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Decompression limits for compressed air determined by ultrasonically detected blood bubbles

MERRILL P. SPENCER
Institute of Applied Physiology and Medicine, Seattle, Washington 98122

SPENCER, MERRILL P. *Decompression limits for compressed air determined by ultrasonically detected blood bubbles.* J. Appl. Physiol. 40(2): 229–235. 1976.—The direct decompression limits for a group of divers over a range of pressure-time air exposures was determined using ultrasonic detection of venous gas emboli (VGE). In addition to dry chamber exposures, ranging from 233 ft for 7 min to 25 ft for 720 min, we exposed six divers to open ocean dives at 165 ft for 10 min. Findings demonstrated a strong individual propensity to form VGE, correlating with susceptibility to bends. No bends developed without the prior detection of precordial VGE. The present concept of no problems after any period of time at 30 fsw was not confirmed. Isopleths of equal percentage occurrence of VGE were computed between 10 and 60%. Open ocean exposures increased the percentage of VGE and bends, when compared to dry chamber exposures. Limiting tissue half times computed from the 20% VGE isopleth suggested that saturation exposures are controlled by a greater sensitivity of the short-half-time tissues than previously appreciated, rather than by additionally extended half times.

Doppler detection; decompression limits; HPO

PRESENTLY USED MODELS for decompression tables were developed as an elaboration of principles of tissue tolerance to excess inert gas laid down by Haldane and Priestley (4). A series of theoretical tissue compartments are each considered to have their allowable supersaturation tolerance, the so-called M values (12). These M values are tested by exposing subjects to hyperbaric respiration followed by a decompression schedule which is calculated not to exceed safe limits. If decompression symptoms develop, a more conservative schedule is tried. Difficulty arises in improving the schedules because the assessment is largely subjective, and the investigator must guess which theoretical compartment is to blame when he makes his adjustment. This report describes a whole-body approach to body tolerance of excess hyperbaric nitrogen using Doppler ultrasonic detection of decompression blood bubbles as an objective end point. The experimental results are related to existing theories, and improvements in present decompression procedures are suggested.

METHODS

Our strategy was to expose human divers to compressed air over a wide range of pressures and lengths of exposure, and to observe the occurrence of venous gas emboli (VGE) for the various exposures.

A specially developed 5-MHz CW Doppler ultrasonic flowmeter was used which operated both a large crystal deep focusing precordial detector (9) as well as a variety of shallow-focusing peripheral vascular detectors (10). The advantage of the precordial detector over other available equipment includes a large focal volume, making positioning less critical, and a wide dynamic range for both faint blood flow signals as well as large amplitude blood bubble signals. In addition, by listening over the heart we have the advantage of detecting all VGE regardless of source.

The precordial transducer is positioned along the left midsternal border. Its focus includes the right atrial appendage, the right ventricular outflow tract, the pulmonary valve, and the pulmonary artery. Optimum positioning is confirmed by locating the closure sound of the pulmonary valve which has a chirping quality similar to some VGE signals, but does, of course, occur regularly in the heart cycle at the end of systole. All VGE signals were recorded on magnetic tape along with voice notations. The tapes were later replayed for confirmation and documentation of VGE signals. If, during the monitoring, precordial VGE were detected, the divers were then asked to flex the limbs—one at a time—while monitoring continued. Bursts of Doppler signals from bubbles, thus dislodged, gave a more specific idea as to the regional source of the VGE. A shallow-focusing Doppler probe was then substituted for the precordial probe and a search was made over left and right femoral, brachial, and jugular veins. Later refinement of the technique resulted in addition of the subclavian veins as a useful peripheral listening site using a new 3.5-cm focal length 5-MHz flooding probe (Fig. 1). During monitoring of the peripheral veins, manual compression of the distal tissues of the limb or neck identified the regional source of VGE. Though arteries were also monitored for possible bubble signals, no arterial VGE were detected in any of our subjects.

