}Kr washout after arterial and local injections of ^{85}Kr solutions (adapted from Häggendal, Nilsson, and Norbäch 1965).

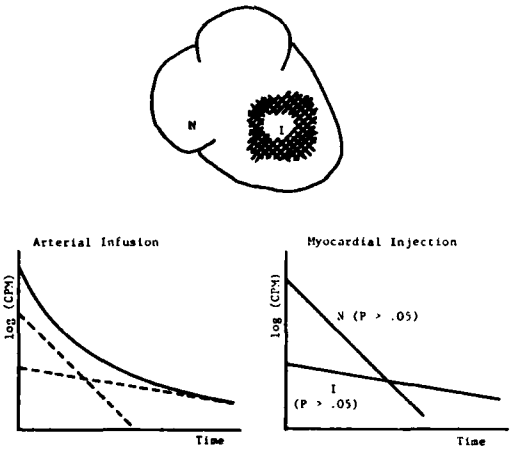

Fig. 2. Myocardial ^{85}Kr and ^{133}Xe washout after arterial and local ^{85}Kr and ^{133}Xe injections (adapted from Linder 1966).

2. Extravascular diffusion. For small inert molecules, diffusion is generally so rapid compared with perfusion that it exerts a negligible influence over blood-tissue exchange (Vann 1976). However, in certain normal avascular tissues, such as bone and articular cartilage, which possess unusually long diffusion distances, diffusion may partially limit the rate of inert gas exchange. This also may be the case in the presence of such pathology as edema or the scar tissue at the site of past injury.

3. Intercompartmental diffusion (Perl, Lesser, and Steele 1960; Rackow, Salanitre, Epstein, Wolf, and Perl 1965; Perl, Rackow, Salanitre, Wolf, and Epstein 1965; Vann 1976). Adjacent regions of tissue that are unevenly perfused or that have unequal inert gas solubilities, i.e., fast and slow compartments, will develop an inert gas tension difference during the course of an uptake or washout. This difference in tension is a driving force that will cause gas to diffuse between tissue compartments. If such compartments are important in decompression sickness, the results of a traditional isolated compartment analysis of decompression data would be misleading. Consider, for example, the case of a tendon surrounded by fat. The tendon itself might be the site of pain, while the fat acted as a reservoir of inert gas that would diffuse into the tendon even as blood flow removed gas from the tendon. Such a transport model also would have implications for the counter-diffusion (or counter-perfusion) of inert gases.

4. Variable blood flow distribution (Jones and Berne 1964; Folkow 1964 a,b; Johnson 1964; Berne 1964; Johnson and Henrich 1975; Haddy and Scott 1975; Vann 1976). The assumption that there is a constant distribution of blood flow to the various tissues of the body over a sustained period of time is questionable. Thus, the use of a series of constant half-time compartments, as in the Haldane model (Boycott, Damant, and Haldane 1908), is probably invalid. To some degree, the variation in blood flow is qualitatively, if not quantitatively, predictable, e.g., muscle perfusion during exercise. However, the less predictable myogenic or metabolic mechanisms (Jones and Berne 1964) may be important during critical phases of decompression when a diver is resting or cold.

These deviations from isolated, parallel, flow-limited compartments are of a local nature and are minor in comparison with the overall importance of blood flow. In fact, many of these deviations are mediated by blood flow. It is therefore concluded that multiple tissue compartments may be used to describe whole body inert gas uptake, with only minor error.

While total body gas uptake is well modelled by multiple tissue compartments, there is no obvious reason other than historical precedent why multiple compartments should apply in the case of pain-only decompression sickness. Indeed, Fig. 3 illustrates that the half time of the ascent-limiting Haldane tissue increases during decompression and indicates that a single tissue with a variable half time (and ratio or M value) is a plausible alternative to multiple tissues with constant half times. It is therefore proposed that a single perfusion-limited tissue is responsible for pain-only decompression sickness. It cannot be said with certainty that deviations from perfusion limitation are not applicable in decompression sickness. However, these deviations can be complex to model mathematically, and it is believed wisest to explore first the applicability of the simpler case of perfusion limitation.

A test of the single-tissue hypothesis would be provided by a set of no-decompression exposure limits determined at a constant level of exercise. Under these circumstances, it would be expected that a single-tissue half time would predict that same surfacing ratio (M value or tissue tension) for each exposure limit. Such a test may perhaps be found in the work of Eaton and Hempleman (1962), who report the no-decompression exposure limits for goats of 50th percentile susceptibility (Fig. 4). If it is assumed that the critical tissue in these animals has a half time of 29 minutes, the surfacing ratio is found to fall in a narrow range between 2.1 and 2.3 (equivalent to tissue tensions of 69.3 to 75.9 fsw). A three-tissue analysis (half times = 10, 20, and 40 minutes) of this data similar to that applied by Eaton and Hempleman gives surfacing ratios of between 2.1 and 3.6. Though this is hardly conclusive evidence, it is nevertheless consistent with the single-tissue hypothesis.

