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An examination of the critical released gas volume concept in decompression sickness

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If P_1 is the pressure breathed by men for a prolonged period, and P_2 is the pressure to which it is just safe to decompress rapidly, then assuming the volume of tissue gas released upon decompression is critical, it is predictable from theoretical considerations that there is a relation between P_1 and P_2 of the form $P_1 = aP_2+b$, where a and b are constants.

The use of men as experimental subjects confirms this theoretical prediction for the case where mixtures of oxygen and helium are breathed. By assuming that the same critical volume of released gas provokes mild attacks of decompression sickness when air, or other respirable gases are breathed, the relevant values of a and b can be deduced, and the values accord well with the known facts for such gases. This analysis also offers an explanation for the changes in signs and symptoms of decompression sickness which can follow changes in the nature of the exposure to pressure.

Introduction

As a result of their attempts to define the conditions necessary to provoke mild attacks of decompression sickness, Boycott, Damant & Haldane (1908) reached the conclusion that 'liquids such as blood, will hold gas in a state of supersaturation, provided the supersaturation does not exceed a certain limit'. They related this allowable supersaturation to the ratio of the absolute pressures involved, 'the decompression being done in stages, but in its being rapid till the absolute pressure is halved and slow afterwards'. These two concepts led them to make the further statement that:

> a pressure of 1 to 1.25 atmospheres above normal corresponds to from 2 to 2.25 times the normal atmospheric pressure; but 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. Hence it seemed probable that, if it is safe to decompress suddenly from two atmospheres of absolute pressure to one, it would be equally safe to decompress from four atmospheres absolute to two, from six atmospheres to three, etc. Our experiments, which are detailed below (page 398), have shown that this is the case. Whether the law holds good for pressures much exceeding six atmospheres is still doubtful, as no experimental data exist.

Obviously this statement immediately suggests a critical ratio hypothesis, which has been one of the basic ideas in decompression calculations since Haldane introduced it, but one must be careful not to ignore that he thought that the potential volume of released gas was the critical factor, rather than supersaturation ratio *per se*. The ratio hypothesis was thus firmly established and any reference to the volume of gas came to be omitted until Behnke (1951) conjectured that, 'It may well be that what appears to be a ratio of saturation tolerance is in reality an index of the degree of embolism that the body can tolerate'. Considerable support for this has come from some pertinent experiments by Hills (1970) who concludes, 'It is far more likely that the quantity of gas separating from solution determines the imminence of decompression sickness rather than its mere presence as determined by a critical limit to supersaturation'. Using ultrasonic techniques, Rubissow & Mackay (1974) and Evans & Walder (1970) have demonstrated that gas emboli are indeed present in blood and tissue following exposure to raised pressures, even when the duration under pressure is insufficient to provoke symptoms of decompression sickness. These observations also provided further support for the suggestion, advanced by Hempleman (1960), that unequal rates of uptake and elimination of gas found during certain forms of exposure to raised pressure could be the result of interference with tissue blood flow caused by the generation of asymptomatic bubbles during decompression. Lever, Paton & Smith (1971), using mice as experimental subjects, introduce another fundamental consideration when they state, 'The most important single factor governing the appearance of decompression sickness seems to be the excess of gas in solution in fatty tissue. Consequently, the critical decompression ratio for a particular gas depends upon its fat solubility'.

Alternatives to the bubble theory as an explanation of the aetiology of decompression sickness have been advanced, for instance by Swindle (1937), End (1939), Cockett, Pauley, Saunders & Hirose (1971). More recently observed changes in the microcirculation associated with observations of disseminated intravascular coagulation following decompression (Lemire, Heinbecker, Chen & Drucker 1970; Philp, Schacham & Gowdey 1971), have led to a belief that the bubble theory is at best only a partial explanation. However, Frattali, Inesada, Robertson & Shackelford (1973), from his observations on the therapeutic value of proteinase inhibitors in cases of decompression sickness of the rat, concludes 'this study cannot support adjuncts or alternatives to the bubble theory that invoke, for example, disseminated intravascular coagulation in the etiopathology of the decompression sickness'.

The working hypothesis can now, therefore, be proposed that after exposure to pressures of air or other respirable gases, there are gaseous bubbles in the tissues, and that the volume of this released gas is responsible for the onset of marginal symptoms of decompression sickness. Furthermore, Hills (1971) has drawn attention to the fact that certain pressure–time courses, when breathing air, predispose the subjects to central nervous system signs rather than the generally

observed simple limb pains (bends). Thus one is posed the additional problem of deciding why released gas gives varying signs and symptoms at the onset of decompression sickness, dependent upon the nature of the exposure to pressure.

