THE PHYSIOLOGY OF NITROX DIVING

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Dr. Vann works at the F.G. Hall Laboratory at Duke University. His experience in both modelling and evaluating decompression tables blends well with his knowledge of diving physiology to produce an overview of the role of physiological principles and experience as they relate to nitrox diving.

ABSTRACT

Vann RD. 1989. The physiology of nitrox diving. In: Proceedings: Harbor Branch Workshop on enriched air nitrox diving. Hamilton RW, Crosson DJ, Hulbert AW, eds. Technical Report 89-1. Rockville, MD: NOAA Office of Undersea Research.

Both efficiency and reliability of decompression can be improved by judicious use of oxygen. One aspect of this is the EAD, or equivalent air depth, a principle which states that only the nitrogen partial pressure need be considered in non-air dives with nitrogen-oxygen mixtures. Limited support for this concept comes from several studies, and none give cause for regarding it as invalid. The choice of decompression tables for EAD diving calls the reliability of available tables into question, but tables reliable for air diving should be good for use with the EAD. Decompression by computer offers an attractive option, but to date none is available for EAD diving.

INTRODUCTION

The most efficient means of improving decompression is by the use of oxygen. Diluting the nitrogen in air by adding oxygen provides a decompression advantage since oxygen metabolized in tissue does not contribute to the bubbles that cause decompression sickness. This is the basis of the Equivalent Air Depth (EAD) theory which assumes that oxygen is totally metabolized and only nitrogen is important in decompression calculations.

THE FATE OF OXYGEN

The EAD is the depth of an imaginary air dive that would have the same nitrogen partial pressure as an actual dive on oxygen-enriched air. The EAD is given by

$$EAD = \frac{(1-F_IO_2)(D + 33\ fsw)}{0.79} - 33\ fsw$$

where D is the actual depth and F_IO_2 is the inspired oxygen fraction. The EAD theory allows existing decompression tables to be used with oxygen-enriched air. If the actual depth is 130 fsw and the F_IO_2 is 0.32, for example, the EAD will be 107 fsw and air decompression schedules for 110 fsw should be used.

How valid is the assumption that oxygen does not contribute to decompression sickness? Metabolism converts oxygen, a relatively insoluble gas, into carbon dioxide which is some 21 times more soluble. The effect of exchanging oxygen for carbon dioxide on dissolved gas tension is illustrated in Figure 1. The x-axis is dissolved gas tension in torr, and the y-axis is dissolved gas content in ml gas per 1000 ml blood. The steeper line shows the relationship between CO_2 tension and content. The slope of this line is the CO_2 solubility. The other line represents the same relationship for oxygen. Its gradual slope reflects the lower oxygen solubility.

Suppose, as indicated by Point 1 in Fig. 1, the oxygen tension were 100 torr. This corresponds to an oxygen content of 3 ml of oxygen per 1000 ml of blood (Point 2). If each O_2 molecule were exchanged for a CO_2 molecule (Point 3), the dissolved gas volume would be unchanged, but the tension would fall to 4.7 torr (Point 4). This is because CO_2 is more soluble than oxygen.

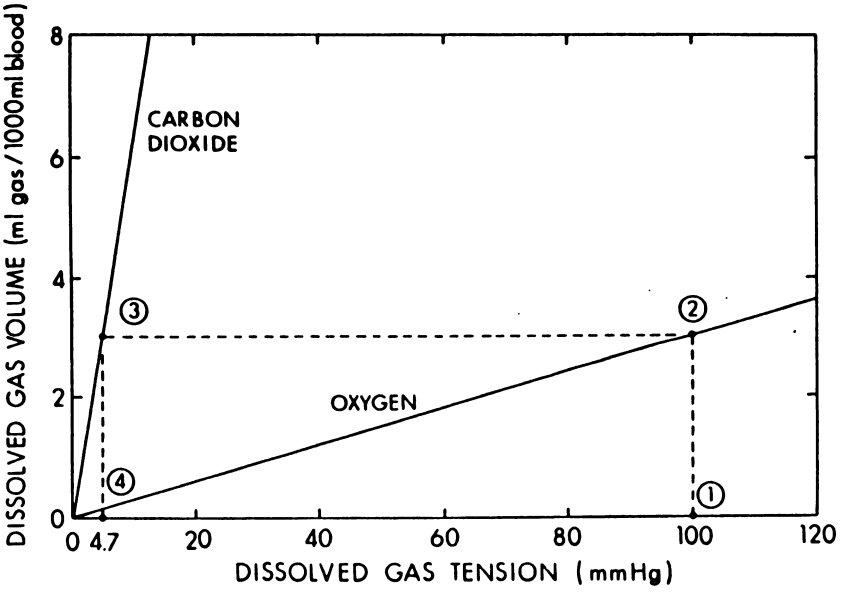

Figure 1. The effects of exchanging oxygen for carbon dioxide on dissolved gas tension. When oxygen is converted into carbon dioxide, the gas tension falls from 100 to 4.7 torr. but the dissolved gas volume remains unchanged because carbon dioxide is more soluble than oxygen (Vann, 1988).

Most of the oxygen carried by blood, however, is chemically bound to hemoglobin, and under normal conditions, only a small fraction is dissolved. Figure 2 shows the total oxygen content of blood in ml oxygen per 100 ml blood, a unit known as volume %, as a function of the oxygen tension. Point A1 is the arterial blood of a diver breathing air at sea level. Hemoglobin is nearly saturated with oxygen under these conditions. As the arterial blood passes through tissue, 5 vol% of oxygen are removed and converted to CO_2. This causes the venous oxygen tension (indicated by Point V1) to fall to 46 torr.

Now consider a diver breathing air at 3.5 ATA. His alveolar oxygen partial pressure is 504 torr, but his arterial tension is only about 450 torr as a result of ventilation-perfusion inequalities in his lungs. This is shown as Point A2 in Fig. 2. When 5 vol% of oxygen are extracted by tissue, the venous tension (point V2) falls to 53 torr.

Now the diver switches to 100% oxygen at 3.5 ATA and his alveolar partial pressure rises to 2,570 torr. Ventilation-perfusion inequalities reduce the oxygen tension in his arterial blood to around 2,000 torr (Lambertsen, et al, 1953). This is shown as Point A3 in Fig. 2. Here, however, the venous tension (Point V3) rises to 380 torr, far above the previous venous values. This unusually high venous tension occurs because the metabolic requirements of tissue are met entirely by dissolved oxygen. Venous hemoglobin remains saturated and on the flat rather than on the steep part of the oxygen content curve.

