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LIMITED SUPERSATURATION VERSUS PHASE EQUILIBRATION IN PREDICTING THE OCCURRENCE OF DECOMPRESSION SICKNESS

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SUMMARY

1. Three experiments have been designed to test the following assumptions underlying the conventional methods for calculating decompression schedules: (a) that there is a critical limit to the supersaturation of a tissue by a gas, and (b) that no gas is formed in the tissues of a subject who does not develop symptoms of decompression sickness.

2. When decompression time was titrated by cutting back upon the time spent at various last stops, it was found that a last stop at a depth of 30 ft was more effective than one at 20 ft which was, in turn, more effective than one at the conventional last stop of 10 ft for each of five schedules. These trials involved fourteen goats and 486 exposures.

3. In a second method, an exposure was selected for each animal from which the 'titrated' stop was now the only one required; but the results were inconclusive since the interval of uncertain diagnosis was now of comparable order of magnitude to the total decompression time.

4. In a third method, symptoms could not be induced in three goats, which had just completed marginally safe decompressions, by exposure to high-intensity ultrasound whose energy would be expected to cause the tissue to exceed any hypothetical metastable limit to supersaturation.

5. It was concluded that it is far more likely that the quantity of gas separating from solution determines the imminence of decompression sickness rather than its mere presence as determined by a critical limit to supersaturation.

6. This is discussed in relation to modifying the diving tables, and the serious implication that conventional schedules are really treating a gas phase in the tissues which does not give rise to symptoms due to the remaining compression afforded by the stopping pressures, rather than preventing the separation of gas from solution in tissue.

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Predictions regarding the likelihood of decompression sickness occurring after residence at high pressure are usually made by calculation. These are almost invariably based upon the concept that the transfer of inert gas to a tissue is a function of the difference in the tension of that gas between the tissue and blood, whether the rate-limiting process is taken as blood perfusion (Kety, 1948), or diffusion, or both (Hills, 1967a).

However, the tissue tension has been generally estimated on the assumption (Schreiner, 1968) that all gas remains in true physical solution during decompressions which do not produce symptoms. Thus the same time functions are conventionally used in dive computations to describe both the uptake and elimination of inert gas by tissues in accord with the original approach of Haldane (Boycott, Damant & Haldane, 1908), and its many subsequent empirical modifications (Hawkins, Shilling & Hansen, 1935; Yarborough & Behnke, 1939; Dwyer, 1956; Workman, 1965). It is also conventional to postulate a critical level (concentration threshold or metastable limit) for the supersaturation of tissue by gas below which it is presumed that no bubbles form because no symptoms are observed.

However, evidence has been collected (Hills, 1966), particularly from aviation medicine (Ferris & Engel, 1951), to suggest that gas may separate out of solution in tissue for decompressions far less severe than any known to produce symptoms. Such data indicate that the zone of supersaturation of tissues by inert gases, implied in conventional calculation methods, may be one of random nucleation similar in concept to the cavitation of liquids *in vitro* and at their interfaces (Hills, 1966). Where there is any formation of the gas phase in a tissue, during any stage of decompression, the equations describing its exchange with blood must change, since only gas in true physical solution can determine the driving force for its transport. This deviation would be greatest in any region sufficiently nucleated to be in phase equilibrium at the imposed ambient pressure, i.e. where all gas in excess of the normal level of saturation has been transferred to the gas phase. In this case the boundary conditions to be applied in the mathematical analysis would differ so greatly from those pertinent to the supersaturation model that very different time functions must result (Hills, 1966), and extensive revision of decompression schedules would be suggested.

Experiments designed to test this basic issue have employed anatomic searches for bubbles in decompressed tissues by physical detection techniques of which the use of ultrasonic beams is currently the most popular (Walder, Evans & Hempleman, 1968). If bubbles are detected by these methods, then there is always the possibility that they were produced, or that their appearance was facilitated by the investigative procedure itself. If bubbles are not detected below the hypothetical critical level of supersaturation, then the presence of the gas phase cannot be ruled out if it forms extravascularly and cannot reach the blood. Moreover, ultrasonics are not likely to detect the irregular masses of gas occasionally seen deep in tissue since these are not likely to resonate and produce the echo enhancement of several orders of magnitude (Horton, 1968), which enables gas to be detected so much more easily when in the form of round bubbles. Even in this form, the smallest bubble which can be resolved by ultrasonic techniques has a diameter of around 100 μ (Buckles, 1968). This is greater than most intercapillary distances and far greater than the value of 2·8 μ predicted theoretically (Hills, 1966) if all the gas separated from solution in a cell were to be coalesced into one bubble.

Any technique employing a physical search for separated gas suffers from the additional disadvantage of the uncertainty in knowing where to try to detect gas, since the tissue type(s)

responsible for marginal symptoms of decompression sickness has not been identified anatomically with any degree of certainty.

Thus, less direct methods seem necessary to determine whether the gas phase is present during a conventional decompression. Moreover, the best assurance that one is dealing with the tissue type responsible for marginal symptoms is to use the occurrence or non-occurrence of marginal symptoms as the gauge for each method employed.

METHODS

First method

One method conforming to the above requirements is based upon the relationships describing the pressure differential, or driving force, for eliminating inert gas from tissue (ΔP_{N_2} for air breathing).

Let us consider an arbitrary stage of any decompression at which the ambient absolute pressure is P, and the critical tissue would be saturated without the gas phase if recompressed to an absolute pressure P_1. If χ is the mole fraction of inert gas in the breathing mixture on a dry gas basis, then the tissue tension of inert gas would be approximately $\chi(P_1-47)$ mmHg, where 47 mmHg is the vapour pressure of water at 37°. This term is included to allow for alveolar dilution of gas by water vapour since χ is defined on a dry gas basis.