In an attempt to clarify these difficulties a re-analysis of experiments by Barnard (1976), using human subjects undergoing prolonged exposures to raised pressure of oxy-helium gas, has been combined with the accumulated evidence from observations on caisson and tunnel workers decompressions and a very limited set of long duration oxy-nitrogen dives recently performed on human volunteers. From the point of view of this analysis the essential common feature of all these observations is that they attempt to define the greatest pressure drop which can be sustained, without the onset of decompression sickness, following prolonged exposure to a constant raised pressure and a relatively sudden change to a new constant lower pressure level. Thus, knowledge of gas transport mechanics in the relevant tissue, or tissues, is avoided by considering only prolonged exposures, approaching equilibration, at constant pressure; and by using only a single step change in pressure, the complex problems arising from bubble growth are also considerably simplified.

The volume of released gas

Suppose that a unit volume of tissue is equilibrated with an inert gas α at a partial pressure $p_{1\alpha}$ under an ambient pressure P_1. After rapid decompression to an ambient pressure P_2, assume that a gas-filled space of volume V is formed and that there is no gas lost via the blood during its formation. This latter assumption that 'the whole excess of gas present in supersaturation were given off' (Boycott *et al.* 1908), implies an eventual equilibration between dissolved and released gas at a partial pressure $p_{2\alpha}$. The value of $p_{2\alpha}$ refers to that partial pressure of dissolved gas which is present in the 'bend' tissue at the threshold of mild decompression sickness and V, being the volume of released gas per unit volume of this tissue is an index of the gas release which can be tolerated. The solubility of the inert gas (S) is taken as the volume of gas, measured at a partial pressure of 760 mmHg (1 atm) and at 37 °C, contained in unit volume of tissue; thus a simple mass balance gives

$$Sp_{1\alpha} = Sp_{2\alpha} + Vp_{2\alpha}. \tag{1}$$

Also, because of the equilibrium between tissue and cavity,

$$p_{2\alpha} + P(O_2, CO_2, H_2O) = P_2 + 2\gamma/r + \delta, \tag{2}$$

where $P(O_2, CO_2, H_2O)$ is the sum of the gas-filled space partial pressure of these gases (assumed approximately constant), γ the surface tension, $2r$ a representative diameter and δ the tissue deformation pressure (Nims 1951).

The above equation is conveniently rewritten as:

$$p_{2\alpha} = P_2 + k, \tag{3}$$

where k is likely to be a constant for a given tissue and released gas volume distribution.

An estimate of k has been made by Hills (1966) to be 74 mmHg, based on a bubble diameter of 2.82 μm, $\gamma = 1.79 \times 10^{-2}$ N/m (Davson 1964), $\delta = 10$ mmHg (Inman & Saunders 1944) and $P(O_2, CO_2, H_2O) = 126$ mmHg.

Eliminating $P_{2\alpha}$ in equation (1) via equation (3) yields

$$p_{1\alpha} = (1 + V/S)(P_2 + k).$$

In the case of a mixture with constant inspired P_{O_2},

$$p_{1\alpha} = P_1 - P_{O_2},$$

whereas if the oxygen content is a constant proportion $(1-f)$ of the mixture,

$$p_{1\alpha} = fP_1.$$

In either case,

$$P_1 = A\,P_2 + B, \tag{4a}$$

where A and B are given by

$$A = 1 + V/S, \quad B = A\,k + P_{O_2} \tag{4b}$$

in the case of a constant P_{O_2} mixture; and in the case of a constant proportion of oxygen in the mixture by

$$A = (1 + V/S)/f, \quad B = Ak. \tag{4c}$$

The critical pressure ratio (R) is defined as

$$R = P_1/P_2.$$

Attempts to calculate trouble-free decompression procedures covering a wide pressure range, have revealed in an indirect manner, that the value of R cannot be regarded as constant (Dwyer 1955). If it can be directly demonstrated with sufficient human experimental data that equation (4a) holds true, as seems to be the case with goat data analysed by Hills (1966), then clearly R would not be expected to remain constant unless $B \ll P_2$.

