Chapter 5

Prevention

Decompression sickness can always be avoided in man if the decompression is sufficiently gradual. The all-important question is how gradual? Other methods of prevention have been attempted and will be described later but none have so far offered any comparison to gradual decompression as the most feasible and effective means of avoiding overt symptoms. Thus the ultimate practical goal in this field is to formulate decompression to the point where the diver or caisson worker is returned to normal pressure safely, yet minimizing the wearisome and non-productive time spent in a chamber or suspended in the ocean during ascent.

However, before spending the rest of the book reviewing the answers which have been proposed to try to optimize this process, it is interesting to see how the problem has been avoided in the creatures which *were* 'designed' to inhabit the ocean.

Natural Inhabitants of the Ocean

Fish

Water-breathing creatures avoid decompression sickness because the concentration of gases in the ocean varies little with depth. Thus their blood—and therefore their tissues—take up no more gas from the water at depth than they do near the surface, so that their total tissue tension of all gases never exceeds the ambient pressure however deep the fish may swim. In fact, just like mammals, there would appear to be an inherent unsaturation (p. 239) in their tissues at the surface which increases with depth, as evidenced by the partial vacuum measured in the rigid gas cavity found within the hollow bone of the cuttlefish (Denton, 1964).

Supersaturated water

However, if the water is supersaturated with gases before inspiration, then the supersaturation can be passed on to the blood across the gill membrane and hence to the tissues of the fish where it can separate from solution. The high oil content of many fish tissues could offer a large hydrophobic surface conducive to bubble inception (see p. 88). Hence, although the inherent unsaturation of living tissue must be subtracted from water supersaturation to obtain tissue supersaturation, the tissues could still present a site much more conducive to gaseous cavitation than the inspired water. This would apply particularly to very clear water free of heterogeneous nuclei which would otherwise precipitate gas from the supersaturated state. It is found in mountain springs (Marsh and Gorham, 1904) and in the still man-made lakes created between the spillways installed in mountain rivers for generating hydroelectric power (Harvey and Smith, 1961; Ebel, 1969). In both cases, trapped air is taken to great depth where it dissolves in the water, later to emerge at the surface as a supersaturated solution. Hence the first sites conducive to bubble formation encountered by the supersaturated gas are within the fish and have led to extensive death amongst many species.

Unfortunately, the fish afflicted by 'bends' do not go deeper where the increased hydrostatic pressure would help to treat the problem

but come to the surface (Renfro, 1963). This writer has found that compression has helped to relieve the distress developed in trout by placing them in non-chlorinated tap-water freshly run from the mains—yet highly supersaturated with air in most town supplies! Anyway the fish is an interesting bends model because it enables the experimenter to change hydrostatic pressure without necessarily changing the inspired tension of any gas present—a point well exploited by Beyer *et al.* (1976).

Liquid-breathing mammals

This ability for liquids to be grossly unsaturated with gases relative to the hydrostatic pressure has been exploited by Kylstra and co-workers as a possible means of avoiding decompression sickness in man. This also applies to all other undesirable effects of hyperbaric exposure except any attributable to pressure *per se*. Thus the diver breathes a liquid containing the oxygen he needs while the same liquid removes carbon dioxide and need not contain any nitrogen—certainly less than would cause bubble formation at the surface. He can then move freely between any depths with no fear of the 'bends'.

This is a most imaginative concept but one where the engineering problems of oxygenating the breathing medium and assisting ventilation of the diver in the water at great depth present a tremendous task in ensuring reliability. There are the minor problems of filling the other body cavities, viz. the sinuses and middle ear, with an incompressible fluid and the problems of changeover when both air and the ventilating liquid occupy the lung.

This writer takes the view that whereas it may not be practicable with present technology for the working diver to breathe liquid on the job, or to perform well with liquid-filled ears and sinuses, liquid-breathing could provide a very effective means for permitting enormous hydrostatic pressure to be applied to bubbles in cases of decompression sickness which have defied other treatments. This ingenious concept would seem much more feasible under the well-controlled conditions of the treatment chamber and is described in more detail in connection with the resolution of symptomatic bubbles (p. 210).

Diving reptiles

The foregoing discussion of fish and liquid-breathing mammals might suggest that man only gets into trouble because he takes down gas *in the gaseous phase* when he descends in the ocean. However, other air-breathing creatures which inhabit the sea seem to avoid decompression problems.

The first example is the marine reptile which can dive with air-filled lungs, turtles reaching depths of 950 fsw (290 m) according to Seymour (1974). The absence of any untoward effects following such remarkable dives has been attributed to right-to-left shunting in the heart, i.e. circulating blood tends to bypass the lungs and so avoids absorbing the air in them.

An ingenious mechanism for eliminating gas during a dive has been proposed for marine sea snakes which can dive with air-filled lungs to depths as great as 130 fsw (40 m) according to Heatwole and Minton (1975). Seymour (1974) has postulated that the increased tension of blood gases on diving provides a diffusion gradient for nitrogen elimination across the skin, since the sea has only surface concentrations of air at all depths. Cutaneous gas transfer has also been observed in turtles (Belkin, 1968).

Diving mammals

The same explanation cannot be invoked for the ability of diving mammals to avoid decompression sickness because the layers of blubber which they lay down as thermal insulation would also act as a diffusion barrier for gases. Weddell seals commonly dive to 1,000–1,300 fsw (300–400 m) according to Kooyman (1966), while a bottle-nosed dolphin has been trained to dive repeatedly to 1,000 fsw (Ridgway *et al.*, 1969).

It has been well demonstrated that, when marine mammals dive, they undergo major shifts in blood flow distribution (Scholander, 1940; Andersen, 1966; Elsner *et al.*, 1966). Overall cardiac output is reduced with blood being diverted towards tissues such as the CNS

which have the poorer anaerobic metabolic capacities.

Most diving mammals are able to expire to relatively low residual volumes for their size, so that they take quite a small amount of air to depth in their lungs. Absorption of this gas by blood is greatly delayed by the ability of the terminal airways to collapse and so minimize the surface area for exchange between air and blood (Kooyman, 1973). Moreover, the upper airways seem overly strong for normal respiration and might well be so designed to provide mechanical restraint against undue compression of the remaining air. Thus less gas would be absorbed by blood than in a lung as uniformly compliant as the human lung. Any nitrogen which did dissolve and reach the circulation could well be 'mopped up' by the large amount of blubber (20–30% of body mass) in which it is five times more soluble than in aqueous tissues (Kooyman, 1973). Hence there is little rise in the nitrogen gas tension in fat or any other tissue and therefore less likelihood of cavitation on subsequent decompression.

It is one thing to divert nitrogen and avoid the 'bends' but how do diving mammals manage to provide their tissues with their oxygen needs?

Oxygen storage

In these marine mammals with large breath-hold capabilities, lung oxygen represents a negligible fraction of total oxygen storage in the body (Kooyman, 1973). The deep-diving mammals have a high myoglobin content for oxygen storage within muscle fibres and a large blood volume with an unusually high content of haemoglobin whose dissociation curve favours reduction to low oxygen contents with maximum release to the tissues. Moreover, the muscles of many diving mammals and other creatures tend to switch from aerobic fat catabolism to glycogen-based fermentation on diving, i.e. resorting to a large anaerobic metabolic capacity, while the limited oxygen stored in blood is diverted to the non-pigmented tissues which need it most, viz. brain. The biochemical nature of these processes has been described in detail by Hochachka and Storey (1975).

Man

By comparison, a diver needs a continuous supply of oxygen regulated on an almost moment-to-moment basis. The foregoing points emphasize just how badly adapted by nature man is to deep diving. For this reason there has long been a school of thought which has considered that man needs to be kept at normal atmospheric pressure by armour plating if he is to venture far beneath the surface of the ocean. Normobaric diving suits were designed as early as 1715 by Letheridge in England (Davis, 1962) where the latest version, nicknamed 'JIM', is designed for a depth limit of 2,000 fsw, yet the articulated joints permit a remarkable degree of manual dexterity (Gisborne and Morrison, 1973). Greater depth capabilities are planned; while the 'armoured gloves' designed to permit almost normal action of the fingers are effective to 500 fsw, at least.

Man's physiological limitations indicate that this form of diving will probably take over for depths in excess of about 1,500 fsw. This writer would like to see it used at lesser depths, not for reasons of the medical hazards as much as for the protection which armour plate affords the diver against mechanical injury—the major killer of divers caused by carelessness or inexperience of any one of the crew. If the cable connecting 'JIM' with the surface became entangled in a pipe section being rapidly raised, or the weights were to fall off a bell, the diver would not suffer by being rapidly hauled to the surface unintentionally.

However, nothing is likely to be quite as effective at moderate depths as the conventional ambient-pressure diver provided he can avoid the many dangers, not the least of which is decompression sickness.

Decompression Methods and the Diving Tables

The prevention of decompression sickness in the regular diver therefore reverts to the all-important problem of how to decompress a man gradually in order to avoid symptoms—in fact, how to prescribe the overall environmental programme which he needs to follow to return to his normal surface atmosphere without taking

unnecessary time or risk of injury. This programme needs to define the optimum relationship between depth, time and composition of the breathing mix appropriate to his exposure.

Means of programming decompression

There are several ways to approach this problem.

(1) Devising decompression schedules purely by trial and error, any procedures which give trouble being modified empirically the next time they are used.

(2) Computing the parameters of the environmental programme using either an empirical calculation method, a mathematical model based upon the likely mechanism or various combinations of these procedures.

(3) Taking a meter to pressure which simulates one of these models or calculation methods and can give the diver a direct indication of how to decompress in the light of his immediate environmental history as accumulated by the device.

(4) Using various techniques to monitor certain physiological or physical parameters of the diver and then to use these direct responses to programme the decompression.

The last is probably the best but is proving by far the most difficult to implement; while virtually all schedules in use today are a mixture of the first two approaches.

It is very difficult to find a purely empirical approach free from 'contamination' by some form of reasoning based on calculation. The only one which this writer has been able to discover is that devised by pearl divers who have been operating around the Northern coast of Australia since about 1880—particularly the Okinawans who venture deeper than the other ethnic groups which dive regularly in those deep tidal waters (see p. 260). It is, perhaps, very significant that these techniques should differ so widely from those devised by calculation, with the possible exception of some recently computed schedules.

The diving tables

Most interest in decompression, however, has centred around schedules with a calculation basis, however extensively they were subsequently modified—either directly or by adjusting the calculation method in the light of practical experience. In practice, it is found that any programme involving the continuous variation of pressure and/or breathing mix is difficult to implement, so pressure changes are generally made in steps. These are not only easier for the diver or chamber operator to follow but offer a simple means of tabulating decompression. Thus the air diving tables simply consist of a list of times to be spent at various stops. When other gases are employed, the times of switching mixes are also indicated. It is conventional to employ intervals of 10 fsw between stops (three or five metres on the metric system) yet, despite the simplicity, it is surprising how often the tables are misread or the prescribed format is not followed because a chamber operator's attention has been diverted or he has fallen asleep during a long decompression.

Is calculation justified?

While there is no foolproof way of avoiding human error in following a particular schedule, there is a great deal that can be done to try to design the best table to be followed. As mentioned earlier, the most popular means of prescribing the deployment of time between the decompression stops invokes calculation of some form but is calculation, or any type of mathematical formulation, justified in the first place?

The early approaches to decompression sickness in the nineteenth century were all qualitative and implicated the separation of gas from solution as 'extricated air' (Lyttleton, 1855) or as bubbles (Hoppe-Seyler, 1857; Bert, 1878; Heller *et al.*, 1900) to quote some of the more prominent publications. In the light of current knowledge of the aetiology

described in Chapter 3, it is most sobering to realize how little our present attitude has changed towards separated gas as the primary insult. However, Chapter 4 revealed just how difficult it was to express nucleation in a quantitative form and how complex were the mathematics describing growth.

Whether one invokes these theoretical arguments or simply returns to practical experience, there is no doubt that the two major parameters determining the imminence of decompression sickness are depth and time—at least for a simple air dive. Moreover, if exposures with no gradual decompression are considered, i.e. 'bounce' dives and the same clinical state such as marginal 'bends' in all cases, then the depths and times provoking this condition are found to be most interdependent. Thus, all other conditions remaining constant, a diver can spend more time shallower but less time deeper. Hence, if this overt state represents the same degree of insult within the body and the same underlying process determines what level the insult-determining parameter reaches on each dive, then the various depth–time combinations for marginal safety should show a continuous relationship when plotted against each other. Accordingly this writer, at least, considers it most significant that such a smooth and well defined depth–time curve (fig. 12) can be drawn to separate 'bends' and 'no bends' points for any individual both for air data (Van der Aue *et al.*, 1951) and a helium: oxygen mix (Duffner, 1958). Behnke (1951) points out that a difference of maybe only 5 fsw in depth can separate serious injury from a state of wellbeing. Not only does this continuity suggest an underlying rationality to the whole bends-provoking mechanism but the comparatively clear demarcation of this relationship warrants the use of quite sophisticated mathematics if simpler functions fail to describe it adequately.

