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Gas phase formation and Doppler monitoring during decompression with elevated oxygen

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Powell MR, Thoma W, Fust HD, Cabarrou P. Gas phase formation and Doppler monitoring during decompression with elevated oxygen. Undersea Biomed Res 1983; 10(3):217–224.—Subjects in 150 man-dives were precordially monitored with a 5-MHz Doppler ultrasound bubble detector. These measurements were made during a series of dives conducted to test decompression tables that utilize changes of breathing mixtures and a time-average P_{IO2} of 1.9 b during the entire decompression period. Precordially detected bubbles at depth were predictive for limb pain in divers approximately 50% of the time; however, 70% of the divers encountered bends problems in the absence of precordially detectable bubbles. Thus, while the presence of venous return bubbles can be associated with a risk factor for bends, the Doppler method appears to lack the specificity needed for personal dive monitoring. During the oxygen-breathing portions of the decompression individual bubbles could not be detected precordially. The amplitude of the Doppler-detected pulmonary artery flow sound increased, however, and possibly indicated the presence of numerous microbubbles.

Dopper ultrasound
decompression tables
decompression sickness
oxygen
West Germany

Diving research has been conducted for the past several years at the Institute for Aerospace Medicine in West Germany on the development of decompression tables for intervention dives (bounce dives) in the depth range of 50 to 220 m. We report here on dry chamber experiments made for 15 to 60 m in the range of 100 to 220 m. The total decompression time for these dives is shorter than those of other published tables of comparable depth and bottom time.

Doppler monitoring was made during many dives to determine possible gas phase formation in the body at the first and subsequent stops of the decompression and to relate this detectable gas to the probability of decompression sickness. We recognize, of course, that phase transitions in individualized microregions in the body are not possible to localize at the present stage of development of Doppler monitoring techniques in human divers; however, many research groups have used Doppler bubble detectors during diving table development with valuable results. In dives with decompression times of 1 to 2 h there does appear to be a statistical inference that human divers with numerous precordially detected bubbles develop decompression sickness (1–4), and a similar finding is true for large animal subjects (4, 5). Saturation

decompression, or decompressions that are ultimately controlled by the long tissue half times, generally produce few detectable bubbles (6, 7) in the latter stages.

If Doppler detectable bubbles are formed during these decompression procedures, we are interested in the depth of formation. Evolution of a gas phase during the initial decompression period could result in 1) a continued growth of that gas still in the tissues as decompression continues to shallower depths (with eventual decompression sickness) and 2) a need to prolong the tables to eliminate this gas phase. It is reported that bubbles generated between the bottom and first or second stage have also been implicated in CNS disturbances (6–8), although we have never seen bubbles in this range in the series described here.

METHODS

Simulated dives

The dives made by these procedures (9), are characterized by the following points:

1. An initial decompression phase with a pressure reduction ratio of approximately 2:1 for 100- to 200-m bottom depths (He-O_2), all for a 30-min bottom time;
2. A long hold at the first or second stop, generally, for 15 to 25 min (from bottom depths of 100–200 m);
3. Use of heliox in the bottom mix and nitrox for later portions of the decompression;
4. Oxygen partial pressures with a time average of approximately 1.9 b during the entire decompression period. With the decompression times required, pulmonary decompression sickness has not yet been encountered. (10)

Decompression times produced by this method are shown in Table 1 with times for other published tables given for comparison.

A total of 150 man-dives were conducted in a dry chamber; these are summarized in Table 2. Work at the bottom was simulated by a bicycle ergometer with an approximate 100-W load. Two divers were compressed per experiment. Depth range was 100–220 m with bottom times between 15 and 60 min. In the study reported here, a total of 36 different dive subjects participated; all were sport divers. Of these subjects, 14 made 5 or more dives (maximum was

TABLE 1
TOTAL DECOMPRESSION TIMES WITH VARIOUS TABLES

Depth m	Bottom Time, min	Decompression Time, min	Institution	Ref. No.
100	20	146	DFVLR*	**
100	30	181	U.S. Navy	11
125	30	211	DFVLR*	**
150	30	314	DFVLR*	12
150	30	727	Duke University	12
150	30	581	Virginia Mason Sub Sea International	12
150	30	790	Int. Underwater Con.	12
153	30	500	Bühlmann	12
200	30	454	DFVLR*	**

*German initials of West German Institute for Aerospace Medicine. **Results not published.