Figure 5 is a single-tissue analysis of the human no-decompression exposure limits for air determined by van der Aue, Kellar, Brinton, Barron, Gilliam, and Jones (1951) and Albano (1970). If a safe surfacing depth of 29 fsw after an infinitely long exposure is assumed (surfacing ratio = 1.484), then the half time associated with each exposure limit may be calculated. Thus, for a range of bottom times varying from

about 10 to 40 minutes, the van der Aue half times vary from about 33 to 62 minutes, while the Albano half times range from 47 to 66 minutes. This analysis suggests that for both data sets, the divers worked hardest during the shortest dives but van der Aue's divers worked harder than Albano's divers.

Very few studies have explored the relationship between work and decompression. Perhaps the best is that of Schibli and Buehlmann (1972), who investigated the effect of work on the bottom upon subsequent decompression time. For three helium/oxygen dives using oxygen during decompression, they found an average decompression time of 292 minutes when the divers rested on the bottom. When the divers worked, however, the average decompression time increased by 28% to 368 minutes. During the resting dives, the average heart rate was 60 bpm, while during the working dives it increased to 100 bpm for 15 minutes of each hour. If it is estimated that the divers' average oxygen consumption was 0.5 liter/min at rest and 0.75 liter/min during work, the decompression time may be roughly estimated for higher oxygen consumptions by linear extrapolation. At a consumption of 2.0 liter/min, it is estimated that the average decompression time will be 876 minutes, or an increase of 200%. However, since gas uptake is not linear, it is probably more reasonable to assume an increase of only 100% over rest.

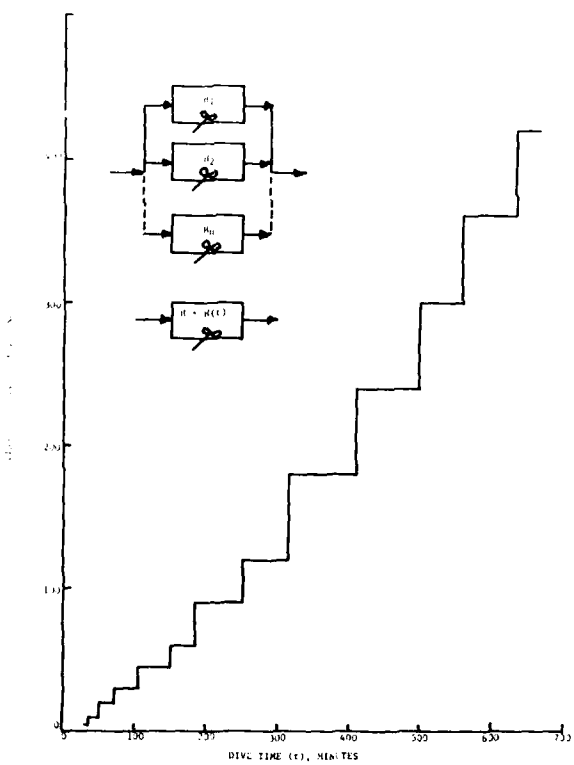

Fig. 3. Tissue half time as a function of total dive time, based upon a Haldane analysis of a 500 fsw/30 min helium/oxygen dive.

PRESSURE REDUCTION

It is generally agreed that pressure reduction leads to bubble formation. However, the magnitude of the pressure reduction necessary to produce bubble formation and the mechanism of bubble formation are questions of some uncertainty. Two principal hypotheses of bubble formation have been offered:

1. Critical supersaturation (or random nucleation)
2. Pre-existing gaseous micronuclei

Boycott et al. (1908) were the earliest proponents of the first hypothesis, still used in modified form today. More recently, however, the experiments of Evans and Walder (1969) with shrimp suggested that the second hypothesis might be more appropriate. In these experiments, three groups of 50 shrimp were