Having justified invoking calculation in the first place, the various options which are available to the decompression designer for selecting his equations can now be considered.

Calculation methods versus models

There are two fundamentally different approaches to determine the equations to be used in the computation of any decompression profile.

(1) Deriving one or more equations to fit known data and then using those equations for other situations. These are really *calculation methods* in which one chooses a time function, preferably one simple to handle such as the exponential, and then selects values for the constants which enable the equation to fit the data. In the case of the exponential, these include both a coefficient related to tolerance (e.g. the decompression ratio or '*M*' value, p. 117) and a constant related to time (e.g. 'tissue half-time'). If one equation proves inadequate, then the designer can either choose a different function (and more constants) or introduce more equations with the same function (and more constants), making sure to state where one takes over from another in determining the ultimate profile. This amounts to the time-honoured practice of 'curve fitting' and leads to a truly empirical calculation method.

(2) Critically reviewing the evidence and selecting the mechanism(s) which the designer considers responsible for the critical insult and, from these, synthesizing a mathematical model. This is then described by an expression whose constants are taken from handbooks of physical and physiological data, where known, or otherwise are determined experimentally.

Calculation methods tend to cope with just a limited range of conditions, so that one which can provide apparently safe decompression schedules for exposures down to 400 fsw may fail completely when extrapolated to 500 fsw. Only the elucidation and mathematical description of the true model can avoid this time consuming, expensive and sometimes dangerous practice.

At first sight, most computational rationales underlying diving tables would appear to be a mixture of the two approaches listed above—the empirical and the synthetic. However, in the publication of a new method of formulating tables, *the equations often do not reflect the words*. There are cases where a method is

described as 'based upon diffusion' but the diffusion coefficient does not appear in the equations nor is reference made to its relation to any one of the constants. There are other instances where a perfusion-controlling mechanism is invoked, yet the accompanying equations do not show a blood perfusion rate nor include a relating reference. Hence many apparent hybrid approaches are really calculation methods in which comments pointing out the compatibility of some feature of the equation(s) with a physical or physiological aspect have been added later. This is particularly true of modified 'Haldane' methods and the designers of some of the most successful methods proven in the field, such as Bühlmann (1975), who do not pretend that theirs are anything other than *calculation methods.*

By the same token, there is no 'model' approach yet available which is purely synthetic and does not at least require some constants to be determined by first applying their equation to practical diving data—if only because the designer cannot be sure of the anatomical identity of the critical tissue(s).

These points serve to outline some of the critiques which can be used to compare the methods and equations actually used to formulate decompression tables.

The 'Haldane' Method

While the early work of the nineteenth century was primarily qualitative, concerned with the overall aetiology of decompression sickness, it was not until the beginning of the twentieth century that the first really quantitative approaches to prevention were conceived. These are credited to the two centres working on these problems in England at the time—the Royal Navy who appointed the celebrated respiratory physiologist J. S. Haldane as consultant to an Admiralty committee on deep diving formed in 1906 (Boycott *et al.*, 1908) and the London Laboratory of Sir Leonard Hill, J. Argyll Campbell and collaborators (Hill, 1912).

Hill argued that a man can be decompressed by a *fixed* amount (ΔP) from his equivalent depth of saturation before bubbles would form and he would subsequently develop symptoms—an approach also adopted by Behnke (1937). However, this approach was largely overshadowed by the 'Haldane' method published a few years earlier which had almost immediate success, such as reducing the death rate among compressed-air workers from over 50% to less than 1% in some of the American railroad tunnels under construction at that time. By contrast, the 'Haldane' approach described the permissible decompression by a pressure ratio rather than the fixed ΔP advocated by Hill (1912).

Decompression ratio

Haldane and his collaborators (Boycott *et al.*, 1908) argued that if a man is decompressed, then his tissues can tolerate a certain degree of supersaturation without forming bubbles. Moreover, he described this critical limit to supersaturation by a decompression ratio—to be designated by much later workers by the symbol M. This is the ratio between the air pressure (tension) reached by the tissue before decompression (p) and the absolute pressure (P) to which the subject is decompressed. Thus the subject should be safe if

$$p/P < M \tag{33}$$

Although the original publication of their calculation method contained no symbols or equations, Haldane and co-workers effectively selected a value of 2 for M. The reason is perhaps best expressed in Haldane's own review of the method (Haldane and Priestley, 1935) where he states:

> The formation of bubbles depends, evidently, on the existence of a state of supersaturation of the body fluids with nitrogen. Nevertheless, there was abundant evidence that when the excess of atmospheric pressure does not exceed about $1\frac{1}{4}$ atmospheres, there is complete immunity from symptoms due to bubbles, however rapid the decompression. Thus bubbles of nitrogen *are not liberated* within the body unless the supersaturation corresponds to more than a decompression from a total pressure of $2\frac{1}{4}$ atmospheres. Now the volume of nitrogen which would tend to be liberated is the same when the total pressure is halved, whether that pressure be high or low.

Formulating a diving table

Before getting too involved in the assumptions

underlying this calculation method and its validity, it would seem as well to summarize the procedure for using the basic 'Haldane' method to formulate an actual diving table.

Haldane considered the body to be composed of a number of hypothetical 'tissues', each of which will take up air on compression of the subject and eliminate it on reduction of the absolute pressure to a value lower than the gas tension (p). The ratio of this parameter to the ambient absolute pressure (P) is then claimed to determine the imminence of symptoms developing if the subject remained at P. If this ratio (p/P) exceeded the critical value of 2·0 for any 'tissue', then that 'tissue' could give rise to symptoms of decompression sickness, i.e. the condition expressed by Equation 33 had been violated.

By applying the same criterion to each of five tissues, tables can thus be compiled in which the duration of each decompression stop is determined by the one whose (p/P) ratio *at that particular time* is closest to the critical value.

If the diver were suddenly decompressed by 10 fsw, then the ratio would be increased to $p/(P-10)$ which could exceed 2. Hence he must remain at P until the highest tension of all tissues (p_m) has been reduced to a level at which

$$p_m/(P-10) \leqslant 2 \qquad (34)$$

when he can move to the next decompression stop 10 fsw shallower than his last. This move then provides an initial increase of 10 fsw in the driving force for gas elimination (or a reduction of 10 fsw in the uptake gradient) in each theoretical tissue.

The tissue tension (p) at an absolute pressure (P) is simply calculated on the basis that the rate of uptake or elimination of gas by any tissue is proportional to the driving force ($P-p$), i.e.

$$\mathrm{d}p/\mathrm{d}t = k(P-p) \qquad (35)$$

where k is the proportionality constant which embodies the effective resistance of that tissue to gas exchange with circulating blood but is often expressed as a time to half saturation ($t_{1/2}$) i.e.

$$k = 0{\cdot}694/t_{1/2} \qquad (36)$$

where $\log_e 2 = 0{\cdot}694$. Haldane advocated the use of five hypothetical 'tissues' to which he allotted half-times of 5, 10, 20, 40 and 75 min to be fairly representative of a continuous spectrum of response times.

The very simple linear relationship depicted by Equation 35 not only renders the 'Haldane' calculation method particularly conducive to programming for a computer but it enables the tissue tension $p(t)$ at any time $(t-t_n)$ after starting the nth stop at pressure P_n to be estimated simply by integrating Equation 35 between these limits,

$$p(t) = p(t_n) + [P_n - p(t_n)] \times \underbrace{[1 - \exp\{-0{\cdot}694(t-t_n)/t_{1/2}\}]}_{\text{(time function)}} \qquad (37)$$

where $p(t_n)$ is the tissue tension at the start of the nth stop. The same expression applies to uptake and to all other stops so that the variation in tissue tension can be calculated throughout any dive remembering that the tissue tension at the end of one stop is still the tension at the start of the next.

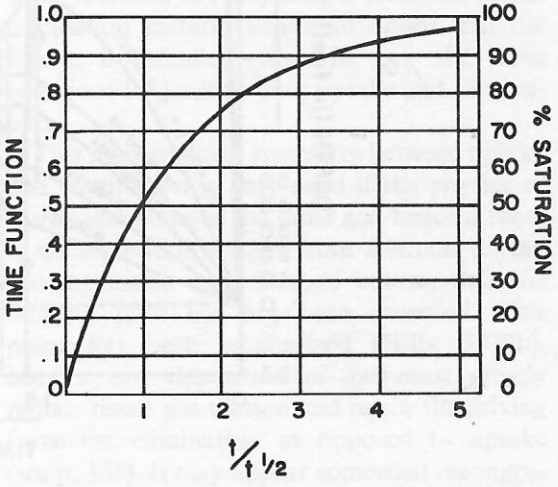


Fig. 32 The time function based upon the exponential (Equation 37) in which the fractional change is related to the 'reduced time', i.e. to the time divided by the half-time ($t_{1/2}$) for that particular 'tissue'. Redrawn from Boycott *et al.* (1908)

The calculation can be simplified even further by reducing all time intervals to fractions of the half-time of the particular 'tissue' for which $p(t)$ is being computed. All that is then needed is to read off the corresponding value of the time function to be used in Equation 37 from the graph originally published by Boycott *et al.* (1908) and reproduced in fig. 32. This graph reduces the exponential function to a convenient form where it can be used for all 'tissues'.

In this way the 'fast' tissues, with large initial gas uptake, tend to control the early phases of decompression; while the 'slow' tissues, with less uptake, but a low rate of elimination, determine the later stops. This is clear in fig. 33 which shows this simple calculation method depicted for Haldane's original value of 2 for the limiting decompression ratio.

The philosophy underlying decompression by this approach is described by Haldane (Boycott *et al.*, 1908) as 'the process of desaturation can therefore be hastened very greatly by rapidly reducing the pressure to half and so arranging the rest of the decompression that the saturation in no part of the body shall ever be allowed to correspond to more than double the air pressure'. Thus the method essentially keeps the diver at the shallowest depth ($P\downarrow$) consistent with the critical value of the ratio (M) for all tissues (Equation 33) in order to maintain the largest driving force ($p - P$) for desaturation [$(-\mathrm{d}p/\mathrm{d}t)$ in Equation 35].

The 'Haldane' method is now seldom used as originally published. However, before discussing the many empirical modifications, it is desirable to know the reasons which Haldane gave for the type of calculation he advocated and what assumptions, if any, the method was hiding.

Reasons and assumptions

The features of the original 'Haldane' method (Boycott *et al.*, 1908) can be listed as follows.

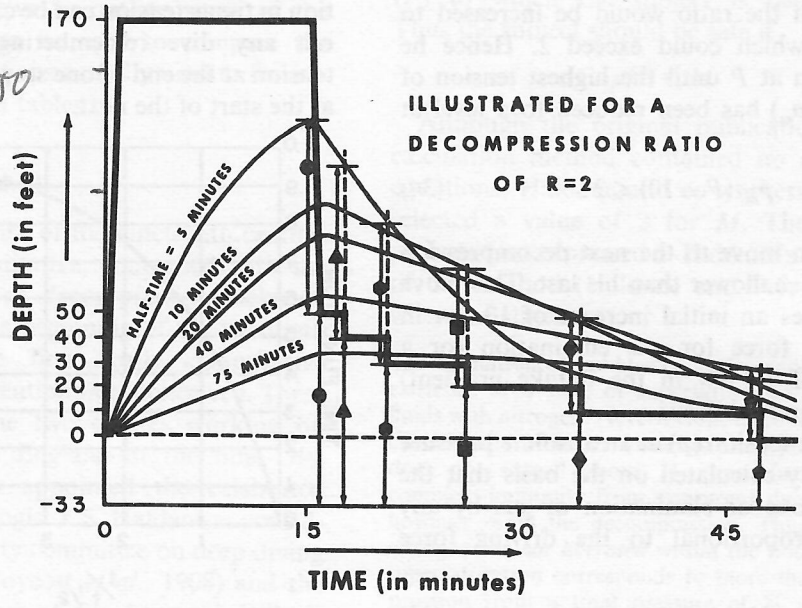


Fig. 33 The 'Haldane' calculation method illustrated using the original value of 2.0 for the decompression ratio for all five 'tissues'. Thus the diver is decompressed to a stop which is not less than one half of the maximum 'footsworth' of air estimated as taken up by any 'tissue' in accordance with the time function shown in fig. 32

(1) The imminence of decompression sickness in any one 'tissue' is determined by the ratio of its tissue gas tension to the ambient absolute pressure, this index not to exceed a critical limit of 2·0.

(2) The rate of uptake or elimination of gas by each 'tissue' is directly proportional to the difference between ambient absolute pressure and gas tension in that 'tissue'.

