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Decompression Theory: British Practice

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The complexities of physics and physiology involved in the aetiology of decompression sickness are so great that attempts to formulate detailed quantitative analyses of the phenomena will only succeed by a happy accident. The size, shape and location of the bubble, or bubbles, in mild decompression sickness are still matters for conjecture. Even were such necessary variables clearly defined, the physics of initiation and growth of the gaseous emboli would require a knowledge of nucleation phenomena and bubble growth in biological media which certainly does not exist. Added to these difficulties is the further one of the collection of reliable data. Given these circumstances it is first necessary to examine the generally accepted facts that have arisen from various sources. Hopefully, these data will lead to the establishment of a set of ideas which can be used to calculate decompression procedures in a wide variety of conditions.

BASIC DECOMPRESSION DATA

Decompression sickness can manifest itself in a number of different ways, varying in severity from mild skin itching and rashes to death. The most commonly observed mild form of decompression sickness is a pain felt in or around a joint, and is commonly termed 'the bends'. It has been established that if attacks of the bends can be avoided, then the more serious forms of decompression sickness become extremely rare events. It is proposed, therefore, to examine principally the factors which evoke this form of mild decompression sickness.

Careful records have been kept of all cases of decompression sickness occurring in the United Kingdom at any major compressed air undertakings. It has been established that if men work for periods of time varying between 8 and 12 hours in compressed air at pressures less than 14 psi gauge (31 ft; 1·95 ATA), and are then decompressed back to atmospheric pressure in not less than 2 min, then decompression sickness requiring therapeutic recompression is rare (less than 0·2% of man-decompressions). Data for exposures to compressed air for periods of time longer than 12 hours are very inadequate. Such evidence as has been accumulated is summarized in a report by Hamilton et al. (1973). It would seem that there is some lowering of the decompression threshold pressure from the value of 14 psi gauge (31 ft; 1·95 ATA), to 12 psi gauge (27 ft; 1·8 ATA). This lowering of the threshold no doubt reflects the fact that the tissues of the body take longer than 12 hours to saturate fully with nitrogen gas, or it may also indicate that some form of physiological adaptation to high pressure is not complete in 12 hours. Similar threshold pressure data are available from the use of oxy-helium mixtures (Duffner & Snyder 1958; Hempleman 1967) and although once again there is a paucity of suitable data, it is possible to make two gross statements from the evidence available. First, the threshold value for no-decompression stoppages breathing oxy-helium is significantly greater than that for air, certainly exceeding 33 ft (2 ATA), and more likely to be in

the region of 36 ft (2·1 ATA) for the more sensitive individuals. Secondly, there would not appear to be a great deal of difference between the threshold performance after only 6 hours on oxy-helium as compared with 24 hours on the same mixture. This is in marked contrast to the findings just noted above on air. There are a few experiments using oxygen–argon breathing mixtures (Hempleman 1967) and these would indicate that the threshold pressure on argon is noticeably lower than that on nitrogen. Thus there would seem to be a progressive lowering of the bend threshold pressure when transferring from helium to nitrogen to argon. This could be related to their respective solubilities or diffusion coefficient or some such similar properties, but the evidence available is far too slight to enable any definitive analysis.

A considerable volume of evidence exists defining the bend threshold for subatmospheric excursions (Fryer 1969). It would seem that nearly all normal men can sustain rapid decompression to 0·5 ATA without ill effects. This threshold value may be compared with the value noted above for compressed air from which two points become obvious. First, whatever is provoking decompression sickness it is not described by a constant partial pressure drop nor, secondly, can it be described by a constant pressure ratio. The precise way in which the permitted threshold pressure drop varies with the absolute pressure of the dive has never been properly established. There are, however, some helpful qualitative observations. Most decompression procedures for compressed air workers in use today consist of a fast phase followed by one or more slower phases. The pressure dividing the fast and slow phases of the decompression used to be based upon the Haldane ratio principle. This created a large number of bends, particularly for pressures in excess of 3 ATA. The State of New York Regulations (1922), which specify a rapid pressure drop to half gauge pressure, were very successful in reducing the number of cases of bends, as was the introduction of an intermediate stage by Catton (1967). These observations imply the necessity of a diminution of the permitted decompression ratio with pressure.

Barnard (1975) completed a fundamental set of exposures to oxy-helium breathing and defined the relationship between permitted pressure changes and the absolute pressure. As with air, so with oxy-helium, it was not possible to describe the change in threshold performance in a simple manner, e.g. constant ratio, or constant pressure difference. However, with deference to the original Haldane concept, it must be noted that the data conform more nearly to a constant ratio than they do to a constant pressure difference. For example, 24 hours exposure at a depth of 69 m (7·8 ATA) can be safely followed by rapid ascent to 47 m (5·7 ATA) giving a decompression ratio of approximately 1·4 and a pressure difference of 22 m (3·2 ATA), whereas rapid decompression following 24 hours at 24 m (3·4 ATA) can only be safely followed by ascent to 10 m (2 ATA), giving a decompression ratio of 1·7 and a pressure difference of 14 m (1·4 ATS).

Nearly all cases of bends are preceded by a latent period during which the diver or compressed air worker is generally unaware of any untoward signs or symptoms. This period of virtually trouble-free waiting, prior to the onset of bends, can vary from a few minutes to several hours and the explanation normally advanced is that silent bubbles are generated during the decompression, and that some time is needed for the growth of these small silent bubbles into large painful ones. This explanation may be an over-simplification, as has been pointed out by Hills (1966) and Hempleman (1975), who both draw attention to the possibilities of coalescence or interdiffusion between bubbles as contributory or even dominant factors.

Whenever marginal cases of decompression sickness are promptly treated by recompression then the pain symptoms are nearly always relieved immediately. If the bends pains are not treated promptly then a chronic condition develops which may not respond to recompression and indeed it is not unknown for such cases actually to become worse upon recompression. Just as the evidence of waiting prior to the onset of bends is considered indicative of bubble growth, so the effectiveness of the recompression in removing the pain symptoms in bends is considered as further evidence that separated gas is responsible for this form of decompression sickness. These deductions are further supported by direct evidence

of the presence of bubbles by several workers using ultrasonic techniques (Rubissow & Mackay 1971; Smith & Spencer 1970). Nevertheless, despite this unanimity of views regarding the causative agent in bends, there are some well-established observations regarding sensitivity to bends pain which render quantitative explanations very difficult. Barnard (1967), for example, described several cases of decompression sickness developing at pressures of about 100 ft (4 ATA) where pains could be relieved or exacerbated by pressure changes of only the order of 1 ft (0·03 ATS). The effectiveness of such small pressure changes at such comparatively high total pressures implies a very sensitive mechanism for the provocation and relief of bends pain.