If the tissue is truly supersaturated at the ambient pressure P at which the blood nitrogen tension is $\chi(P-47)$ mmHg, then the driving force for tissue desaturation (ΔP_{N_2}) is given by:

$$\Delta P_{N_2} = \chi(P_1-47)-\chi(P-47) = \chi(P_1-P) \qquad (1)$$

On the other hand, for any tissue region which is in a state of phase equilibration, it has been predicted by physico-chemical analysis (Hills, 1966) that:

$$\Delta P_{N_2} = P(1-\chi)+47\chi-k \text{ mmHg} \qquad (2)$$

where k is a small constant of about 133 mmHg. This expression has been shown to hold experimentally for constant volume cavities deposited subcutaneously in rabbits (Hills & LeMessurier, 1969) and for various gas pockets at constant pressure in rats (Van Liew *et al.*, 1965).

The vital difference between the above expressions is that ΔP_{N_2} decreases with P in equation (1), but increases with P in equation (2). This provides a particularly convenient method for determining the thermodynamic state of the tissue, since it suggests that the elimination of nitrogen is faster at greater depth if the gas phase is present, but slower if it is not. In terms of decompression procedures this would mean that for phase equilibration (equation 2), time spent at a deeper last stop (at perhaps 20 or 30 ft) should prove more effective in avoiding decompression sickness than that spent at a conventional last stop of 10 ft.

This provides a simple means of determining the physical state of the relevant tissue(s) by 'titrating', or cutting back to a bends point the time spent at last stops of 10, 20 and 30 ft before 'surfacing' directly. If the total decompression time is less when surfacing direct from 10 ft than from 20 ft, and less from 20 ft than from 30 ft, then the conventional concept of limited supersaturation is correct. However, if the reverse trend is observed, then the gas phase must have been present during the decompression whether symptoms occurred or not. Only those subjects were used who did not develop symptoms when given each of the full schedules

calculated upon a conventional supersaturation basis. It was then known that the constants used in deriving that schedule were safe, even if they were not optimal for those subjects, so that no gas should have separated from solution in their tissues if the concept of limited supersaturation is correct.

The method has been tested (Hills, 1968) and shown to be feasible using the U.S. Navy (1954) decompression schedule for a dive of 60 min at 160 ft. Since the effect of acclimatization was found to be small relative to total decompression time, and insufficient to change the scores between 10, 20 and 30 ft last stops, 'titrations' have not been repeated in the ensuing application of the first method. Also, in these preliminary trials, 20 ft proved to be significantly more effective as the last stop than 10 ft, while there was no clear preference between 20 and 30 ft. However, it might be argued that, since the U.S. Navy diving tables advocate the shortest total decompression times of any published, they may not have included sufficient allowance for contingencies in the values they took for the constants in the conventional calculation methods, or that these may not apply for the particular dive tested. Hence, the experiment has been performed using U.S. Navy schedules for other dives and, for the same dive, using initial stages advocated by other tables similarly based upon the supersaturation concept but employing more conservative constants.

General procedure. Large female goats (110–130 lb), have been used as the subjects for all methods described here, since they are comparable with men in their susceptibility to decompression sickness (Davidson, Sutton & Taylor, 1950) as well as in body mass and percentage of blood volume (Altman & Dittmer, 1964). It is immediately apparent when these animals are experiencing bends since they simply lift a hoof, taking their weight off the limb which is giving pain.

Each goat was compressed individually in a separate pressure chamber, at least 2 days being allowed between successive runs upon the same animal. Air was used as the breathing mixture in all cases. The chambers were intermittently flushed to keep the carbon dioxide partial pressure below 3 mmHg. Immediately any symptoms were confirmed, that animal would be recompressed upon oxygen to an equivalent depth of 50 ft, held there for 50 min, and then gradually decompressed to normal atmospheric pressure over a period of 2 hr. This was found to cure all cases, and appeared to have no effect upon the next run.

Procedure for the first method. Each goat was given a fixed exposure to compressed air followed by a decompression formulated upon the conventional concept that there is a critical limit to supersaturation by gas which is best expressed by a decompression ratio whose values are constant for each theoretical tissue considered. This standard procedure was then repeated in every run—apart from the following modifications to the later stages.

The normal 10 ft-stop was changed by large time intervals (about 30 min) until there were two distinct points—one from which direct 'surfacing' gave bends, and another from which no bends developed within 24 hr of decompression. This interval was then roughly halved in successive trials until it was no longer possible to make a clear diagnosis. The maximum decompression time for a clear bends case (B) and the minimum for a clear no-bends case (N) were then recorded as encompassing the titration point. In most titrations the uncertainty interval was only 2–10 min.

The decompression procedures used for a dive of 60 min at 160 ft included one calculated from the original method of Haldane using a decompression ratio of 2·0 (Boycott *et al.*, 1908), a standard Royal Naval schedule and two intermediate tables. Titrations were not repeated

since the comparison of 10, 20 and 30 ft last stop titrations had not been influenced by the relatively small effect of acclimatization found during the use of the U.S. Navy schedule for this dive (Hills, 1968)

The U.S. Navy tables have been used for other dives, i.e. 60 min at 170 ft and 60 min at 180 ft. Details of the schedules and exposures used are given in Table 1.