We recorded the time of first occurrence of the precordial bubble signals following decompression and an estimate of their duration, as well as a graded indication of their frequency along with the absence or presence, severity, and location of any symptoms of decompression sickness. The grading system for the venous return bubbling rate heard over the right heart consisted of a five-point scale: zero is taken to indicate a complete lack

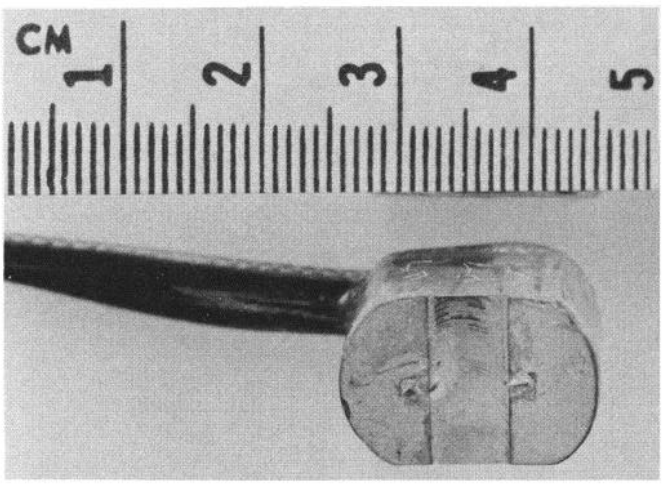

FIG. 1. Miniature dual crystal detector for monitoring of both peripheral and precordial sites.

of bubble signals; grade 1 indicates an occasional bubble discernible within the cardiac motion and with the majority of the cardiac periods free of bubble signals; grade 2 is designated when many, but less than half, of the cardiac cycles contain bubble signals; grade 3 is designated when most of the cardiac periods contain bubble signals, but not overriding the cardiac motion signals; grade 4 is the maximum detectable bubble signal, heard continuously throughout systole and diastole of every cardiac period and overriding the amplitude of the cardiac motion signals.

Exposures. All of the results presented in this paper refer to what we call "direct decompression" exposures. Direct decompression is defined as return from hyperbaric exposures to 1 atmosphere absolute (ATA) at the rate of 1 f/s without staged decompression stops. The term direct decompression (D-D) is used here as a more appropriate term for what is commonly referred to as No-Decompression (No-D) (11). The body is subjected to a square-wave input in the form of a constant known pressure (D) for a known period of time (T).

Twenty-six expert male scuba divers residing in the Seattle area, ranging in age from 21 to 37 yr (avg 27 yr), volunteered for the study. Their weights averaged 80.8 kg ranging from 71 to 102, and the heights averaged 179.7 cm ranging 170 to 188 cm. These experimental subjects, including three full-time instructors, frequently performed scuba dives in the waters of the Puget Sound area and were well adapted to decompression stresses. In each of the exposures the subjects were resting quietly in a sitting or semirecumbent position throughout the exposure.

Several predictors of the locus of the VGE direct decompression limits were used. These included the US Navy No-D table for air (12), the experimental results of Hawkins et al. (5) and Albano (1), as well as theoretical predictions from nitrogen elimination data of Behnke (2). Animal data of Hempleman (6), Smith and Johanson (8), and Gillis (3) were also taken into account.

Precordial predive control recordings were initially made on all divers 15 min prior to compression. Then immediately before, and for 1–5 h after decompression, the divers were monitored with the precordial blood bubble detector for the presence of VGE. Peripheral veins were checked with special Doppler detectors if precordial VGE were found. The predive recordings gave a base line with which to compare the postdive recordings and also made certain that all the divers were bubble free prior to compression. None of the subjects had been diving within 24 h of the experiments. Postexposure precordial recordings were made at 5, 10, 15, 25, 40, and 60 min during the first hour and at 30-min intervals during a subsequent 60- to 300-min period, depending on the specific dive profile. This sequence was followed if Doppler ultrasonic signals of VGE were not detected over the precordium. Extended monitoring periods were used when VGE developed.

Subjects were recompressed for therapy if bubbles occurred in more than grade 3 quantities, or if bends pain developed. Recompression treatment in the 30-ft dives was to 60 ft on the US Navy treatment schedule V. For the remaining dives, recompression treatment was for 1 h at 30 fsw with the subject breathing 100% O_2. In many instances, where grade 1 bubbles were detected early and rapidly progressed to grade 2 quantities, the subject breathed 100% oxygen at one atmosphere until the bubble signals diminished or disappeared. Our conservative approach to treating grade 3–4 bubbles with HPO, no doubt, reduced our incidence of bends, but the use of surface O_2 in grade 2 bubbles probably did not reduce bends because surface O_2 was only used when the subject had stabilized at grade 2 without bends.