Anyone who has taken part in experimental diving realizes that a dive can result in a persistent low level pain ('niggle') in a joint, and that such a niggle can cause intermittent trouble over a period of 2 or 3 days. An exaggerated example is provided by the Ocean Systems diver who was still sensitive to flight in an unpressurized aircraft several days after completion of his saturation helium dive (Hamilton et al. 1966). It is inferred from such observations that there is a tissue-bubble complex formed as a result of diving and that the physics of this new situation is not representative of the physics of the tissue when the bubble is not occluding the circulation. Once this concept of a tissue-bubble complex is accepted, then grave doubts arise about whether or not such complexes are, to a lesser degree, also influencing the whole decompression problem.

In a double approach to this problem, the present author considered that he had succeeded in showing that in normal diving procedures the rate of acquisition of gas was not the same as the rate of loss of gas. It is necessary to emphasize the phrase 'in normal diving procedures', where large initial pressure changes are involved. It may be possible by such techniques as are advocated by Behnke (1967) and Hills (1966) to avoid the formation of a gas phase. These unequal rates of uptake and elimination of gas were demonstrated by the following experiments.

First it was shown in a population of goats that the time required to equilibrate all relevant body tissues to a constant raised pressure of air was of the order of 6 hours (Hempleman 1967). Secondly, it was shown that if goats were exposed for 6 hours to a pressure of air P_1 and then rapidly decompressed in 150 sec to pressure P_2 where a threshold bend is obtained, then the ratio (r) P_1/P_2 is virtually constant over a large range of P_1 values, but for lower P_1 values r increases noticeably (Hempleman 1967). If it is true that uptake of gas by all relevant tissues is the same as the release of gas, then the following experimental sequence should be quite trouble-free.

1. Expose for 6 hours to pressure P_1.
2. Decompress rapidly to P_2, such that no decompression sickness occurs.
3. Wait 6 hours at P_2 to equilibrate all the tissues to the new pressure level.
4. Decompress to P_3 such that

$$\frac{P_1}{P_2} = r = \frac{P_2}{P_3}.$$

It is found experimentally that procedure (4) is unsafe if the value of r is near to the critical threshold value and the conclusion reached is that the 6-hour period at pressure P_2 does not permit ascent to pressure P_3 because some change has taken place as a result of the first pressure drop from P_1 to P_2. Presumably this change is concerned with the appearance of silent bubbles and these interfere with tissue gas exchange.

Proof that a physical change in the body has occurred as a result of decompression is not given by the above experiments. The results could have been obtained from a change in physiological responses caused by, for instance, breathing raised pressures of air for such prolonged periods. In order to attempt to investigate the physics and physiology of this situation, a second entirely different series of experiments was performed. In outline these experiments were as follows. First, the decompression sickness threshold pressures were obtained for several animals. They were rapidly compressed to pressure P for a time t. At the end of time t they were decompressed back to atmospheric pressure in 150 sec. Certain fixed values of t were selected and the P values were obtained which gave mild threshold bends on return to atmospheric pressure.

Assume now that there exists a set of tissues with half-times of 5, 10, 20, 40, and 80 min. The

choice of these particular tissues is purely arbitrary but this does not affect the validity of the subsequent reasoning. It is now possible, as a result of performing a range of exposure times, to assign permitted ratios to each of these hypothetical tissues for each of the animals. As the threshold 'bends' are all obtained at atmospheric pressure the possibility of altered physiological factors interfering with the experimental results is eliminated.

Following this series of experiments, a third series was performed using the same animals in order to compare each animal with itself. In the third series each individual experiment consisted of a double exposure to pressure of 25-min duration, with a surface interval of 90 min between them. For the first 25-min exposure, the animal was compressed rapidly to pressure P_1 which was a pressure exactly 10 ft (0·3 ATS) less than the threshold bend pressure for that particular animal and for that particular exposure time. The animal was then decompressed back to atmospheric pressure in 150 sec and left at atmospheric pressure for 90 min. There was no risk of decompression sickness from this procedure because, as mentioned, the animal had been exposed to a pressure well below the 'bend' threshold value for this particular duration of exposure. Nevertheless, a large amount of dissolved gas had been acquired in the body tissues as a result. During the 90-min wait at atmospheric pressure all tissues are losing this excess dissolved gas, and it is a simple calculation to follow this process using the Haldane-like tissues with their appropriate exponential time course.

The second exposure consisted, as before, of a rapid pressurization to some pressure P_2 and stay at this pressure for 25 min, followed by decompression back to atmospheric pressure. P_2 is the pressure at which a threshold 'bend' just occurs when the animal returns to atmospheric pressure for the second time. It is now quite easy to calculate the excess gas present in the various tissues at the time of reaching atmospheric pressure following this double pressure excursion. The 'bend' threshold values for the various tissues for the single and double dives can now be compared as in Table 18.1 where goat 34 is given as a typical example of this experimental series. Here the 5-min tissue had shown in the dive (A) of 25 min to 130 ft (4·9 ATA) that it was capable of performing a decompression ratio of 4·81 on return to atmospheric pressure. Anything less than a 4·81 ratio should not affect this tissue, as these values represent its threshold performance. Examination of the double dive data reveals that this particular tissue only performed a 4·08 ratio drop. This difference is very great and must mean that the 5-min tissue played no part whatsoever in the bend produced by the double dive.

In a similar manner, all other proposed tissue half-times can be eliminated.

Six other goats were used in these experiments. Each tissue half-time on every goat showed disparities similar to the example given above. Thus it is quite impossible to use the same reasoning for the uptake of the gas as for the elimination. It is suggested that a tissue-bubble complex is formed during decompression. The sole question remaining is whether such a complex situation forms from every decompression, or whether there are ways of avoiding this.

The findings just discussed refer, of course, to individual animal results. Even if it were possible to avoid the formation of a tissue-bubble complex during the course of a decompression it would still be necessary, for practical purposes, to regard the rate of acquisition of gas as being faster than the rate of elimination of dissolved gas. This arises from the following elementary considerations. In any population there will be those subjects who absorb gas rather more rapidly than others, and there will also be those who eliminate gas more slowly. In order, therefore, to deal adequately with a population of animals or men it will be necessary to introduce unequal rates of uptake and elimination of inert gas in order to cover adequately the envelope of the behaviour of the whole group.

Whenever men are exposed regularly to hyperbaric conditions they seem to develop a marked resistance to attacks of bends in many cases. This effect is particularly noticeable in caisson and tunnel workers (Golding et al. 1960). This form of adaptation or acclimatization to hyperbaric exposures can be seen to occur both when breathing compressed air or oxy-helium mixtures. It is by no means certain that all forms of diving exposures will lead to adaptation.