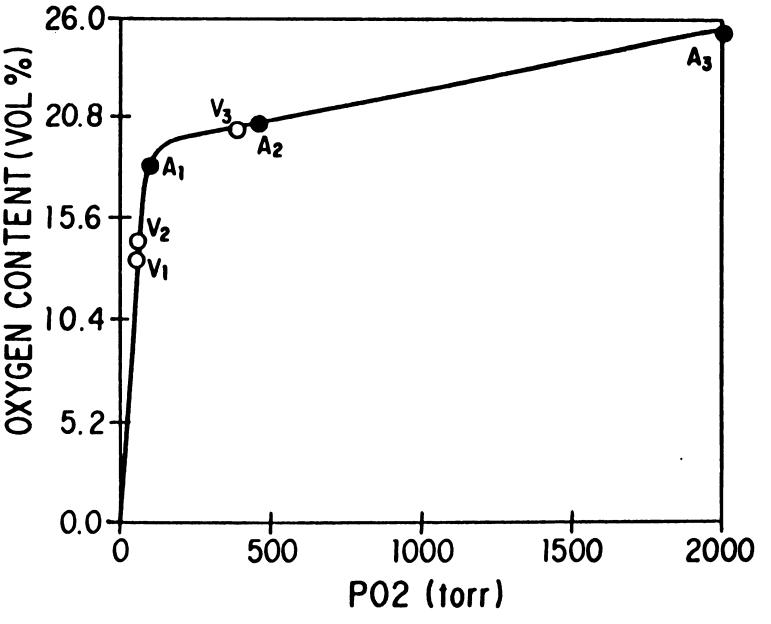

Figure 2. The total blood oxygen content in vol % (ml 02 /100 ml blood) as a function of blood oxygen tension. Total content is the physically dissolved oxygen plus the oxygen chemically bound to hemoglobin. The points marked A1, A2, A3 and V1, V2, V3 are approximate arterial and venous oxygen tensions during air breathing at sea level, during air breathing at 3.5 ATM, and during oxygen breathing at 3.5 ATM. The oxygen extraction from blood is taken as 5 vol %.

Tissue oxygen extraction is a function of both metabolism and blood flow. The oxygen extraction was 5 vol% in Fig. 2, but this is not true throughout the body. Table 1 lists a range of oxygen extractions for various tissues and organs and indicates that the extraction can be as high as 10 and low as 1.3 vol%.

Table 1. Oxygen extraction (ml/100 ml blood) in various tissues and organs (Folkow and Neil, 1971).

Heart	10
Brain	6
G.I. tract	6
Resting muscle	5
Kidney	1.3
Remainder	5

The effect of oxygen extraction on venous tension is shown in Fig. 3 as a function of arterial tension. The lowest curve, which represents an extraction of 6 vol%, indicates that the venous tension rises gradually at arterial tensions of up to 2,000 torr. At extractions of 5 and lower, however, the venous tension increases precipitously. At the lowest extractions, the venous oxygen tension can contribute more than an atmosphere to the dissolved gas tension. When added to nitrogen already dissolved in tissue, it is easy to imagine that oxygen might potentiate bubble formation and lead to a condition which Donald (1955) called "oxygen

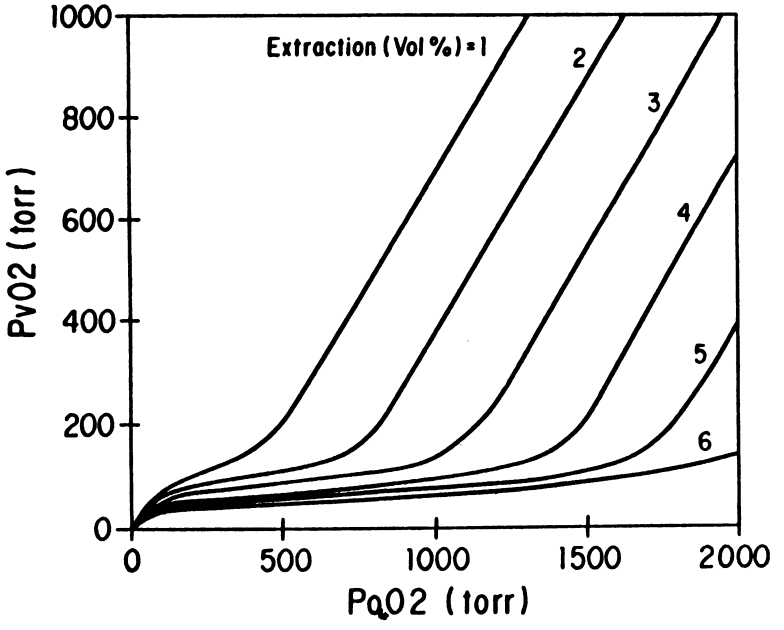

Figure 3. The effect of oxygen extraction on venous oxygen tension (P_vO_2) as a function of arterial oxygen tension (P_aO_2). At higher oxygen extractions, P_vO_2 remains relatively constant as P_aO_2 rises. In tissues with lower extraction, P_vO_2 rises steeply at high P_aO_2. This increase begins sooner at lower extractions. Since elevated $P_vO_2$contributes to bubble formation, the error in the EAD theory becomes greater as P_vO_2 rises.

bends." The EAD assumption that oxygen is totally metabolized, while only slightly in error at low inspired partial pressure, fails badly at high partial pressure.

"OXYGEN BENDS"

What insight concerning oxygen bends does experiment provide? Berghage and McCracken (1979a,b) determined the pressure reduction which produced a 50% DCS incidence (or ED50) in rats exposed on helium-oxygen to various pressures, times, and P_IO_2's. If there were no error in the EAD theory, an increase in P_IO_2 would lead to an equal increase in pressure reduction, or

$$[(P_IO_2)2 - (P_IO_2)1] = [\text{ delta P2 - delta P1}]$$

If these quantities are not equal, however, their difference represents an error in the EAD theory. Dividing this difference by the larger P_IO_2 defines that part of the oxygen which contributes to decompression sickness. Expressed as a percentage, this quantity will be called the % EAD Error.

$$\%\text{ EAD Error} = 100\% \times \frac{[(P_IO_2)2 - (P_IO_2)1] - [\text{delta P2 - delta P1}]}{(P_IO_2)2}$$

Figure 4 presents the % EAD Error calculated from data of Berghage and McCracken (1979a) as a function of P_IO_2. All EAD Errors are defined relative to the lowest P_IO_2 and are widely scattered over both positive and negative values. The heavy line, which shows the mean error, suggests that the EAD theory holds reasonably well at low P_IO_2's but becomes progressively worse at higher P_IO_2's.

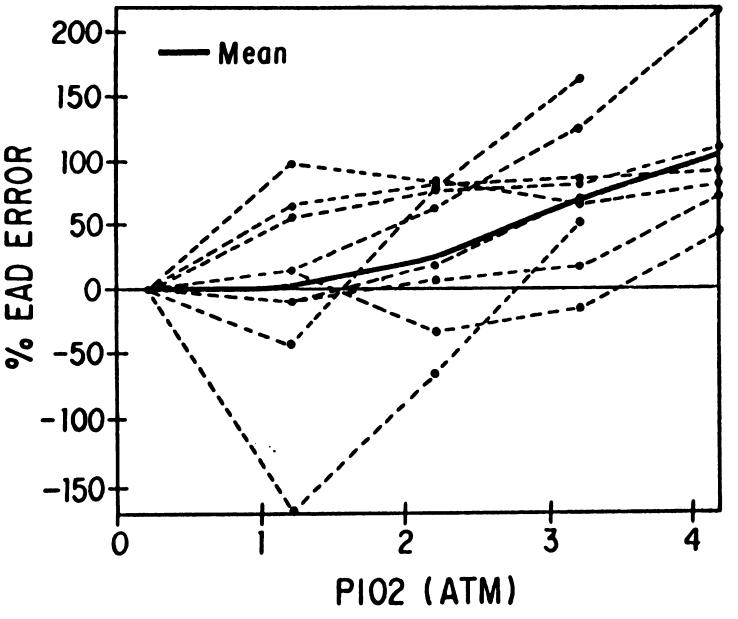

Figure 4. The % EAD Error as a function of P_IO_2 for the data of Berghage and McCracken (1979a). Saturation pressures were 10, 25, and 40 ATA with exposure times of 30, 60, and 120 min.