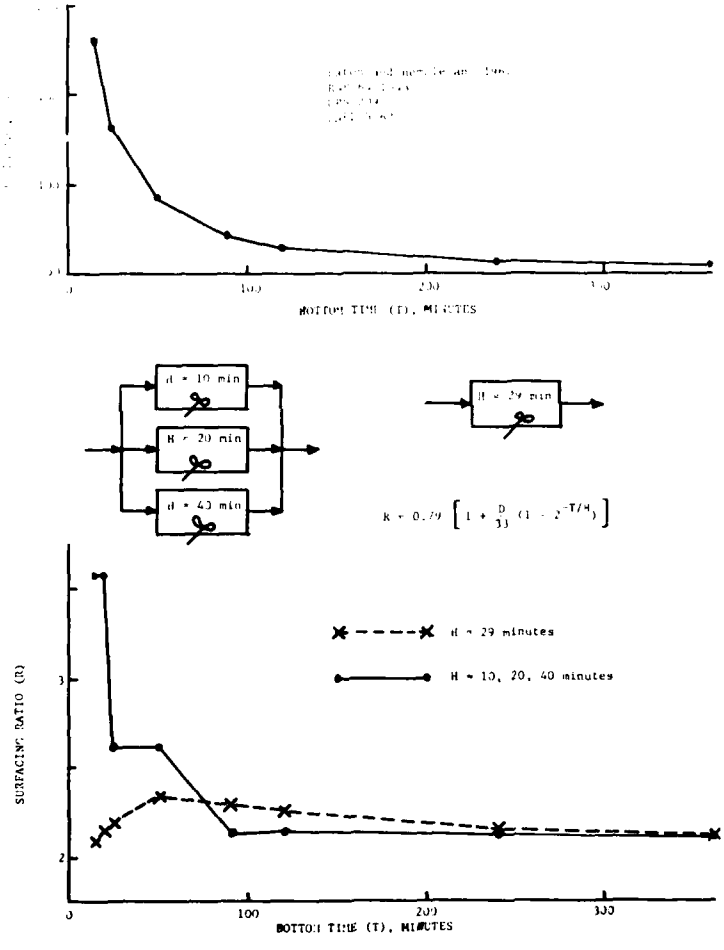

Fig. 4. No-decompression exposure limits (air) for goats of 50th percentile susceptibility (from Eaton and Hempleman 1962). Lower half of figure illustrates how the surfacing ratio varies with exposure limit for a single-tissue and three-tissue model.

exposed to altitude and observed for the appearance of bubbles within their transparent shells. Bubbles were seen in 48 of the first group. The second group made a rapid compression to more than 400 ATA prior to altitude exposure, and bubbles were observed in only four of the 50 shrimp. The third group also made a rapid compression but was stimulated electrically to produce movement prior to altitude decompression. In this group, bubbles were observed in 16 shrimp. These experiments are consistent with the concept of gaseous micronuclei.

To test this concept further in animals that are closer to men on the evolutionary scale, air-breathing rats were exposed on the three dive profiles illustrated in Fig. 6 (Vann, Grimstad, Neilsen, and Carey 1978). The final part of all three profiles consisted of a two-hour exposure at 240 fsw. On the second and third profiles, the time at 240 fsw was preceded by rapid excursions to 600 and 1000 fsw, respectively. The numbers of rats exposed on the profiles were 200, 195, and 153. Figure 7 shows how the bends incidence for each profile (expressed as the number of animals tumbling in a rotating cage divided by the total number

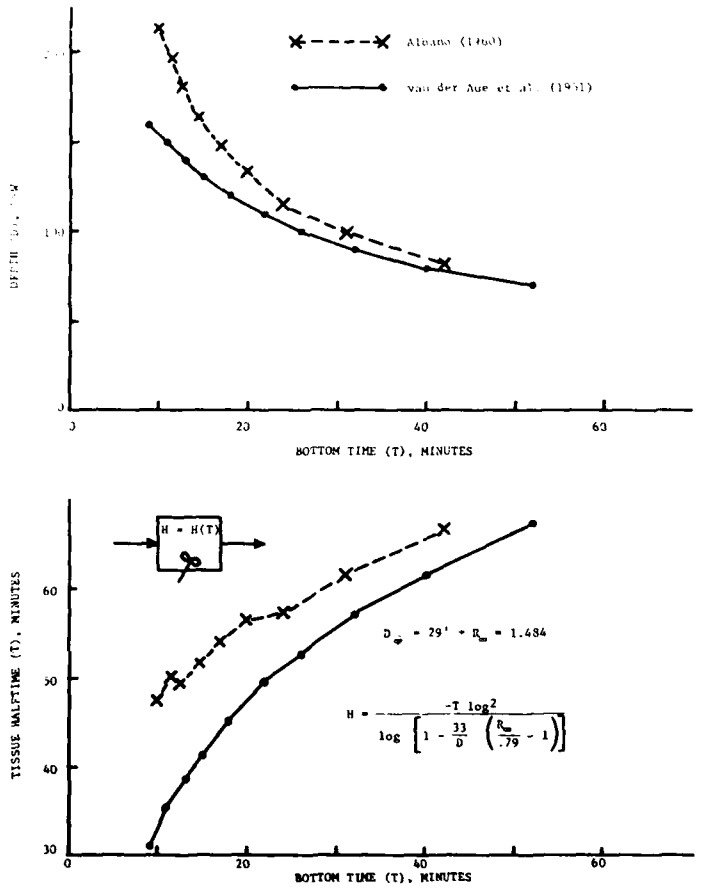

Fig. 5. No-decompression exposure limits (air) for humans determined by van der Aue et al. (1951) and Albano (1970). Lower half of figure illustrates how a single-tissue half time varies with exposure limit.

exposed) undergoes large fluctuations until over 100 animals have been exposed. The final incidences were 83% for the no-excursion profile, 74% for the 600-fsw excursion profile, and 64% for the 1000-fsw excursion profile. After making corrections for weight and time of day of exposure, the differences between the no-excursion and 600 and 1000 fsw excursion profiles were found to be significant at $P_{600} \leqslant 0.025$ and $P_{1000} \leqslant 0.001$. These results are consistent with the concept of gaseous micronuclei and inconsistent with the concepts of critical supersaturation or random nucleation.