TABLE 2
SUMMARY OF 150 DIVES IN DRY CHAMBER

Depth, m	No. of Man Dives	Compression Time, min	Bottom-Time Range, min	Oxygen in Bottom Mix, %	Cases of Decompression Sickness
100; 105	10	4	15–30	14.5; 13.9	1
100	4	4	60	14.5	0
120; 130	10	4	17–30	12.3; 11.4	0
120; 130	8	4	48–60	12.3; 11.4	0
140; 150	32	4	17–30	10.7; 10.0	6
150	12	4	60	10	2
160; 170	18	5	30	9.4; 8.9	2
180	14	5	30	8.4	1
200	40	5	30	7.6	13
220	2	5	30	7.0	1

See text for details.

14) with the 3-yr study permitting an interdive comparison with respect to the tendency to develop bends or bubbles, or both.

To accommodate the required rapid sequential gas changes, all breathing mixtures were administered through an SMS-I diving apparatus (Drägerwerke, Lubeck, W. Germany) and oxygen was supplied with an SAA I oxygen-breathing apparatus (Drägerwerke) equipped with a snorkel mouthpiece instead of a breathing mask. During the entire decompression, elevated oxygen partial pressures (time-average = 1.9 b) were used, and inert gas mixtures (helium-oxygen, nitrogen-oxygen) and pure oxygen were sequentially used to effect relatively short but safe decompression times. Despite the numerous mixtures, evidence of counterdiffusion effects (e.g., vestibular decompression sickness) was not found. A typical profile is shown in Fig. 1. Changes were made in the tables during the development phase. For example, the bottom time was lengthened to test the safety of the tables. The bends rate reported in this paper is that occurring during the development phase.

Treatment recompressions were according to U.S. Navy Table 5 (11). Problems with pulmonary oxygen toxicity following treatment were not encountered.

Doppler monitoring

Monitoring of the divers was accomplished with a 5-MHz continuous-wave Doppler bubble detector developed by the Applied Physics Laboratory (APL) of the University of Washington in Seattle (13). The transducer was placed 1 to 2 in. (2.5–5.0 cm) to the left of the midsternal border and adjusted around this area to give the maximum amplitude of the pulmonary artery flow sound. Monitoring sessions on the two subjects of any given dive experiment were initiated at the 60-m stop and at approximately 25-min intervals thereafter. After surfacing, monitoring was conducted at 15- to 20-min intervals for 1 h. Generally, two researchers, who were experienced at bubble detection with this system, listened simultaneously to the original, unfiltered signal.

A tape recording was made for future reference. We noted that the APL instrument produced a clear pulmonary artery flow sound with little cardiac motion background noise.