To test the relationship between chamber exposures and actual open ocean dives, we exposed six divers to identical profiles in the chamber and in the open water at Cobb Seamount, 270 miles off the Pacific coast of Washington State. Efforts were made to keep the depth-time (profile) relationships as close to identical as possible. The chamber exposures were monitored by both mechanical and electrical pressure transducers. A water depth gauge was used whose calibration agreed with the chamber gauges. Ascent and descent rates were maintained at 60 ft/min in the chamber, whereas ascent and descent in the water was estimated to be the same by pulling one hand over the other, on a descending line, in 1-ft increments while counting "one-thousand one, one-thousand two, etc." The chamber exposures represented a warm, comfortable environment with no work load other than respiration of a more dense medium, while the open ocean exposures necessitated working from an open boat, swimming in swelling seas running at slightly more than 1 knot, and water temperature at 55°F. All divers on the open ocean exposure wore wet suits, bouyancy compensating vests, weight belts, and twin 72-ft^3 air tanks.

RESULTS

Chamber exposures. The grade of precordial bubbles and corresponding symptoms for each dive and each exposure are listed in Table 1. In this table, for example, *diver SC*, on the top row of the table, made 12 dives. On the dive for 7 min at 233 ft (in upper left corner of the table), grade 4 precordial bubbles were detected and he

TABLE 1. *Results from direct decompression chamber experiments*

Diver	Avg Grade	Group A						Group B				Group C	
		Ft/Min						Ft/Min				Ft/Min	
		233/7	230/8	200/7	165/10	150/15	150/10	70/70	70/60	70/50	60/60	30/720	25/720
SC	2.0	4 D*	2 B	0 —	2 B	1 BC†	0 B	2½ B	3 B†	2 AB	2½ B	2 BC	
					3 B								
MP	1.6	4 BD*	3 B†	1 B	0 B	3 BC†		1 B	0 B	3 BC	1 B	1 B	
PM	1.2	0 B	2 B	0 B	1 B	2 B†		3 BC		0 B	0 B	3 D*	
MR	1.0	2 B*	1 B	0 B	0 B	2 B†		0 —	0 B	0 —	2½ B	4 B*†	3½ —
AG	0.86	0 B	1½ B	0 —	1 B	3 B*		0 B	0 B	1 B	0 B	3 A*	0 —
DJ	0.60	2 C	0 B	1 B	0 —	0 B	0 B	0 —	0 —	0 —	3 C	0 B	
					0 B	0 B				0 —		3 BC†	
AF	0.45	0 B	1 B	0 B	1 B	0 B		0 B	1 —	1 B	0 B	1 —	0 —
BA	0.40	0 B	0 B	0 —	0 B	2 BC†		0 B	0 B	0 B	0 B	2 B	
JE	0.33	0 B	0 B	0 —	0 B	0 —		0 B	0 —	0 B	0 —	1 A	3 C†
					0 B								
KP	0.13	0 B	0 B	0 B	0 B	1 B		0 —	0 B		0 B		
GA	0.09	0 B	1 B	0 —	0 —	0 B		0 —	0 —	0 —	0 —	0 —	
					0 —								

Columns represent feet water pressure (gauge)/time in minutes. 0, 1, 2, 3, 4 = maximum grade of precordially detected bubbles. A, B, C, D, E = decompression symptoms: A = vague uneasy feeling; B = skin itching; C = mild pain; D = moderate pain; E = severe pain (none found). Fifteen additional subjects made 22 exposures each, four or less in number. The results on all 26 subjects are summarized in Table 3. *Recompression Rx on O_2. †Rx with surface O_2.

reported moderate pain. He was treated by recompression on O_2. On another dive, for 8 min at 230 ft, *diver SC* developed grade 2 bubbles and skin itching. The table shows the spread in propensity to produce VGE ranged from *SC*, who produced VGE in 10 out of 12 dives, to *GA*, who produced VGE in only 1 out of 11 dives. The average grade of VGE formed by each subject exposed more than three times is distributed from 2.0 for *SC* to 0.09 for *GA*.