Further evidence for the existence of gas nuclei was provided by Unsworth, Dowson, and Wright (1971), who demonstrated cavitation bubbles in cracking knuckle joints at normal atmospheric pressure. It is unquestionable that such bubbles will grow in size if they are present in supersaturated tissue during decompression. Could these bubbles be responsible for the various symptoms of decompression sickness? The earliest reference to bubbles in decompression sickness appears to be that of Boyle in 1672. Most subsequent work in the calculation of decompression schedules has followed the Haldane concept of critical

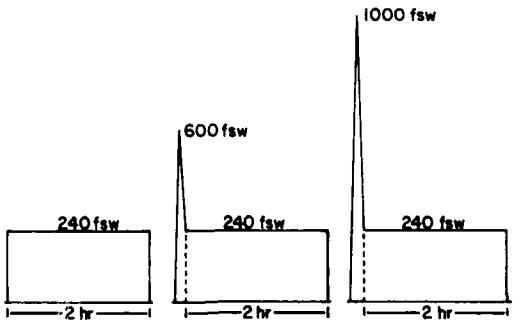

Fig. 6. Dive profiles on which air breathing rats were exposed to test for evidence of gaseous micronuclei.

Fig. 7. Results of tests of Fig. 6; bends incidence (% of animals tumbling) is illustrated as a function of number of animals exposed.

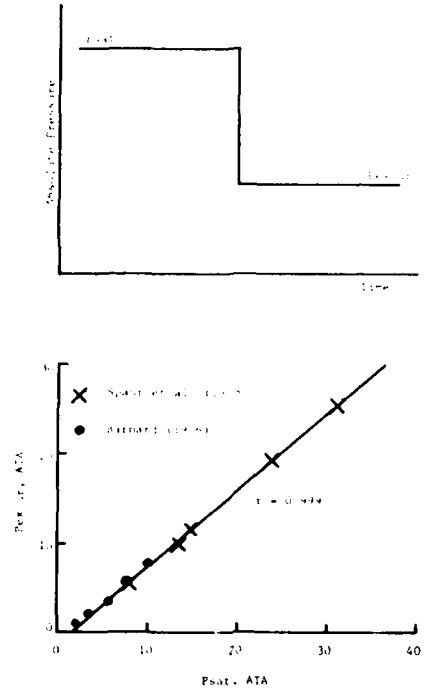

Fig. 8. Saturation excursion profile in which a diver saturated at a pressure P_{sat} makes maximum safe upward excursion to pressure P_{excur}. Lower half of figure plots helium-oxygen saturation excursion data of Barnard (1976) and Spaur et al. (1978).

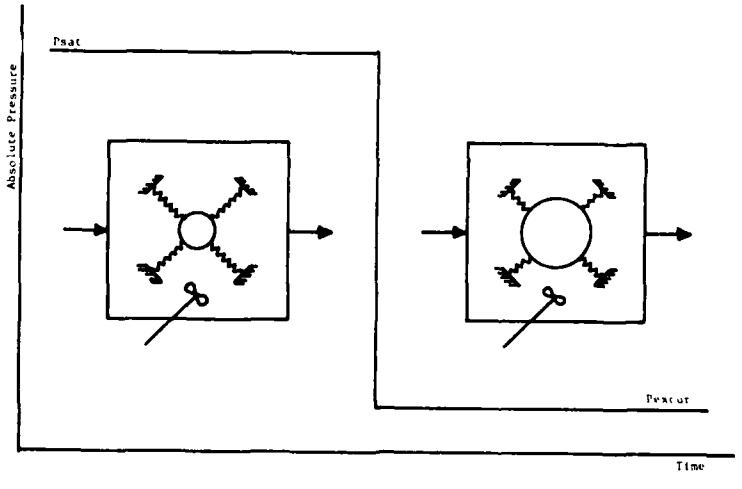

Fig. 9. Response of proposed decompression model to an upward excursion from a saturation exposure is illustrated. A micronucleus in a perfusion-limited tissue is stabilized by the elastic properties of tissue (represented by springs). Micronucleus increases in volume as a result of change in pressure.