TABLE 18.1

Examples of bend threshold experiments

Goat no.	Dive no.	Time (in min)	Gauge depth reading ft	Tissue half-times (min)					Tissue half-times (min)					Remarks
				5	10	20	40	80	5	10	20	40	80	
				Total air pressure ft (abs)					Tissue ratios					
	A	25	145	173·2	152·5	117·0	83·9	61·2	5·249	4·620	3·545	2·540	1·855	Bend
	B	50	120	153·0	149·0	132·0	102·5	75·1	4·636	4·515	4·000	3·106	2·276	Bend
	C	90	100	133·0	133·0	128·6	112·0	87·1	4·030	4·030	3·879	3·394	2·639	Bend
33		25	135	163·5	144·2	111·2	80·4	59·2	4·956	4·370	3·370	2·437	1·794	No bend
	D	80 min on surface +												
		25	135	163·5	144·2	113·2	88·1	69·8	4·956	4·370	3·430	2·670	2·115	Bend
	E	360	60	93·0	93·0	93·0	93·0	90·4	2·818	2·818	2·818	2·818	2·740	No bend
Maximum attained in single dives				173·2	152·5	132·0	112·0	90·4	5·249	4·620	4·000	3·394	2·740	
Maximum attained in double dives				163·5	144·2	113·2	88·1	69·8	4·956	4·370	3·430	2·670	2·115	
Difference				−9·7	−8·3	−18·8	−23·9	−20·6	−0·293	−0·250	−0·570	−0·724	−0·625	
	A	25	130	158·7	140·1	108·3	78·6	58·3	4·809	4·246	3·282	2·382	1·767	Bend
	B	50	95	128·0	124·9	111·3	88·0	66·3	3·878	3·785	3·372	2·667	2·010	Bend
	C	90	85	118·0	118·0	114·3	100·2	79·0	3·576	3·576	3·463	3·036	2·394	Bend
34		25	120	149·0	132·0	102·5	75·1	56·3	4·515	4·000	3·106	2·276	1·706	No bend
	D	80 min on surface +												
		25	105	134·5	119·5	95·6	76·7	62·8	4·076	3·622	2·897	2·324	1·903	Bend
	E	360	45	78·0	78·0	78·0	78·0	76·0	2·364	2·364	2·364	2·364	2·303	No bend
Maximum attained in single dives				158·7	140·1	114·3	100·2	79·0	4·809	4·246	3·463	3·036	2·394	
Maximum attained in double dives				134·5	119·5	95·6	76·7	62·8	4·076	3·622	2·897	2·324	1·903	
Difference				−24·2	−20·6	−18·7	−23·5	−16·2	−0·733	−0·624	−0·566	−0·712	−0·491	

The simplest form of non-saturation diving occurs when the diver exposes himself to raised pressure and then without any decompression stoppages returns to atmospheric pressure. Such dives are called 'no-stop dives'. Two groups of workers have attempted to define with controlled experimentation the no-stop diving limit (Hawkins, Shilling & Hanson 1935; Albano 1960). Unfortunately, however, in the light of modern knowledge these attempts can be seen to represent only rough estimates of the normal diving population. No account could be taken of acclimatization or adaptation to regular diving during the experimental series. It is likely, therefore, that the more sensitive results were obtained from the shallow diving data and that as the experiments continued with greater and greater depths the men became more and more resistant. Furthermore, these two sets of results available in the literature are considerably at variance with one another. In these circumstances the results from experiments using large animals are the only available indicators of human performance.

Hempleman (1963) completed a large series of no-stop dives using goats as the experimental subjects and his data will first be used as indicators of possible human performance. It was observed that if the median average value of the no-stop bend threshold pressure was multiplied by the square root of the exposure time to pressure then a constant value was obtained, which remained similar in value until the time of exposure exceeded about 100 min. To find such a simple relationship is extremely encouraging and it is now necessary to enquire whether such a relationship will apply to the human situation.

In view of the conflicting experimental results available, one may resort to the findings from many hundreds of thousands of field trial exposures using the US Navy Air Diving Tables. For a dive to 100 ft the no-stop duration is given as 25 min, yielding a $P\sqrt{t}$ value of $100\sqrt{25}=500$. Reference to the 50 ft no-stop dive durations shows a value of 100 min and for this dive $P\sqrt{t}=50\sqrt{100}=500$. In fact, without elaborating further examples, the relationship $P\sqrt{t}=500$ quite satisfactorily describes the no-stop diving curve as currently used in the US Navy, for all values of t less than 2 hours. For values of t greater than 2 hours it is clear that this relationship becomes increasingly less accurate. Taken to the opposite extreme of the time scale, this relationship predicts that a dive to 500 ft (16 ATA) which was just safe would be of 1 min duration. Reference to experience with submarine escape by the buoyant ascent method using air as the breathing medium will confirm this prediction.

There is such a dearth of statistically valid information using oxy-helium for no-stop diving that it is not possible to test this simple relationship on another gas mixture. In any case, due to the rate with which helium will equilibrate in body tissues, the relationship would only hold for some 30 to 40 min.

It is relevant to note that there are several other forms of mathematical analysis which can be used to fit the no-stop data on both air and helium with an accuracy quite adequate for the available data. The most used of these is an extension of the Haldane multi-exponential analysis which was so successfully exploited by Dwyer (1956) in his calculations for the current US Navy Air Diving Tables. There is no particular merit in any form of mathematical analysis except in so far as it leads to a simple, elegant, and versatile way of encompassing the known facts and accurately predicting some new ones, and also whether it leads to some insight into the aetiology of the particular form of decompression sickness.

It has been known for some years (Donald 1956), that oxygen can play a role in the onset of limb bends, and a recent study by Eaton and Hempleman (1973) confirms and extends some of these findings using both oxygen–nitrogen and oxygen–helium breathing mixtures. Making allowances in the calculations for the presence of high partial pressures of oxygen in brief non-saturation dives is difficult and will almost certainly, in the present state of knowledge, have to be done somewhat arbitrarily.

Saturation, or steady state diving presents in many ways a much simpler problem than discussed previously. Nevertheless, not a great deal of systematic work has been performed using steady state or saturation diving techniques. The reason is not far to seek, and concerns the fact that the collection of data is extremely time consuming

and often hazardous. Sometimes attempts are made to avoid the time-consuming work required to define the rate of saturation of the body with inert gases. This was done, for example, in certain preliminary experiments to the Tektite project, where a method was evolved to achieve total saturation in reduced time in the slowest tissues. For the purpose of this experimentation it was assumed that doubling the desired nitrogen partial pressure gradient will produce the required saturation of the slowest (360 min) tissues within 1 tissue half-time (Edel 1971a). One has the utmost sympathy with attempts to reduce the tedium necessary for defining saturation exposure requirements, but unfortunately any short-cut does need a theoretical basis and, when assumptions of a theoretical nature become necessary, the objective validity of the experiment is immediately endangered. In the complex situation pertaining in diving experiments there seem to be no short-cuts available.