Examination of experimental evidence

In conformity with present British diving practice pressures will be expressed either in metres of sea water (m s.w.) or bars. By definition 10 m s.w. is 1 bar and transferring between these two systems of pressure measurement is extremely simple. To avoid any possibility of confusion absolute pressures will be used throughout, and gauge pressures can be obtained to a sufficient order of accuracy for these data by subtracting 10 m s.w. (1 bar).

There have been no independent tests of formula (4a) over a suitable range of saturation exposures and gases. However, the results of Barnard (1976) constitute a limited, but valuable, set of experimental determinations of P_1 and P_2, and a brief description of his experimental method is as follows.

Human volunteers were compressed, three at a time, to a pressure P_1 for a

period of not less than 24 h followed by a relatively rapid decompression at rates varying between 0.1 bar/min and 1.2 bar/min to a 'bends threshold pressure' P_2 (see below). The gas breathed throughout the experimental period consisted of 0.22 bar oxygen with remainder helium, and the temperature was controlled for the comfort of the experimental subjects, in the range 26–29 °C.

It is important to describe his experimental design in some detail because, as will be seen, the results obtained are inter-dependent and have a greater significance as a consequence of this. The series commenced by confirming that 24 h at 20 m s.w. could be safely followed by decompression to 10 m s.w. The second set of experiments concerned establishing the threshold value (P_1) which could be sustained for 24 h and safely followed by decompression to 20 m s.w. This 'just-safe' value was found as follows.

An initial value was chosen for P_1 which experience would indicate as just safe when followed by decompression to 20 m s.w. Twenty-four hours were spent at this estimated P_1 value and upon decompression to 20 m s.w. the subjects remained a further 24 h before being decompressed to 10 m s.w. Thus in starting to define the threshold value of P_1 this experiment was also re-testing the validity of the observation that 20–10 m s.w. was a safe pressure change. Following this safe exposure to P_1, the next exposure was to 1.1 P_1 for 24 h followed by rapid decompression to 20 m s.w., which if safe, was again followed by 24 h at 20 m s.w. and reduction to 10 m s.w. Barnard changed his P_1 values by ratio units of 0.1 when searching for the value which gave an attack of decompression sickness. The value of P_1 which gave an attack of the bends on rapid ascent to 20 m s.w. proved to be 37 m s.w. The bend threshold ('just safe') value of 34 m s.w., i.e. the previous trouble-free exposure of the series, was then used as a P_2 value to search for a new P_1 value in the next experiment.

In searching for this new P_1 value there were several safe ascents to 34 m s.w., and these were followed by 24 h at 34 m s.w., 24 h at 20 m s.w. and finally ascent to 10 m s.w. Thus, once again, in defining the new P_1 value the experiments were reconfirming the validity of the results established at lower pressures. By using this experimental design Barnard produced two sets of data for consideration in this analysis. The first set of values for P_1 and P_2 could be taken as those established from the bend or no-bend pressure values and these are linearly related (correlation coefficient 0.984). However, Barnard concluded after further experimentation that these data, obtained by searching in rather coarse steps of 0.1 P, needed refinement and he altered one of the stops from 57 to 52 m s.w., using the latter value in all his subsequent work. This single alteration affects two of the points plotted in figure 1. It is these two adjusted points which were thought to be more soundly based, and hence were used, together with two unadjusted points, to obtain the regression line drawn in figure 1, which can be represented by the equation (correlation coefficient 0.999):

$$P_1 = 1.397P_2 + 5.7 \quad \text{m s.w.} \tag{5}$$

The regression line runs close to the point P_1 = 118 m s.w. and P_2 = 79 m s.w., which was not established as a result of provoking an attack of the bends at a value of $1.1P_1$, i.e. 129 m s.w. This pressure level (P_1, 118 m s.w.) when followed by rapid decompression began to give results not typical of the previous experiments in the series. Apart from the lack of a clear end-point in the form of an attack of the bends shortly after the decompression from P_1 to P_2 there were other