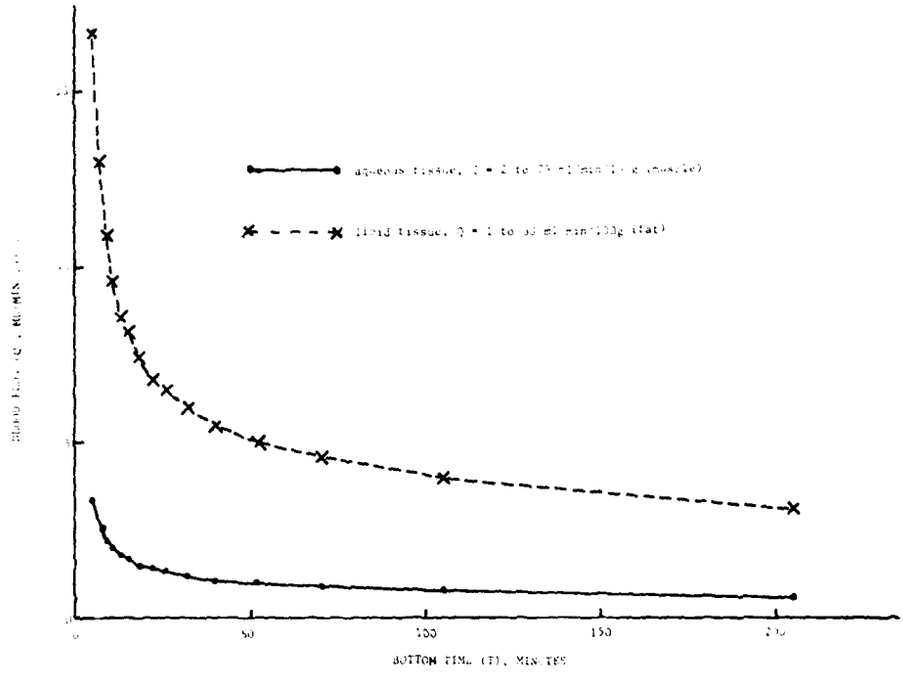

Fig. 10. Relationships between critical tissue blood flow and no-decompression exposure limits of van der Aue et al. (1951) are shown for both aqueous and lipid tissues.

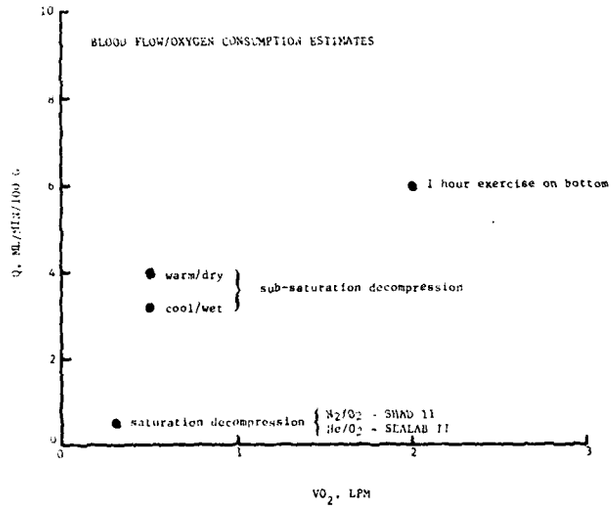

Fig. 11. Estimates of relationship between critical tissue blood flow and diver oxygen consumption.

supersaturation, although Piccard (1941) and Behnke (1951) suggested that the critical parameter might be gas volume. Nims (1951) was the first investigator to give the bubble theory a quantitative form, followed by Hills (1966), who developed it further. More recently, Hennessy and Hempleman (1977) applied bubble theory to the helium/oxygen excursion data of Barnard (1976), which appear to describe a straight line (Fig. 8). By assuming that all supersaturated gas leaves solution and forms a bubble, they were able to demonstrate theoretically that there is indeed a linear relationship between saturation (P_{sat}) and excursion pressure (P_{excur}).

Spaur, Thalmann, Flynn, Zumrick, Reedy, and Ringelberg (1978) have extended Barnard's work with a slightly higher oxygen partial pressure (P_{O_2} = 0.4 atm instead of 0.22 atm). Figure 8 shows that both sets of data appear to fall very near the same straight line. However, this may be misleading because the excursions chosen for test by Spaur et al. were apparently guided by Barnard's work. An analysis of the combined data has been conducted based upon the assumption of a tissue volume containing a pre-existing micronucleus (Fig. 9). This analysis is similar to that of Hennessy and Hempleman (1977), with two exceptions: 1) a gas nucleus is considered to be present in the tissue prior to decompression; 2) the tissue is assumed to have the elastic properties described by Vann and Clark (1975). Tissue elasticity or some other mechanism is necessary to stabilize the nucleus against surface tension and the inherent unsaturation (Hills 1966) or partial pressure vacancy (Behnke 1967) of tissue.*

The analysis of the helium/oxygen excursion data and of similar (but less extensive) nitrogen/oxygen data leads to basic constants for the model. This analysis suggests that the critical tissue is predominantly lipid, because both the inert gas solubility and the shear modulus of the tissue are close to those properties for olive oil or fat. The lipid tissue hypothesis also is supported by a blood flow analysis of the no-decompression exposure limits of van der Aue et al. (1951). The tissue half time corresponding to each of the limits may be calculated as shown in Fig. 5. This half time can be converted into blood flow by using the relation

$$Q, \text{ml/min/100 g} = \frac{100 \ln 2}{(\alpha_b/\alpha_t)\ \text{half time}}$$

where α_b and α_t are the blood and tissue nitrogen solubilities. Choosing 0.67 atm^{-1} for lipid tissue solubility and 0.137 atm^{-1} for both aqueous tissue and blood solubilities, the relationships between blood flow and exposure limit can be constructed for lipid and aqueous tissues, and are shown in Fig. 10. For aqueous tissue, the blood flow ranges from a maximum of 3.4 ml/min/100 g to a minimum of 0.6 ml/min/100 g. For lipid tissue, the flow ranges from 16.6 to 3.1 ml/min/100 g. From Folkow and Neil (1971) and Hansen and Madsen (1976), a blood flow range for muscle can be found to be 2 to 75 ml/min/100 g and for fat to be 1 to 30 ml/min/100 g. A comparison of these ranges with those derived from the data of van der Aue et al. suggests that the critical tissue is more likely to be lipid than aqueous.