The data of Table 1 also demonstrate the great variability in response of the same diver from day to day. For example, *DJ* formed VGE after decompression from 60 min at 60 ft, but did not form them after 50, 60, and 70 min at 70 ft. Also of interest is *JE*, who formed VGE only after the long shallow exposures 25/720 and 30/720, but formed no bubbles on any of the other exposures, including the 150/15 exposure which caused the greatest difficulty of all the protocols.

Table 2 shows the time of onset of the first detectable VGE. In spite of the wide scatter in time of onset, there is a definite trend for a sooner bubble release into the veins after deep exposures and a later release after shallow exposures. Table 3 shows the percentage of subjects with precordial detected bubbles computed for each exposure and for each group of exposures. Because of the great variability between divers, and within the same diver from day to day, the exposures were lumped into three groups designated *A*, *B*, and *C*. The mean pressure and time for each group was computed in each group as shown in Table 4.

Graphical analysis. The results were analyzed with a graphic method to develop isopleths of equal occurrence of VGE and bends pain. Figure 2 illustrates the method. The average pressure and time for each group of exposures is plotted on log-log coordinates, and the percentage of subjects with precordial VGE is entered beside each respective point. The isopleths are computed from the plot with the following assumptions over the range of the data: *1*) that each isopleth is a power function which is represented by a straight line on log-log coordinates, and *2*) that the distance between isoembolic isopleths is in direct ratio to the differences in their percentage occurrence of VGE.

TABLE 2. *Time of onset of first detectable VGE (minutes after surfacing)*

Exposure ft/min	Times of 1st Bubbles	Average Time Of 1st Bubbles		No.	$\bar{x}$	$s_{\bar{x}}$	$s_{\bar{x}}$
233/7	3, 3, 5, 13	6.0					
230/8	3, 8, 10, 12, 15, 70	19.7					
200/7	6, 12, 20	12.7	Group A	25	12	±13	2.6
165/10	3, 4, 5, 10, 12, 15	8.2					
150/15	2, 5, 13, 15, 15, 15	10.8					
150/10	No bubbles						
70/70	No data						
70/60	10, 35, 40	28.3					
70/50	10, 10, 30, 30	20.0	Group B	11	25	±17	5
60/60	7, 12, 30, 60	27.3					
30/720	6, 15, 15, 30, 30, 90	31.0					
25/720	3, 17, 19½, 53, 186	55.7	Group C	11	42	±54	16

The locus of the three isopleths representing the three different percentage occurrences are located by finding the straight lines with spacing representing assumption *2*, i.e., the distance between the 29% isopleth and the 38% isopleth should be 24% of the distance between the 29% and 67% isopleths. By trial and error, we therefore find the unique set of three lines which fit these criteria. The slope constant K was determined to be -0.51.

Figure 3 shows the results of applying this graphical method to determine three isopleths representing the fractional grade of VGE and the percentage occurrence of bends pain. It is seen that the isopleths represent both sets of data, i.e., a 19% pain isopleth represents the 1.4 fractional grade isopleth, the 8% pain isopleth represents the 0.71 grade isopleth and the 6% pain isopleth represents the 0.56 grade. The slope constant K in both these sets of data is -0.525.

Figure 4 represents a group of VGE isopleths interpolated over the bubbles gradient data shown in Fig. 2. Also represented are the extrapolated 10 and 20% isopleths. In addition, the table shows the superimposed

TABLE 3. *Analysis of direct decompression experiments*

	Group A						Group B				Group C	
	233/7	230/8	200/7	165/10	150/15	150/10	70/70	70/60	70/50	60/60	30/720	25/720
No. of subj	12	12	12	18	12	2	12	12	11	13	11	9
No. of exposures	12	12	12	21	13	2	12	12	12	13	12	9
No. of subj with VGE	4	7	2	7	7	0	3	3	4	4	9	5
Percent with VGE	33	58.3	16.7	33.3	54	0	25	25	33.3	31	75	56
Avg grade	1	0.96	0.17	0.55	1	0	0.63	0.42	0.58	0.69	1.58	1.06
No. with bends	3	0	0	0	3	0	1	0	1	1	3	1
Percent with bends	25	0	0	0	23	0	8.3	0	8.3	7.6	25	11

Note: Bends have not been found to occur in the absence of VGE.