(3) It is necessary to consider five 'tissues' of equal susceptibility and half-times of 5, 10, 20, 40 and 75 min.

The first of these (the decompression ratio) is discussed in the next section in connection with its experimental validation by later workers, while the other two features essentially provide the means for incorporating the effect of time.

In his verbal description, Haldane introduced the effect of time by reasoning that blood (saturated at the ambient pressure) will equilibrate with tissue upon each 'round' of the circulation, so that the amount transferred to the tissue each time it passes will get steadily less. In this way he argues that 'the progress of the saturation of the body with nitrogen is thus a logarithmic curve of the form shown' (in fig. 32). Thus he assumes that the uptake and elimination of gas is controlled purely by the blood perfusion rate and goes on to quote Zuntz's (1897) derivation of this time function for a circulation-limited system, where the time constant (k) is related to the blood perfusion rate ($\dot{Q}$) and the partition coefficient of the gas between blood and tissue (λ) as

$$k = \lambda \dot{Q} \qquad (38)$$

Inversion of the 'logarithmic curve' proposed by Haldane leads to the exponential function as depicted in Equation 35 for relating tissue gas tension to the past history of compression on air.

His text refers to nitrogen but, since the *calculation* allows tissue gas to 'saturate' in time, i.e. to attain the same value as the absolute ambient pressure, the pressure parameter (p) really represents an equivalent tension of air—or so many 'footsworth' of air. In other words, although Haldane specifically mentions that nitrogen comprises 80% of the air, this is not reflected in his method of calculation. Hence he is effectively regarding air as though it were one gas for computational purposes. However, the decompression table would be exactly the same if p did refer to nitrogen only and the critical value of the ratio were reduced accordingly—i.e. to $0{\cdot}8 \times 2 = 1{\cdot}6$.

Haldane also fully appreciated that the distribution of blood flow throughout the body was not uniform, so that the better perfused tissues would 'saturate' faster than the others. This was envisaged as a continuous spectrum of response times which could be approximated quite well by considering only five 'tissues' the time constants of which differ by roughly equal logarithmic steps; hence his selection of values for his five tissue half-times (5, 10, 20, 40 and 75 min).

Validity of the decompression ratio

Haldane frequently refers to supersaturation but does not define whether he is using this word in its true thermodynamic sense of representing a state of suppressed transformation (p. 77) or whether he simply means the overall gas content of tissue in excess of saturation, whatever form the gas may take. However, when it comes to computing a table, his actual calculation method leaves no doubt that the former is intended, since he uses the same equations to describe both uptake and elimination.

This mathematical symmetry between uptake and elimination is only valid if the physics of the system remains the same and hence if there is no separation of gas from solution during decompression—provided of course, that the limiting ratio has not been exceeded. This point has been emphasized (Hills, 1970b), because any deposition of gas must greatly reduce tissue gas tension and hence the driving force for elimination as opposed to uptake (see p. 138). It may appear somewhat incongruous, therefore, to read Haldane's own explanation for his threshold value of two for the decompression ratio. This reads 'the volume (not the mass) of gas (measured at the existing

pressure) which would be liberated if the whole excess of gas present in supersaturation were given off is the same, whether the absolute pressure is reduced from two to one atmospheres, or from four to two or from eight to four' (Boycott *et al.*, 1908). Such a statement could be interpreted as implying that it is the volume of gas liberated rather liberation *per se* which determines the outcome of a dive. However, his subsequent statement (p. 110) leaves no doubt that this was not intended, i.e. he claimed that no bubbles should be formed for $p/P < 2$, so that, provided this interpretation is correct, Haldane was perfectly justified in assuming mathematical symmetry. Moreover, his statement concerning volumes need not be regarded as incompatible with his calculation method when it is remembered that there are several thermodynamic energy functions which could be used to describe a metastable limit to supersaturation (e.g. Equation 10)—all logarithmically related to pressure or volume and hence consistent with the pressure ratio concept. Although Haldane could have offered better justification for the decompression ratio by quoting contemporary references in the physical sciences (p. 77), there is no doubt that his actual calculations invoke the ratio as an index of *true supersaturation*. Thus the critical limit of 2, or any other value taken for M in Equation 33, represents a 'trigger point' for bubble formation—or the metastable limit to suppressed transformation in physico-chemical parlance (p. 77).

Thus the original 'Haldane' calculation method has many of the attributes of a true model in view of the fundamental interpretations offered for the ratio as an index of limiting supersaturation and the exponential as an expression of gas transfer limited by the circulation. Whether these basic axioms are valid is quite another question and one which will be discussed in great detail later, since they are so important in calculating diving schedules; but the 'Haldane' approach is physically consistent within itself—despite early claims that it was inconsistent with the laws of physics (End, 1938; Shilling, 1941). It is largely the subsequent empirical modifications which have detracted from the 'Haldane' approach as a fundamental model.

U.S. Navy modifications to the 'Haldane' method

The introduction of the 'Haldane' method of formulating preventive decompression undoubtedly represents the greatest single advance in avoiding decompression sickness and provided the basis of Royal Naval tables (Miles, 1962). These proved particularly successful for air diving to maximum depths which Behnke (1951) considered to be in the region of 120 fsw. The same method was also used for greater depths and, if any table gave trouble, the decompression was extended empirically, often simply by adding time to the last (10-foot) stop.

The inadequacies of the 'Haldane' method were not fully realized until a few years before the Second World War when it became strategically desirable to be able to dive deeper than 200 fsw. While the Royal Navy continued to modify directly any table where theory and practice differed unduly, the U.S. Navy preferred to modify the calculation method on which the table was based. This writer finds it particularly difficult to trace the reasons for the actual changes made to the decompression tables published in successive U.S.N. diving manuals simply by reading their reports. However, there are several significant papers advocating major modifications to the 'Haldane' method which do seem to be reflected in the published tables.

The first modification was suggested by Hawkins *et al.* (1935) who allotted different ratios to the different Haldane 'tissues', ranging from $M = 5{\cdot}5$ for the fastest ($t_{1/2} = 5$ min) to $M = 1{\cdot}7$ for the slowest ($t_{1/2} = 75$ min) on the basis of their analysis of over 2000 dives to 100–200 fsw. This was compatible with the later feelings of Haldane himself who thought that some reduction in the ratio was required for dives in excess of 6 ATA (165 fsw) (Haldane and Priestley, 1935).

This introduced a basic difference to the calculation procedure in that the imminence of decompression sickness in any one 'tissue' was now given by the index (p/MP) rather than (p/P), where M now had a unique value

for each response curve (i.e. each $t_{1/2}$ value). Hence the 5 and 10 min 'tissues' with M values of 5·5 and 4·5 respectively tended to give small values for the index (p/MP), so that they rarely became the controlling 'tissues' for most practical air exposures, even at the start of decompression.

For this reason, Yarbrough (1937) ignored these two fast 'tissues' in calculating a set of tables which, when tested and revised, became the first set of U.S. Navy Standard Air Decompression Tables derived by the U.S.N. themselves and hence the first to diverge from Royal Naval practice which they had previously adopted. The need to allow for the effects of exercise resulted in some modification to the 'resting' M-values originally derived by Hawkins *et al.* (1935), so that the final tables produced by Yarbrough reflected ranges of $2.45 \leqslant M \leqslant 2.8$ for the 20 min 'tissue' and $1{\cdot}75 \leqslant M \leqslant 2{\cdot}0$ for the 40 and 75 min 'tissues'. Further modifications to the ratios were advocated by Yarbrough and Behnke (1939), while Dwyer (1956) suggested the use of four 'tissues' with half-times of 20, 40, 80 and 120 min to which he allotted variable M values (see p. 117).

However, it may be argued that a comprehensive calculation method should also be able to predict the no-stop decompression curve, i.e. if it is valid in prescribing decompression, it should also be capable of predicting the exposure limits beyond which gradual decompression becomes necessary. For this reason it was somewhat disturbing to find that, in the analysis of their bounce-dive curve for air exposures, Van der Aue *et al.* (1951) showed rather higher M values than advocated by Yarbrough for table formulation, ranging from 2·8 for $t_{1/2} = 20$ min to $M = 2{\cdot}06$ for $t_{1/2} = 75$ min and $M = 2{\cdot}0$ for $t_{1/2} = 120$ min. This tended to confirm an earlier viewpoint that more conservative M values are needed for programming decompression stops than apply to surfacing ratios (Behnke, 1947). Moreover, it became apparent with experience of longer and deeper air exposures (Des Granges, 1957) that further slower 'tissues' with lower M values needed to be invoked (Workman, 1957) if the U.S.N. were to retain the basic 'Haldane' rationale for calculation.

Helium–oxygen tables

At the suggestion of the eminent physical chemist J. H. Hildebrand, Sayers and Yant (1925, 1926) showed the advantages, during the subsequent decompression, of substituting helium for nitrogen in the breathing mixture of guinea pigs exposed to pressure. These advantages were largely substantiated in human trials by End (1937) and Behnke and Yarbrough (1938) and soon led to the first set of helium : oxygen decompression tables (Momsen and Wheland, 1939). Calculations to revise these tables (Molumphy, 1950) were again based upon the 'Haldane' rationale with tissue half-times of 5, 10, 20, 30, 40, 50, 60 and 70 min. However, the introduction of a period of breathing pure oxygen at the last (40-foot) stop made it imperative to switch to partial pressures of inert gas in computing diving tables. Thus the ratios of 5·0 to 1·7 which Molumphy allotted to his eight tissues now refer to (P_{He}/P) so that they are rather higher than the values for air diving when these are reduced to a partial pressure basis, i.e. the original 'Haldane' ratio of 2·0 now gives the same condition for safety as $P_{N_2}/P \leqslant 1{\cdot}6$ (see p. 113). The underlying assumption that oxygen did not contribute to total tissue gas tension had been realized but not acted upon by Haldane (Haldane and Priestley, (1935).

Current U.S. Navy calculation methods

The advent of helium : oxygen mixes and the need to dive to much greater depths led to the introduction of more and more 'tissues' into the 'Haldane' calculation method which U.S. Navy workers continued to follow almost without exception. Thus additional 'tissues' with half-times of 120, 160, 200 and 240 min were needed just to compute the decompression for an exposure of 120 min at 300 fsw. The basis for current U.S. Navy tables was provided by Workman (1969) who, at the last count, was using as many as fourteen 'tissues' each with a given half-time and each allotted a unique

ratio, i.e. 28 empirically selected constants. Thus any designer using this technique had twenty-eight degrees of freedom by which to modify the calculation method if any experimental data did not fit.

It must be mentioned that the U.S. Navy tables also contain direct modifications made in the light of experience in using a particular table; but one is advised to use caution in interpreting their published bends rates as an index of the success of their calculation method. If they state that their overall incidence of decompression sickness over the last year has been much less than one per cent, this is undoubtedly true and genuinely refers to use of the U.S.N. schedules by their divers. However, their figures include many no-stop exposures and when they do need to invoke gradual decompression their divers tend to 'move over' two columns in selecting the decompression for their particular exposure, i.e. not using the schedule advocated for their actual exposure but using one corresponding to a 10- or 20-min longer stay on the bottom. When the U.S.N. tables are used as printed, Canadian sources (Kidd *et al.*, 1972) have indicated a bends rate in the region of 14% and many commercial organizations who have tried them seem to agree with this as a 'ball park' figure. When U.S.N. divers 'move over' two columns, their total decompression time (and bends incidence) show little difference from standard Royal Naval tables which have been generally regarded as very conservative. While R.N. tables have only been available to approved parties, the American tables have represented one of the few comprehensive sets of decompression schedules freely available to civilians for many years—a fact which has tended to focus much attention upon the U.S. Navy in diving and their vigorous research programme in this area.

The 'Haldane' rationale also seems to be the basis for Russian calculations, their table designers discussing permissible supersaturation in terms of pressure ratios (Brestkin, 1965; Aleksandrov and Brestkin, 1965; Brestkin *et al.*, 1965; Zal'tsman and Zinov'eva, 1965); while the French 'G.E.R.S.' and the Swedish Royal Naval tables also tend to be based upon the same underlying concepts. The West German Naval Medical Institute (Cabbarou *et al.*, 1975) has recently revived interest in the 'Haldane' decompression format by claiming a number of bends-free dives in which there were large initial pressure drops—long first 'pulls' towards the surface indicative of high M values previously discarded by other proponents of this calculation method.

However, with these few exceptions, the inadequacies of naval practice have forced commercial enterprises to develop their own methods over the last decade in their rush to dive much deeper and more economically in exploiting offshore resources.