In summary, a decompression model has been developed that has the following properties:

1. There is a single critical tissue, which is predominantly lipid.
2. Gas exchange is perfusion limited as long as the gas remains dissolved.
3. Inert gas is conserved, i.e., a mass balance is maintained for gas entering and leaving the tissue or gas nucleus.

*Though the tissue elasticity concept is adequate for this purpose, it is by no means certain that it is the mechanism by which nuclei are stabilized. An alternative mechanism is offered by Yount, Kunkle, D'Arrigo, Ingle, Yeung, and Beckman (1977), but the question of stabilization remains one of the most important unresolved issues in decompression theory.

4. Bubbles grow from pre-existing micronuclei that are stabilized by tissue elasticity.
5. The depth of the first decompression stop is controlled by blood flow on the bottom.
6. The length of a decompression stop is determined by inspired oxygen partial pressure and blood flow during decompression.

GAS ELIMINATION

Inert gas is carried by the blood to the lungs in solution or as bubbles. It is assumed here that in the critical tissue for pain-only bends, any gas that comes out of solution will be trapped within the tissue and must re-dissolve before it can leave. The driving force for removing inert gas from tissue is the inherent unsaturation (partial pressure vacancy) plus the "inflation" pressure in the gas nucleus caused by tissue elasticity (Vann and Clark 1975). The rate at which dissolved gas is removed from the tissue depends upon the inspired oxygen partial pressure (PI_{O_2}) and the blood flow. The PI_{O_2} is easily measured, but the blood flow during decompression is a more difficult parameter to determine. It is affected by both immersion and temperature and appears to decrease as the diver cools. This is consistent with the findings of Balldin (1973).

An effort is being made to relate blood flow to oxygen consumption, which is an observable measure of exercise. Figure 11 illustrates some of the preliminary estimates that have been made. These estimates are based upon analysis of data in the literature and of dives conducted at Duke University, under the sponsorship of the Office of Naval Research and the Naval Medical Research and Development Command. The dives are conducted with the Mk 15 Underwater Breathing Apparatus (Biomarine CCR-1000), which maintains 0.7 atm PI_{O_2} in an N_2/O_2 mixture at any depth. Thus the inspired oxygen percentages at the 30, 20, and 10-fsw decompression stops are 37, 44, and 54%. Figure 12 illustrates the seven decompression profiles that have been tested at Duke, plus the Equivalent Air Depth (EAD) profile (not tested), and a profile tested at the Navy Experimental Diving Unit (NEDU). The long decompression times necessary at an oxygen consumption of 2 liters/min are in general agreement with the increase in decompression time found with the extrapolation of the data of Schibli and Buehlmann (1972) from rest to a consumption of 2 liters/min. It is also of interest to compare Profiles #4 and #5, which are identical except that in #4 the diver is removed from the water at 30 fsw and is warm and dry for the remainder of the decompression. Profile #4 produced no bends in 12 dives, while Profile #5 produced one Type I bend in 5 dives. During these 5 dives, the divers' oral temperatures were noted to have dropped about 1° to 2°C as a result of immersion. It is surmised that these moderately hypothermic divers sustained a reduction in blood flow that retarded gas elimination during decompression.

After the dives shown in Fig. 12, the divers were examined for intravascular bubbles with precordial Doppler instrumentation and by a phased array ultrasonic scanner developed at Duke that produces a cross-sectional view of the heart or major vessels. The Doppler recordings were made with the diver lying on his back and sequentially exercising each limb. The audio signals were scored according to the subjective grading system of Spencer (1976). The post-dive bubble scores of two divers on 5 dives that produced high bubble scores (Profiles #4 and #5) are shown in Fig. 13 . The bubbles generally did not appear until after the diver had showered. They reached a maximum score at 1 to 2 hr post dive, and could still be detected in lesser quantity at 4 hr or more post dive. This is consistent with the observations of Pilmanis (1976). As Fig. 13 illustrates, one diver bubbled equally in both legs, while the other showed more bubbles in the left leg. Similar findings have been reported by Spencer (1976). The divers of Fig. 13 were totally symptomless, as were many other divers in whom bubbles were detected. However, bubbles were detected in all divers who developed Type I bends symptoms (no Type II symptoms occurred).

The two-dimensional ultrasonic scanner permits real-time imaging of bubbles and appears to be somewhat more sensitive in the detection of bubbles in the heart or inferior vena cava. Moreover, in one symptomless diver, a few bubbles were observed in the left side of the heart. Subsequent tests ruled out the possibility of atrial or ventricular shunting, and it was concluded that the bubbles had reached the left heart by passing through the lungs.

In Fig. 14, it is postulated that critical tissue for Type I bends is in parallel with the tissue or tissues responsible for intravascular gas bubbles. This tissue probably includes the muscles that can take up large volumes of gas during exercise on the bottom (bubbles are not detected in resting divers on these profiles) and then release some of this gas into the venous blood after decompression. The lungs can apparently filter limited quantities of these bubbles, but their filtering capacity can be overloaded, at which time some bubbles cross the lungs and enter the systemic circulation. While the systemic circulation may be able to absorb some of these bubbles without the development of (overt) symptoms, an over-abundance or a critically located arterial bubble may well cause Type II decompression sickness symptoms.