From the point of view of defining the boundary conditions of the decompression sickness problem the most useful experiments are those where the appearance or non-appearance of decompression sickness is used to assess the limits of any experimental procedures. Using these criteria the following two facts emerge. First, as the diving becomes deeper, and particularly at depths greater than around 200 m (21 ATA), there is a change in the nature of the decompression sickness which presents itself from minimal provocation. At lower depths if one exceeds certain rates of pressure decrease, or perhaps takes too great a pressure change on the Haldane-like stage decompression procedure, then the first presenting form of decompression sickness is nearly always a mild limb pain. At greater depths this does not seem to hold true and the new first presenting form of decompression sickness is generally concerned with the vestibular apparatus, or the hearing. Hills (1971) has already drawn attention to the fact that certain pressure-time courses, when breathing air, predispose the subjects to present CNS symptoms rather than simple limb bends. Thus the assumption often made by decompression calculators that if one avoids mild limb pains then one will also avoid nearly all other more serious forms of decompression sickness, is in need of some modification depending upon the type of diving envisaged. Secondly, decompression from saturation or steady state exposures is very sensitive to the partial pressure of oxygen being breathed by the subject. Partial pressures of oxygen not exceeding 0·22 bar will not allow trouble-free decompression procedures from depths greater than about 120 m (13 ATA) however long those procedures. Changing the oxygen partial pressure to that used by the US Navy, namely 0·3 to 0·35 bar oxygen partial pressure, will give somewhat more satisfactory results but still yields far too many cases of mild bends, particularly when decompressing from depths in excess of about 150 m (16 ATA). Changing to 0·4 bar oxygen partial pressure transforms the nature of the diving and yields trouble-free diving in subjects certainly for all depths down to 250 m (25·8 ATA) when using decompressions of the more conservative type, as adopted by the US Navy. When employing even high partial pressures of oxygen it should be noted that Bühlmann completed a number of successful dives at the Royal Naval Physiological Laboratory, Alverstoke, with comparatively short decompression times, from depths as great as 300 m (1000 ft; 31 ATA). In support of a relationship between oxygen partial pressure and total exposure pressure is the work of Berghage, Conda and Armstrong (1973). Whilst acknowledging that their results were obtained using small animals as experimental subjects and that it is unwise to transfer such results 'in toto' to the human situation, there is nevertheless a strong indication that, in this particular case, the human and small animal results have a large measure of qualitative agreement.

Although divers can be satisfactorily protected from attacks of acute decompression sickness, and can be given effective therapy for these disorders, providing treatment is prompt, it is unfortunate that a number of cases of dysbaric osteonecrosis do nevertheless seem to occur. Fortunately, the vast majority of these osteonecrotic lesions are asymptomatic but the avoidance of these bone changes is of prime concern to anyone concerned with decompression procedures. The months which must elapse before detection of the lesions and the difficulty of obtaining sufficient reliable 'follow-up' data, over a period of several years,

are proving formidable barriers to progressing towards an understanding of this disorder.

One or two facts are, however, quite clear. Osteonecrosis can follow from only a single exposure to raised air pressure (Swain 1942), although, not unexpectedly, the chance of developing a lesion is related to the number of entries into hyperbaric conditions (Walder 1970). Some men who have had several attacks of mild decompression sickness are quite free from osteonecrotic lesions, whereas others who have had no overt attacks of bends nevertheless develop bone lesions.

A great mass of data is available concerning the non-appearance of decompression sickness, for instance in excursion diving from saturation or steady state conditions (Bornmann 1971). Whilst such facts are unhelpful for defining the boundaries of the decompression problem they do form a gross first-order test of any quantitative ideas.

A summary of the diving data

A general summary of the major points which have just been outlined will now be given prior to an attempt to see whether one can make a quantitative analysis.

1. There are many forms of decompression sickness. The simplifying assumption will be made that if mild bends pains can be avoided then all more serious forms of decompression sickness will become extremely rare. It is noted that there are some exceptions to this general rule, particularly when deep oxy-helium dives are being undertaken.

2. Following prolonged exposure to pressure it is observed that the bend threshold is greater when breathing helium–oxygen than nitrogen–oxygen (air) and greater again than argon–oxygen.

3. Prolonged exposures to pressure, followed by rapid decompression to a new just-safe level are feasible up to great pressures, at least 350 m (36 ATA). The relationship between the pressure of exposure and the pressure to which one decompresses just safely is not a simple one.

4. In most cases of mild bends there is a trouble-free waiting period prior to the onset of bends which can vary from a few minutes to several hours.

5. All mild bends cases will resolve if treated promptly by recompression.

6. In a population there are people who absorb gas rapidly on compression and there are those who eliminate gas more slowly on decompression. There is thus an asymmetry in the uptake and elimination of inert gas when viewing the population as a whole during the compression and decompression phases of a dive. Equally it has been established that using normal diving procedures there appears to be an asymmetry between the uptake and elimination of gases during compression and decompression procedures even for the individual.

7. Certain forms of diving can lead to adaptation or acclimatization which increases the resistance of the divers to attacks of mild decompression sickness.

8. The no-stop dive data using air as a breathing medium can be simply explained on a $P\sqrt{t}$ relationship providing t does not exceed about 100 min.

9. The outcome of decompression from a saturation or steady state dive, is very sensitive to the oxygen partial pressure being breathed. This sensitivity is also related to the pressure of the dive.

10. Osteonecrosis occurs as a result of hyperbaric exposures in a small proportion of men. Men without any history of overt attacks of the bends can, nevertheless, show osteonecrotic lesions; conversely, men who have had numerous attacks of the bends are sometimes quite free from any such bone disorders.

11. A mass of 'safe' diving data is available and useful as a gross preliminary test of ideas, e.g. excursion dives, surface decompression, etc.

A PHYSICAL MODEL FOR CALCULATION PURPOSES

All systems of calculation seem to have only one point of common agreement, namely that a bubble (or separated gas) is the primary aetiological agent causing limb bends. The following further hypotheses regarding the formation of the gas phase are quite tenable in the present state of knowledge, and it would be an unprofitable exercise to defend one rather than another.

1. A gas nucleus is always present, either in a 'crack', or due to tribonucleation phenomena, or

perhaps vortices in the heart. No decompression can, therefore, be undertaken following a dive according to this hypothesis without a gas phase being present in the tissues or circulation.

2. There is a small pressure drop permissible without gas formation due to the partial unsaturation of tissues caused largely by the oxygen content of the gas being breathed. Thus, provided the decompression is kept within certain limits it is possible to avoid forming a gas phase in the body, and this should lead to a much safer decompression.