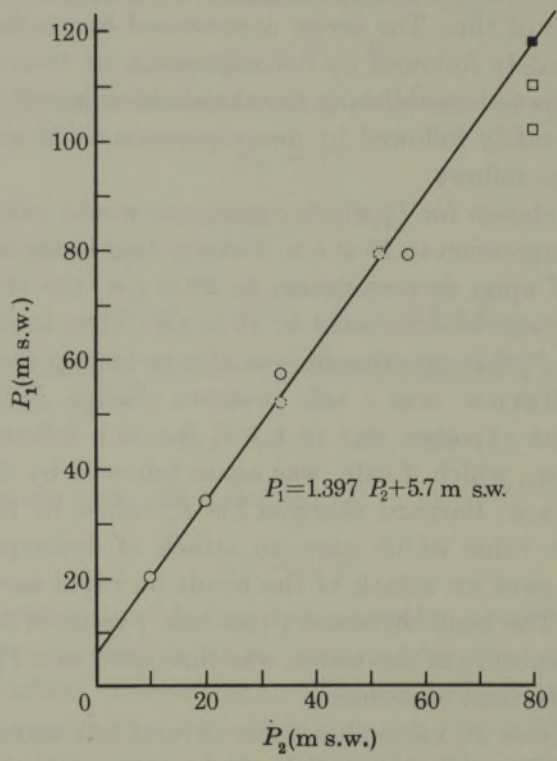

FIGURE 1. Critical pressure curve on normoxic helium, data from Barnard (1976). ○, One day exposure at P_1 followed by decompression to a just-safe pressure P_2 for one day; ◌, adjusted point (see text); □, bends free first stop; ■, marginal bends, first stop.

more serious signs and symptoms beginning to appear, such as transient deafness, or attacks of vertigo accompanied by nystagmus. Continuing to use human volunteers to define end-points in these circumstances was unacceptable and the experiments were terminated. This final point and the two previous attempts at lower P_1 levels are plotted in figure 1. The point P_1 118 m s.w., P_2 79 m s.w. is termed a marginal bends point in figure 1 because there were indeed minor aches and pains characteristic of a mild attack of the bends, but the important observation to note is that other forms of decompression sickness are also occurring and the significance of this is discussed later. In view of these altered experimental findings the point P_1 118 m s.w., P_2 79 m s.w. was not used in obtaining the regression curve. In figure 1 the regression line is extrapolated into this higher pressure zone to give a visual presentation, for later discussion, of the fact that the

slope of the line established with such precision at lower pressure levels will not account satisfactorily for findings at pressures greater than around 100 m s.w. There is possibly a small error at the point where $P_1 = 20$ m s.w. and $P_2 = 10$ m s.w. as this was not a fully proven threshold point on the 0.22 bar oxygen mixture, and was estimated from the threshold bends pressure of 2.4 bar, when subjects breathed a gas mixture consisting of 20 % oxygen, 80 % helium (Hempleman & Trotter 1965).

From equation (4*b*) and taking 1 bar as 10 m s.w.,

$$V/S = 0.397,$$

$$k = 2.5 \text{ m s.w.} \quad \text{or} \quad 188 \text{ mmHg}.$$

If $P(O_2, CO_2, H_2O)$ is estimated at 126 mmHg and δ as 10 mmHg (Hills 1966) then the contribution by surface tension is 304 mmHg, considerably higher than Hills value of 190 mmHg. A simple estimate using values of γ as 2.7×10^{-2} N/m (oil) to 7.2×10^{-2} N/m (water) suggests a bubble diameter of 2.7–7 μm. However, it is not possible to identify the tissue type or critical volume since the solubility is unknown.

The formula predicts (equation (4*c*)) that prolonged exposure to a pressure equivalent to 22 m s.w., breathing 20 % oxygen, 80 % helium could be safely followed by rapid ascent to the surface (P_2 10 m s.w.), and may be compared with the experimental bend threshold value of 24 m s.w. for P_1 put forward by Hempleman & Trotter (1965), when using this particular breathing gas mixture and the same P_2 value.

Air diving

A check of validity of formula (5) can be made by calculating the equivalent constants on air. The solubilities of helium in olive oil and water are taken respectively as 0.0150 and 0.0087 ml/ml at 760 mmHg, 37 °C. Equivalent nitrogen values used are 0.0670 and 0.0127.

Hence by equation (4*c*), in the case of fatty tissue

$$P_1 = 1.361P_2 + 3.4 \text{ m s.w.}, \qquad (6a)$$

and aqueous tissue

$$P_1 = 1.604P_2 + 4.0 \text{ m s.w.} \qquad (6b)$$

It is also possible to attempt a mixed tissue analysis, that is supposing helium bends occur in watery tissue, nitrogen bends in fatty tissue, or vice versa. The latter case is not realistic since it predicts too low a value for the sub-atmospheric bend threshold, and indeed, equation (6*b*) above fails here also. However, the former case can be considered as a possibility.