Before discussing commercial approaches to decompression problems, as far as the proprietary nature of their findings will permit, a milestone in the switch of the focus of attention from the U.S. Navy to civilian efforts was, perhaps, the world-record dives to as deep as 1,000 feet made by the Swiss mathematician Hannes Keller between 1960 and 1962. Keller was the first to popularize the use of multiple inert gases during a dive and one of the first to use the computer to generate decompression schedules. Although the subsequent publication of the underlying calculation method (Keller and Bühlmann, 1965) revealed no new principles in computing tables, his achievements proved a great stimulation to both military and private interests in the field.

Commercial calculation methods

The modifications to the 'Haldane' method introduced by the U.S. Navy were quite conservative by comparison with the commercial changes to follow. The proliferation of 'tissues' greatly accelerated after Schreiner and Kelley (1967) published their computer programme. There are numerous arguments in the literature concerning the M value to be allotted to a particular 'tissue' but in practice, every time a calculated diving table proved unsatisfactory in the field, a new 'tissue' was 'pulled out of the hat' to accommodate the difference. Schreiner and Kelley (1971) very aptly called this a 'pragmatic' approach.

However, if the calculation method is so inadequate that it has to be modified to comply with every result, then it becomes useless for formulating a table for a new depth or time of exposure. If a table proves unsafe then, with an effectively infinite number of degrees of freedom, there are as many ways in which one can go about improving that table and so one does not know where to start.

In an attempt to reduce the number of possibilities, yet still retain the 'Haldane' rationale, this writer tried differentiating the time function with respect to the time constant (k in Equation 35) and equating to zero, i.e. finding the maximum tissue gas tension (Hills, 1966). In this way the designer selects from an infinite spectrum only the tissue with the worst possible half-time for the particular dive history up to that point. However, this approach proved unsuccessful unless a purely arbitrary spectrum of M values was adopted; so one is back to the infinite number of degrees of freedom which this writer found unacceptable. It also raises a basic theoretical point largely avoided in the early U.S.N. modifications of the 'Haldane' method: how can 'tissues' with a continuous spectrum of blood perfusion rates have different inherent susceptibilities as reflected by their M values? If the hypothetical tissues of the 'Haldane' method simply reflect local differences in circulation *within the same tissue*, then they should have the same M value—as, indeed, was allotted by Haldane. If, on the other hand, they represent different anatomical identities, then the same insult to different organs should produce very different clinical manifestations and yet, in practice, the same limb bends seem to occur whether one violates the 'trigger points' for the 5, 20, 80 min or any other 'tissue' (p. 31). It was the lack of satisfactory answers to questions such as these which led this writer, at least, to abandon the 'Haldane' method.

The mathematical simplicity of this rationale, however, and the ease of programming linear systems for the computer has led to further empirical modifications until few workers in the commercial field make any pretence that the calculation routines evolved bear any resemblance to the model originally conceived by Haldane.

If one has introduced an effectively infinite number of 'tissues', each allotted a different ratio, and the method has still not proven satisfactory, one is finally faced with the prospect of changing constants into variables—either the time constants or the M values or both.

It would seem perfectly reasonable to expand the time base with exercise, such as using a factor of 1·5–2·0 by which to multiply the actual bottom time of a diver who has been working (Workman, 1969).

One is then left with the prospect of allowing the ratio to vary as a function of any environmental parameter, particularly depth, since it is conceivable that any 'trigger point' to bubble formation could be a function of absolute pressure as well as intrinsic factors related to tissue constitution.

Variable ratio

The frequent need to revise ratios after trials of a new set of decompression tables to a greater depth has led to various ways of trying to predict the relevant values beforehand. The first attempt was probably that of Dwyer (1956) who related the maximum safe tissue pressure of nitrogen (M) to the surfacing ratio (M_s) and the depth ratio at the decompression stop (M_d) as

$$M = 33(M_s/M_d)^{10} + M_d - 1 \qquad (39)$$

Trials of tables using these depth-dependent M values showed a significantly lower incidence of bends than previous air tables (Doll, 1965).

More recently a compromise between a ratio and a fixed ΔP has been invoked in procedures used by caisson workers (Hempleman, 1969) to give an M value dependent upon the absolute pressure (P) expressed in psi as

$$M = 400/(P + 180) \qquad (40)$$

Thus the permitted ratio for air exposure decreases with depth from $400/(15 + 180) = 2{\cdot}05$ at normal atmospheric pressure but obviously

cannot apply when pressures approach 220 psia at which $M = 1{\cdot}0$, i e. no decompression would be allowed.

Such limitations in the use of equations for M have led Bühlmann (1969) to adopt empirical relationships between the permissible decompression ratio and depth. Moreover, he works in terms of partial pressures and takes the tensions of nitrogen and helium in the same tissue as additive, i.e. his condition for safety is simply

$$P_{N_2} + P_{He} < M' \cdot P \qquad (41)$$

where M' refers to tissue *partial pressures.*

In the presentation of his further modification of the 'Haldane' rationale, Bühlmann (1969) presents two curves relating $(P_{N_2} + P_{He})/P$ to $(P_{N_2} + P_{He})$, one for slow and the other for fast 'tissues'. Recent revisions (Bühlmann, 1975) have split each of these curves into two, to end up with four empirical curves, one for 'tissues' with a helium half-time of 105–240 min, a second for 45–90 min, a third for 10–30 min and the fourth for the five-minute helium 'tissue'. These can be equally well presented as M' versus P and are shown in fig. 34 in this form for ease of comparison with other calculation methods advocating a depth-dependent ratio.

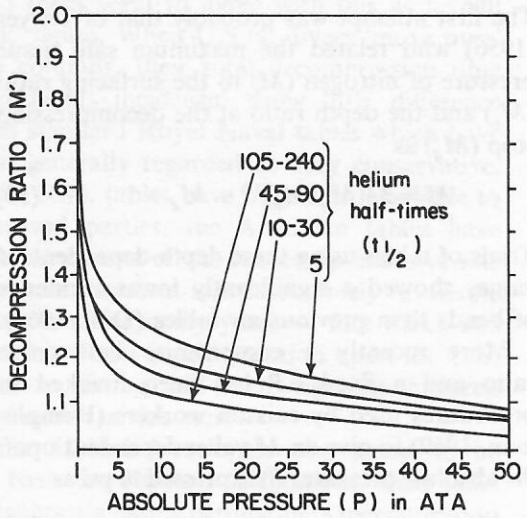


Fig. 34 The decompression ratio (M' in Equation 41) expressed as an empirical function of pressure for each of four ranges of 'tissue' half-times employed in the calculation method developed by Bühlmann. Data from Bühlmann (1975)

Bühlmann now advocates the use of sixteen 'tissues' with helium half-times ranging from 5–240 min as an effective cover of the spectrum. He then takes the nitrogen half-time as 2·65 times the helium half-time of the same 'tissue', i.e. the 240-min helium 'tissue' has a nitrogen half-time of 635 min. This implies diffusion-controlling transport since, by Graham's law relating diffusion coefficients to molecular weights:

$$D_{He}/D_{N_2} = \sqrt{(28/4)} = 2{\cdot}65 \qquad (42)$$

After applying this diffusion ratio to the actual time, the time response is still generated using the exponential function retained from the original 'Haldane' method—yet derived in that approach on a perfusion-controlling basis! However, Bühlmann makes no pretence that his decompressions are based on anything other than an empirical calculation method and, of all the published tables, his have certainly achieved an outstanding degree of success in the field.

His lead has encouraged other workers to adopt depth-dependent ratios with Smith (1975) allotting a different ratio to each 'tissue' at each depth on a purely empirical basis. With at least 16 'tissues' and 10-foot depth intervals, this leads to massive tables of M or M' values, many of the combinations tabulated falling on Bühlmann's original curves. Thus Bennett has presented a table with 736 empirical constants and, even with these 736 degrees of freedom to make the 'Haldane' method fit the data, it was necessary for Vann to compute the major portion of the decompression profile from a fundamental model before the hybrid of the two approaches gave an acceptable bends rate for a dive of 30 min at 500 fsw (Bennett and Vann, 1975).

Results such as these suggest that, whereas the 'Haldane' format may provide adequate interpolation between known data points, it is inadequate for extrapolation. Moreover, the huge number of degrees of freedom which some proponents of this calculation rationale now

need to invoke in performing a calculation cause this writer to query why they calculate at all. Unless a calculation method provides a large degree of constraint in formulating a decompression, one might just as well start by drawing one's last successful or unsuccessful profile on a clean sheet of graph paper and proceed to change it by intuitive feeling or simply by trial and error.

Validity of the ratio

Long before the original 'Haldane' calculation method was extended to these extreme degrees of empiricism, there were those who queried whether the pressure ratio concept really provided the most relevant index of the imminence of decompression sickness, irrespective of the underlying mechanism. Thus Hempleman (1957) undertook a most painstaking series of trials on goats which has, unfortunately, received much less attention than its elegant simplicity would warrant. He exposes a goat to compressed air at an absolute pressure P_1 for 12 hours, when the animal must be very close to attaining a steady-state condition. It is then rapidly decompressed to another absolute pressure P_2 and left at that pressure for several hours to see whether or not it develops a bend (fig. 11). The procedure is repeated several days later on the same goat exposed to the same pressure P_1 but rapidly decompressing to a slightly higher or lower pressure P_2 depending upon whether the animal 'bent' or not during the previous trial. This is repeated until values of P_2 are obtained for both 'bends' and 'no bends' points. This whole 'titration' of the final pressure P_2 is then repeated for other values of P_1. Hempleman's results for three goats are plotted in fig. 35. It can be seen that the straight line through the origin ($P_1 = P_2 = 0$), representing a constant ratio, offers a good locus for the titration points.

This result certainly supports the pressure ratio as an index of the imminence of marginal symptoms and suggests that it is not particularly depth-dependent—at least, not for the slowest 'tissue' over the range 0–300 fsw. If there are other faster 'tissues' which need to be considered, then it also seems to apply to them. Hemple-

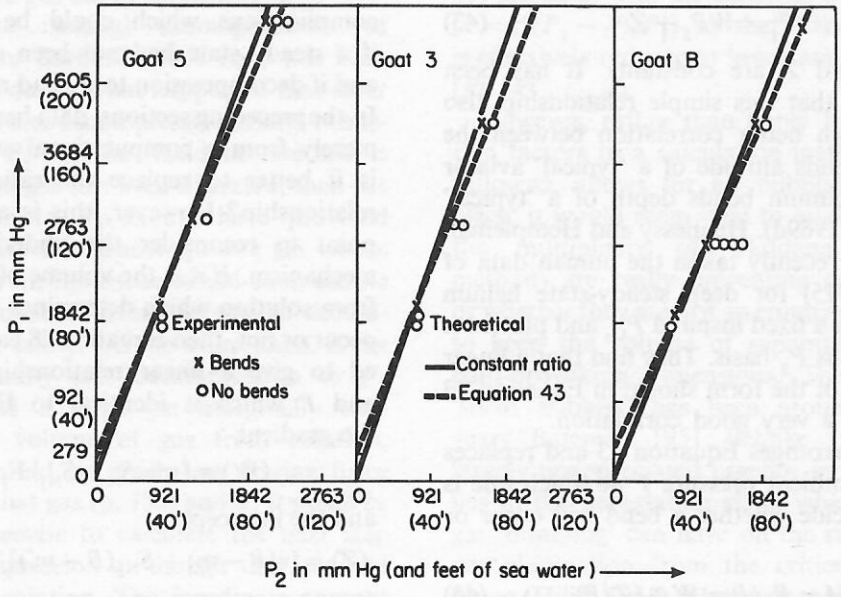


Fig. 35 Depicting the absolute pressure (P_2) to which the subject is rapidly decompressed after allowing 12 hours to reach steady state at P_1 while breathing air. Redrawn from Hills (1969d) using data from Hempleman (1957)

man found that the ratio offers an equally good interpretation of goat trials where the animal was not allowed to attain a steady state at P_1 but has remained at that pressure for a short interval before rapid decompression to P_2. Subsequent titration of P_2 for the same value of P_1 and the same time at this pressure showed that a straight line still offered the best relationship between P_1 and P_2 though it was steeper due to the lesser time for taking up nitrogen. For the same 'bottom time', the same 'tissue' of the 'Haldane' spectrum should determine the outcome of the dive, so this particular 'tissue' is no more depth-dependent than the slowest—if, indeed, they are different.

Linear $P_1 : P_2$ relationship

While a constant ratio offers a good fit for Hempleman's data, Hills (1969d) has shown that a straight line not passing through the origin, i.e. a linear relationship between P_1 and P_2, affords a complete separation of all 'bends' from 'no bends' points, so offering a better correlation of the same data. Thus the equation for a constant ratio ($P_1 = MP_2$) is replaced by

$$P_1 = WP_2 + Z \qquad (43)$$

where W and Z are constants. It has been pointed out that this simple relationship also offers a much better correlation between the minimum bends altitude of a 'typical' aviator and the minimum bends depth of a 'typical' diver (Hills, 1969d). Hennessy and Hempleman (1977) have recently taken the human data of Barnard (1975) for deep steady-state helium exposures on a fixed inspired P_{O_2} and plotted it on a P_1 versus P_2 basis. They find that a linear relationship of the form shown in Equation 43 again offers a very good correlation.