3. There is a permissible decompression ratio. Anything less than this permitted decompression ratio has a vanishingly small risk of causing decompression sickness. Any greater ratio has a near certainty of causing decompression sickness. Most decompression schedules in use today depend upon this idea.

Coupled with these three concepts are two major dichotomies of view: namely that bubbles are intravascular or that bubbles are extravascular—and there are sub-divisions of these opinions.

4. The relevant bubbles are intravascular.

(a) The intravascular bubbles are first formed in the arterial circulation.

(b) The intravascular bubbles are first formed in the venous circulation.

5. The relevant bubbles are extravascular.

(a) The extravascular bubbles are formed in interstitial fluid.

(b) The extravascular bubbles are formed in intracellular material.

Added to these various aetiological pathways are two other possibilities:

6. The rate of elimination of dissolved gas from the relevant tissue is largely dependent upon the circulation.

7. The rate of elimination of the dissolved gas from the relevant tissue is largely dependent upon the rate of diffusion of the dissolved gas through the tissue spaces.

There are several paths through the hypotheses mentioned above and these represent only the initial difficulties for anyone attempting a physical picture of events leading to an attack of the bends. Many divergent views are held by highly intelligent and well-informed workers in this field. This serves to illustrate the fact that if anyone found an accurate physicomathematical analysis, it would be an astonishing piece of good fortune. What has happened in the history of the development of attempts to formulate a theoretical structure from which to calculate decompression procedures is that certain basic facts have been used as a reasonable foundation for an approach, and this approach has then been tested in areas of diving not covered by the original observations. The approach has been seen to be only partially successful and has been modified in the light of the new evidence and the cycle of refinement started again. When firm theoretical concepts are eventually established by these methods it will be possible to re-examine the plethora of possibilities regarding the aetiology of decompression sickness. Meantime, hopefully someone will develop a technique, e.g. ultra-sound, which will unequivocally establish where the symptomatic bubble is, and then accurately monitor its growth and resolution, in a large variety of differing circumstances.

The point to be made is that a precise knowledge of the aetiology of the bends is not a necessary prerequisite for successful decompression table calculations. The basic diving data must, of course, be incorporated into any system of calculation which is employed, but a knowledge of the underlying physiology involved can largely be ignored. The author would now like to advance the following scheme for calculating decompression procedures which has met with considerable success and is sufficiently versatile in overall concept to allow further modifications with the advance of more reliable data. There are two principal assumptions and the quantitative aspects of these assumptions will first be described as they relate to the calculation of air decompression procedures. In the first assumption it is considered that if after a long period at P_1 a man is to be decompressed rapidly to some lower pressure P_2, then the permitted decompression ratio $P_1/P_2=r$ varies with P_1 in the following manner.

$$\frac{P_1}{P_2} = r = \frac{27{\cdot}5714}{P_1+12{\cdot}407}$$

where the pressure is measured in bar (ATA approx.).

Thus as P_1 increases, the permissible pressure ratio r decreases according to the above relationship. This expression attempts to cover quantitatively the well-established observations described previously. It predicts that after very long exposures to compressed air at an absolute pressure of 1·92 bar, rapid decompression to an absolute pressure of 1 bar can be undertaken without any ill effects. In the light of the latest evidence from the Tektite exposures, this value of 1·92 may be slightly too high. There must be diminution in the permitted ratio as the pressure increases. For man the problem is open to some doubt as to how much diminution is necessary, because there is a lack of appropriately controlled experimentation, but with animals the matter is beyond dispute and a cut-back in this ratio with increase of pressure is easily and quantitatively demonstrable. It is estimated by analogy from large animal experiments that for man the permitted ratio will change approximately from 1·9 to 1·6 over the range of gauge pressures of 0·96 bar to 3·4 bar. Once again it must be noted that this is just an estimate from animal work and it may be necessary to consider even more cut-back in the permitted decompression ratios as experience dictates. The second assumption concerns the observation that the shape of the pressure time curve for the onset of decompression sickness seems to be the same as that of the curve for the uptake of nitrogen by the whole body, and this in turn is the same shape as a curve describing the quantity of gas diffusing into a slab of material when only one of the faces is exposed to the pressure of gas. Thus there is an exact and well-known physical analogy to describe the uptake of gas curve, or the acquisition of danger curve. The particular equation being used is:

fractional saturation

$$= 1-\frac{8}{\pi^2}\left\{e^{-Kt}+\frac{1}{9}e^{-9Kt}+\frac{1}{25}e^{-25Kt}+\cdots\right\}$$

where $K=D\pi^2/4b^2$, D=diffusion coefficient, and b=thickness of the slab exposed on one side.

This is a solution to Fick's law

$$-\frac{dc}{dt} = D\frac{d^2c}{dx^2}$$

where c is the gas concentration (or partial pressure (at some distance x inside the slab, for the particular conditions of thickness b, the slab being initially free of nitrogen and then having one face suddenly exposed at time $t=0$ to a fixed raised pressure of nitrogen or other gas.

This equation has been put into a computer for the particular value of $K=0{\cdot}007928$, which value gives a 30% saturation after 22 min and was considered to be the most realistic fixed point on which to base the shape of this curve. Using this K value implies that in about 9 hours the body is 99% desaturated with gas. This order of time may not be enough in the light of new facts and the K value may need adjusting in the not too distant future from the value which has just been given. As will become apparent in the discussion which follows, the success of these systems of calculation is such that, for the moment at any rate, drastic changes are not warranted.

Bearing in mind that decompression tables are meant to protect the more sensitive men in a large population of healthy divers, the following scheme was evolved. It was assumed that the rate of uptake of gas is 1·5 times faster than the rate of elimination. That the uptake of gas and the elimination of it by a group of healthy men will not be symmetrical is beyond dispute but the assessment of the factor as being 1·5 is, of course, just an estimate and like several other of the previous estimates may need modification in the light of experience.

As an example of the type of calculation involved let it be supposed that one has to decompress a tunnel worker who has been exposed to a gauge pressure of 50 psi, i.e. 3·4 bars for 30 min. In view of the fact that the bar seems to be becoming accepted as a major international pressure unit this will be used throughout in the calculation but, of course, the principle is exactly the same whether feet, metres of seawater or psi gauge are involved.