TABLE 1. Details of the exposures and corresponding decompression schedules before modification for titration

	Exposure		Decompression format								
				Time at stops (min)							
Procedure	Depth (ft)	Time (min)	Source	70 ft	60 ft	50 ft	40 ft	30 ft	20 ft	10 ft	Total*
I	160	60	Haldane Method	1	4	6	10	16	27	69	135
II	160	60	Royal Navy	—	3	5	20	25	40	50	145
III	160	60	Empirical 'a'	—	—	10	12	21	31	44	120
IV	160	60	Empirical 'b'	—	—	–	20	25	35	50	132
V	170	60	U.S. Navy	—	—	2	15	22	37	74	152
VI	180	60	U.S. Navy	—	—	5	16	19	44	81	167

* 2 min added in all cases for time in decompressing from maximum exposure depth to the first stop.

Second method

'Titration' by reducing total decompression time can be regarded as a process of 'cutting into' the safety margin incorporated into each schedule. Denuded of the uncertainty of this margin in reality, the total decompression time then affords a quantitative index of the effectiveness of the overall format in removing gas from the body.

In the first method, titration was effected by reducing the schedule from the end, so 'cutting into' the safety margin as applied to the last stage only. An alternative means of titration would be one of reducing the theoretical safety factor to all stages simultaneously. However, there are many ways in which this can be done in view of the many constants necessary to compute a conventional decompression—any or all of which can be varied to adjust the margin. There is the additional problem that a small minority of advocates of the supersaturation concept (Hill, 1912; Rashbass, 1955), prefer to express the metastable limit as a tension differential. However, this predicament can be avoided if a dive can be selected which would require only one decompression stop. By all criteria which have been used to define a metastable limit, this stop would be at 10 ft for an effectively infinite exposure to a depth of $(D_\infty + 10)$ ft, where D_∞ is the maximum bends-free depth for that subject. Hence, the titration of time required at single stopping depths of 10, 20 or 30 ft should enable one to determine whether there is a critical limit to the supersaturation of tissue by gas.

The maximum bends-free depth (D_∞) for immediate (no-stop) decompression was determined by subjecting each of four animals, for 6 hr, to depths increasing by intervals of 5 ft with successive runs. A 6 hr exposure time was used since goats are at least 97% equilibrated with respect to nitrogen in this period (Davidson *et al.*, 1950). The results of these 'calibrations' are shown in Fig. 1 for the further four goats (F, G, H and J) used in these titrations of decompression time.

The exposure used for the comparative trials was 6 hr at a depth 10 ft greater than the maximum for no bends (D_∞) determined for that particular animal for no-stage decompression. After this period, the animal would be rapidly decompressed to a depth of 10, 20 or 30 ft, held there for 32 min, and then rapidly reduced to normal atmospheric pressure. The procedure would then be repeated at least 2 days later with the exception that only 16 min could be spent at the same stop if the previous run had proven safe. The time required at a particular stopping depth was thus titrated by repeating the procedure, and successively halving the interval between previous bends and no-bends decompression times, until no clear diagnosis was possible.

The tiration was then repeated for the other two of the three stopping depths—10, 20 and 30 ft. Different orders were used upon different animals to allow for any acclimatization. For the same reason the first of each series of titrations was repeated for some animals.

Third method

If the theoretical basis of conventional decompression schedules is correct, then all gas should remain in true physical solution in a tissue supersaturated to just below the critical value (metastable limit) for cavitation. However, it should precipitate some of that gas if supplied with sufficient energy. Thus a subject who has just received a decompression which is marginally safe should display symptoms when exposed to a high-intensity ultrasonic beam. Symptoms are the necessary manifestation of the presence of the gas phase in the critical tissue type(s) required by conventional supersaturation theories (Schreiner, 1968). On the other hand, if phase equilibration is relevant to the situation, then symptoms should not be induced in such subjects since the high intensity ultrasonic beam cannot precipitate more gas in regions which are already at equilibrium. By this approach it is postulated that the imminence of symptoms of decompression sickness is determined by the quantity of separated gas rather than by its mere presence in the critical tissue type(s) (Hills, 1966).

This is a major departure from previous applications of ultrasonics in this field since here they are used to induce the gas phase rather than to detect it.

Hence this provides a convenient third method for differentiating between limited supersaturation and phase equilibration by techniques which use the occurrence of symptoms to avoid the need to identify the critical tissue type anatomically.

In this series of trials three more large female goats (X, Y, and Z) were 'calibrated' by determining their minimum bend depths (P_∞) for direct surfacing—see Fig. 2. These animals were then exposed to pressures of ($P_\infty - 10$) ft for 6 hr after which they were rapidly decompressed to atmospheric pressure.

Within 30 sec of decompression, one hoof of each goat was held in contact with the bottom of an ultrasonic cleaning bath (Blackstone model SG3) full of water and set at maximum intensity. After 2 days the procedure was then repeated for another hoof until all four hooves of the three animals had been subjected to ultrasonic irradiation.

RESULTS

First method

Fourteen goats were used in this series of trials from which fifty-five titrations were completed from a total of 486 individual exposures. In view of the large number of runs, details of

TABLE 2. Titration of ten goats for last stop depths of 10, 20 and 30 ft after an exposure of 60 min at 160 ft
(This table summarizes the results of 486 exposures)