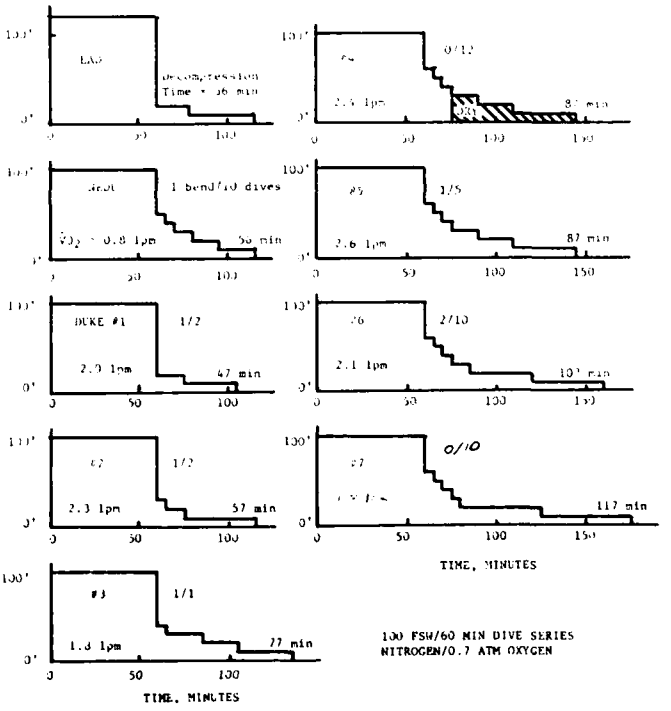

Fig. 12. Decompression profiles tested at Duke University.

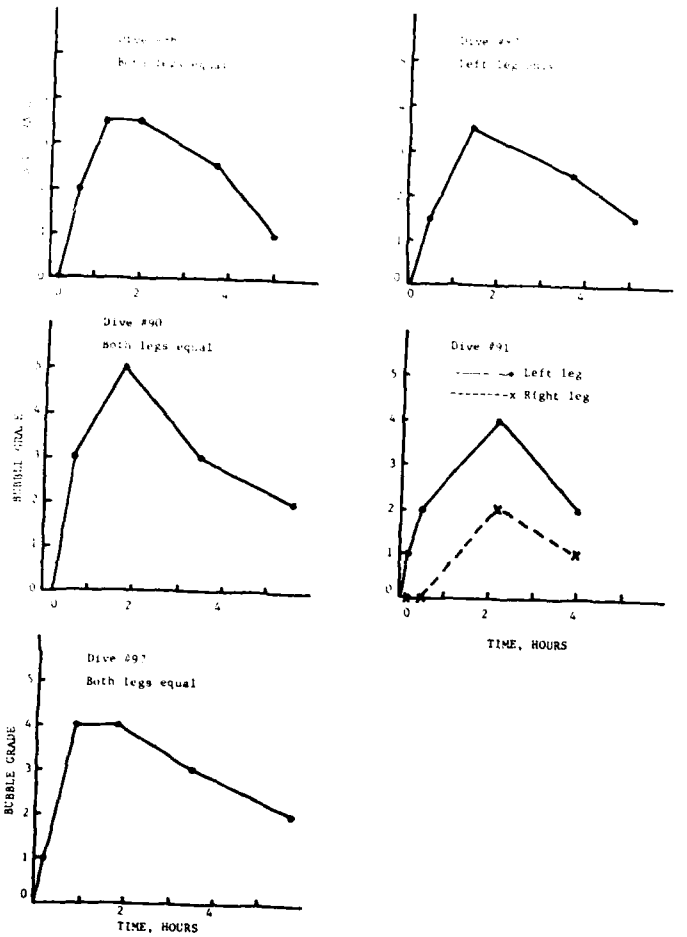

Fig. 13. Post-dive Doppler signals occurring in two divers after Profiles #4 and #5 of Fig. 12. Both divers were symptomless.

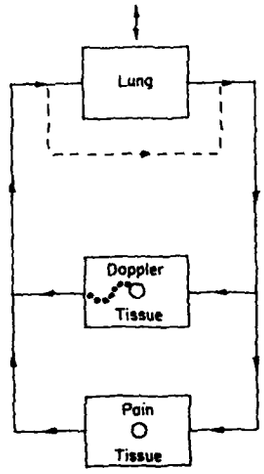

Fig. 14. Postulated relationship between tissues responsible for decompression pain and for intravascular bubbles.