If one re-arranges Equation 43 and replaces P_2 by the ambient pressure P at which one is trying to decide whether a bend will occur or not, then

$$M = P_1/P = W + (Z/P) \qquad (44)$$

Hence the subsequent analyses of these classical experiments of Hempleman and Barnard would predict that a table designer committed to the ratio concept would need to allow for his M values to decrease with depth ($M\downarrow$ as $P\uparrow$ in Equation 44). It is also particularly interesting to see how the linear relationship lies intermediate between the constant ratio originally advocated by Haldane (Boycott *et al.*, 1908) and the fixed ΔP advocated by Hill (1912).

In passing, it is interesting to note that if gas were incompressible, the foregoing derivation of Equation 43 would give a value of unity for the gradient (W). By the same token, if the undesirable pressure differentials causing bends were provided by fluid shifts rather than bubbles, then the plot of P_1 versus P_2 should have a gradient of one—as found for vestibular symptoms in practice (fig. 20). Hence it is tempting to speculate (p. 72) that vestibular manifestations of decompression sickness may involve dysbarically induced fluid shifts rather than bubbles.

Vital question

The great theoretical advantage of analysing Hempleman's and Barnard's data is the opportunity to isolate a single decompression from the complex effects of time and the many other complications which could be put forward if a steady state had not been attained at P_1 and if decompression to P_2 had not been rapid. In the preceding sections, data have been viewed merely from a computational standpoint, i.e. is it better to replace the ratio by a linear relationship? However, this is an appropriate point to reconsider the underlying physical mechanism. *If* it is the volume of gas separated from solution which determines whether bends occur or not, then Equation 28 can be rearranged to give a linear relationship between P_1 and P which is identical to Equation 43 if the gradient

$$(W) = (v + S_{N_2})/S_{N_2} \cdot F_{IN_2} \qquad (45)$$

and the intercept

$$(Z) = [v(B - m) + S_{N_2}(B - m')]/S_{N_2} \cdot F_{IN_2} \qquad (46)$$

for air exposures. Moreover W and Z are constants if the volume of separated gas per unit volume of tissue (v) is now a constant for

marginal bends, since m, m', B, S_{N_2} and F_{IN_2} are all constants (see p. 103).

This still leaves open whether the critical tissue in question is predominantly (97%) aqueous (Hills, 1969d) when v and S_{N_2} are both small, or predominantly lipid (Hennessy and Hempleman, 1977) when v and S_{N_2} are both higher, S_{N_2} being the net solubility of nitrogen in the whole tissue, i.e.

$$S_{N_2} = fS_{FN_2} + (1-f)S_{AN_2} \qquad (47)$$

where f is the fractional lipid content of the tissue, while S_{AN_2} and S_{FN_2} are the solubilities of nitrogen in the aqueous and the fatty portions respectively. This issue is discussed in detail on p. 193.

However, there is a more important question left unanswered. Although the linear plot of P_1 versus P_2 for marginal bends looks highly compatible with the predicted volume of separated gas, this relationship could still represent the 'trigger point' to bubble formation—the concept essential to the whole 'Haldane' rationale, even to the ultimate levels of empiricism; but is this the case? In other words, *can a decompression which fails to violate any pressure ratio or linear pressure relationship still precipitate gas out of solution?*

Unlike the isolated decompressions of Hempleman or Barnard which have just been considered in detail, what happens if that diver attempts a further decompression after a bends-free stop at P_2? If the 'Haldane' method is correct and no gas has been liberated, then the decompression to P_2 should have provided a good gradient for removing more gas whose elimination from the tissue would soon enable the diver to make a further yet smaller decompression with safety. On the other hand, if the initial marginally safe decompression to P_2 had deposited a large yet marginally subsymptomatic volume of gas from solution, then there would be much less driving force to eliminate that gas (p. 138) and so *it would be particularly unwise* to calculate the next step in the decompression as though that gas had remained in solution. The inordinate amount of time which Barnard (1975) found necessary before his diver could attain another steady state at P_2 certainly points towards an appreciable 'dumping' of gas for sub-symptomatic decompressions. As a further example, there is the case of an 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). Hempleman (1969) points out that, by the 'Haldane' rationale, it would be necessary to assign a half-time to the tissue involved but this would then need to be some quite absurd value.

There have been several empirical attempts to allow for gas elimination during decompression being slower than uptake for the same estimated driving force, i.e. mathematical asymmetry. Notable among these are various unpublished factors by which to elongate dive time during decompression only. The same effect has been achieved by the 'bubble regression' factor of Crocker (1957) and, unwittingly, by the physical asymmetry afforded by using gas flow through orifices to simulate tissue gas exchange in the analogue designed by Kidd and Stubbs (see p. 129). On the other hand, Albano (1960) assumes that the rate constant for blood : tissue gas exchange (k in Equation 35) increases with the extent of decompression $[k = k'(P_1 - P)/P_1]$ so that 'tissue' half-time is effectively reduced at lower ambient pressure ($P\downarrow$, $k\uparrow$, $t_{1/2}\downarrow$).

However, rather than apply bubble correction factors to a calculation method which, if followed, allows for no bubbles in the first place, it would seem vital to establish whether the multiplicity of 'Haldane' calculation methods are really preventing gas separation or whether they are just an empirical concoction to keep the volume of separated gas below pain-provoking dimensions. The concept of 'silent bubbles' has been around for many years (Bateman, 1951; Behnke, 1951), but the largely unappreciated urgency in this question lies in the devastating effect which premature gas 'dumping' can have on the rate of overall gas elimination from the critical regions in tissue (Hills, 1966). However, before devoting the whole of Chapter 6 to the scientific evidence on this vital issue, it is necessary to look at

attempts which have been made to *model* bends provocation and hence prescribe preventive decompression on a more fundamental basis.

Models

In the days before commercial interests dominated the diving scene, the U.S. Navy chose to try to improve the 'Haldane' method as already described. On the other hand, the Royal Naval Physiological Laboratory opted for a new approach; although much of their work has not been reflected in the official R.N. tables until quite recently. This group of workers was basically dissatisfied with the extent of empiricism needed to force the 'Haldane' rationale to fit and the lack of suitable answers to such questions as the one asked earlier concerning why a theoretical insult to different hypothetical tissues (with different inherent susceptibilities according to their M values) showed no obvious differences in the symptoms observed. Clinically, there would seem to be no need to invoke more than one tissue to explain limb bends.

Single tissue model

Thus Hempleman (1952) introduced the concept that a single tissue may be responsible for marginal symptoms of decompression sickness. However, mathematically, the use of only one tissue in the calculation means that there is only one equation to use in designing a decompression and hence only that same equation for predicting the limits to no-stop decompression—i.e. for reproducing the whole bounce-dive curve. The linear time response (Equation 35), therefore, so popular in all decompression calculations, could not be used since no single exponential function offers even a moderate fit to the bounce-dive curve (see p. 36). In his search for a more appropriate time function, Hempleman therefore made a further major divergence from previous thinking in this field by abandoning the concept that blood : tissue gas exchange is limited by the circulation and that tissue is effectively fully-stirred (see p. 168). Instead, he selected a linear bulk diffusion model by adopting the approach of A. V. Hill (1928) in regarding extravascular tissue as a flat parallel-sided slab bathed on both sides by blood at arterial gas tensions. Hill had actually used this model to predict the transient uptake of oxygen by muscle on the start of heavy exercise by athletes but ignored metabolic oxygen consumption in deriving a $\sqrt{(t)}$ relationship which was therefore more appropriate for describing the transfer of inert gases.

The $\sqrt{(t)}$ relationship

Hempleman therefore incorporated this time function for overall gas uptake into his single-tissue model to give a single equation relating depth (H) to bottom time (τ) for a single exposure followed by no-stop decompression. Thus bends can occur if

$$H\sqrt{(t)} > \text{constant} = 475 \text{ fsw (min)}^{1/2} \quad (48)$$

This very simple expression has provided a particularly good fit to the experimental bounce-dive curves both for air diving up to 100 min and the use of 80 : 20 helium : oxygen mixes. If a constant of 500 fsw $(\text{min})^{1/2}$ is used, this fits the U.S.N. no-stop limits very well. Moreover, if minor allowances are made for the different contributions made by metabolic and inert gases to total separated gas volume (Hills, 1975a), then the $\sqrt{(t)}$ relationship shows a near-perfect fit to both air and heliox no-decompression limits for bottom times up to 180 min (see p. 189).

Assuming the principle of superposition, Crocker and Taylor (1952) used the Hempleman equation (48) to formulate a set of tables for short-duration dives. These again resulted in a better correlation of bends incidence than offered by standard 'Haldane' methods available at the time, the greatest exposure tested being 25 min at 180 fsw.

The $\sqrt{(t)}$ relationship also provides a very good description of the initial phase of nitrogen wash-out from the whole body during oxygen breathing (Eggleton *et al.*, 1945). These are the type of data which, yet again, require multiple equations and many more degrees of

freedom if analysed using the more conventional exponential functions (Jones, 1951). It has also been pointed out that the uptake expression for bulk diffusion into any shape approximates to a $\sqrt{(t)}$ for small values of time (Hills, 1966)—i.e. for minimal gas penetration, any surface can be taken as effectively flat.

However, while the $\sqrt{(t)}$ relationship may provide a good fit for relatively short time intervals, it must eventually break down for large values of t, since $\sqrt{\infty} = \infty$. In other words, tissue cannot go on taking up inert gas indefinitely but must eventually reach a steady state. In order to prolong the fit to higher values of t, the U.S. Navy suggested empirical modifications by replacing $\sqrt{(t)}$ with a power function, t^{μ}, with μ ranging from 0·38 to 0·75 (Duffner *et al.*, 1959) but this must still become infinite for $t = \infty$.

Thin-slab model

Instead, the group at R.N.P.L. preferred to modify their model, or rather, to apply their mathematics a little more rigorously to their initial model. After all, by adopting the equation of A.V. Hill, they had effectively assumed an infinite thickness to their parallel-faced tissue slab. This modification was made by Rashbass (1955) who retained the first three terms of the Fourier series of the linear bulk diffusion equation (p. 246) to give the mean tissue tension $\bar{p}$ at time (t) following an increase in ambient pressure from P_0 to P_1 as

$$\bar{p} = P_1 - (P_1 - P_0)\frac{8}{\pi^2}\left[\exp(-k''t) + \frac{\exp(-9k''t)}{9} + \frac{\exp(-25k''t)}{25}\right] \quad (49)$$

Rashbass then compares this mean tissue gas content with the ambient absolute pressure (P) on the basis of the fixed ΔP originally advocated as the 'trigger point' for bubble formation by Hill (1912). Thus he reasons that bends will not occur if

$$\bar{p} - P \leqslant 30 \text{ fsw} \quad (50)$$

The method still invokes the concept of a metastable limit to true supersaturation, assuming no separation of gas from solution provided the condition expressed by Equation 50 is not violated. The critical limit to supersaturation is derived by reasoning that a 'weak' diver has a minimum bends depth of 33 fsw, to give $\bar{p} = P_1 = 66$ fsw in Equation 49 for steady state $(t = \infty)$ at P_1. Thus for decompression to the surface, $(P = 33$ fsw), $P_1 - P = 33$ so that, after allowing a 10% margin for error, 30 fsw represents a conservative threshold or 'trigger point' to bubble formation.

Unfortunately the sea trials of the air tables calculated by this method were disappointing (Crocker, 1957) but led Crocker to retain the format simply as 'a convenient mathematical instrument' to which he then applied his empirical 'bubble regression factor' already described. The Hempleman–Rashbass concept of determining the outcome of a dive by calculating the total amount of gas taken up by a uniform bulk of extravascular tissue and then comparing this value with the ambient pressure, was then extended to a radial model by Wittenborn (1963).

Bulk diffusion

These 'bulk diffusion' models are particularly attractive theoretically, since they permit the whole bounce-dive curve—and hence the exposure limits for gradual decompression—to be predicted *by one equation*. Moreover, this correlation is much more meaningful than any 'Haldane'-type 'fit' because there are only two constants to select empirically—one related to inherent susceptibility (e.g. 30 fsw in Equation 50) and the other to the time base (e.g. k'' in Equation 49). With only two degrees of freedom, the comparison with practical data therefore offers a much more rigorous test of the model and hence the resulting basis for formulating tables.