Procedure:	I						II						III						IV					
Last stop:	10 ft		20 ft		30 ft		10 ft		20 ft		30 ft		10 ft		20 ft		30 ft		10 ft		20 ft		30 ft	
No-bends/ Bends:	N	B	N	B	N	B	N	B	N	B	N	B	N	B	N	B	N	B	N	B	N	B	N	B
Goat																								
K	68	—	41	—	43	38	*95*	*85*	*65*	*57*	*58*	*52*	76	—	45	—	35	—	*92*	*84*	*80*	*74*	*82*	*74*
L	66	—	39	—	35	—	95	—	55	—	50	45	*106*	*97*	*45*	*40*	*40*	*35*	90	84	60	55	35	—
M	66	—	44	39	44	39	95	—	69	64	40	35	76	—	65	—	35	—	84	—	47	—	41	35
Q	68	—	41	—	35	—	95	—	55	—	35	—	76	—	45	—	35	—	84	—	47	—	35	—
R	*66*	*60*	*61*	*56*	*43*	*38*	95	—	65	60	63	58	80	—	77	69	84	74	—	132	80	74	120	114
S	*68*	*60*	*69*	*64*	*50*	*45*	*97*	*95*	*100*	*95*	*60*	*55*	80	—	80	74	77	68	99	89	112	104	—	132
T	—	—	—	—	—	—	*100*	*95*	*103*	*95*	*105*	*100*	78	—	62	57	35	—	116	107	—	—	—	—
U	66	—	—	—	—	—	106	100	57	—	42	37	*95*	*90*	*74*	*64*	*75*	*65*	—	—	—	—	—	—
V	—	—	—	—	—	—	*119*	*113*	*100*	*95*	*70*	*65*	—	120	88	80	69	64	—	—	—	—	—	—
W	—	—	—	—	—	—	*101*	*95*	*100*	*95*	*90*	*85*	—	—	—	—	—	—	—	—	—	—	—	—
Average	67	60	49	53	42	40	100	97	77	80	61	59	83	102	65	64	54	61	94	99	71	77	63	89

N—denotes minimum total decompression time (in minutes) for no-bends; B—denotes maximum total decompression time (in minutes) for bends.

symptoms are omitted. The results are summarized in Table 2 for an exposure of 60 min at 160 ft and in Table 3 for other exposures.

TABLE 3. Titration of four goats for last stop depths of 10, 20, and 30 ft after initial staging according to U.S. Navy tables

Procedure:	V						VI					
Last stop:	10 ft		20 ft		30 ft		10 ft		20 ft		30 ft	
No-bends/Bends:	N	B	N	B	N	B	N	B	N	B	N	B
Goat												
A	167	—	44	—	—	—	—	—	—	—	—	—
B	151	143	140	132	83	76	—	—	—	—	—	—
C	—	—	—	—	—	—	88	80	43	—	44	—
D	—	—	—	—	—	—	104	92	84	78	66	60

N—denotes minimum total decompression time (in minutes) for no-bends; B—denotes maximum total decompression time (in minutes) for bends.

In assessing the relative effectiveness of 10, 20 and 30 ft as the last decompression stop, the following comparisons can be drawn from the above results:

1. The average bends (B) and no bends (N) decompression times lie in the order (10 ft)> (20 ft)>(30 ft) for each of the six procedures tested.

2. Taking the averages for only those combinations of animals and procedures where all six values have been determined, i.e. both N and B values obtained for 10, 20 and 30 ft, they are:

99·5–92·4 min for 10 ft
85·1–78·8 min for 20 ft
68·5–62·5 min for 30 ft

The values from which these averages have been taken are in italics in Table 2.

3. Considering the eighteen chances in the data to compare 10 and 20 ft last stops, e.g. goat L on Schedule IV, 20 ft proved more effective than 10 ft in twelve cases and vice versa in one case (goat S on Schedule IV), while the bends/no-bends intervals overlapped in the remaining five (e.g. goat T on Schedule II).

4. Taking the twenty-four chances to compare 20 and 30 ft last stops, 30 ft was more effective in twelve cases and 20 ft in one case.

5. Considering the nineteen chances to compare 10 and 20 ft last stops, 30 ft was more effective in sixteen cases and 10 ft in two.

While forty of the above comparisons favoured a deeper last stop, three out of the four cases which indicated the reverse occurred for goat 'S' using procedure IV. This animal reverted to the general trend for all other procedures where definite comparisons were possible.

All five of the above methods of comparing the results agree, and leave little doubt that 30 ft is more effective than 20 ft, and that 20 ft is more effective than 10 ft as a last decompression stop for each of the schedules tested. This conclusion is exactly consistent with equation 2 in which a higher value of P, corresponding to a deeper last stop, gives a higher value for (ΔP_{N_2}) and hence a greater driving force for nitrogen elimination at greater depth. This is the exact reverse of the supersaturation condition expressed by equation 1, and must

add strong support for the belief that the gas phase was present during each decompression whether it proved safe or not.

While the above comparisons have been restricted to the last stop, it is interesting to note that procedure I proved more efficient than any other—whether one takes overall average decompression times or those for individual animals (K, L, M, Q, R or S in Table 2). From Table 1 it can be seen that procedure I allocates more time to deeper initial decompression which, once again, would be predicted as more efficient on the basis of equation 2.

Second method

The results are listed in Table 4, together with details of symptoms and the number of days elapsed between the run and the first time that particular animal was used in these trials. The titrations are plotted diagrammatically in Fig. 1, and summarized in Table 5.