As a further practical test of the effectiveness of the predictions made from the Barnard helium data, two experiments with human subjects breathing 0.22 bar

oxygen and the remainder nitrogen were performed. In the first exposure three men remained 48 h at a constant raised pressure of 30 m s.w. and were then decompressed at 1 m s.w. per minute to 20 m s.w., where they remained to assess the effectiveness of this relatively sudden change in pressure. From fat solubility

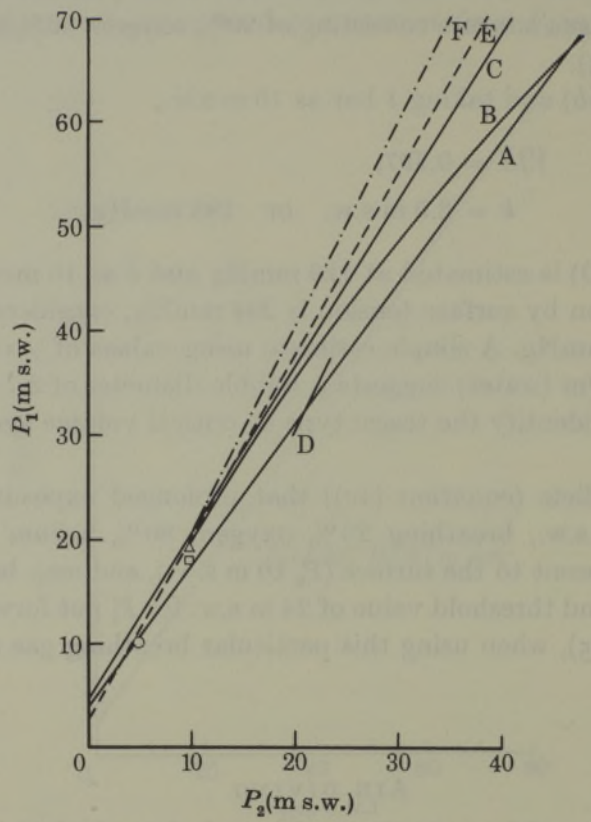

FIGURE 2. Critical pressure curves on air from various sources shown in table 1. The formulae of the Workman M-value$_{240}$ (1965), Nishi & Kuehn (1973), Bühlmann (1969), Des Grange (1956) and the linear regression of Hempleman's formula (1969) are graphically indistinguishable from the predicted curve for lipid tissue A. ○, Aerial decompression threshold point; □, caisson worker threshold point; △, divers threshold point; A, lipid tissue; B, Hempleman (1969); C, aqueous tissue; D, caisson workers' formula; E, Hills (1966); F, Haldane (1908).

data for both nitrogen and helium the critical pressure formula using 0.22 bar oxygen with nitrogen added is:

$$P_1 = 1.089P_2 + 4.9 \text{ m s.w.} \qquad (6c)$$

Taking $P_1 = 30$ m s.w. the threshold value of P_2 is 23.0 m s.w. Decompressing to 20 m s.w. would be expected to present 'bends' in a proportion of the men. In fact, one man from the three had to be treated for an attack of 'bends'. In a second experimental dive three men attempted a decompression from prolonged exposure (48 h) at 30 m s.w. to 25 m s.w., which should be quite safe according to (6c), and indeed this pressure change was achieved without incident.

In view of the possible versatility and accuracy of the calculation based upon the helium data the various sets of formulae are given in table 1 and some have been plotted in figure 2 for comparison purposes. Three separate points have also been added. The threshold for a 'just-safe' pressure to which one may decompress from atmospheric pressure without any decompression sickness developing in normal healthy young males is shown as 0.5 bar or 5 m s.w. (Fryer 1969).