Figure 5 shows data from Berghage and McCracken (1979b) and from Rashbass and Eaton (1957). There is less scatter in the second Berghage study although fewer P_IO_2's were tested. The heavy line, indicating the mean values, suggests that over half the oxygen at 2.2 and 3.2 atm contributes to decompression sickness.

The lowest line in Fig. 5 is from Rashbass and Eaton (1957) who found the ED50 of rats compressed for 20 min to various depths and P_IO_2's prior to decompression to sea level. With this experimental design, the % EAD Error is given by the increase in P_IO_2 less the increase in ED50 pressure.

$$\% \text{ EAD Error} = 100 \times \frac{[(P_IO_2)2 - (P_IO_2)1] - [PB2 - PB1]}{(P_IO_2)2}$$

Rashbass and Eaton's data indicate that the EAD theory is valid between 0.2 and 2 atm and in error by 20% at 3.5 atm.

Lillo (1988) exposed rats to 141 fsw of nitrogen and 1, 2, or 3 atm of oxygen for various bottom times. Figure 6 shows how the percent non-fatal DCS and percent fatal plus non-fatal DCS increased with P_IO_2 after saturation exposures. The 50% increase for both end-points between 1 and 3 atm of oxygen indicates a substantial EAD error.

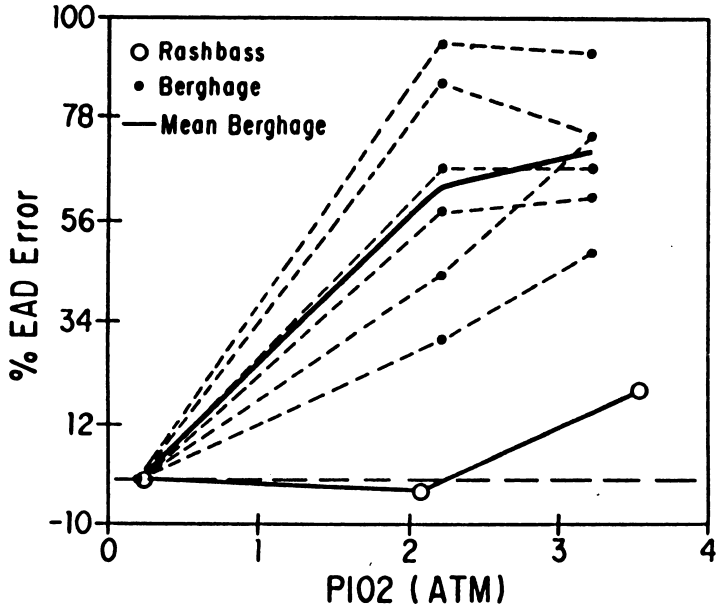

Figure 5. The % EAD Error as a function of P_IO_2 for data from Berghage and McCracken (1979b) and from Rashbass and Eaton (1957). Berghage and McCracken (1979b) used a saturation pressure of 15 ATA and exposure times of 1, 5, 10, 20, 40, and 80 min. The P_IO_2 after the pressure reduction was 0.5 ATM. Mean values are indicated by the heavy line. Rashbass and Eaton (1957) compressed rats from sea level to pressures ranging from 7 to 11 ATA for 20 min at various P_IO_2's in nitrox. Decompression back to sea level took 15 sec.

Donald (1955) was the first to address the question of oxygen in the EAD theory. He exposed 7 goats to a 60 min air dive at 50 fsw with a P_IO_2 of 0.53 atm. None of these animals developed DCS. When the P_IO_2 was raised to 3.53 atm with the same nitrogen content, however, 6 of 7 animals developed serious but transient symptoms. Five recovered spontaneously at sea level and one needed recompression. These results, shown in Fig. 7, indicate that oxygen is not an innocuous gas at 3.53 atm. Nevertheless, the spontaneous recovery from serious symptoms suggests that oxygen bends are probably less harmful than nitrogen bends. Presumably, the excess oxygen in bubbles is rapidly absorbed upon

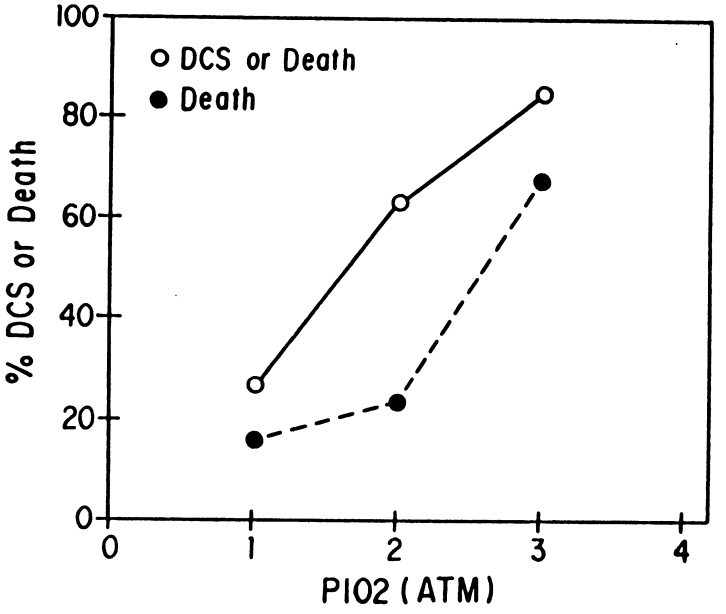

Figure 6. Fatal and non-fatal DCS for rats compressed to 141 fsw of nitrogen with 1, 2, or 3 ATM of oxygen. From Lillo (1988) based upon his analysis at saturation exposure.

decompression to sea level and return to normal P_IO_2.

Eaton and Hempleman (1973) decompressed goats from elevated pressure to sea level to determine the bends threshold pressure. They tested various P_IO_2's during 18, 50, and 180 min nitrox dives and during 120 min heliox dives. Figure 8 shows the % EAD Error as a function of P_IO_2. The errors varied from 10 to 20% for nitrox but were negative for heliox.

Two studies have investigated the EAD theory in humans. Logan (1961) tested 15, 30, 60, and 180 min nitrox dives at high and low P_IO_2's but the same nitrogen partial pressure. The DCS incidences from these tests are shown in Fig. 9 as a function of P_IO_2. Three studies were inconclusive because DCS did not occur on any dive. During 2 studies, one incident occurred in 5 trials at the lower P_IO_2 and 2 incidents occurred in 5 trials at the higher P_IO_2. These results are reflected by the increase in incidence from 20 to 40%.