TABLE 4. Calibration of four goats for susceptibility to decompression sickness and their titration for single decompression stops of 10, 20 and 30 ft

No.	Goat	Time since first dive (days)	Exposure depth (ft)	Last stop		Symptoms	Onset time (min)
				Depth (ft)	Time (min)		
1	F	0	50	0	—	Bend–RF	43
2	F	5	45	0	—	Bend–LF	30
3	F	7	40	0	—	Nil	—
4	F	12	50	10	32	Nil	—
5	F	19	50	10	16	Nil	—
6	F	21	50	10	8	Bend–RF	3
7	F	26	50	10	12	Nil	—
8	F	28	50	10	10	Nil	—
9	F	33	50	20	32	Mild bend–RF	11
10	F	35	50	10	32	Nil	—
11	F	39	50	20	32	Nil	—
12	F	41	50	20	16	Nil	—
13	F	43	50	20	8	Bend–LF	34
14	F	46	50	20	12	Nil	—
15	F	50	50	20	10	Bend–RF	18
16	F	53	50	30	16	Nil	—
17	F	55	50	30	10	Bend–RF	64
18	F	60	50	30	13	Nil	—
19	F	69	50	0	—	Bend–RH	45
20	G	0	50	0	—	Nil	—
21	G	5	55	0	—	Nil	—
22	G	7	60	0	—	Bend–RH and LH	33
23	G	12	65	10	32	Nil	—
24	G	19	65	10	16	Nil	—
25	G	21	65	10	8	Nil	—
26	G	26	65	0	—	Nil	—
27	G	28	70	0	—	Nil	—
28	G	33	75	0	—	Nil	—
29	G	35	80	0	—	Bend–RF and LF	20
30	G	38	85	20	32	Nil	—
31	G	40	85	30	16	Nil	—

TABLE 4 (*continued*)

No.	Goat	Time since first dive (days)	Exposure depth (ft)	Last stop		Symptoms	Onset time (min)
				Depth (ft)	Time (min)		
32	G	42	85	30	8	Bend–LH	29
33	G	45	85	30	12	Nil	—
34	G	49	85	30	10	Bend–RH	17
35	G	52	85	20	16	Nil	—
36	G	54	85	20	8	Bend–LH	18
37	G	59	85	20	12	Nil	—
38	G	61	85	10	16	Nil	—
39	G	68	85	10	8	Bend–LH	20
40	G	70	85	10	12	Nil	—
41	H	0	55	0	—	Nil	—
42	H	2	60	0	—	Mild bends	30
43	H	14	65	10	32	Nil	—
44	H	21	65	10	16	Mild bends	180
45	H	23	65	10	24	Nil	—
46	H	28	65	10	20	Nil	—
47	H	32	65	10	18	Nil	—
48	H	37	65	20	32	Bend–RF	27
49	H	39	65	20	40	Nil	—
50	H	42	65	20	36	Nil	—
51	H	44	65	30	32	Nil	—
52	H	46	65	30	16	Nil	—
53	H	49	65	30	8	Nil	—
54	H	53	65	30	4	Nil	—
55	H	56	65	30	2	Nil	—
56	H	58	65	0	—	Nil	—
57	H	63	70	0	—	Nil	—
58	H	72	75	0	—	Bend–RF	7
59	J	0	65	0	—	Nil	—
60	J	2	70	0	—	Bend–LF	19
61	J	9	75	10	32	Nil	—
62	J	16	75	10	16	Mild bends	59
63	J	18	75	10	24	Nil	—
64	J	23	75	10	20	Bend–RH	43
65	J	25	75	10	22	Nil	—
66	J	30	75	20	32	Nil	—
67	J	32	75	20	16	Nil	—
68	J	36	75	20	8	Nil	—
69	J	40	75	20	4	Bend–LH	5
70	J	42	75	30	32	Nil	—
71	J	45	75	30	16	Nil	—
72	J	49	75	30	8	Bend–RF	10
73	J	52	75	30	12	Nil	—
74	J	54	75	30	10	Bend–RH	43
75	J	59	75	20	8	Bend–LH	—
76	J	61	75	10	20	Nil	—
77	J	64	75	10	16	Nil	—
78	J	66	75	10	10	Bend–RH	29

N.B. All exposure times at maximum pressure are 6 hr. RF—right foreleg, RH—right hindleg, LF—left foreleg, LH—left hindleg.

It can be seen that goat H acclimatized to the extent that its minimum bends depth has increased by about 15 ft during the trials. Hence the results of this animal can no longer afford a valid comparison. It can also be seen (Fig. 1) that decompression time was about equally effective at 10, 20 or 30 ft for goats F and G, while the results upon goat J indicate that