TABLE 1. CRITICAL PRESSURE FORMULAE ON AIR

formula (m s.w.)	no-stop threshold pressure (m. s.w.)	source	application
$P_1 = 2P_2$	20	Boycott, Damant & Haldane (1908)	original Admiralty table
$P_1 = 2P_2 - 10$	—	Catton (1967) $30 < P_1 < 40$ m s.w.	caisson workers formula
$P_1 = 1.518P_2 + 4.6$	19	Des Grange (1956) 120 min tissue	U.S.N. standard air tables
$P_1 = \frac{275.714P_2}{P_1 + 124.072}$ $= 1.366P_2 + 5.6$*	19	Hempleman (1969) $19 < P_1 < 70$ m s.w.	R.N.P.L. tables
$P_1 = 1.375P_2 + 5.2$	19	Workman (1965) M-value 240 min tissue	U.S.N. mixed gas tables
$P_1 = 1.385P_2 + 4.2$	18	Nishi & Kuehn (1973)	Canadian tables
$P_1 = 1.401P_2 + 4.7$	19	Bühlmann (1969) 240 min tissue $10 < P_1 < 70$ m s.w.	Swiss tables
$P_1 = 1.361P_2 + 3.4$	17	fatty tissue equation (6*a*)	—
$P_1 = 1.604P_2 + 4.0$	20	aqueous tissue equation (6*b*)	—
$P_1 = 1.766P_2 + 2.3$	20	Hills (1966)	—

* A least squares regression line passing through the no-stop threshold depth of the formula.

The other two points show the maximum pressure of air which can be breathed for an indefinitely prolonged period, followed by rapid decompression to atmospheric pressure, without provoking decompression sickness. This maximum pressure has been termed 'no-stop threshold pressure' in table 1 or simply 'threshold point' in figure 2. For divers and compressed air workers the no-stop threshold values are given as 19.0 m s.w. and 18.3 m s.w. respectively (Hempleman 1969; Kidd, Stubbs & Weaver 1971). Unfortunately there are no well-defined values at a variety of pressure levels such as those available from the helium diving.

The graph, however, appears to show that the formula, $P_1 = 1.361P_2 + 3.4$ m s.w. based on a fatty tissue conversion of the Barnard helium data is feasible. The caisson workers rule of thumb of halving the gauge pressure after a near saturation exposure in the range 3–4 bar agrees well with the above formula and Hempleman's empirical formula. In fact, it seems desirable to replace the latter by a simple straight line fit in the air diving range. Indeed, if this formula is replaced by a least-squares fitted straight line constrained to go through the point $P_1 = 19.24$ m s.w. and $P_2 = 10$ m s.w., i.e., the no-stop threshold point of the formula, then the relation

$$P_1 = 1.366P_2 + 5.6 \text{ m s.w.} \tag{7}$$

is obtained, which is extraordinarily close to the predicted relation

$$P_1 = 1.361P_2 + 3.4 \text{ m s.w.} \tag{6a}$$

In view of the Kidd *et al.* (1971) results, equation (6*a*) is seen to be probably more accurate. When the Workman (1965) concepts are examined it is found that his M-values, for the tissue with the longest half-time, yield the relation:

$$P_1 = 1.375P_2 + 5.2 \text{ m s.w.} \tag{8}$$

There is also the pressure ratio relation proposed by Stubbs and used by Nishi & Kuehn (1973), which when converted into the mathematical form and units of measurement being used here gives

$$P_1 = 1.385P_2 + 4.2 \text{ m s.w.} \tag{9}$$

Once again there is a striking agreement with equation (6*a*).

Des Grange (1956) and Bühlmann (1969) use empirical safe ascent curves plotted as P_{N_2}/P_2 against P_{N_2} for each tissue. Since $P_{N_2} = 0.8P_1$, it is a simple task to replot their curves in terms of P_1 against P_2. Remarkably, all such curves turn out to be practically linear. The results, for the longest halftime in each case, are approximately

Des Grange

$$P_1 = 1.518P_2 + 4.6 \text{ m s.w.}; \tag{10}$$

Bühlmann

$$P_1 = 1.401P_2 + 4.7 \text{ m s.w.} \tag{11}$$

It is significant that these safe ascent curves, developed and experimentally adjusted under the dominating influence of a pressure ratio approach are far more simply related by $P_1 = AP_2 + B$.

Taking the previously quoted value for sub-atmospheric decompression, that 5 m s.w. is a 'just-safe' P_2 value when $P_1 = 10$ m s.w., the values obtained from equations (7) to (11) are 3.2, 3.5, 4.2, 3.6 and 3.8 m s.w. respectively, which are all too low, whereas equation (6*a*) yields 4.8 m s.w. and is close enough for practical purposes.

An estimate of the critical volume of released gas per unit volume of tissue is only possible if the number of bubbles per mm^3 and their diameter is known.