After 30 min the worker's body is 42·9% saturated, this value being obtained by reference to the basic equation, and thus the quantity of gas in the relevant tissue(s) (or slab analogy) is the same as if he had been at a gauge pressure of 1·46 (i.e. 3·4 times 42·9%) bar for an indefinitely long period. The permitted ratio which can be used with this quantity of gas is obtained from assumption

(1), given above, and is 1·85. Hence the first stage of the decompression is at an absolute pressure of 2·46 divided by 1·85 = 1·3 bar or a gauge pressure of 0·3 bar. For a safe decompression, therefore, the fast phase should end at a gauge pressure of 0·3 bar. It is convenient to place the decompression stages at 0·2 bar intervals and in this case would mean stopping the fast phase at a gauge pressure of 0·4 bar. The calculation continues as follows. The exposure to a gauge pressure of 3·4 bar for 30 min followed by a rapid drop in pressure to a gauge pressure of 0·4 bar may be regarded as equivalent to adding together two exposures, one of which carried on indefinitely absorbing gas at a gauge pressure of 3·4 bar, followed after 30 min by a negative exposure of 3·4 minus 0·4, i.e. gauge pressure of 3·0 bar, which also carried on indefinitely. Ultimately the two curves would become asymptotic and the gas left in the body would be at a gauge pressure of 0·4 bar; but it is only necessary to wait long enough to make the next pressure drop to a gauge pressure of 0·2 bar. At a gauge pressure of 0·2 bar, the permitted excess gas pressure is easily calculated from assumption (1), as 1·26 bar. The superimposition of the ingoing and outgoing curves is continued until it can be seen that an excess gas pressure of 1·26 bar is left. At this point a drop to a gauge pressure of 0·2 bar is permitted. The duration of this stage is calculated by superimposing a pressure drop of 0·2 bar (negative) curve on the existing two curves. The calculation is continued in this simple manner until it is safe to reach atmospheric pressure with an excess gas pressure of 0·92 bar remaining. It must be borne in mind when doing these decompression calculations that the fast graph is used for estimating gas uptake and that at the moment of decompression there is a discontinuity when the slow elimination graph is employed.

All of this simple but somewhat tedious mathematical analysis is best left to the computer. Merely by the insertion of appropriate factors it is possible to calculate quite easily any decompression procedure in any units. Accordingly a set of decompression procedures for use by caisson workers has been in use since 1966 with pounds per square inch as principal units of pressure, whereas since 1968 Air Diving Tables using feet of seawater with the final stop placed at 20 ft (1·6 ATA) have been under active testing. More recently in 1972, as a result of experience with these 1968 Air Diving Tables, a revised metric version, using 5 m (0·5 ATS) increments of depth for decompression stages has been calculated and issued for use.

The decompression ratios estimated from assumption (1) are based on experiments using air as the breathing medium, and consequently in the calculations air was regarded as a single gas. This did not lead to a sufficient cut-back in the permitted decompression ratio and an allowance in the calculations is now made for high oxygen partial pressures. Whenever the partial pressure of oxygen in air (or mixture) exceeds 0·6 bar then it is considered that significant amounts of dissolved oxygen are present in the tissues and that there is an increased decompression risk. This is estimated by adding 25% to the dive depth, and proceeding with the calculations as just outlined using assumption (1). An oxygen first stop depth is thus obtained, and 5 min is spent at this depth to allow for metabolic usage of the excess dissolved oxygen gas. Following this 'oxygen stop' the calculations proceed as outlined above.

Calculation of oxy-helium tables follows exactly the same principles but the real difficulty with these calculations is that assumption (1), concerning the permitted ratios to be used at varying depths, is only obtainable after considerable diving experience and such experience has not yet been gained. Consequently, as with the air diving, the relationship between permitted ratio and dive pressure is somewhat uncertain.

TESTING DECOMPRESSION SCHEDULES

One can examine the literature of compressed air diving and caisson work and read such statements as 'only 10 cases of bends occurred from 10,000 exposures, giving an incidence of 0·1%'. Such statements unaccompanied by a proper further analysis are valueless and misleading. In the first place the nature of the exposures to pressure should be clearly stated. For example, 10 exposures to 30 psi gauge (67 ft; 3 ATA) followed by 10 bends and then 9990 trouble-free exposures to

12 psi gauge (27 ft; 1·8 ATA) would give the results quoted above, but there is very obviously something amiss with the first decompression procedure despite the very low overall figure. For diving tables and sometimes for decompression meters, great claims are made with regard to success in protecting the diver from mild decompression sickness. Almost invariably the type of diving which forms the basis for these claims is very largely of the no-stop variety. Dives such as 15 min at 90 ft (3·7 ATA) or 40 min at 60 ft (2·8 ATA) dominate the statistics with thousands of results whereas exposures of 1 hour duration at 160 ft (5·8 ATA), where the adequacy of the decompression procedures is really revealed, represent a minute proportion of the overall numbers.

Assessing decompression procedures from field trial results is an extremely difficult problem. Very rarely in practical circumstances does a diver proceed exactly to the limits of a particular decompression profile. It would be rare to find a diver exactly at 100 ft, for example, for exactly 20 min and decompressing exactly along the procedures laid down for this particular dive by any recommended decompression schedule. Thus the adequacy of the decompression routines is never properly evaluated in actual diving circumstances. It is also well known that divers add on safety factors in both time and depth in areas of the decompression tables which they have discovered from actual practice are not offering them adequate security. Now whilst this is a readily understandable and indeed quite sensible practice for the working diver, it is not conducive to reliable statistical evidence for analysis by those principally concerned with assembling objective data.

Bearing these points in mind, it is now necessary to examine how the tables based upon the above calculations have fared when put to stringent controlled testing. With regard to air diving, using the 1968 calculations whereby the diver was decompressed in 10 ft (0·3 ATS) increments except for the final stage which was placed at 20 ft (1·6 ATA), the following has now emerged. Both Royal Navy and Royal Canadian Navy divers were tested for 1 hour at 160 ft (5·8 ATA) on the Air Tables issued either by the US Navy or the Royal Navy, and a disappointingly high percentage of bends were obtained on both these air diving schedules. Following these trials, the 1968 Air Diving Procedures were tested by RN divers and there were no cases of decompression sickness either in the laboratory dry chamber tests or in the subsequent wet tests in the laboratory and in the open sea. Random trials have since continued over a period of 4 years using acclimatized and unacclimatized personnel for the tests, and there has been an increase in confidence that these particular profiles represent a distinct advance on the currently available Air Diving Tables. However, when dives of the order of 1 hour at 200 ft (7 ATA) were performed at sea on 10 different men, there were no cases of bends, but subsequent testing in the dry chambers in the laboratory yielded one or two 'niggles' and one mild bend, which occurred upon surfacing. This is an indication that once again these ideas have an area of usefulness but that when pushed to extremes they will not offer adequate protection for the diver. From a practical standpoint, this limitation is of no great moment because a survey of all diving activity showed that only extremely rarely were such dives as 1 hour at 200 ft (7 ATA) ever undertaken and indeed 200 ft is now being regarded as beyond the suitable limit for air diving due to the increased respiratory problems encountered at this depth when attempting heavy exercise. A small modification was, nevertheless, made to the 1968 tables before issuing the metricized version in 1972 and this was an acknowledgement of the fact that the no-stop curve calculated for the 1968 tables was somewhat overconservative. For example, the no-stop dive at 100 ft (30 m; 4 ATA) was given as 15 min and this can be seen to contrast quite markedly with the 25 min given in the USN no-stop dive for this depth. Consequently, a change was made to bring the no-stop diving limits close to the RN diving table limits, as at present published in the *RN Diving Manual* and, for example, at 100 ft (30 m; 4 ATA) this would be 20 min. Other than this minor modification dictated by experience, the 1972 calculations are on exactly similar lines to the 1968 tables.