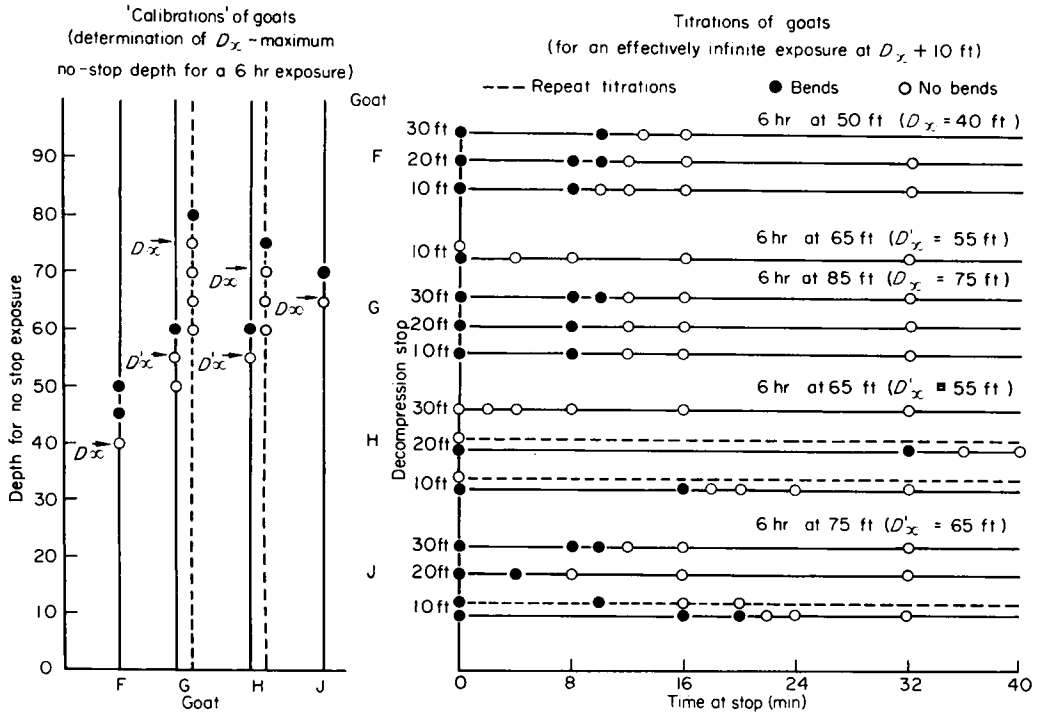

FIG. 1. Results for the determination of the maximum bends-free depth (D_∞) by titration of the pressure to which the goats have been exposed for 6 hr followed by direct 'surfacing'. Also shown are the titrations of decompression time spent at single stops of 10, 20 and 30 ft following exposure for 6 hr to a pressure of ($D_\infty + 10$) ft for the corresponding animal. D'_∞ is the value of D_∞ before the extensive acclimatization noted in the particular cases of goats G and H.

time spent at a last stop of 20 ft is appreciably more effective than at one of 10 ft. However, the fact that only one animal out of four gave a clear differentiation between supersaturated and phase equilibrated states indicates an experimental limitation to the second method. This is set by the relatively short time (4–32 min) necessary for one stop decompression compared with the interval of uncertainty separating clear bends cases from clear no-bends (2–10 min).

Third method

In none of the twelve runs using ultrasonic irradiation (four hooves of three animals) was there any trace of symptoms at any time (Table 6). These results are illustrated in Fig. 2.

TABLE 5. Comparison of titration times at depths of 10, 20 and 30 ft

		Exposure		10 ft stop		20 ft stop		30 ft stop		
		Equivalent depth		Maximum no-bend	Minimum bend	Maximum no-bend	Minimum bend	Maximum no-bend	Minimum bend	Order of titration
No.	Goat	(ft)	(hr)	(min)	(min)	(min)	(min)	(min)	(min)	1–2–3–4
1	F	50	6	8	10	10	12	10	13	10 ft–20 ft–30 ft
2	G	85	6	8	12	8	12	10	12	30 ft–20 ft–10 ft
3	J	75	6	20	22	4	8	10	12	10 ft–20 ft–30 ft–10 ft
4	repeat J	75	6	10	16	—	—	—	—	
5	average J	75	6	15	19	4	8	10	12	
Average titration time										
lines 1, 2 and 3				12·0	14·7	7·3	10·7	10·0	12·3	
lines 1, 2 and 5				10·3	13·7	7·3	10·7	10·0	12·3	

N.B. Data for goat H omitted since it had acclimatized by at least 10 ft before all three titrations could be completed (Fig. 1).

On the other hand, nineteen out of twenty test-tubes of bubble-free distilled water, supersaturated with air to a pressure of 33 ft (1 atmosphere gauge), bubbled when placed in the ultrasonic bath set at the same intensity as used for the goat hooves. This test used twenty out of a

TABLE 6. Determination of minimum bends pressure (P_∞) for three goats and results for their exposure to (P_∞–10) ft

No.	Goat	Exposure pressure (ft)	Time since first trial (days)	Limb subjected to ultrasonics	Symptom	Onset time (min)
1	X	55	0	None	Bend–RH	9
2	X	50	2	None	Bend–RF	21
3	X	45	4	None	Nil	—
4	X	40	7	RF	Nil	—
5	X	40	9	LF	Nil	—
6	X	40	11	RH	Nil	—
7	X	40	14	LH	Nil	—
8	X	45	18	None	Nil	—
9	X	50	21	None	Bend–RF	26
10	Y	55	0	None	Bend–RH	4
11	Y	50	2	None	Bend–LF	3
12	Y	45	4	None	Bend–LF	11
13	Y	40	7	None	Bend–RF	20
14	Y	35	9	None	Nil	—
15	Y	30	14	RF	Nil	—
16	Y	30	18	LF	Nil	—
17	Y	30	21	RH	Nil	—
18	Y	30	23	LH	Nil	—
19	Y	35	25	None	Nil	—
20	Y	40	30	None	Nil	—
21	Y	45	32	None	Bend–RH	17
22	Z	55	0	None	Nil	—
23	Z	60	2	None	Bend–RF	8
24	Z	50	4	RF	Nil	—
25	Z	50	7	LF	Nil	—
26	Z	50	9	RH	Nil	—
27	Z	50	11	LH	Nil	—
28	Z	55	14	None	Nil	—
29	Z	60	18	None	Bend–LF	11

N.B. All exposure times at maximum pressure are 6 hr.

RF—right foreleg, RH—right hindleg, LF—left foreleg, LH—left hindleg.

total of twenty-four identical tubes each containing 10 ml of water which had been kept at 1 atmosphere gauge pressure for 1 week. The water in the other four tubes showed bubbles soon after decompression, so these were omitted from the ultrasonic tests.