TABLE 4. *Direct decompression exposures incidence and fractional grade of precordial bubbles*

	Group A	Group B	Group C
Weighted avg of groups	190 ft/9.6 min	67.4 ft/60 min	28 ft/720 min
No. of exposures	72	49	21
% Subj with VGE	38	29	67
Fractional grade bubbles	0.71	0.56	1.40
% Occurrence bends pain	8	6	19

Total no. of exposures: 142; range depth and time: 233/7 min–25/720 min.

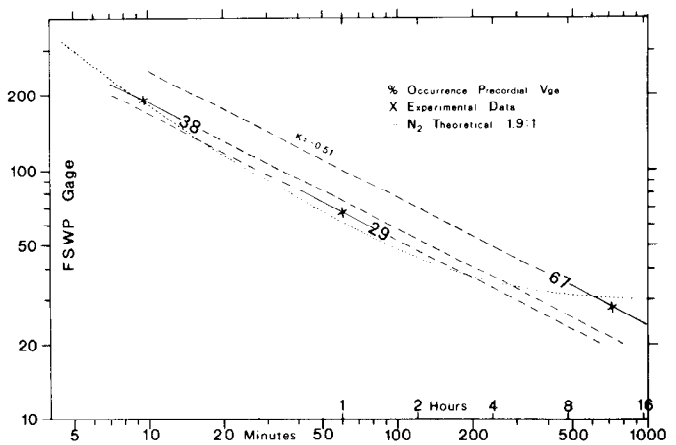

FIG. 2. Graphically calculated isopleths of equal percentage occurrence of precordial venous gas emboli (VGE) (fswp = feet seawater pressure): × 38% = average occurrence for *group A* exposures; × 29% = average occurrence for *group B* exposures; × 67% = average occurrence for *group C* exposures. Dotted line represents theoretical direct decompression limits based on whole-body nitrogen elimination and a 1.9:1 excess nitrogen ratio.

5% bends isopleth and the direct decompression limits recommended by the US Navy.

Peripheral VGE. The regional source of VGE was explored in all subjects displaying precordial signals. The veins most frequently carrying VGE were the subclavian and femoral, but the jugular, brachial, and popliteal were also found with VGE signals after many exposures. Local compression or muscular contraction was useful in regional localization of VGE sources including the neck, upper arm, forearm, thighs, or calf. In all cases where jugular VGE were detected, compression of the neck or face tissues elicited a sudden increase in VGE without accompaniment of cerebral or visual complaints. Although frequently checked for, no arterial gas emboli signals were found. Recompression treatment never produced arterial VGE.

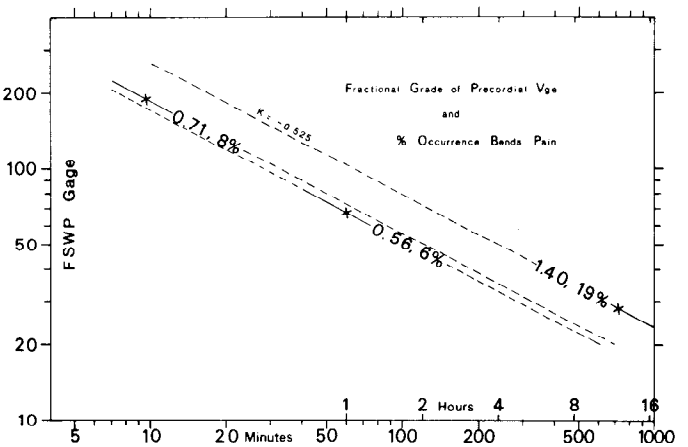

FIG. 3. Graphically calculated isopleths of equal grade of precordial bubbles and equal occurrence of bends pain: × 0.71, 8% = average occurrence of precordial grade bubbles, and bends, respectively, for *group A* exposures; × 0.56, 6% = average occurrence of precordial grade bubbles, and bends, respectively, for *group B* exposures; × 1.40, 19% = average occurrence of precordial grade bubbles, and bends, respectively, for *group C* exposures.