Weathersby et al (1986) tested 30, 60, and 240 min nitrox dives at various depths and P_IO_2's. The mean DCS incidences at high and low P_IO_2 are shown in Fig. 9 for each bottom time. Each point represents the average of 76 to 82 dives. The incidence increased slightly at the higher P_IO_2 in one study and decreased in two studies. This could indicate either higher or lower risk at elevated P_IO_2, but neither conclusion was supported statistically. A lower risk might be explained by decreased tissue perfusion and nitrogen uptake due to oxygen-induced vasoconstriction.

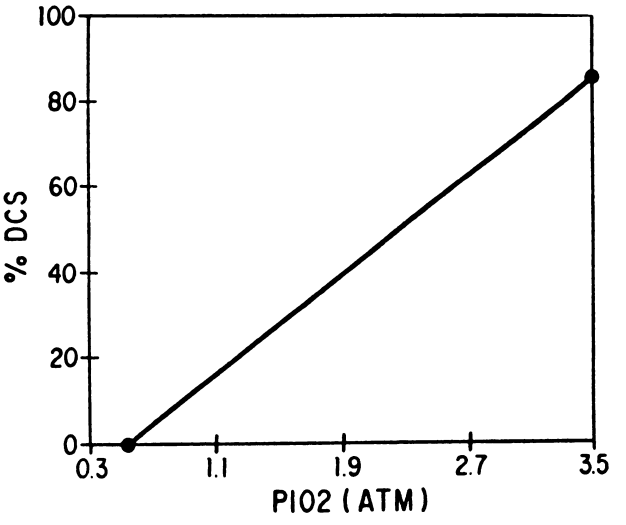

Figure 7. The DCS incidence of 7 goats with 2 ATM of nitrogen and 0.53 or 3.53 ATM of oxygen (Donald, 1955).

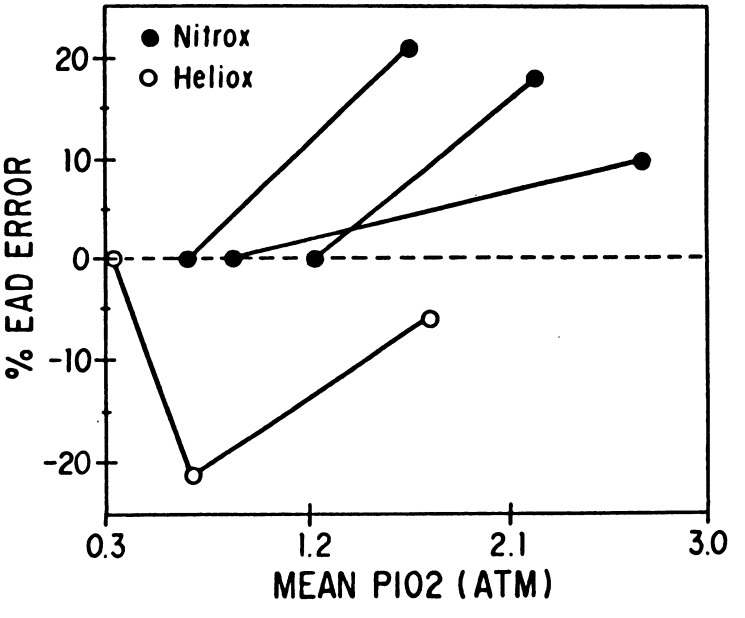

Figure 8. The % EAD Error as a function of P_IO_2 for goats decompressed to sea level after exposure to various pressures for 18, 50, and 180 min nitrox dives and 120 min heliox dives (Eaton and Hempleman, 1973).

Species differences, differences in experimental design, rapid decompression rates, and severe symptom end points may render some of the previous studies inapplicable to humans. Qualitatively, these studies show that EAD errors are smallest at low P_IO_2 and larger at high P_IO_2. This is consistent with the nature of oxygen transport in blood. Of the human studies, Logan

concluded (on the basis of too few experiments) that oxygen partial pressures between about 1.2 and 1.6 atm make a small, but not statistically significant, contribution to decompression risk and that this risk does not warrant abandoning the EAD theory. The Weathersby data does nothing to contest this conclusion, at least up to oxygen partial pressures of 1.3 atm.

NITROX DIVING AND DECOMPRESSION

How effective is nitrox diving at reducing decompression risk and time? We studied this question during 60 min dives to 100 and 150 fsw (Vann, 1982). A goal of at least 20 safe trials was set for each decompression schedule tested although as many as 30 trials were conducted. While this is more testing than is frequently used, the binomial probability theorem still indicates less statistical confidence than is desirable. With 20 safe tests, for example, the confidence level that a schedule will not produce more than a 5% DCS incidence is 64%. With 30 safe tests, the confidence level is 79%. To achieve a 95% confidence level, 60 safe tests are required. A schedule which caused decompression sickness was usually not tested again because more trials are needed to achieve the same confidence level than for a schedule tested without incident.

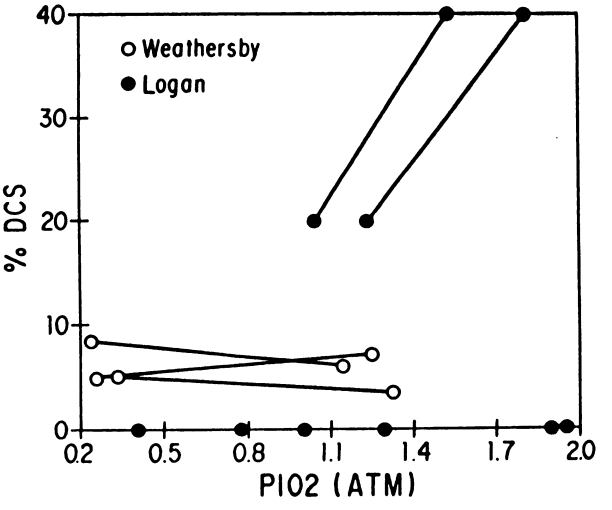

Figure 9. The DCS incidence as a function of P_IO_2 for humans from studies of Logan (1961) and Weathersby, et al. (1986).

The dives took place in a wet pot with the divers exercising at depth at an oxygen consumption of 1 lpm and resting during decompression. The water temperature was 20-25C, and full wet suits were used. The divers were Navy SEAL's, and the breathing apparatus was the Navy's Mk 15 UBA. The Mk 15 is a closed-circuit, mixed-gas scuba which controls the P_IO_2 independent of depth to a set point of 0.7 atm. Modifications to one Mk 15 made the set point adjustable and allowed the diver's oxygen consumption to be measured in real time as an index of workload.

The use of elevated oxygen partial pressure is limited by CNS oxygen toxicity. During one 100 fsw dive with a 1.6 atm set point, an oxygen convulsion occurred after 40 min of moderate to heavy work. The diver later reported, and bench tests confirmed, that the breathing apparatus had excessive respiratory resistance. This can cause CO_2 retention leading to an early onset of oxygen toxicity (Lambertsen, 1974; Piantadosi et al, 1979). Accordingly, the apparatus was modified to reduce resistance, and the divers were instructed not to "overbreathe" the equipment or work to the point of dyspnea. The oxygen set point was lowered to 1.4 atm in subsequent trials, and no further symptoms of oxygen toxicity occurred during exposures of up to 160 min.