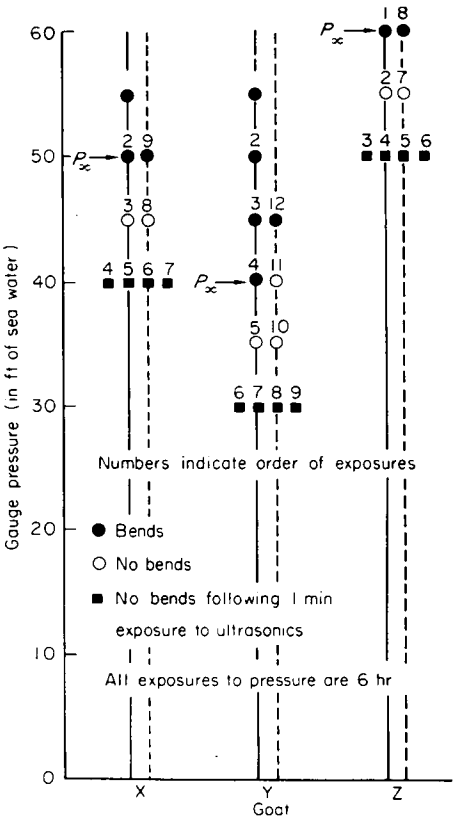

FIG. 2. Results for the determination of the minimum bends depth (P_∞) by titration of the pressure to which the goats have been exposed for 6 hr followed by direct 'surfacing'. Also shown are the results of ultrasonic irradiation of each limb of each animal upon rapid decompression after 6 hr exposures to ($P_\infty - 10$) ft.

DISCUSSION

In view of the ease with which the same high-intensity ultrasonic source was able to induce cavitation *in vitro*, there would seem to have been a very good chance that the same energy supplied to the goat hooves would have enabled the critical tissue(s) to exceed any metastable limit for a phase change—if such a limit exists. Immersion and the good mechanical contact obtainable between the hoof and the vibrating base must have provided an excellent means of transmitting vibrations, via the bone structure and water outside of the limb, to a critical tissue type known to be closely associated with the locomotor system (Ferris & Engel, 1951). There would seem to be two possible explanations for the result:

1. That it is the quantity of gas separating from solution which determines the imminence of decompression sickness, and not its mere presence as postulated in conventional theories. The quantity of separated gas per unit volume of the worst possible (fully nucleated) regions should not be influenced by ultrasonic irradiation, since it is already maximal by virtue of phase equilibration.

2. That the large amount of ultrasonic energy was still not equivalent to the small concentration deficit from the critical degree of supersaturation, although this deficit could be no greater than 10 ft.

While the second would seem unlikely, it may still be argued that a source of even higher intensity may have been necessary to supply the additional energy for the tissue to attain the hypothetical metastable limit. This was not possible since the level of irradiation was already approaching the region in which the ultrasonics could cause pain of their own accord without any previous pressure exposure of the tissue.

The experiment cannot be claimed to be as conclusive as if the outcome had been reversed, and it had been found possible to induce symptoms ultrasonically. However, as they stand, the results must favour the hypothesis that it is the quantity of separated gas per unit volume of tissue, and not its mere presence, which is the criterion for symptoms.

While no conclusions can be drawn from the results of the second method, those of the first leave little doubt that gas can be removed from the critical tissue(s) more effectively at greater depth. This is predicted by equation 2 and can hold only if the gas phase is present during the conventional decompressions tested, since it is most unlikely that any change in the narcotic action of nitrogen could cause the effects recorded. Moreover, it cannot be attributed to increased vasodilation at greater depth, since vasoconstriction is more likely for the increased alveolar oxygen partial pressure.

The above deductions have serious practical implications. If a diver has been on the ocean floor for sufficient time, his critical tissues will contain more gas than the maximum quantity which can be tolerated at the surface without giving rise to decompression sickness—whatever its phase distribution and whichever theoretical criterion is applied to assess the occurrence of symptoms. Hence the excess gas must be removed by gradual decompression which is often termed 'staging'. In order to remove this excess most rapidly, and hence to reduce the wearisome and non-productive time spent by the diver (or caisson worker) in decompressing, it is desirable to maintain the maximum driving force for tissue desaturation (ΔP_{N_2}) appropriate to each depth.

In conventional decompression tables, based upon equation 1, the diver is given the greatest first 'pull' to the surface and thereon kept at the shallowest depth permitted by whatever pressure ratio(s) or tension differential is used to describe the hypothetical safe limit of supersaturation. However, if the deductions drawn from the foregoing experimental work are essentially correct and there is no metastable limit to supersaturation, then equation 2 would provide a better description of ΔP_{N_2}. This would suggest that the excess gas can be removed more effectively at greater depth—which is the exact reverse of conventional reasoning. A wealth of human practical experience is available to support this statement in the form of the empirically-derived techniques of Okinawan pearl divers (LeMessurier & Hills, 1965) who employ initial stops much deeper than advocated in conventional tables, and surface directly from 25–35 ft in appreciably shorter total decompression times.

According to the above argument, standard decompression schedules which prove safe represent a treatment for a latent gas phase which does not become manifest as symptoms by virtue of the remaining compression in accordance with Boyle's law. This should be particularly true of the U.S. Navy (1954) tables where their very long first 'pull' to the surface should cause a large molar quantity of gas to separate from solution in tissue. Moreover, when the above argument was assumed in a mathematical analysis (Hills, 1966, 1969) of thirteen published sets

of diving trials using conventional decompressions, it showed a significantly better correlation of bends cases then predicted by the standard 'supersaturation' methods used to compute those schedules.

While the tables based upon conventional calculation methods may be safe, or have been made so by empirical modification, the above discussion would indicate that the deployment of decompression time in these schedules is far from optimal. The extent of this deviation can be appreciated from a comparison of the very different forms of equations 1 and 2.