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SYMMETRY OF INERT GAS UPTAKE AND ELIMINATION

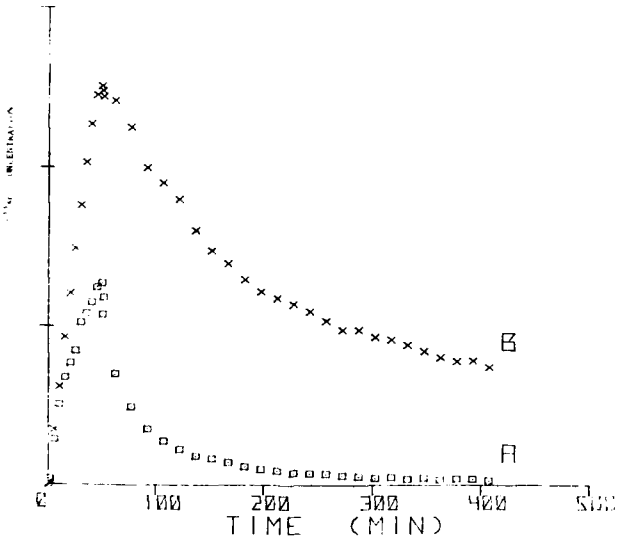
P. K. Weathersby

I will restrict my remarks to a quick presentation of data on one of the points in question, the symmetry of inert gas uptake and elimination.

The experiments we have to report are limited in scope: they were performed with a very dilute gas (radioactive ^{133}xenon), at a constant pressure of 1 atm and on a single type of animal preparation (anesthetized dogs). They included 9 different runs using 6 different animals. The total time of gas measurement was 6 to 7 hours. The radioactive gas was breathed for periods of 10 min to 50 min, followed by room air breathing for a subsequent 6 hours. During both uptake and elimination, the gas concentration in roughly 1/3 of the dogs was measured with a gamma camera with a computer that stored the data according to 4000 different areas (sites) in the dog.

The study produced approximately 8,000,000 gas measurements, which are still being analyzed. At this point, several hundred curves of the type shown in Fig. 1 have been analyzed. The two curves shown are from a single run in which gas in the shoulder region (upper curve) and brain (lower curve) rises to a peak at the end of the uptake phase and decreases over the next six hours. For clarity, only every fifth data point is shown.

The data have been analyzed by a number of procedures. Among them are series of exponential functions fitted to the combined uptake and elimination data. Sample results are shown in Fig. 2. Curve A of the first slide has been fitted with 1, 2, 3, or 4 exponential terms. It is clear that two or more exponential terms are excellent approximations to the data. The same conclusion is reached after a similar analysis of many other data sets.



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This work demonstrates that under very "gentle" conditions (dilute isobaric exchange in an anesthetized animal) inert gas uptake and elimination can be characterized by the same response function.

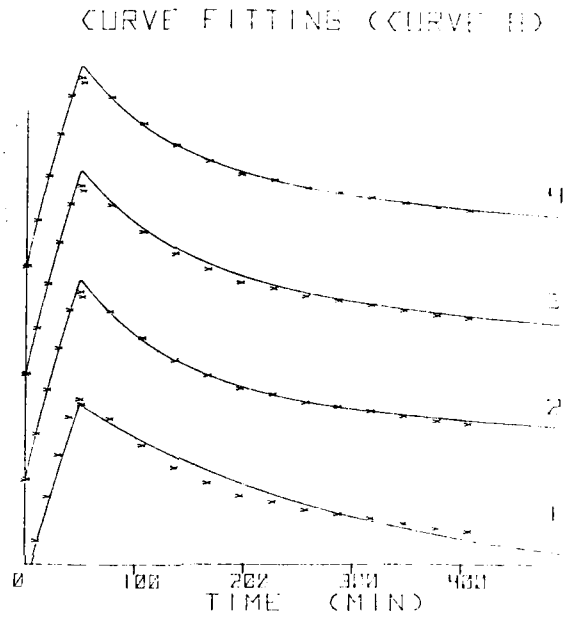

Fig. 2.

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Decompression - Theory - Tissue: compartments, saturation, uptake - Inert gas: uptake, elimination, partial pressure - Oxygen - Nitrogen: saturation, residual, elimination - Helium: saturation, residual, elimination.

20. ABSTRACT (Continue on reverse side if necessary and identify by block number)

Multiple tissues or tissue compartments must be used to describe whole-body gas exchange.
Tissue gas uptake is described by the following: exposure time, pressure differential, and tissue half time.
Only the inert gas partial pressures need be considered in the decompression problem.
Breathing multiple inert gases appears to provide some decompression advantages.

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Compression procedures have no effect on decompression.
Tissue supersaturation limits are best described by a series of pressure-reduction ratios.
The pressure-reduction ration for nitrogen-saturated tissues increases with increasing depth; the ratio remains constant for helium-saturated tissues.
Tissue supersaturation limits increase with increasing depth.
Pressure differentials during decompression can be maintained indedinitely.
Gas uptake and elimination are symmetrical relationships.
Oxygen breathing enhances gas elimination by increasing the pressure differential between the tissues and alveolar air.
The concept of residual nitrogen time is sufficient to nandle repeated exposures.
The first section of the report presents a brief history of the development of the U.S. Navy's version of the Haldane model, along with a description of some of the diving environments in which the model has not worked. The second section deals with the 12 neo-Hardanian assumptions and presents the best current thinking with regard to their variability; it also serves to meet the objectives of the Workshop. The third section of the report contains the individual manuscripts presented by the Workshop attendees. This section provides amplifying remarks that can be used for interpretation of the comments made in the second section.


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