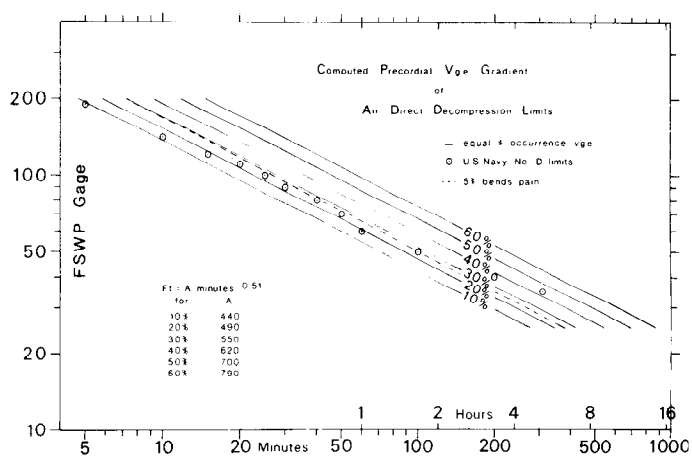

FIG. 4. Interpolated percentage occurrence isopleths for precordial VGE compared with the 5% bends isopleth and present US Navy No-D limits.

Recompression treatment. Oxygen breathing at 30 fsw for 1 h was highly successful as treatment for bends and to dissipate decompression gas emboli. VGE, as well as symptoms, always disappeared within ten minutes after recompression. Surface oxygen was often effec-

tive in dissipating grades 1 and 2 VGE, and usually required 20–30 min. Surface oxygen was not always effective in grade 3 or 4. No doubt, the rapid effectiveness of both treatment methods was related to its immediate application after the onset of symptoms. There was great advantage in being forewarned by VGE signals indicating when and in whom bends was likely to occur.

Open water exposures. The possible differences in results obtained from laboratory chamber and the open water exposures was investigated by comparing responses in six dives to the same pressure and time exposures in both laboratory chamber and open ocean. Six expert and regular divers were subjected to 165 fswp for 10 min in the chamber on the same day. Two weeks later, the same divers made a carefully controlled 165/10 dive on the slopes of Cobb Seamount in the North Pacific Ocean, 270 miles due west of Grays Harbor, Wash.

Table 5 shows that the open water dives produced an increase in both percentage occurrence and average grade of precordial VGE. Three men who did not bubble in the laboratory exhibited VGE after the ocean dives, but did not bend. (One of them with grade 2 was treated preventively with surface oxygen.) The two divers who bubbled in the laboratory increased their open water VGE grade and developed bends pain requiring recompression treatment. *Subject SC* was among the bubble-prone group of divers in the larger laboratory studies with an average grade of bubbles of 2.0. *Subject GA,* who was the most bubble resistant in the larger chamber study group, was the only ocean diver who did not produce detectable VGE. It is possible that the percentage occurrence of VGE and bends is elevated in these data because one of the men is known to be bubble prone, giving the group as a whole a loading factor in favor of bubbles and bends. The differences between the chamber and ocean exposures, however, are considered to be qualitatively correct and support the idea that dive schedules developed from chamber exposures may not be safe unless a factor can be applied to make them more applicable to open water dives.

TABLE 5. *Comparison of chamber and water exposures to precordial bubbles*

	Chamber		Ocean	
	Grade, 0–4	Time, min	Grade, 0–4	Time, min
Diver				
SC	3	10	4*	4
TL	2	5	4†	4
PMcK	0		2‡	20
JN	0		1	10
JE	0		1	12
GA	0		0	
% Occurrence	33		83	
Avg grade	0.83		2.0	
% Bends pain	0		33	

Time refers to the onset of the first detectable VGE following surfacing. *Rx on Table 5. †Rx 45 min on O_2 at 30 fsw. ‡Rx 60 min surface O_2.

TABLE 6. *Power curve fits for various direct decompression limits*

Source	Experimental		Calculated	
	T range	D range	*a*	*k*
US Navy	5–310	35–190	377	−0.43
British Navy	8–135	40–150	403	−0.47
Hawkins et al. (5)	13–38	100–200	1,229	−0.71
Albano (1)	10–44	82–213	1,054	−0.68
Hempleman (7)	55–100	47–200	475	−0.50
Hempleman (goats) (6)	15–250	57–180	549	−0.44
Spencer (20% VGE)	10–720	28–190	490	−0.51

$D = a\,T^k$, where D = depth in feet gauge and T = time in minutes. Coefficient of correlation is greater than 0.97 in all data.