While equation 2 advocates deeper staging, the limit must be set by the conditions for creating the gas phase. This, in turn, must be determined by the position of phase equilibration which is also the physical state upon which equation 2 was derived. This condition represents the 'worst possible' from the double standpoint of representing both the maximum volume of gas which can separate from solution and the minimum driving force for the subsequent elimination of that gas. On a random nucleation basis, this condition may apply to only one in many million possible micro-regions of tissue where cavitation could occur, the rest retaining their supersaturation. However, as the 'weakest links in the chain', the equilibrated regions are those for which the decompression should be designed, since they contain the greatest volume of separated gas tending to bend or otherwise distort a nerve ending beyond its pain-inducing threshold.

Optimal formats for the depth versus time profiles have been derived from equation 2 (Hills, 1967b). When tested *in vivo*, these formats enabled total decompression time to be reduced by as much as 35% below that advocated in conventional schedules simultaneously 'titrated'.

Hence the foregoing observations are all consistent with the concept that it is the local quantity of gas separating from solution per unit volume of tissue which determines the imminence of symptoms of decompression sickness and not its mere presence as predicted on the conventional basis of a hypothetical limit to the supersaturation of tissue by gas. However, whether the above deductions are valid or not, the above results have one immediate application in that decompression time now spent at a last stop of 10 ft could be employed more effectively if added to the 20 ft stop. This has the added practical advantage of avoiding a 10 ft stop where the diver is frequently awash when there is any appreciable swell upon the ocean.

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REFERENCES

ALTMAN, P.L. & DITTMER, D.S. (1964) *Biology Data Book*. Federation of American Societies of Experimental Biology, Washington.

BOYCOTT, A.E., DAMANT, G.C.C. & HALDANE, J.S. (1908) The prevention of compressed air illness. *Journal of Hygiene*, **8**, 343–443.

BUCKLES, R. G. (1968) The physics of bubble formation and growth. *Aerospace Medicine*, **39**, 1062–1069.

DAVIDSON, W.M., SUTTON, B.M. & TAYLOR, H.J. (1950) Decompression ratio for goats following long exposure and return to atmospheric pressure without stoppage. Medical Research Council (U.K.) Report U.P.S. 110.

DWYER, J.V. (1956) Calculation of air decompression tables. U.S. Navy Experimental Diving Unit Project NS 185-195, Washington.

FERRIS, E.B. & ENGEL, G.L. (1951) The clinical nature of high altitude decompression sickness. In: *Decompression Sickness* (Ed. by J. F. Fulton). Saunders, Philadelphia.

HAWKINS, J.A., SHILLING, C.W. & HANSEN, R.A. (1935) Suggested change in calculating decompression tables for diving. *Naval Medical Bulletin (Washington)*, **33**, 327–338.

HILL, L. (1912) *Caisson Sickness and Physiology of Work in Compressed Air*. Arnold, London.

HILLS, B. A. (1966) *A Thermodynamic and Kinetic Approach to Decompression Sickness*. Library Board of South Australia, Adelaide.

HILLS, B.A. (1967a) Diffusion versus blood perfusion in limiting the rate of uptake of inert non-polar gases by skeletal rabbit muscle. *Clinical Science*, **33**, 67–87.

HILLS, B.A. (1967b) A thermal analogue for the optimal decompression of divers. *Physics in Medicine and Biology*, **12**, 437–454.

HILLS, B.A. (1968) Relevant phase conditions for predicting occurrence of decompression sickness. *Journal of Applied Physiology*, **25**, 310–315.

HILLS, B.A. (1969) Thermodynamic decompression: an approach based upon the concept of phase equilibration in tissue. In: *The Physiology and Medicine of Diving and Compressed Air Work* (Ed. by P. B. Bennett and D. H. Elliott). Baillière, Tindall & Cassell, London.

HILLS, B.A. & LEMESSURIER, D.H. (1969) Unsaturation in living tissue relative to the pressure and composition of inhaled gas and its significance in decompression theory. *Clinical Science*, **36**, 185–195.

HORTON, J.W. (1968) Maximum sonar echoes from a gas bubble in blood. *Proc. 21st., A.C.E.M.B. (Houston)*, p. 84.

KETY, S.S. (1948) Quantitative measurement of regional circulation by the clearance of radioactive sodium. *American Journal of the Medical Sciences*, **215**, 352–353.

LEMESSURIER, D.H. & HILLS, B.A. (1965) Decompression sickness: a study of Torres Strait diving techniques. *Hvraldradets Skrifter, Norske Videnkaps Akad, Oslo*, **48**, 54–84.

RASHBASS, C. (1955) Investigation into the decompression tables–VI. New Tables. Medical Research Council (U.K.), Report U.P.S. 151.

SCHREINER, H.R. (1968) Safe ascent after deep dives. *Review of Subaquatic Physiology*, **1**, 28–37.

U.S. NAVY (1954) *Diving Manual*. Bureau of Ships, Government Printing Office, Washington.

VAN LIEW, H.D., BISHOP, P.P., WALDER, D. & RAHN, H. (1965) Effects of compression on composition and absorption of tissue gas pockets. *Journal of Applied Physiology*, **20**, 927–933.

WALDER, D.N., EVANS, A. & HEMPLEMAN, H.V. (1968) Ultrasonic monitoring of decompression. *Lancet*, **i**, 897–898.

WORKMAN, R.D. (1965) Calculation of decompression schedules for nitrogen-oxygen and helium-oxygen dives. U.S. Navy Experimental Diving Unit Research Report 6-65, Washington.

YARBOROUGH, O.D. & BEHNKE, A.R. (1939) The treatment of compressed air illness utilizing O_2. *Journal of Industrial Hygiene*, **21**, 213–218.