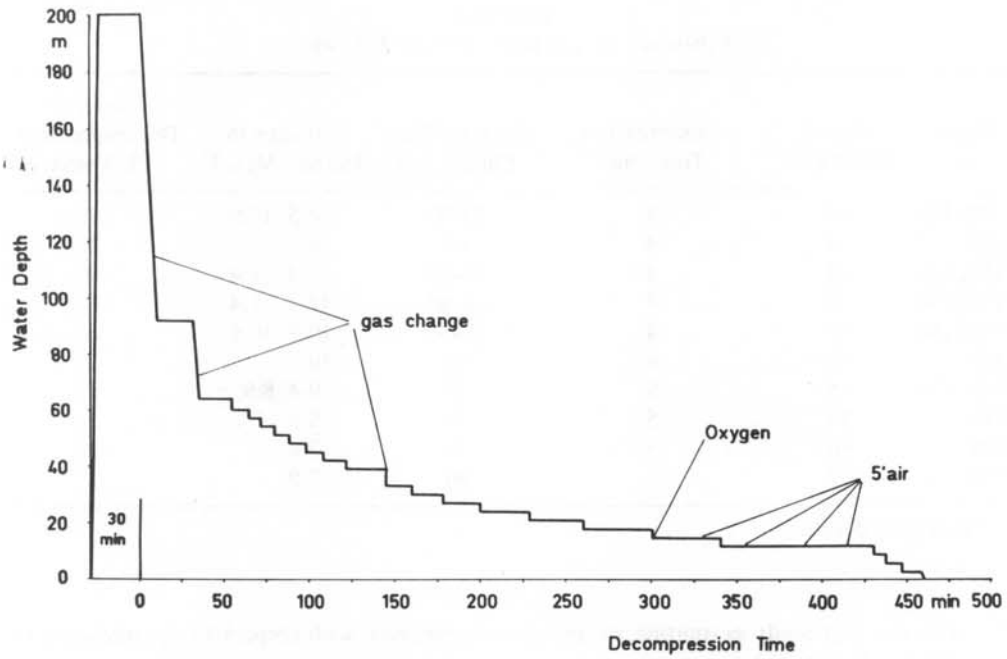

Fig. 1. Profile of a typical decompression.

Divers remained seated during the monitoring periods. Any movement, such as standing at a given time in the decompression phase, produced a short burst of detectable bubbles in a few subjects, whereas none had been heard when they were quietly seated. A persistent bubble-signal increase was not noted in moving subjects (when compared with motionless ones) in cases where such a difference was sought (11 subjects in the 200-m series).

Bubble grades according to the system of Spencer and Johanson (14) were noted and compared to the final outcome of the dive with respect to the occurrence of decompression sickness. Briefly, this system may be divided into grades: Grade 0, absence of any detectable bubbles; Grade 1, occasional bubble signal discernible; Grade 2, less than half of the cardiac cycles contain bubbles; Grade 3, all cardiac cycles contain bubbles; Grade 4, strong bubble signals apparent throughout systole and diastole.

The decompression profile was not varied during the dive if detectable bubbles were found, except for one case in which the final portion was lengthened.

RESULTS

During the development of the tables, 26 cases of Type I decompression sickness (mild joint pain) were encountered in the 150 man-dives. Table 3 shows these frequencies. The percentage of divers experiencing joint pain was slightly higher for the group demonstrating precordially detectable bubbles in Grades 2 to 4. This table gives the results for bubbles and joint pain occurring both at depth and after reaching the surface; 9 of 26 cases of bends (35%) occurred after surfacing. A high percentage of false-positive results (bubbles but no joint pain) as well as false-negative results was encountered during the series. Table 4 gives an analysis of the depth of the first detectable bubbles of the 31 cases in the "with bubbles, Grades 2–4" entry

TABLE 3
NUMBER OF DIVERS WITHOUT AND WITH BENDS, AT DEPTH AND AT SURFACE

Doppler Findings	No. of Cases	Dive Results	
		Without bends	With bends
Without bubbles	119	100 (84%)	19 (16%)
With bubbles, grades 2–4	31	24 (77%)	7 (23%)

See text for explanation of bubble grades. Divers with grade 1 bubbles were also noted; they experienced no problems with limb pain.

of Table 3. Of the divers with bubbles detected at depth, 6 of the 13 later developed joint pain (the only problem that occurred in this dive series).

As is evident from the last column in Table 4, bubbles that were first detected after the subject reached the surface were of little predictive value under this decompression procedure. Of the 18 cases of "first bubbles" on the surface (that is, after leaving the chamber), only one diver developed joint pain. These divers accounted for the majority of cases of the false-positive results.