DISCUSSION

Table 6 represents a comparison of our results with the data of others. The coefficient and slope constant for power function curve fits for each investigator's data is shown. The coefficient of correlation is greater than 0.97 in all of the data analyzed and reinforces our assumption that a power curve can truly represent the direct decompression limits.

Though the overall US Navy recommended direct decompression limits are well fitted by a power function, they wander curiously around the computed fit, as do most of the limits found by others. There is no biologically reasonable explanation for deviation from a smooth curve and the deviations may represent either an unsafe or an inefficient limit.

It is remarkable how close the slopes of our data for bends and VGE approximate the $P\sqrt{t}$ relationship noted by Hempleman (7). The tolerance of his goats to bends appeared higher than our data with a $P\sqrt{t}$ value ranging around 700–900, instead of 510–570 for our human bends pain occurrence of up to 29%. This difference is, no doubt, explained by our caution in detecting slight degrees of bends in humans who can verbally complain, versus the necessity with goats of depending on objective clinical signs and behavior changes.

The explanation for the difference in our results and the theoretical predictions of Spencer (10) from nitrogen elimination curves of Behnke (2) lies in the shift in controlling tissue between the short deep exposures and the long shallow ones. The theoretical limits are based on a single ratio of excess inert gas for the entire body, but if we consider as a requirement that lower surfacing ratios are necessary in the longer-half-time tissues, then the limits will be straighter on log-log plots, i.e., become a power function instead of a multiple exponential one, as predicted by nitrogen elimination. The success of Workman's tables (12), no doubt, rests to a great degree on the use of M values, which consider decompression from long dives to be controlled by longer half times. Such an adjustment in the direct decompression exposure limits would tend to change those limits from curvilinear to linear ones as they appear on log-log plot. The linear log-log plot, i.e., limits described by a power function such as $(t^{-0.5})$, does not necessarily mean that the ideal decompression model is a diffusion limited one, for it may be a perfusion limited one controlled by different surfacing ratios in various controlling body tissues.

Practical recommendations. Safe limits for direct de-

compression after exposure to pressurized air require zero occurrence of severe symptoms of decompression sickness but for practical reasons some incidence of VGE must be accepted. We believe a 20% occurrence of bubbles and less than 5% occurrence of mild bends pain (i.e., that which can be relieved by two aspirin) represents a reasonable compromise. The body appears to quickly recover from the effects of nonsymptomatic grade 1 bubbles, but beyond those quantities the source and consequences of the VGE become overriding considerations. There are bends-prone and bubble-prone individuals who, if they dive, should do so only in very shallow water, i.e., less than 30 ft in depth. Candidates for scientific, commercial, or caisson work, as well as the sports diver, should be screened for rejection of those who are the most bends and bubble prone.

Extrapolating the experimentally determined isopleths to 20% VGE, we find the direct decompression limits between 25 and 200 ft to be defined by the equation

$$\mathrm{Ft} = 490\, t^{-0.51}$$

where Ft equals the pressure in feet of seawater gauge and t is the bottom time in minutes. For practical operations, we recommend an alternate easily calculated bottom time for each work depth as

$$\text{minutes} = \left(\frac{465}{\text{feet}}\right)^2$$

Using the simpler squaring function and an a constant of 465, instead of 490, 10–20% VGE may be expected and a safer margin provided for the short deep dives where an error of 1–2 min may be dangerous. These findings and recommendations differ from those in the US Navy Diving Manual by being slightly less conservative in the short deep dives, but more conservative in both the middle-range and the long shallow exposures.

Bubble nucleation. For depths shallower than 20 ft our definition loses its meaning because at these shallow depths the gas bubbles may never enter the circulation and may never grow in size to produce symptoms regardless of the duration of exposure. No doubt, some "threshold" depth must be exceeded in order to produce VGE after very long shallow exposures. We assume that in these shallow ranges any excess inert gas stresses the existing nucleation sites, and if they are small gas pockets, the stress merely enlarges them without their rupture and production of secondary bubbles. At depths greater than our study range, arterial aeroembolism may become the principal problem rather than bends pain.