Regarding the applicability of these calculations to caisson work, there is little doubt that they represent a good step forward and that the bends percentages over most of the pressure–time combinations encountered in tunnel and caisson work

have been lowered. Nevertheless, there are clear indications that if the working pressure is sufficiently great and the exposure time sufficiently long then these ideas are inadequate to offer satisfactory protection to the tunnel worker. By 'unsatisfactory protection' is meant a bends percentage not in excess of 2·0%. It is difficult to assess the true reason for the inadequacies which appear, but undoubtedly working for several hours at pressures of 3 ATA or more exposes men's lungs to raised pressures of oxygen which may have some effect upon the subsequent decompression, although, as noted earlier, it is also probable that the K value being used for the elimination of nitrogen is too large and is leading to the view that nitrogen is eliminated from the body rather more rapidly than turns out to be the case in a proportion of the population.

With regard to the incidence of dysbaric osteonecrosis it can be said that after 7 years, and 42 000 entries into compressed air at pressures in excess of 14 psi gauge (1·9 ATA), using these decompression procedures there have been no cases of this disorder of sufficient severity to warrant surgical intervention. Whilst this is, of course, very encouraging, it will be some years yet before one can declare unequivocally whether serious forms of dysbaric osteonecrosis are non-existent using these tables. It is somewhat unfortunate that similar large-scale systematic observations are not available from prolonged and exclusive use of other air decompression tables, as it would then be possible to make objective statements regarding the effectiveness of various pressure-time courses. The incidence of dysbaric osteonecrosis has now become one of the major measures of the effectiveness of decompression tables.

Regarding helium diving, all schedules for short-term diving down to 500 ft (16 ATA) depth for 15 min duration have been given very thorough laboratory and seagoing tests. Once again it is clear that a steady inadequacy is apparent as the dives progress to greater depths. For example, a typical set of recent tests show 0 bends from 98 dives at 200 ft (7 ATA), 2 mild bends from 115 dives at 300 ft (10 ATA) and 4 mild bends from 22 dives at 450 ft (14·6 ATA). The original tests, performed to establish these schedules as suitable, gave 0 bends at either 300 ft or 500 ft (10 or 16 ATA). It is now apparent, however, that for these early tests the divers had been diving regularly and were indubitably fully 'acclimatized' or 'adapted' to helium diving of this short-duration type. Furthermore, the tests were conducted in the obvious manner, by commencing at the shallow depth and gradually working deeper. This meant that fully 'worked up' men were tried on the more difficult 450 and 500 ft (14·6 and 16 ATA) schedules. Modifications are now in hand to make the deep schedules more suitable for general diver use. Nevertheless even these deep schedules offer a very good security to the diver at very considerable depths, and the worst result from their use seems to be a mild limb pain occurring at, or close to, surface pressure.

AETIOLOGY OF 'THE BENDS'

It is possible, by observing the various decompression schedules which have been used with varying success to decompress divers and caisson workers, to reach preliminary ideas on the aetiology of the bends.

If one examines the no-stop air or oxy-helium diving curves, the following gross fact becomes apparent. Short dives can be performed safely at great depths and long-duration dives can only be performed safely at shallow depths. There arises, therefore, the idea that a quantity of gas is involved in the provocation of limb bend pains. This idea has received considerable support from some very pertinent experiments by Hills (1970) who concludes, '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'.

It is well known that very serious neurological signs and symptoms can sometimes be presented as a result of decompression, and yet when prompt recompression is given there is generally complete and dramatic relief. As Barnard (1965) pointed out, such remarkable reversibility can only occur if the nervous tissue is substantially undamaged during the course of the attack. If bubbles formed extravascularly in the cell substance then the mechanical damage from the presence of these bubbles necessary to produce a hemiplegia, for example,

would be severe, and would hardly be rapidly reversible. However, if one assumes that the bubbles are intravascular, then partial occlusion of the circulation could cause widespread depression of activity which is quite rapidly reversible if not maintained over too long a period. The same argument can be advanced for the milder form of decompression sickness, namely limb pains (bends), which is often accompanied by neurological signs such as diminished reflexes (Barnard 1965), and all such signs are removed completely by recompression.

Following prolonged exposure to compressed air, workers can be decompressed along one of two quite different types of pressure-time profiles in use today. These two different decompression procedures are illustrated by considering the Washington State Regulations and the Blackpool Tables as used in the United Kingdom. The former set of schedules use a continuous decompression procedure and the latter use the more conventional Haldane stage decompression procedure. If these two time courses are compared it can be seen that they are quite dissimilar. The important point to be noticed from the results of operating these two quite dissimilar routines is that from a decompression sickness point of view the outcome is very similar. Here are two totally dissimilar pressure-time courses offering good protection from attacks of limb pains.

The same diversity of decompression paths is becoming apparent when considering decompression from long exposures to raised pressures of oxy-helium. Prolonged excursions from saturation or steady state pressure are now an everyday occurrence and it is clear that large sudden pressure changes are feasible even at depths as great as 300 m (31 ATA; Bühlmann et al. 1970). The systematic experiments of Barnard (1975), already referred to in the text, further illustrate that decompression can be successfully undertaken employing the classic Haldane stage method. Thus, as with air, so with oxy-helium, the time course offered to the diver for his protection can vary quite markedly and yet still be successful. Originally the findings of Barnard (1974) on stage decompression from saturation/steady state dives were considered to have demonstrated that this stage method was only useful at pressures of 100 m (11 ATA) or less. It has since been realized that the partial pressure of oxygen used (0·22 bar) will not allow successful decompression, on other quite different pressure-time courses at pressures in excess of about 120 m. There is a synergistic effect between oxygen partial pressure and absolute pressure, which has been mentioned previously.