Table 4 shows that first bubbles were not detected in the depth range of 15 to 1 m. Pure oxygen is breathed in this range. Table 5 shows, with two examples from a bottom depth of 200 m, the effect of inert gas percentage on Doppler-detectable bubbles. These 2 divers developed Doppler-detectable bubbles, one at 45 m and the other at 42 m. Bubble grade, depth, inert gas percentage, and $P_{I_{O_2}}$ are indicated. It is evident that the presence of a Doppler-detectable gas phase in the pulmonary artery is not dependent on the oxygen partial pressure (during decompression) but depends instead on the fraction of the inert gas diluent. With no inert gas supplied in the breathing mixture in the range of 15 m to the surface, the equilibrium diameter of bubbles would be reduced and difficult to detect. Therefore, first bubbles would not be expected in this final decompression phase, and indeed none were found.

A gradual increase in the amplitude of the Doppler blood-flow signal was noted during the final oxygen-breathing portion of the decompression. As the intensity of the signal is very dependent on transducer position and angle, a more definite characterization of the signal was not possible under our experimental conditions. This amplitude increase was noted in more than 75% of all divers monitored. Its physiological significance is not known at the present time, but it may represent microbubbles that cannot be individually distinguishable by Doppler techniques. A similar amplitude increase with an upward shift of the mean frequency was reported by Nishi and Livingstone (15) in decompression experiments with implanted probes on rabbits. In our Doppler study we found no relationship between this amplitude change and the dive outcome with respect to joint pain or other subjective feelings.

TABLE 4
ANALYSIS OF DEPTH OF FIRST DETECTABLE BUBBLES

	Depth of First Detected Bubbles, m			
	60–31	30–16	15–1	Surface
Number of cases	6	7	0	18
Number of joint pain cases*	2	4	0	1

*Usually developed in range of 10 m to surface.

TABLE 5

DOPPLER-DETECTABLE BUBBLES AS A FUNCTION OF INERT GAS FRACTION AND OXYGEN PARTIAL PRESSURE, AFTER 200 M FOR 30 MIN

Depth, m	Inert Gas, %	$P_{I_{O_2}}$, (b)	Doppler Bubble Grade*
Subject DH: no decompression problems			
45	70	1.6	2
30	50	2.0	2+
24	50	1.7	3
20	50	1.5	3
15	0	2.2	0
12	0	2.2	0
6	0	1.6	0–1
3	0	1.3	0–1
0	80	0.2	3
Subject TS: mild joint pain; recompressed			
42	70	1.6	3
39	50	2.5	3
30	50	2.0	3
21	50	1.6	3
18	50	1.2	3
15	0	2.5	3
12 (early)	0	2.2	3
12 (late)	0	2.2	1–2
9	0	1.9	0
6	0	1.6	0
3	0	1.3	0

*See text for Doppler bubble grades.

TABLE 6

JOINT PAIN AND DOPPLER-DETECTABLE BUBBLES IN DIVERS MAKING MORE THAN 5 DIVES

Diver	Cases of Bubbles / Total number of dives	Cases of Bends / Total number of dives	Coincidence*
D.W.	4/12	0/12	0
K.L.	3/8	0/8	0
R.B.	2/7	0/7	0
G.H.	5/6	0/6	0
D.H.	7/14	1/14	1
H.S.	1/13	2/13	0
H.-O.S.	2/6	1/6	0
K.H.	1/5	0/5	0
B.F.	0/5	0/5	0
C.-D.O.	0/5	0/5	0
R.L.	0/5	0/5	0
W.M.	0/7	5/7	0
F.S.	0/6	3/6	0
B.H.	0/9	2/9	0

*Coincidence indicated that both events occurred during the same dive.