Table 2 shows the results of decompression trials with oxygen set points of 0.7 and 1.4 atm. After the 100 fsw dive at 0.7 atm, one DCS incident occurred in 11 trials of an 80 min schedule. A 90 min schedule was tested safely 29 times. With the 1.4 atm set-point, a 20 min schedule was tested safely in 27 trials. The 150 fsw dive was attempted unsuccessfully at 0.7 atm in one trial of

a 195 min long schedule, but a 100 min schedule was tested safely in 20 trials at 1.4 atm. There was one DCS incident in 11 trials of a 90 min schedule. Very few intravascular bubbles were detected by precordial Doppler after the safe schedules.

Table 2. The effect of oxygen partial pressure on decompression during 60 min dives at 100 and 150 fsw (Vann, 1982). The divers did light work (1 lpm oxygen consumption) at depth and rested during decompression.

Depth (fsw)	PIO2 (ATM)	Decomp Time	DCS/ Dives	Mean Doppler	EAD (fsw)	USN Decom time (min)
100	0.7	80 min	1/5	1.0	106	56
	0.7	90	0/29	0.4		
	1.4	20	0/27	0.1	77	20
150	0.7	195	1/1	4.0	169	152
	1.4	90	1/11	0.5		

The last decompression stop during the 1.4 atm schedules was placed at 20 rather than 10 fsw to take advantage of the larger oxygen window and accelerate nitrogen elimination (Vann, 1982). The 1.4 atm set-point could not be maintained at 20 fsw, however, without exhausting gas from the Mk 15 breathing loop into the water and depleting the oxygen supply. This problem was largely overcome by reducing the set-point at 20 fsw from 1.4 atm (87% oxygen) to 1.3 atm (81% oxygen). Placing the final stop at 20 fsw rather than at 10 fsw also allows better depth control during open water diving.

Table 2 shows the EAD's for the these dives and the corresponding Navy Standard Air Decompression times. At 100 fsw and 0.7 atm, the time for safe decompression was about twice that of the Navy tables. At 1.4 atm for both the 100 and 150 fsw dives, the decompression time was nearly the same as the Navy EAD time. These results suggest that the corresponding Navy schedules may be less than satisfactory when used with an air breathing medium.

DECOMPRESSION TABLES

The most common nitrox diving technique employs a mixture with a fixed oxygen percentage rather than a fixed partial pressure. The U.S. and Royal Navies had standard nitrox mixes of 32.5, 40, and 60% oxygen for use with semi-closed circuit scuba. The U.S. Navy provided a list of Equivalent Air Depths so that these mixtures could be used with the Standard Air Decompression Tables. They also provided and taught the equation for calculating the EAD at any oxygen percentage (U.S. Navy Diving Manual, 1963).

The new French national decompression tables developed by COMEX provide EAD lists for mixtures ranging from 25 to 50% oxygen in 5% increments (Imbert and Bontoux, 1987). These mixes can be used with any decompression table, including in-water oxygen decompression and surface decompression. Other than the additional training and care needed to split, mix, and analyze gas, and the operational problem of depth control, there appears to be no reason against and sufficient precedent for using the EAD theory with any gas mix at oxygen partial pressures of up to at least 1.3 atm and possibly as high as 2 atm.

A much more difficult problem, however, is the choice of a decompression table to use with the EAD theory. The first axiom pertaining to this choice is that all decompression tables which allow useful work are associated with a finite risk of decompression sickness. What constitutes acceptable risk has yet to be agreed upon, but somewhere between 0.1 and 1% may not be an unreasonable number. Note that the term "acceptable risk" applies to dives at the full extent of permitted depth and bottom time. As most dives do not approach these limits, bends incidences in the literature such as 0.03% or 0.01% (Bove, 1987) do not accurately measure table safety.

A second axiom in selecting a decompression table is that there is little data on which to base a risk assessment and much of this data is concentrated around no-stop and single exposure dives. Existing evidence concerning the widely used Navy tables, for example, suggests the no-stop limits and short decompression dives are reasonably safe for single exposures, but dives requiring decompressions of 30 min or more have progressively higher decompression risks (Weathersby et al, 1985; Thalmann, 1985).

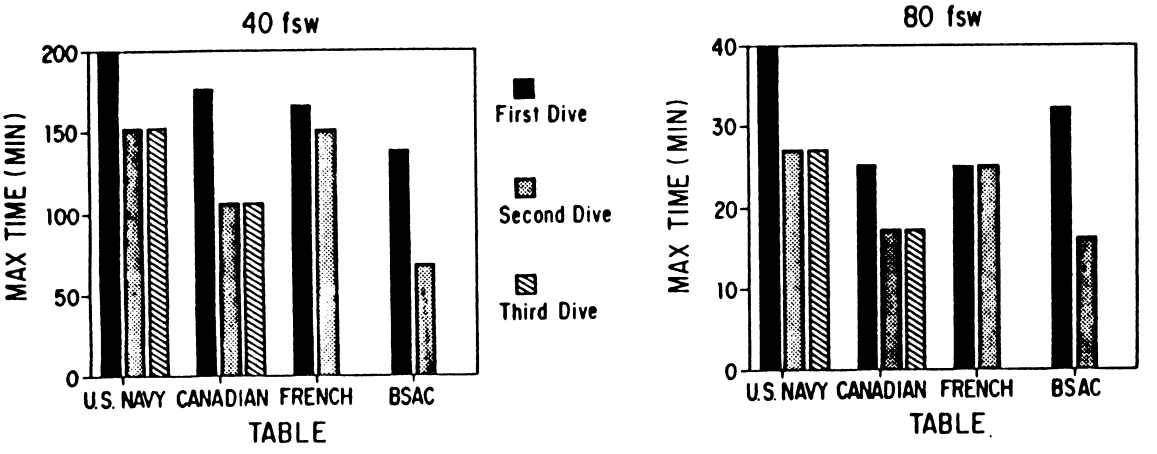

Figure 10. A comparison between no-stop repetitive dives to 40 and 80 fsw with a 4 hr surface interval for the U.S. Navy, Canadian, French, and BSAC air decompression tables.

The safety of the important repetitive dive tables is an even greater mystery. The Navy repet tables appear overly conservative for one or two repetitive dives with Repetitive Groups near the beginning of the alphabet, but multiple repet dives, particularly over many days, may lead to increased risk (Thalmann, 1985).

The uncertainty concerning both single and repetitive dives is illustrated in Fig. 10. The no-stop repet dive limits for 40 and 80 fsw dives with 4 hr surface intervals are compared for the U.S. Navy, Canadian (UDT, 1987), French, and the older BSAC (Hazzard, 1980) tables. The height of each bar represents the maximum no-stop exposure limit. The solid bar in each table grouping indicates the first dive, the diagonally-hatched bar indicates the second, and the cross-hatched bar indicates third and subsequent dives. The agreement between tables is not striking, particularly for repet dives. Both the Navy and Canadian tables allow multiple repetitive dives of equal length for the same surface interval. The French and BSAC tables, however, consider more than one repetitive dive to be unsafe. The surface interval after which a second dive is no longer repetitive is 12 hrs for the French and Navy tables, 16 hrs for the BSAC tables, and 18 hrs for the Canadian tables.