However theoretically attractive these bulk diffusion models may appear in interpreting the bounce-dive curve, it was subsequently pointed out that the constant needed to give a realistic time base implies a diffusion coefficient (D) several orders of magnitude lower

than values generally accepted for gases in water or in gross tissue sections (Hills, 1966). The time constant (k'') is related to D by $k'' = \pi^2 Dt/4b^2$, so that the value considered by Rashbass to give the best fit of practical diving data ($k'' = 0{\cdot}00793$ min^{-1}), implies that $D/b^2 = 5{\cdot}4 \times 10^{-5}$ sec^{-1}. Even for a large intercapillary distance of 40 μm ($b = 2 \times 10^{-3}$ cm), this implies a diffusion coefficient of $2{\cdot}1 \times 10^{-10}$ cm^2 sec^{-1}—many orders of magnitude smaller than those in the region of 10^{-5} cm^2 sec^{-1} used for water or tissue (p. 182). Similar differences are implied in the constant in Hempleman's $\sqrt{(t)}$ equation (48) and to the value of $(D/a^2) = 0{\cdot}129$ min^{-1} needed in the radial model (Hills, 1966), where a is the capillary radius. Despite the remarkable fit which these bulk diffusion equations offer for the bounce-dive curves, the general physiological literature assumes that blood : tissue gas exchange is limited by the circulation. Hence apparent discrepancies in D values of such magnitude cannot be ignored, particularly in approaches which are claimed to be more fundamental. This second vital issue concerning the relative contributions of diffusion and blood perfusion in controlling gas uptake is discussed in detail in Chapter 7.

Sub-symptomatic decompression

In the bulk diffusion approaches described so far, bounce dives are analysed, in essence, simply by estimating the total gas assimilated by the single tissue at the maximum depth of exposure. If this estimate does not exceed a critical constant, then the diver can return to the surface rapidly and safely. The fact that one equation can provide such a good fit to such a unique no-stop decompression curve without invoking more than one or two degrees of freedom in the calculation really indicates that the time function is particularly relevant (Hills, 1969e). However, it in no way validates the rest of the equation, such as the P in Equation 50, which would always be constant for the unique aspects of this particular set of data—i.e. immediate decompression to the surface.

Purely from a calculation standpoint, the total gas taken up at maximum depth would be the only relevant variable whether bends were determined by the volume of gas or by critical supersaturation in which almost any reasonable equation could be used to describe the 'trigger point' under these unique conditions. In other words, the analysis of bounce-dive data avoids the vital issue concerning what happens after a sub-symptomatic decompression in the tissue of the diver who has exceeded the no-stop exposure limits. Any inadequacy of the remainder of the expression (Equation 50), i.e. the terms independent of time, would only show up in regular staged decompression and could explain the disappointing sea trials of tables based on the combination of Equations 49 and 50.

Reasoning such as this led Hills (1966) to propose that, since cavitation is essentially a random process (Chapter 4), decompressions should be programmed according to the 'worst possible case', i.e. to the few micro-regions of the critical tissue where there is local thermodynamic equilibrium between the phases. This critical micro-region where suspended transformation (p. 77) has broken down is regarded as the 'worst possible' not only because it represents the maximum number of gas molecules which can separate from solution but this 'dumping' of gas leaves the minimum driving force for its subsequent elimination from the tissue as a whole. This 'thermodynamic' approach is discussed in more detail later (Chapter 9) but departs radically from previous theories by advocating much deeper decompression stops with more time to be spent at the deeper stops than prescribed in the more conventional tables based upon the 'Haldane' rationale and the other methods described so far.

Primary event versus critical insult

The models and calculation methods described so far contain no differentiation between the primary event in inducing decompression sickness and the critical level of insult needed to precipitate clinical symptoms. These terms have been defined in more detail earlier (p. 48) and considerable attention has been paid to this difference in discussing mechanisms qualita-

tively. However, in actual calculations, all of the foregoing methods assume that the criterion for bubble formation coincides with that for the eventual onset of bends. Thus the same 'trigger point' is used for optimizing as for analysing decompressions. Indications that this may not be valid are numerous, one of the first being the different set of *M* values needed by early U.S.N. workers to design air-diving tables compared with the higher values obtained from analysing the air bounce-dive curve (see p. 115). Most later designers argued that, since the no-stop decompression analysis referred to bends on the surface while most stops in a programmed decompression are deeper, this simply reflected a depth-dependent ratio as already described.

However, a fundamentally different attitude was taken by Hills (1966) whose 'thermodynamic' approach makes a distinction between *the primary event* as the point of bubble inception and *the critical level of insult* as the volume of gas needed to separate from solution to provoke pain or other symptoms. Thus allowance for 'silent bubbles' is made in the mathematics. At least these criteria are used for analysing dives based upon any other model or calculation method, while their use for designing a decompression leads to two simultaneous computations:

(1) calculating the volume of gas which would separate from solution if the subject were to surface directly from that point in the profile but taking no action on the result of this continually updated calculation until the predicted volume was less than that known to provoke pain;

(2) using the phase equilibration (true saturation) criterion to avoid bubble inception as the basis for programming decompression up to the stage where (1) indicates that it is safe to surface.

This leads to profiles where there is deeper initial staging but the diver can make a relatively large jump at the end when, according to this theory, he is permitted to 'dump' gas but to just below the pain-provoking amount. This 'drop-out depth' is usually in the region of 20–30 fsw, its selection being described in more detail in Chapter 9.

This writer is of the opinion that, in the immense empirical modifications to the 'Haldane' method, many table designers are effectively making much the same distinction between the primary event and the critical level of insult. After all, in many of these popular tables, the diver may only make a 10 fsw decompression when the designer knows quite well that the man would run no risk of bends if brought 20 fsw closer to the surface. This is contrary to the basic 'Haldane' philosophy where a 20 fsw decompression would have provided more driving force for gas elimination than a 10 fsw decompression. This inconsistency in the 'Haldane' rationale is expounded in more detail later as a method for differentiating between critical supersaturation and phase equilibration as the relevant criterion for bends (see p. 158).

Seek the peak

If the concept of phase equilibration is taken to its limit, then the criterion for gas separation should be applied to the highest local value. Thus Hills (1966) further diverged from previous bulk diffusion models by applying his critical conditions to the peak of the gas distribution rather than to the mean total gas tension averaged over the whole tissue (see Chapter 9). This increases the mathematical complexity by introducing bulk diffusion equations which are functions of both time *and distance*, but does not invoke any more degrees of freedom and so retains the immense advantages of all bulk diffusion models based upon the single-tissue concept.

Mixed perfusion: diffusion models

Studies to try to estimate the relative contributions of both bulk diffusion and blood perfusion to blood:tissue gas exchange (Hills, 1967b; Vann *et al.*, 1976) and accompanied by 'transient' analyses which can allow for both, have led to recent modifications in which a circulation-based resistance is placed in series with the tissue bulk (Chapter 9). This avoids the assumption previously underlying bulk diffusion models that venous blood leaves

tissue at arterial tensions of the inert gases. These modifications confine the blood to a constant volume compartment in which arterial blood flows in and venous blood is effectively the overflow. If this 'tank' is assumed to be effectively fully-stirred, this represents a simple linear resistance in series with the tissue bulk in which the height of the peak is still the decompression-determining parameter. This 'fully-stirred tank' has been used in series with the flat parallel-faced slab (Krasberg, 1976) or its surface may be envisaged as cylindrical (Hills *et al.*, 1976) when it is geometrically compatible with the radial diffusion model. However, the introduction of a circulation resistance adds two degrees of freedom to the model: the rate of blood flow and the capacity of the tank.

An assumption made in all of these models is that the rate of growth, or the coalescence of 'dumped' gas into its pain-provoking form, does not significantly affect the outcome of the dive. In fact, all of the models and calculation methods described so far in this chapter effectively terminate the calculation at the moment that the diver reaches the pressure at which the designer needs to know the imminence of decompression sickness. At least, the computation may continue but subsequent calculation does not influence the decision taken to move to that pressure. However, this is not true of one other class of model where the rate of growth of the gaseous phase to its pain-provoking form is considered to be the major factor.

Growth models

U.S. Air Force workers during the Second World War were not only interested in predicting whether a bend would occur but when it would occur. Thus Nims (1951) and Bateman (1951) consider the rate of transfer of gas molecules from solution in tissue to the gaseous phase to be the rate-limiting process determining the onset of symptoms. They regard the manifestations of decompression sickness as a matter of the position and size of bubbles whose initiation is not a limiting factor while, according to Nims, it is not even rate-contributing. In justification of this view, Harris *et al.* (1945a), Harvey (1945) and Whitaker *et al.* (1945) are quoted as having observed gaseous cavitation in animals provoked by exercise *without change of pressure.*

Nims derives an equation to relate the pressure of gas in a bubble to time and to the degree of supersaturation of the surrounding fluid by the gas. When this bubble pressure exceeds the pain threshold according to the concept of Inman and Saunders (p. 59), a bend should occur. Thus Nims offers a good correlation between the extent of aerial decompression and the recorded onset times for symptoms. However, this approach assumes a fixed bubble radius during growth limited by diffusion and yet regards tissue as effectively fully stirred when estimating blood:tissue gas exchange, i.e. then ignoring these concentration gradients around the bubble. Perhaps the largest assumption, though, arises from the way in which the deposition of gas into the bubble is ignored in determining mean tissue tension and hence the driving force for gas elimination *via* the circulation.

These objections are circumvented to some degree by Bateman (1951) who considers bubbles growing in a well-stirred fluid. His growth equation is derived from the *in vitro* experimental studies of Bateman and Lang (1945) who measured the overall expansion of decompressed gas solutions dilatometrically. Hence it is probable that his growth equation is also a function of some nucleation in addition to gas transfer across the blood: bubble interface.

Bateman's correlation of threshold altitudes for various periods of pre-oxygenation is as good as the very random scatter of experimental values permits. However, in attempting to formulate the staging of a diver, his theory completely breaks down, leading to his very honest admission that his equations 'have been painstakingly examined only to be completely demolished by the final demonstration of fundamental inadequacy' (Bateman, 1951). Re-examination of his calculations by this writer indicates that, for diving, his critical tissue ran out of driving force for gas elimination to blood soon after decompression passed the

position of phase equilibrium, so that too much gas was theoretically 'dumped' too soon. However, if air is not assumed to be one gas, i.e. the principle of the inherent unsaturation is invoked to provide that driving force (see p. 239), then his method gives reasonable values for the onset of bends in divers in addition to aviators.

Critical size of nucleus

Another approach which is based upon the existence of preformed nuclei is that of Albano (1960). His justification is better described in his own words as 'les mouvements, l'activité fonctionelle des différents systèmes, l'existence des cavités trés hydrophobiques dans les tissus permettent la formation et la stabilité des noyaux'. Albano is primarily concerned with the dependence of nucleus radius (r_n) upon ambient absolute pressure (P), pointing out that $r_n \propto P^{-1/3}$ on the basis of Boyle's Law. Thus a nucleus reduced in size by going to greater depth is less likely to be activated into growth.

Decompression Meters

No set of diving tables can be sufficiently comprehensive to cover all combinations of 'bottom' depth and exposure time or to account for variation in pressure due to tide or the vertical travel often required in completing an underwater task. For this reason the diver employs the decompression schedule computed for the minimum exposure which encompasses his actual exposure. Accordingly, if he has reached a maximum depth of 191 fsw for however short a period during an 11-minute dive, then he must use the table for 20 min at 200 fsw—if following the U.S.N. manual for instance. Clearly this is uneconomical if decompression is costing money. It might be possible to have a 'topside' computer produce an immediate schedule for each individual exposure but, even if the dive history could be relayed to the surface, this must remain a pipe-dream until these devices are much cheaper, much easier to operate and much more resistant to physical abuse.

What is therefore needed is a robust meter which the diver carries with him to assimilate the complete history of the exposure and then give him an immediate indication of what to do in order to follow a decompression especially optimized by the device for that particular exposure. However, this ideal situation has two potential shortcomings:

(a) failure of the mathematical model or calculation method to simulate the actual processes *in vivo* which are inducing decompression sickness;

(b) failure of the analogue system adopted in the meter to follow that model or calculation method due to inadequate mechanical or electrical design.

The first of these applies equally to table formulation as already expounded in the foregoing part of this chapter. However, the second has led to much criticism of meters which is most unfortunate because, as a concept, they would appear to offer the most efficient and convenient means of prescribing decompression. Not only do they allow for unique exposures but they can be 'tailored' to the individual diver so that a less susceptible individual can be decompressed more rapidly if facilities permit.

The shortcomings in the early meters stemmed largely from the use of pneumatic systems, any engineer knowing how difficult it is to predict the flow of gases at low rates and how sensitive these are to a number of parameters (Coulson and Richardson, 1965). Before describing actual designs, however, it might be useful to view the whole range of systems which can be invoked as analogues for the decompression process *in vivo*.