DISCUSSION

The conclusions from the data gathered during the experiments to develop these dive tables are that in the bottom depths studied here one might expect a later appearance of decompression sickness with the detection of bubbles from 60 to 16 m (in our case, 6 out of 13; Table 4). In our dive series gas bubbles detected at the surface following decompression appeared to have little prognostic value for joint pain. Of the 19 divers in whom bubbles were first detected at the surface, bends appeared in only one case, and the remaining 18 divers were subjectively well even in cases where numerous bubbles (Grades 3–4) were found.

We noted, with respect to the ultrasound results, that some divers were more prone to form Doppler-detectable precordial bubbles than others. These divers, however, were not the ones most susceptible to decompression sickness; Spencer (16) reported opposite findings in his studies with no-decompression dives. In all subjects making 5 or more dives we have noted that the two groups were distinct, that is "bubbles-prone" but not "bends-prone" and "bends-prone" but not "bubbles-prone." This is shown in Table 6, where the last column (Coincidence) indicates the divers in whom both joint pain and Doppler-detectable bubbles occurred during the same dive. The few cases of bends pain and bubbles appearing together in the same dive (as seen in Table 3) resulted from divers who made fewer than 4 dives. They were possibly the most sensitive, and they later withdrew from the program; a follow-up of the "bubbles-prone, bends-prone" tendency was thus not possible for these individuals. The false-positive results of Table 3 consisted of the group of "bubbles-prone, not bends-prone" divers. It is this lack of coincidence that provides one problem for the control of bends incidence through Doppler monitoring.

An evaluation of the overall effectiveness of a decompression schedule can be made to some degree by means of Doppler monitoring, inasmuch as we noted a tendency of some profiles to produce an increased number of detectable bubbles while at the same time resulting in a general increase in the incidence of joint pain decompression sickness. As pointed out by Masurel (7), Doppler monitoring is the only way of evaluating asymptomatic decompression profiles at the present time.

We interpret our results in light of the pathophysiology of gas phases present in the body during decompression. Following decompression a gas phase develops in the microvasculature that can be detected by Doppler devices when released. It is by no means clear, however, that the gas responsible for limb-pain decompression sickness is intravascular and thus always Doppler detectable; the noxious gas phase may well be extravascular in a joint space or in tendon tissue near a joint. Furthermore, bubbles come from body tissues in a way related to the total tissue mass and its ability to dissolve gas; these bubbles are pooled in the vena cava (17, 18). There is presently no way to use the Doppler precordial technique to differentiate a few bubbles coming from bends-producing tissue (hypothetically in or near joints) from many bubbles coming from muscle or adipose tissue.

While for some dives of short duration there may exist a statistical correlation between the precordial bubble grade and pain-only decompression sickness (4), this correlation should be kept distinct from a cause-and-effect relationship.

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Powell MR, Thoma W, Fust HD, Cabarrou P. Formation de la phase gazeuse et surveillance Doppler durant la décompression avec un mélange riche en oxygène. Undersea Biomed Res 1983; 10(3):217–224.—Des sujets humains ont été suivis au niveau précordial avec un détecteur de bulles ultrasonore à effet Doppler de 5 MHz dans 150 plongées. Ces mesures ont été accomplies au cours

d'une série de plongées effectuées pour vérifier des tables de décompression ou des changements de mélanges respiratoires et une P_{IO2} de 1.9 bar, exprimée en moyenne de temps, ont été utilisés pendant toute la période de décompression. Les bulles détectées en profondeur au niveau précordial ont permis de prédire la douleur dans les membres des plongeurs dans près de 50% du temps. Cependant, 70% des plongeurs ont souffert de troubles de décompression en absence de bulles détectables au niveau précordial. Ainsi, même si la présence de bulles dans le retour veineux peut être associée avec un "facteur de risque" pour la maladie de décompression, la méthode à effet Doppler ne possèderait pas le degré de spécificité requis pour la surveillance de plongée personnelle. Durant la partie de la décompression où un mélange riche en oxygène fut respiré, les bulles individuelles ne purent pas être détectées au niveau précordial. Cependant, l'amplitude du signal du débit artériel pulmonaire détectée par l'effet Doppler augmenta, indiquant possiblement la présence de nombreuses bulles microscopiques.