Each table has a different method for finding repetitive dive bottom time. The Canadians use a modification of the Navy procedure, the French provide a separate table for each surface interval, and the BSAC uses an average of the first and second dive times weighted according to surface interval. There is little question that a shorter dive is safer, but how short is safe enough? When dealing with risks of less than 1%, individual susceptibility may be more important than a small change in bottom time. At present, data are insufficient to determine which repetitive dive method is safe enough but not overly conservative.

While decompression tables will be with us for a long time, the ultimate solution for repetitive diving is the diver-worn decompression computer. As with any decompression procedure, however, DCS risk is greater for certain types of diving and depth-time ranges. The Divers Alert Network is currently studying the nature and risk of diving with both tables and computers. Table 3 shows preliminary results of this study for 38 computer cases and 91 computer and table cases. Dive profiles were examined in 4 categories: No-stop or decompression; square or multi-level; single or repetitive; and single-day or multi-day. For both computers and tables, decompression sickness was twice as frequent for repetitive and multi-day dives as for a single or single-day dives. Since the first dive of every repetitive dive is a single dive and the first day of every multi-day dive is a single-day dive, repetitive and multi-day dives are significant risk factors.

This observation appears contradictory to the effect known as "adaptation" in which DCS risk is decreased by daily pressure exposure. Adaptation was best documented in caisson workers exposed to relatively low pressures for up to 8 hrs (Walder, 1968). The most widely accepted explanation for adaptation is that daily pressure exposure depletes the nucleation sites from which bubbles form (Vann, 1987). A more recent explanation argues that decompression sickness is mediated by bubbles which activate the complement system and that depletion of complement during repetitive diving leads to reduced DCS susceptibility (Ward et al, 1987).

Multi-day diving differs from caisson work in that pressure exposures are shorter, deeper, and more frequent. Such diving may lead to an accumulation of bubbles which require longer than 12 hrs to be absorbed. It is for this reason that the BSAC and Canadian tables have adopted 16 and 18 hrs as the surface interval to clear the effects of a previous dive. An alternative explanation is derived from animal experiments which demonstrated that venous bubbles in the alveolar circulation can pass through the pulmonary filter and enter the arterial circulation during repetitive diving (Gait et al, 1975).

Table 3. Preliminary analysis of DCS cases collected by the Divers Alert Network.

	Computer Only 38 Cases ('84-'87)	Computer & Tables 91 Cases ('87)
Multi-Day	66%	70%
Repetitive	84	67
Multi-Level	47	27
Decompression	71	27

Table 3 indicates that bends are more common during square rather than multi-level dives for both computers and tables, but more multi-level bends occur with computers. The greatest difference between computers and tables is that decompression sickness occurs more frequently during decompression dives with computers and more frequently during square dives with tables. One explanation for this observation argues that divers are more likely to decompress when using a computer. Another

explanation holds that the decompression risk is greater for table diving because the no-stop limits are longer.

Without knowledge of the number of dives conducted, a distinction between these explanations is difficult. Fortunately, this knowledge, which is essential to the assessment of DCS risk, may be available in the future with the help of the decompression computer used as a depth recorder. Figure 11 shows a dive profile recorded during a Divers Alert Network Doppler research trip by a Suunto decompression computer made in Finland. The dashed line is the profile which the diver recalled from memory. The actual profile read from the computer memory is shown by the solid line which was drawn from depth-time points recorded at 3 min intervals.

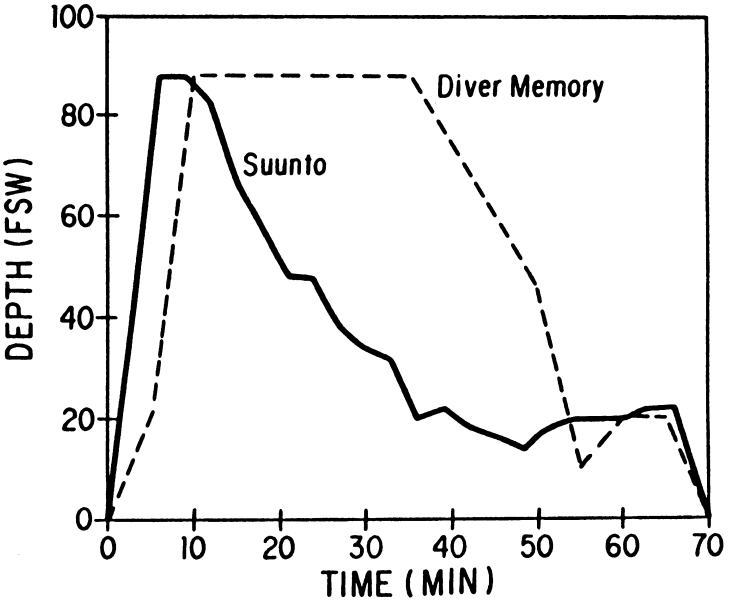

Figure 11. Dive profiles recalled by a sport diver and stored in a Suunto decompression recorder/computer. From data taken during a Divers Alert Network Doppler research trip.

Dive profiles of this nature and the results of both safe and unsafe dives can now be analyzed for decompression risk by a statistical method known as maximum likelihood. This method can be used to determine how well a decompression model explains the results of a dive series, and thus, it allows a quantitative comparison between models. The most successful model I have yet found assumes that before bubble formation, inert gas exchange is limited only by blood flow while after bubble formation, gas exchange is limited by both blood flow and diffusion (Vann, 1986). In Fig. 12, diffusion resistance is imposed by a diffusion barrier surrounding the bubble.

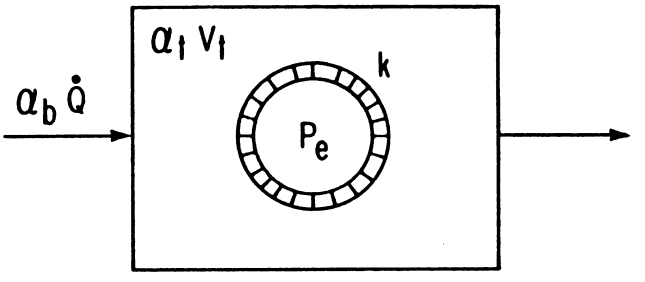

Figure 12. A decompression model simulating blood flow limited inert gas exchange in tissue and diffusion limited inert gas exchange in a bubble (Vann, 1986).

Such a representation of gas exchange makes it possible to predict the frequently observed delayed onset of DCS symptoms. This delay is illustrated in Fig. 13 for a 60 min air dive to 150 fsw. Most decompression models, including the Haldane model, predict that the greatest decompression risk occurs immediately upon surfacing. When bubble growth is limited by diffusion, however, the maximum risk occurs in the post-dive period--2 hrs after surfacing in the example shown here. Since bubble resolution as well as growth is limited by diffusion in this model, a bubble could persist and expand during a series of multi-day dives. This might explain the increased risk observed during multi-day diving.