The present concept of no problems to be expected after any period of time at 30 fswp (13.3 psig) is not confirmed by our results. With only 12 h exposure at 25 ft, we produced one case of moderate bends pain among nine subjects, and this group sustained a 56% occurrence of VGE. Further studies will be required for better definition of saturation and caisson working conditions at these shallow depths.

It is not known whether decompression VGE arise from extravascular nuclei and pass through the capillary membrane to reach the blood, or whether they first form in the capillaries and small venules draining the out-gassing tissues. Of interest here is our frequent observation that air injected into the subepicardial tissues of the anesthetized dog passes immediately into the pulmonary artery and is detected there with Doppler ultrasound flowmeters.

Safety of ultrasound. A valid question exists, concerning the safety of using ultrasound on divers and compressed air workers, as to whether the ultrasonic energies used can produce or promote bubble formation. No definitive study has been made, but theoretical and experimental considerations (11) indicate that present energies used produce no added clinical insult, and that the usefulness of the information for monitoring the safety of the diver, far exceeds any probably deleterious effects which might be expected. The availability of the precordial and peripheral VGE detectors certainly alerted us during our experimental exposures to those subjects which might progress on to bends; as a consequence, early O_2 and/or recompression treatment prevented any serious problems. Ultimately, the decision must be made as to whether or not the diver is safer with the ultrasound information and the remote possibility of ultrasound damage than he is without the knowledge that is forming VGE.

Computation of limiting half-time tissues. The separate half-time tissue tolerances which are related to a given percentage occurrence of VGE can be computed from the direct decompression limits. This is performed by computing for each depth the half-time compartment which is 98% saturated at each limiting exposure time. For example, the half time that is 98% saturated after 10 min at 150 ft is found to be 1.8 min. Likewise, if a 40 ft for 135 min dive is considered, the half time which is 98% saturation after that exposure is 25 min. By plotting a series of these limiting half times on log-log coordinates with the 20% VGE isopleth, we find the following relationships

$$h = 40{,}000\ \mathrm{Ft}^{-2.0}$$
$$\mathrm{Ft} = 200\ h^{-0.50}$$

where h equals the limiting half time and Ft equals the limiting air pressure. These relationships apply to the diving depth ranges between 25 and 200 ft. These limiting half times appear far more conservative than those used by Workman (12), especially for the long shallow exposures. The longest controlling half time for our recommended limits lies between 64 and 100 min for a 20- or 25-ft exposure, and suggests that instead of requiring consideration of longer half times for long D-D exposures, these relatively short h times for decompression exposures will lead to safer and more efficient decompression tables

Limitations of the Doppler ultrasonic methods. The venous gas embolism detection method allows a new objectivity in defining decompression limits, but there is some uncertainty in recognizing grade 1 bubbles. The cardiac motions produce a clutter background signal against which the random bubble signals must be recognized. Peripheral VGE are more clearly heard against the background venous flow signal, but here we are limited by the unpredictable regional source of VGE,

and the fact that all peripheral veins are not available for monitoring. The best technique presently calls for use of both precordial and peripheral listening and development of a degree of skill comparable to stethoscopic auscultation of heart murmurs. Without reasonable skill and diligent monitoring, one cannot say that detectable VGE are not present. The Doppler system used here probably does not pick up the smallest decompression bubbles, but we estimate that it detects bubbles with diameters of 100 μm.

Bends pain does not always develop following the occurrence of Doppler detected VGE. The signals, however, alert one to the possibility so that preventive measures may be taken. In our experience, limited to air exposures, bends pain does not occur in the absence of detectable VGE. Bends pain surely does not result from gas emboli themselves, but is considered to be due to ischemic obstruction in the peripheral tissues from gas bubbles which are not released into the venous return. The Doppler ultrasound detection scheme requires that bubbles be in motion and it cannot, therefore, pick up static bubbles before their release from the capillaries or spaces in which, undoubtedly, they form first. The method has not been applied to long saturation exposures where it may be that tissue bubbles form slowly to produce bends and never enter the venous return to the heart. It still remains to be studied what the relationship is, if any, between VGE and central nervous system bends, visual symptoms, and deafness. Skin bends have not, in our experience, been associated with VGE.

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