Simulation of gas transfer in vivo

Whether gas transfer *in vivo* is limited by blood perfusion or diffusion (Chapter 7), the rate is taken as proportional to the tension gradient (or *local* gradient when simulating bulk diffusion). Hence any analogue for this system requires the extensive entity providing the flux (simulating tissue gas) to be transferred

Table 7 Corresponding parameters in bulk diffusion and physical systems which can be used to simulate it

Basic physical process	Diffusion	Thermal conduction	Electrical conduction	Fluid flow
Extensive quantity transmitted	dissolved gas	heat	charge	fluid volume
Intensive parameter (driving force)	gas tension	temperature	potential	pressure
Resistance factor	diffusion coefficient (D)	thermal conductivity (κ)	resistance (R'')	flow resistance (Ω)
Time base (dimensions: time^{-1})	(D/a^2)	($\kappa/s\rho a^2$)	$R''C''$	$\Omega C'$
Other constants needed	capillary radius (a)	core radius (a) specific heat (s) density (ρ)	capacitance (C'')	chamber capacity (C')

in direct proportion to the driving force which, in turn, is determined by the distribution of the corresponding intensive parameter. Various combinations of extensive and intensive parameters are given in Table 7. The major skill in deriving analogue systems comes in selecting the components to ensure temporal similarity to tissue and geometric similarity where appropriate. The second of these simply involves appropriately scaling the capacities but the time base may prove much more elusive to define.

One convenient method involves combining the constants into a group with dimensions of time only (Hills, 1966). Thus any tissue has a time base given by $\lambda\dot{Q}$ (Equation 38) if uptake is perfusion limited, $\pi^2 D/L^2$ (p. 124) if there is linear bulk diffusion, D/a^2 (p. 247) if there is radial bulk diffusion, etc. where each group has the dimensions of (time)$^{-1}$. These must be equated to the corresponding group of the analogue system, such as RC for a combination of electrical resistance (R) and capacitor (C) in series or $\kappa/(b')^2$ for a radial thermal conductor, etc., where each group of constants must also have the dimensions of (time)$^{-1}$. If the analogue is required to generate schedules rather than to act as the basis for a meter, then a high ratio of the analogue to tissue time base can be selected to accelerate table production.

The systems actually exploited so far are either electrical, pneumatic, hydraulic or thermal. Each is primarily based on one of the calculation methods or models for prescribing decompression already discussed earlier in this chapter.

'*Haldane*' *models*

The concept of a physical model to simulate the 'Haldane' rationale has been traced back to the Scripps Institute by Howard and Bradner (1976). In a proposal submitted to the U.S. Office of Naval Research in 1953, Groves and Munk described five hydraulic schemes employing fluid flow through capillaries or porous slabs into one or two chambers, while the Foxboro company produced a prototype of a two-chamber 'parallel' model (Fredrickson, 1956).

The first commercial unit (S.O.S./Scubapro) was designed by De Sanctis in Italy and consisted of a watertight chamber fitted with a Bourdon tube (pressure gauge) separated from a distensible gas-filled bag by a porous plug (Alinari, 1964). This pneumatic resistor (fig. 36) was selected to enable the pressure to rise in the chamber with a half-time of the order of 30 min. This rise was then registered by the pressure gauge, the scale of which was designed to incorporate a decompression ratio in indicating the minimum depth to which it would be safe for the diver to ascend. Such a system would represent a one-tissue 'Haldane' model if it were not for the deviation from linearity between gas flow and pressure difference which occurs in using *compressible* fluids, unless one

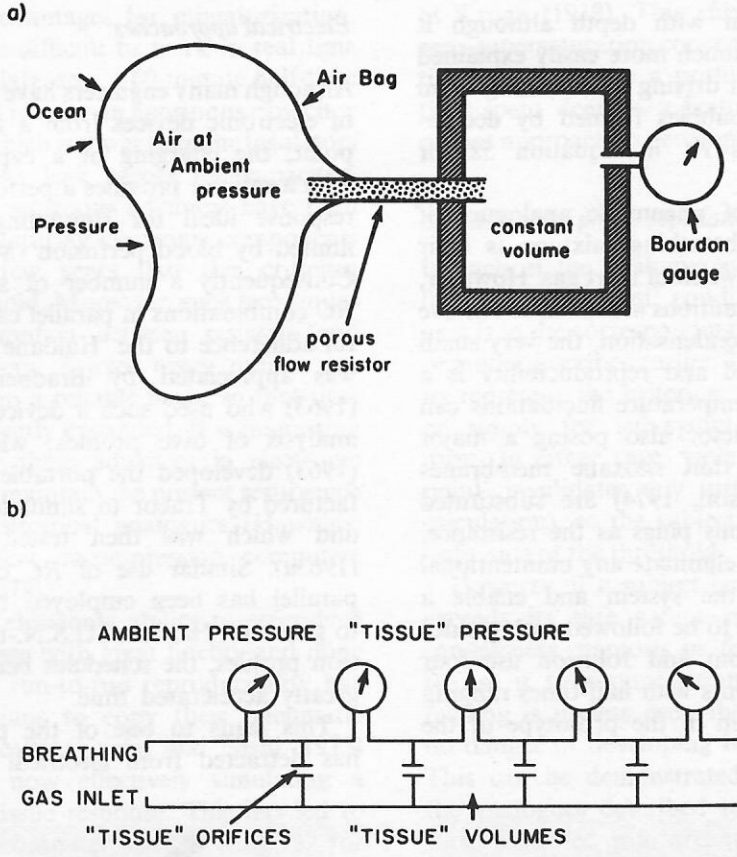


Fig. 36 Schematic diagrams of two pneumatic decompression meters which have been produced commercially: (a) the simple S.O.S. meter (Italian patent No. 624, 174) and (b) the 'series' version (MK III P) of the Canadian meter designed by Stubbs and Kidd (1965a)

is working within the Knudsen flow range, i.e. with small pressure drops. This means that there is mathematical asymmetry between uptake and elimination with gas following a slower time course in coming out of the watertight chamber than in going in. If gas separates from solution for typical M values, then this deviation from a true Haldane 'tissue' would be an advantage (Hills, 1966).

This also applies to the Canadian pneumatic analogue designed by Stubbs and Kidd (1965a, b) with four rigid chambers in parallel, each with its own orifice size, to simulate four Haldane 'tissues'. These were subsequently placed *in series* as shown in fig. 36 with some empirical adjustment of the relative capacities and orifice sizes (Stubbs and Kidd, 1967). Moreover, it was shown that this revised model gave a lower bends incidence than the U.S.N. tables which it was designed to simulate in the original model (Kidd *et al.*, 1972). However, it is interesting how these successful modifications have led to a model which simulates bulk diffusion rather than any 'Haldane' rationale (Hennessy, 1973b).

A feature of the Canadian meter is mathematical asymmetry which undoubtedly arises from not working within the Knudsen flow range, although symmetry was implied in their original 'parallel' design. This has led the designers to the conclusion that gas is eliminated from the critical tissue(s) in accordance with a slower time course than it is taken up. Weaver (1967) has attributed this to an increase in the

diffusion coefficient with depth although it would seem to be much more easily explained by the reduction in driving force arising from sub-symptomatic bubbles formed by decompression ($\Delta P_{N_2}\downarrow$ as $P\downarrow$ in Equation 52 for *separated* gas).

An advantage of pneumatic analogues, if connected to the breathing mixture, is their ability to 'sense' a switch of inert gas. However, unless extreme precautions are taken to remove dust and prevent condensation, the very small orifices get plugged and reproducibility is a major problem. Temperature fluctuations can be annoying, a factor also posing a major difficulty if very thin siloxane membranes (Borom and Johnson, 1974) are substituted for orifices or porous plugs as the resistance. However, these do eliminate any unintentional non-linearities in the system and enable a 'Haldane' rationale to be followed with greater fidelity. Thus Borom and Johnson use four parallel compartments with half-times ranging from 44 to 144 min in the prototype of the G.E. meter.

Electrical approaches

Although many engineers have a deep suspicion of electronic devices from a reliability standpoint, the charging of a capacitor in series with a resistor provides a perfectly exponential response ideal for simulating tissue uptake limited by blood perfusion (Mapleson, 1963). Consequently a number of suitably selected *RC* combinations in parallel can provide faithful adherence to the 'Haldane' rationale. This was appreciated by Bradner and Mackay (1963) who used such a device for laboratory analysis of dive profiles; while Wittenborn (1963) developed the portable version manufactured by Tracor to simulate U.S.N. tables and which was then tested by Workman (1963a). Similar use of *RC* combinations in parallel has been employed by Todd (1969) to generate 'Haldane'/U.S.N.-type decompression profiles, the schedules being produced in greatly accelerated time.

This leads to one of the problems which has detracted from electrical meters despite

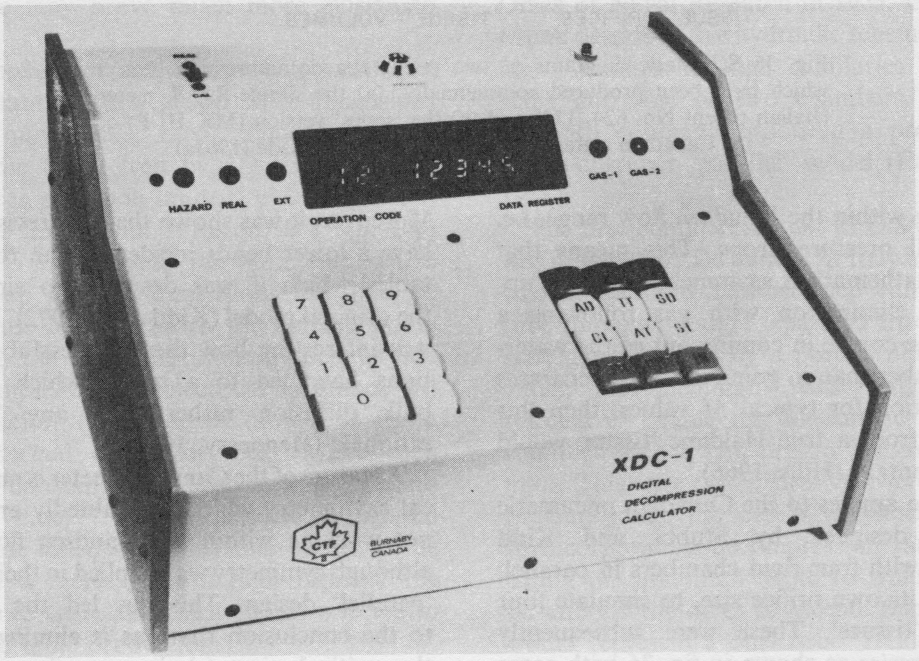


Fig. 37 The digital decompression calculator based upon the Canadian 'series' pneumatic meter (fig. 36(b)). Reproduced by permission of Canadian Thin Films Ltd., Toronto

their obvious advantages for miniaturization. It is much more difficult to work in real time because to simulate, say, a 60-minute half-time 'tissue', it would require an enormous capacitor or a resistance which is lower than the insulators around it or the leak across the capacitor. However, such enormous advances have been made in manufacturing electronic components over the last few years that this criticism may no longer hold. Moreover, such techniques as restricting contact between resistors and capacitors to, say, 3 μsec every minute are now available on a reliable basis, so that time scales can be greatly expanded. It is interesting to see whether these advances in electronic technology will maintain the present resurgence of interest in electrical analogues (Hawkins, 1974) and pocket decompression computers (Seiple *et al.*, 1973).

This current electronic ability to reproduce any time response with great fidelity and none of the fears of run-to-run reproducibility has led the Canadians to copy their pneumatic meter electronically (Kuehn and Nishi, 1975) and they are now effectively simulating a simulation of tissue response. This has led to the handsome computer shown in fig. 37 for instantaneously generating a decompression table according to the time response semi-empirically evolved in their 'series' pneumatic meter. While this represents a deviation from Haldane traditions in the tissue transport model it adopts, a total departure is effected in two systems which also dispense with the ratio by invoking thermodynamic criteria for gas separation (Chapter 9).