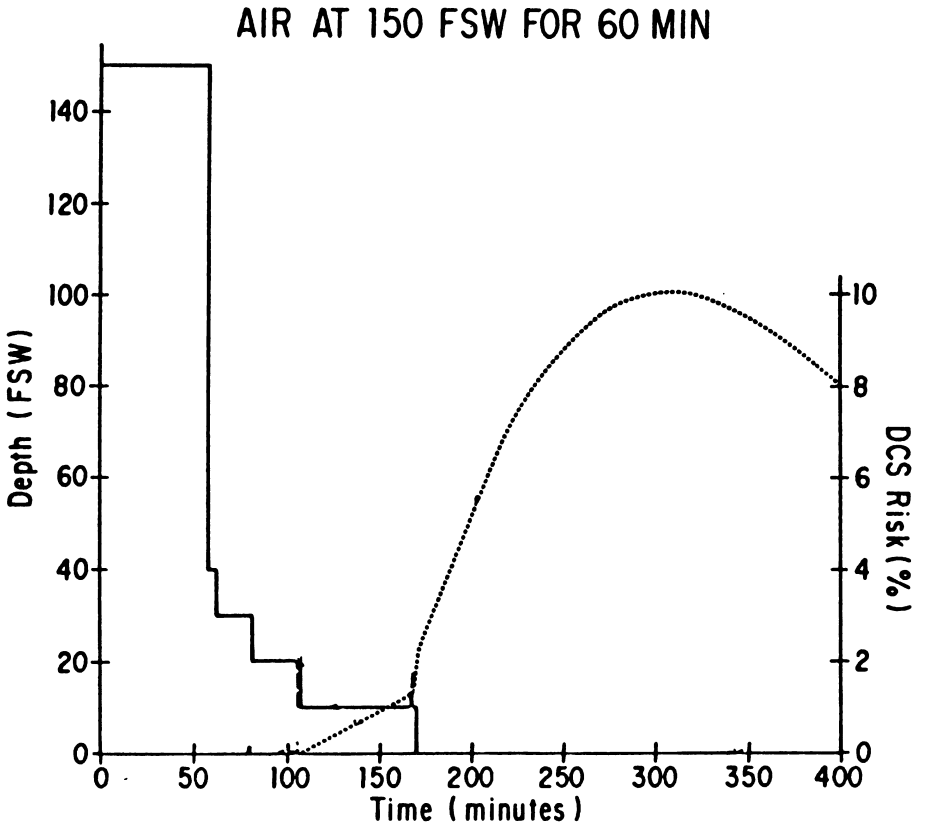

Figure 13. DCS risk after a 60 min air dive to 150 fsw as predicted by a decompression model in which gas exchange is blood flow limited in tissue and diffusion limited in bubbles (Vann, 1986).

CONCLUSION

The most effective method of increasing decompression safety and efficiency is the judicious use of oxygen. Oxygen-enriched air, supplied either as a constant percentage or a constant partial pressure, is equally useful in reducing nitrogen uptake at depth, but a constant partial pressure mix is preferable during decompression as it allows faster nitrogen elimination. Alternatively, 100% oxygen breathed during the shallow stops will provide the greatest decompression safety and shortest decompression time.

Experimental evidence pertaining to the validity of the EAD theory is mixed and further study is certainly warranted, but evidence from human studies does not contraindicate its use in unmodified form, at least up to an oxygen partial pressure of 1.3 atm.

The most significant problem for nitrox diving is a lack of confidence in existing decompression tables, particularly repetitive dive methods. The solution to this problem lies in empirical testing and statistical analysis. Dive computers are expected to play an important role in decompression procedure validation through their data logging capability and to provide a practical solution to the thorny issues of repetitive and multi-day diving.

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APPENDIX TO DR. VANN'S PAPER

Vann RD, Dovenbarger J, Wachholz C, Bennett PB. 1988. DCS and decompression meters. Undersea Biomed Res 15(Suppl):64.

101. DCS AND DECOMPRESSION METERS. R.D. Vann, J. Dovenbarger, C. Wachholz, P.B. Bennett. Hall Lab & Anesth. Dept., Duke Medical Ctr., Durham, NC.

38 DCS incidents with decompression computers were reported to the Divers Alert Network. 33 involved males (mean age 37 yrs). 5 involved females (mean age 36 yrs). 8 had pain, 11 had serious symptoms, and 19 had pain and serious symptoms. Dive profiles were categorized as: no-stop or decompression; square or multi-level; single or repetitive; single or multi-day; maximum depth on day of incident. Individual risk factors were examined. DCS increased with depth, decompression, repetitive, and multi-day diving. Individual risk factors were prominent in the least stressful categories. Dives in the upper left corner of the table may have been safe or may not have been conducted.

Max Depth	<60	60-69	70-79	80-89	90-99	100-109	110-119	120-130	>130
NoStpSquare									
DecomSquare									2(1*)
DecomMltLev							1*	1*	
NoStpMltLev				1(1)+					1(1)
NoStpRepet	1*				1*&(1)				
NoStpMltRpt			1(1)*		2(1*)	1(1)		1(1)	2(1*)
DecomMltRpt				3(3)&	1(1)	1(1)		1(1)%	1*(1)
DecomRepet		1(1)*		1*&(1)	2(1)	4$(3)	1(1)	2	5(2)

Risk factors:

() - 25 multi-day dives
* - 14 two incidents in same diver (high susceptibility)
& - 8 back trouble (spinal stenosis, diskectomy, fracture, etc.)
$ - 4 CNS disease or previous injury at DCS site
\+ - 1 vestibular DCS with a history of vestibular trouble
% - 1 obesity

Discussion following Dr. Vann:

DR. VANN: An abstract for the 1988 UHMS meeting which gives a little information about decompression sickness and diver-carried decompression computers is included as an appendix to this paper. This particular abstract does not do a comparison with tables but it looks into the effect of depth.

Also, we took into account the individual risk factors which, as I indicated, seem to be important for very low risk dives.

DR. LAMBERTSEN: In the course of investigating the role of oxygen, dissolved oxygen in tissues in relation to whether decompression sickness occurs, one has to distinguish between whether bubbles are going to form anyhow; in other words, the nature of the decompression itself. Is that not right, that there is an inert gas component? It is not just oxygen that the animals are being exposed to?

DR. VANN: That is correct, yes, but it is the total dissolved gas situation that is important, oxygen plus inert gas plus CO_2.

DR. LAMBERTSEN: You are exactly right. Now, if you then recognize that and want to investigate whether oxygen actually can produce bends, not just contribute to whatever bends have happened, that is a different situation, is it not? Are there not two separate circumstances?

DR. VANN: It is sort of a compendium, but I think I would agree with that end of your compendium.

DR. LAMBERTSEN: If you wanted to see if there is such a thing as "oxygen bends", which is what the terminology says....

DR. VANN: Donald's term.

DR. LAMBERTSEN: That is what I said. We must not repeat something that is not right. Oxygen would be a certain thing if you were to compress on oxygen and stay there for a time and then decompress on oxygen to see whether you got oxygen bends. If you then wanted to find out if you got some influence of oxygen on bends, you would do a mix situation. Most all of these, as I see them, are mixed gases, are they not?

DR. VANN: Absolutely.