The matter may be restated as follows. If a diver is exposed to a pressure P_1 and then wishes to decompress himself to a lower pressure P_2, the possibilities ahead of him are as follows. If P_2 can be reached in a short period of time without causing decompression sickness, then clearly this is the most economical path. If P_2 cannot be reached without provoking decompression sickness then two major possibilities present themselves for his consideration. First, he can decompress rapidly to some intermediate pressure, P_3, such that the sudden change from pressure P_1 to P_3 does not provoke decompression sickness, but he will now have to stay at pressure P_3 until such time as he can make the next large move towards pressure P_2. Alternatively, he may attempt to decompress himself quite slowly from P_1 in order to stay away from provocation of bends as much as possible. He will thus spend time travelling from P_1 to P_3, which could have been achieved on the previous system much more speedily. However, the hope is that through spending this extra time, and being more conservative, he will have a safer and possibly speedier decompression. There are practical and theoretical advantages in both systems of decompression and experience dictates which to choose for certain circumstances. For example, no-stop diving is a discontinuous or stage system of decompression, but no-one would be foolish enough to suggest that this form of diving should be abandoned in order to avoid formation of some, perhaps even non-existent, silent bubbles. Whereas at great depths on oxy-helium mixtures, there is a distinct risk that sudden large changes in pressure may provoke rather more serious forms of decompression sickness than mild limb pains. Consequently, there is an understandable reluctance to employ this form of pressure-time profile at great depths. From a practical standpoint the fact that one can decompress quite safely from both saturation or non-saturation dives using stages in much the same order of time, or in many

cases much less time than by any other system, means that the relevant tissues behave 'as if' supersaturation was possible.

In actual fact of course, bubbles may be present in the relevant tissues, but perhaps due to their position in the blood vessel, or to the little understood growth patterns of very small bubbles, it may be that no serious interference with tissue gas exchange takes place until quite a substantial pressure change occurs. This problem was also answered in a different manner in Chapter 11 of the first edition of this book by Behnke, who said:

> (1) A metastable condition of gas transport probably did not extend through successive decrement of pressure.
>
> (2) What appeared to be a condition of supersaturation was a reflection of tolerance of the vascular system to bubble accumulation.

However, use of the phrase 'What appeared to be a condition of supersaturation' concedes the practical fact that one can consider decompression 'as if' supersaturation did exist. Nevertheless, the experiments of Hills (1970) prevent anyone supposing that there are fixed supersaturation limits, as proposed by Haldane and used in modified form by most groups since. This idea, and the numerous variations of it, must now be considered inadequate. In support of the Hills findings the author must mention that the last stop on the air diving tables, calculated as outlined above, was at 20 ft (1·6 ATA) and not the usual 10 ft (1·3 ATA), and this departure from normal practice has been highly successful.

'De-canting' and 'surface decompression' procedures have been practised with complete safety for many years. If one decompresses to atmospheric pressure following an exposure to pressure which will obviously lead to decompression sickness, there is nevertheless a 'safe period' at atmospheric pressure before the onset of trouble, and it was discovered by Edel (1971b) that for the Tektite I depth levels a 15-min surface interval could be safely tolerated.

If one examines the idea of Hills (1970) that in the 'worst possible case' all excess inert gas is released at once upon decompression, then clearly the critical volume which causes decompression sickness is not immediately effective in causing symptoms. One must invoke the further idea that this volume of gas takes time to re-arrange itself, via coalescence, into a pain-provoking shape. As mentioned above, from many thousands of observations with surface decompression procedures, and the onset of mild bends from other forms of diving it is possible to state that the chances of such coalescence occurring on surfacing, i.e. instantaneous or 'worst possible' coalescence, are negligible and to be realistic one must introduce a mechanism offering a very low probability at zero time. Thus one is forced to accept a probability/time function for coalescence.

This causes re-examination of the statistical aspects of bubble formation, leading to the original idea of phase equilibration, and limited supersaturation.

Given infinite time, a gas-saturated solution will spontaneously form a gas nucleus. In a reasonable period of time, say 1 hour, the probability of this event occurring is so small as to be unworthy of consideration. If the solution is decompressed, and a state of supersaturation supervenes then it is obvious that the probability of bubble formation is dependent upon two variables, the degree of supersaturation and the total time involved at that level of supersaturation. It may be objected at this point that mechanical factors such as tribonucleation, muscle contraction etc. will invalidate this probability concept, but if one once invokes such precipitating factors then for the 'worst possible' case one must abandon inherent unsaturation, oxygen window, etc., because if these mechanical factors are stressing the solutions and causing cavitation, they can cause bubble formation even in undersaturated solutions. Thus, if one ignores mechanical factors, the limited supersaturation and the phase equilibration theories, although apparently at variance, do in fact converge when it is realized that the level of sustained supersaturation is a critical variable. A complex set of functions describing the probability of nucleation for various levels of supersaturation from zero time to infinity, are necessary. Simple phase equilibration takes the probability after infinite time, i.e. 1·0. whereas simple supersaturation theory takes the probability after a very short time; both views are therefore extremes of a much more complicated situation. The enormity of the

computational task confronting the schedule calculator is now becoming very apparent.

Finally, it is possible to explain the fact qualitatively in a very different way. It may be supposed that bubbles are always present regardless of the nature of the decompression. Dissolved gas in the tissue(s) has two paths for elimination during decompression, first via the blood stream and secondly into the bubble, and the bubble itself is subject to expansion according to Boyle's Law. Thus three factors are determining the outcome of the pressure-time profile. The prospects of rendering this model quantitative are indeed daunting, especially when the difficulties of understanding the growth of small bubbles are appreciated.

Regarding the site of origin of these bubbles, one must agree with Behnke (1971) that the evidence is heavily in favour of it being intravascular. Furthermore, bubbles are almost certainly first found in the arterial circulation, as various observers over many years attest, e.g. Wagner (1945), Lever et al. (1966), Hempleman (1968) and Buckles (1968).

Bubbles in the arterial circulation would be thrust into the capillary bed and, if large enough, would cause blockage, whereas such bubbles generated on the venous side would be prone to being dislodged by the blood flow. Bubbles occluding the arterial side of the tissue circulation would not grow against the arterial pressure, but they would extend down the pressure gradient into the venous side and it is this apparent venous occlusion accompanied by bubble generation which can be mistaken for true venous origin and blockage.

Further evidence that the arterial vessels are implicated as sources of 'separated gas' is gleaned from the observations that when dead animals, with no circulation, are given exposures to high pressures of air or other gases the bubbles seen upon decompression are always in the arterial system (Hempleman 1968; Smit-Sivertsen 1975).

CONCLUSION

Bearing in mind the complexity of events leading to 'bends' pains it is astonishing to find that simple calculations, such as those described earlier, can lead to quite successful practical procedures over quite a wide range of pressures and times. All current theories have incorrect or grossly oversimplified underlying assumptions, and a good deal of further experimentation is needed to reduce the extent of our ignorance.

Although attention has largely been directed towards avoidance of the 'bends' it must be reemphasised that avoidance of bone damage is equally, if not more, important.

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