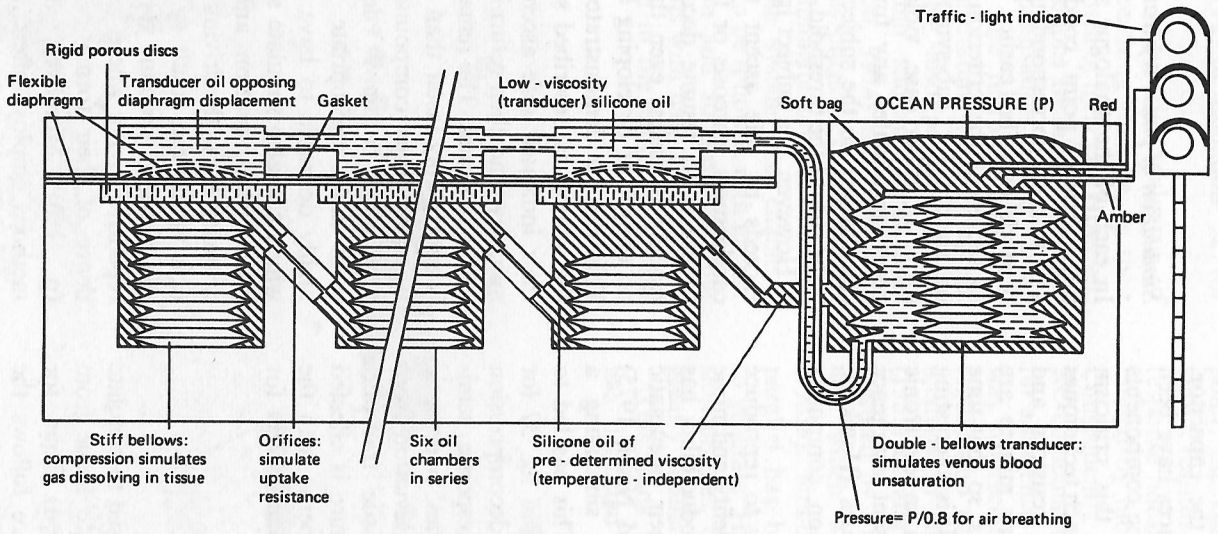
Thermal analogue

Thermal conduction has been used to simulate the radial diffusion of gases into tissue from a blood capillary (Hills, 1967c,d). Thus, for air diving, the core temperature follows the inspired nitrogen partial pressure. Decompression is optimized by decreasing the absolute pressure until its equivalent temperature coincides with the peak temperature monitored by thermocouples placed in the large block of asbestos simulating the classical tissue model of Krogh (1918). This effectively invokes the zero-supersaturation concept for bubble formation and produces a profile with much more time spent deeper—a feature so characterstic of that approach to decompression (Chapter 9).

Simulation of phase separation

In each of the analogue systems described so far, a 'trigger point' condition is applied just as it is in the corresponding calculation method or model described earlier. This may be intended to represent the criterion for bends to occur or simply for sub-symptomatic bubbles to form. In either case, violation of the 'trigger point' invalidates any further simulation (or calculation), so the subject is kept just on the 'safe' side of the threshold.

However, if a subject ignores his meter and exceeds its safe ascent rate for reasons of carelessness, narcosis or fear but later wishes to use it to resume decompression, then its reading is useless even though he may be in no danger of developing bends (Hills, 1975b). This can be demonstrated by taking any of the analogues described so far and exceeding their indicated safe ascent by 10 fsw for 10 min, say, and then returning to obey what the device indicates. The remaining decompression is appreciably faster than if there had been no violation, thus compounding the error at a time when a *slower* decompression would seem more appropriate. Consequently, it is highly desirable to have an analogue system which can also simulate separation of the gas phase from solution and so allow for the dramatic fall in the driving force for gas elimination which can result (p. 137). There is also the need to account for 'silent' bubbles in repetitive diving where there is abundant evidence of their formation between exposures (p. 150). Moreover, different calculation methods employ different 'trigger points', so that a profile which would prevent phase separation by one rationale may violate the thresholds of another. Hence it is also desirable to be able to allow for bubble formation in analysing one format by the criteria pertaining to another.



Decompression Meter

Fig. 38 The hydraulic decompression meter designed to allow for the fall in the driving force for eliminating gas if the diver ignores the optimal procedure indicated by the instrument and inadvertently causes gas to separate from solution in the critical tissue. From Hills (1975b)

Arguments such as these led this writer to use rigid chambers not unlike those shown in fig. 36 but to connect each to a cylinder closed with a piston to which the absolute pressure of the diver is applied to the remote side. Thus, if the gas pressure in any chamber (simulating the total gas tension at the corresponding point in tissue) exceeds the absolute pressure, then the piston will move until there is equal pressure on both sides. The increase in effective chamber volume is proportional to the volume of gas 'dumped' at that point and also cuts off the driving force for gas elimination (fig. 83). However, the 27-compartment analogue (Hills, 1967e) suffered from some of the maladies common to other pneumatic systems, while oil-leaks around pistons restricted its use to laboratory analysis of profiles.

These problems have been overcome in a recent 'hydraulic' version (Hills, 1975b) where a viscous silicone oil is used as the transfer medium to reduce temperature effects, to reduce size and to offer much wider engineering tolerances in orifices. The compression of a sealed evacuated bellows is used to simulate gas dissolving in tissue, i.e. Hook's law is invoked as an analogue for Henry's law. A leak-proof diaphragm replaces the pistons for simulating phase separation, their displacement activating a traffic-light indicator selected as a very simple display for a practical diving meter. The version based on the thermodynamic approach (Chapter 9) is shown in fig. 38 but the same principles of simulating gas transfer and separation can be applied to most other approaches.

One reason for avoiding electronics in allowing for 'silent' bubbles is the difficulty of storing the charge (used to simulate gas) at a fixed potential (representing pressure). However, with modern methods of facilitating analogue–digital conversion, it may now be feasible to devise an electronic analyser.

Oxygen analogue

All of the meters described so far have been concerned with optimizing depth against time

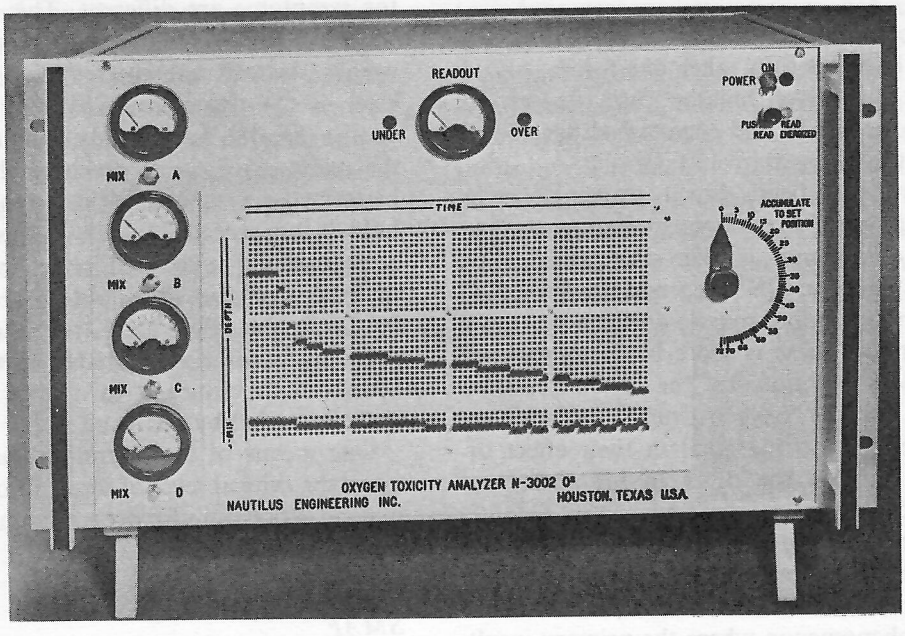


Fig. 39 A desk-top instrument for testing a decompression profile and automatically indicating the imminence of oxygen convulsions at any point in any dive which involves no more than four different breathing mixes. Reproduced by permission of Aquanaut Developments Inc. and Nautilus Engineering Inc., Houston

with the imminence of decompression sickness dictating the outcome. However, where the composition of breathing mix can be varied, convulsions can be equally undesirable, the substitution of oxygen for inert gas greatly accelerating decompression. Quantification of this process is described in Chapter 8, leading to a simple calculation method for estimating the imminence of oxygen toxicity (particularly the CNS variety). This has now been put into the form of an electrical desk calculator (fig. 39) where the cumulative oxygen toxicity index (COTi in Equation 79) can be immediately estimated at any point selected on any decompression profile fed into the device, provided no more than four gas mixes will be employed on the dive. This can be used either for optimizing the use of oxygen or for estimating the safe dosage to prescribe as treatment for a case of bends occurring *during* decompression, thus minimizing the risk of a convulsion.

Pharmacological Approaches to Prevention

The 'bends pill'

Pharmacological approaches to decompression sickness have basically taken two forms:

(a) let the diver breathe pure oxygen to avoid the bends and then use drugs to try to prevent oxygen toxicity, or

(b) prevent the bends directly by using plasma expanders, anticoagulants, hydration, surface active compounds, cardiac stimulants, ganglionic blockers, CNS depressants, bronchodilators or smooth-muscle antagonists.

The first of these is more likely to provide a complete solution to the decompression problem but the 'oxygen drugs' produced so far are either too marginal in their effect or seriously impede the diver in his ability to work underwater.

The second approach has not yet shown more than marginal success largely because, in the opinion of this writer, it is difficult to avoid a phenomenon where the primary insult is essentially *physical* by employing *Chemical* means. However, there are a number of pharmacological agents which can sometimes provide a cure for a marginal bend and, when such medication is taken *prior to* the predicted onset of mild symptoms, it would appear to have just as good a chance of preventing their appearance in the first place. These techniques will therefore be described later in more detail in connection with treatment (see Chapter 8), notable among them being the administration of low-molecular-weight dextran (Cockett and Nakamura, 1964) and heparin (Barthelemy, 1963).

Effect upon models

The action of pharmacological agents is ignored by most proponents of models and calculation methods for formulating diving tables. Their mild preventive action and the impression that they are really affording some degree of *treatment* to an underlying abnormal state is certainly much more compatible with 'silent bubble' theories than with any based on a 'trigger point' to bubble formation. Hence this impression also favours the attitude that the primary event and the critical level of insult for symptoms are different. This writer takes the view that agents such as low-molecular-weight dextran act by reducing the overall level of the tissue insult over its whole time course (fig. 18). In this way, administration of the agent prior to its reaching the peak can be termed prevention if it can avoid the threshold being exceeded; while it is successful as a treatment if it can reduce an insult already in excess of the threshold to a sub-critical level. On the other hand, potentiating agents such as serotonin probably act upon the threshold to reduce it to a level more likely to be exceeded by the insult.

One means of incorporating such an effect upon the critical level of insult into the mathematical expressions for programming preventive measures is described on p. 58.

SMAF

However, a preventive pharmacological approach which is more difficult to interpret as simply a treatment for 'silent bubbles'

or their side effects is based upon the smooth-muscle activity factor (SMAF). Chryssanthou *et al.* (1964) showed that bradykinin given immediately after a one-minute decompression from an air exposure of six hours at 90 psi markedly decreased the survival time of obese mice. It was therefore argued that an antagonist for bradykinin administered before decompression might prevent decompression sickness. However, tissue extracts from decompressed mice showed no evidence of bradykinin activation but did reveal elevated levels of a previously unidentified smooth-muscle activity factor (SMAF) in lung (Chryssanthou, 1974). SMAF elicits slow, weak contractions of smooth muscle causing broncho-constriction, increases vascular premeability, potentiates bradykinin and has now been found in blood, kidney and other organs.

SMAF also increased the susceptibility of *thin* mice, particularly if administered before decompression (Chryssanthou *et al.*, 1971). This potentiation of decompression sickness was eliminated by prior doses of compounds, such as PPCH, with antagonistic activities towards smooth-muscle-stimulating substances. However, when used to prevent decompression sickness without potentiation of the disease by SMAF, PPCH offered considerable protection—but only in obese mice (Chryssanthou *et al.*, 1964). The switch to other agents active in countering bradykinin and ones already approved for clinical use such as cyproheptadine and dimethothiazine, offered no protection 'at a single dose level' (Chryssanthou, 1973). It did, however, offer a 'striking' preventive action at an elevated dose range.

No positive evidence of any beneficial effects of these antagonists in preventing decompression sickness in larger mammals has yet been forthcoming. Whether SMAF, bradykinin or any other smooth muscle stimulant is directly responsible or not, this work again suggests the involvement of a humoral factor in some forms of decompression sickness (p. 56). However, this work was largely based upon trials scored on the basis of death or recovery of the animals, so that a different threshold could be involved by comparison with limb bends in man.

Primary event

These findings of Chryssanthou and co-workers must convey a word of caution to many of us who tend to become enraptured with physical models to the exclusion of the truly biological element. Even so, it can easily be argued that the unique symptoms of decompression sickness are not produced without decompression or some other phenomenon, such as counterdiffusion supersaturation, which is likely to induce gaseous cavitation in tissue. Hence the primary event is still most likely to be bubble inception, whether the critical insult to the tissue which this initiates is mediated physically, humorally, or whether one mode potentiates the other. The major problem is still one of preventing any separation of gas from solution until the end of decompression—if then. Therefore, the major vital issue is still: when does bubble inception occur—at the position of phase equilibrium or at some critical limit to supersaturation?