DR. LAMBERTSEN: I am trying to bring out here that if you want to talk about oxygen bends, then it ought to be oxygen. To do that you would end up producing oxygen poisoning, and you would be decompressing an oxygen poisoned animal, because you have to go to very high O_2 pressures to do that.

What I was saying is that there has not been a pure situation; if we are going to talk about oxygen bends, we ought to know what we mean and make sure it is oxygen. Then when you get through doing the experiment, you should be able to separate it from the oxygen poisoning. That is the kind of experiment that is needed.

DR. VANN: I think the experiment that is needed is to look at EAD theory, and most profitably and most importantly to do it in humans. Regarding the terminology of whether you call it oxygen bends, EAD theory, or whatever, we know what we are talking about, the concept of "Does oxygen contribute?" at this or that partial pressure. That is the issue.

You are right. The term oxygen bends has confused people, because people have generally said that any increased oxygen partial pressure is a problem. I think Ed Thalmann did this; he jumped on the Eaton and Hempleman data, and because of that he put it into his model. Whereas, if you look at all of the data, they are so scattered that, as Jim pointed out, you can go any way you wish. But if you look at the human data and particularly the study by Weathersby--who expected, I think, to find a bad EAD effect there--the best evidence just does not support it.

DR. LAMBERTSEN: I think that is a good way to state the circumstances with a mixed situation of oxygen and inert gas. So where we are at the moment, then, with this discussion is that nobody has done a demonstration of oxygen bends. There have been many studies in animals and some

examinations of human decompression with mixed oxygen and inert gases, and you do not find anything there, statistically. Isn't that what you just said?

DR. VANN: Yes. And I think what you are doing is to correct a historically incorrect semantic term.

DR. LAMBERTSEN: That is the idea. Now we have gone two thirds of the way through the story, okay.

The next third of the story is, as I pointed out to you before, whenever you expose someone, animals or otherwise, to oxygen at very high pressures, you actually create a tissue hypercapnia. This then does things to the whole of the body physiology that are so complicated and uninvestigated that you cannot just say that we have just examined an effect of physically dissolved oxygen on bubbles, when the events that have been produced by oxygen at high pressure are multiple events and not just increases in physically dissolved oxygen.

DR. VANN: That is true, but you should not fall into our own oxygen trap. It is a function of the PO_2 itself. At low PO_2's there is very little difference and the higher you get, the greater that effect will be.

DR. LAMBERTSEN: If you again want to say that oxygen may influence the factors that lead to bends, then the story becomes a little more accurate; but to call it oxygen bends or bends aggravation by physically dissolved oxygen, that is not right. You have to really watch out for that one or else the experiments get designed wrong.

MR. GALERNE: I have a question. Can you tell now the possibility of CO_2 participation in bends?

DR. VANN: Well, there is a recent study that I think examined that effect. This was done over in England; it was called "Islander," I believe. They were looking at nitrox/air saturation, at 25 fsw give or take a few feet, and they did it with CO_2 and without CO_2. I can not remember, was it 2% CO_2, Claude?

> Reference: Bell PY, Harrison JR, Page K, Summerfield M. 1986. An effect of CO_2 on the maximum safe direct decompression to 1 bar from oxygen-nitrogen saturation. Undersea Biomed Res 13(4):443-55.

CAPT HARVEY: They had a couple of different levels.

DR. VANN: In any event, they did not find any increase in bends as a result of CO_2. The implication was there that there may have been fewer intravascular bubbles with CO_2, but the changes were so small that you could not really draw any significant conclusion.

There is a lot of information in the old literature, particularly from tunnel work, concerning the effect of CO_2 on bends which says, yes, it is important, and then this study says, no, it is not important. Perhaps it is a condition of the situation. Perhaps it does not make any difference.

DR. HAMILTON: You do not know what else is going on in addition to the higher CO_2. There is really no causal relationship that has been shown.

DR. VANN: Correct.

DR. HAMILTON: The British group did an experiment that tried to clear everything else away and look at just that, and I thought it was pretty good.

DR. VANN: Yeah, it seemed to answer the question.

DR. HAMILTON: On the slide in which you showed Weathersby's data, with no EAD deviation at all, you showed some points by Logan. If I recall what you said, they were something like two out of six or four out of six.

DR. VANN: One out of five and two out of five.

DR. HAMILTON: That is not a difference. You cannot draw that kind of a line and compare it with Weathersby's data on that.

DR. VANN: I think that is true. That is right.

DR. HAMILTON: Weathersby used lots of dives, and furthermore he was trying to find a difference.

DR. VANN: He was trying to find it and was disappointed when he did not find it.

DR. HAMILTON: That is the best evidence. All of the rest of the evidence you more or less dismissed, did you not?

DR. VANN: Well, you know, something is going on, and certainly the higher the PO_2, the more oxygen is likely to have an effect.

DR. HAMILTON: That is what Dr. Lambertsen said, because oxygen is a poison or a very physiological substance. We do not know enough about what it does at 2 atmospheres. The effect may be more one of physiology than its acting as an inert gas.

CAPT HARVEY: I would like to reemphasize something that you said in the paper, because I think it is important. We are looking at a relatively new (or revised) technique in using this enriched air or oxygen additive or whatever you want to call it. When we do that and we use the EAD concept, we are now relying on tables which have incidences of bends associated with them. We do not know what that incidence is.

With the Navy tables, frequently they do not dive them right up to the mark, if you will. Particularly if there is hard work done or cold exposure, they will "slide" a table.

So when you try to analyze the statistics, it is very difficult to really say what the bends incidence is for any given Navy table. I would caution everyone not to blame the technique of adding the oxygen. If we start seeing decompression sickness or a problem, remember the table itself may be the problem that you are finding. Keep that in mind.

DR. VANN: The evidence indicates that the problems are going to be with the tables, not the EAD theory.

MR. NISHI: I would like to comment on what you said about the bubble intensity increasing at about a 2 hour maximum.

DR. VANN: That was just in a particular example.

MR. NISHI: From some of our doppler studies we find that bubbles do not appear right away and we get the maximum bubbles about one to two hours after a dive. So this might correlate with your calculations.

MR. RUTKOWSKI: I do not know if this is the time or the place, but if possible, could you just comment a little bit on the status and the validity of the new PADI 60 minute half time "tissue" for repetitive dives?

DR. VANN: As you said, this is neither the time or the place.

DR. HAMILTON: Come on, it is a good time and a good place. Go ahead.

DR. VANN: No, I am not going to say anything about that. There are a couple of issues that are being sorted out, and this is not an appropriate time to discuss it right now.

MR. RUTKOWSKI: I just thought maybe we could catch you in a weak moment.

DR. VANN: Nice try.

DR. CROSSON: What would you say would be the next major step for looking at it (enriched air nitrox) in the broadest scope, looking at research?

DR. VANN: Well, I have a modest proposal that I plan to make. I do not know if I should discuss it this widely but I think there is a lot that can be done. I think we are perhaps at the right moment in space and time to make some improvements and some changes and perhaps this group will catalyze it. I mean, basically, let's see what we can do to improve decompression safety and efficiency by using oxygen-enriched gas mixtures, oxygen enriched air. I think a lot can be done. As to your specific program, I have some ideas in mind but I really want to think about it a little bit more before commenting.