ultrason à effet Doppler
tables de décompression
maladie de décompression
oxygène
Allemagne de l'Ouest

REFERENCES

1. Powell MR, Johanson DC. Ultrasound monitoring and decompression sickness. In: Shilling CW, Beckett MW, eds. Underwater physiology VI. Proceedings of the sixth symposium on underwater physiology. Bethesda: Federation of American Societies for Experimental Biology, 1978:503–510.
2. Neuman TS, Hall DA, Linaweaver PG Jr. Gas phase separation during decompression in man: ultrasound monitoring. Undersea Biomed Res 1976; 3:121–130.
3. Nashimoto I, Gotoh Y. Relationship between precordial Doppler ultrasound records and decompression sickness. In: Shilling CW, Beckett MW, eds. Underwater physiology VI. Proceedings of the sixth symposium on underwater physiology. Bethesda: Federation of American Societies for Experimental Biology, 1978:497–501.
4. Powell MR. Physiological significance of Doppler-detected bubbles in decompression sickness. In: Pearson R, ed. Early diagnosis of decompression sickness, #7-30-77, Undersea Medical Society, Toronto, May 1977:184–210.
5. Powell, MR. Doppler ultrasound monitoring of venous gas bubbles in pigs following decompression with air, helium, or neon. Aerosp Med 1974; 45:505–508.
6. Gardette B. Correlation between decompression sickness and circulating bubbles in 232 divers. Undersea Biomed Res 1979; 6:99–107.
7. Masurel G. Ultrasonic detection of bubbles in medical aspects of diving accidents. Luxemburg: Commission of European Communities, 1979.
8. Spencer MP, Powell MR. The etiology of convulsions after hyperbaric exposures. Undersea Biomed Res 1977; 4:A23.
9. Cabarrou P, Müller KG, Fust HD, Oser H, Krekeler H, Finkeldey U. Development and testing of heliox dives in excess of 100 meters. In: Shilling CW, Beckett MW, eds. Underwater physiology VI. Sixth symposium on underwater physiology. Bethesda: Federation of American Societies for Experimental Biology, 1978:383–388.
10. Krekeler H, Cabarrou P, Fust HD. Untersuchung zur Vertraglichkeit der Sauerstoffatmung im Bereich von 2,2 bis 2,8 bar (abstr). DFVLR-Forschungsbereicht 78-10, Institut fur Flugmedizin, Bonn-Bad Godesberg, 1978.
11. U.S. Navy. U.S. Navy Diving Manual, Washington, DC: U.S. Dept. of the Navy, 1979.
12. Hamilton RW Jr, ed. Decompression procedures for depths in excess of 400 ft. Undersea Med Soc Workshop Feb. 28, 1976. 271 p.
13. Hougen DP, Belcher ED. Hyperbaric decompression blood flow monitoring. Final Report N0001-69-C-0402. Seattle: Univ of Washington, Appl Physics Lab, APL-US, 7609, 1976.
14. Spencer MP, Johanson DC. Investigations of new principles for human decompression schedules using the Doppler ultrasonic blood bubble detector. Washington, DC: US Navy, 1974. O.N.R. Tech Rep N00014-73-C-0094.
15. Nishi RY, Livingstone SD. Intravascular changes associated with hyperbaric decompression: theoretical considerations using ultrasound. Aerosp Med 1973; 44:179–183.
16. Spencer MP. Decompression limits for compressed air determined by ultrasonically detected blood bubbles. J Appl Physiol 1976; 40:229–235.
17. Powell MR. Leg pain and gas bubbles in the rat following decompression from pressure: monitoring by ultrasound. Aerosp Med 1972; 43:168–172.
18. Powell MR. Gas phase separation following decompression in asymptomatic rats: visual and ultrasound monitoring. Aerosp Med 1972; 43:1240–1244.