Rubissow & Mackay (1974) have observed that on initial decompressions 2–10 bubbles per mm^3 appear in fatty tissue, with diameter 2–5 μm, yielding an upper estimate of $V/S = 10^{-5}$ for nitrogen. This value is about 10^4 times too small but it is not known if this bubble distribution had attained a critical volume. The value can only be approached by considering either bubbles of larger diameter or a denser bubble distribution. Either are possibilities, but in the former case the effect of surface tension would be negligible and thus the tissue deformation pressure must be large to explain a k value of 188 mmHg.

It seems clear that a simple critical pressure formula of the form $P_1 = AP_2 + B$ can be applied to both air and helium exposures with considerably greater accuracy than a pressure ratio. The larger the value B the more apparent will a ratio appear to decrease with increasing pressure. The slope of the line (A) depends on the solubility of the gas in the tissue and thus it is likely that gas in fatty tissue is responsible for simple threshold bends at moderate exposures.

Other tissue types

The question arises whether or not this analysis could apply to other tissues in the body capable of causing different forms of decompression sickness. Thus, one may suppose that there are at least two responsible tissue types A, B, one with a smaller critical volume $V_B (V_A > V_B)$, and the critical pressure curves for these two tissue types would be of the form $P_{1A} = a_1 P_{2A} + b_1$ for tissue A, and $P_{1B} = a_2 P_{2B} + b_2$ for tissue B, where a_1, b_1, a_2 and b_2 must be positive constants if they are to possess practical significance. Thus the two curves will intersect at a common cross-over pressure P_C, which must also be positive and less than the maximum value of P_1 attainable by divers. These practical constraints lead to consideration of the particular possibility $a_1 > a_2, b_1 < b_2$. This implies $V_A/S_A > V_B/S_B$, giving $a_1 > a_2$, and if surface tension is considered the dominant effect for excess pressure and is much greater in tissue B then $b_1 < b_2$ is a practical possibility.

Thus

$$P_{2B} < P_{2A} \quad \text{if} \quad P_1 < P_C$$

$$P_{2B} > P_{2A} \quad \text{if} \quad P_1 > P_C.$$

From these simple considerations it is seen that in decompression from moderate exposures, $P_1 < P_C$, tissue type A will, if allowed, produce ill-effects which are normally treated well before type B becomes manifest. On deeper exposures the reverse will be true with a consequent lowering of the safe decompression ratio. That is, decompression sickness will occur in tissue B before symptoms appear or are expected in tissue A.

It is suggested that two such tissue types could be the fat pads in joints (tissue A) and the fluid of the vestibular and auditory organs (tissue B). It is known that the latter organs appear to be more prone to decompression accidents on deeper exposures (Farmer & Thomas 1973).

An estimate of the appropriate critical pressure formula may be based upon the vestibular decompression incident for helium saturation at 467 m s.w. with 0.45 bar oxygen partial pressure (Royal Naval Physiological Laboratory Report 1, 1971). The diver woke with symptoms at 394 m s.w. The decompression rate was 12 m s.w./h and a somewhat arbitrary depth of 398 m s.w. may be chosen as not far removed from the critical pressure P_2. If it is also assumed that the tissue is considered primarily aqueous with a similar bubble distribution as in tissue type A, it can be shown that

$$P_1 = 1.113P_2 + 24.1 \text{ m s.w.} \tag{12}$$

The cross-over pressure P_C occurs at approximately 96 m s.w. Thus, for a prolonged exposure to 260 m s.w. on helium and 0.45 bar oxygen, the first stop should be not less than 211 m s.w. to avoid type B decompression incidents, whereas type A will occur only if rapid decompression occurs to below 182 m s.w.

During the U.S. Navy Experimental Diving Unit program to develop excursion diving tables, ended June 1976, it was found that when subjects breathed a gas mixture consisting of 0.4 bar oxygen, remainder helium for a period of 24 h at 437 m s.w. or 315 m s.w., then the 'just safe' pressures to which they could rapidly ascend were 366 m s.w. and 257 m s.w. respectively. Equation (12) (0.45 bar oxygen) predicts safe threshold pressures of 371 m s.w. and 262 m s.w. respectively.

In the case of subjects breathing 20 % oxygen, 80 % helium, a rapid decompression from saturation depths greater than 32 m s.w. to the surface (10 m s.w.) will also predispose to type B attacks.

Thus, the result of Hills (1971) where it was found that c.n.s. symptoms as distinct from ordinary limb bends, could be induced in goats by including a surface excursion, before the eventual decompression, can readily be explained by the concept of two or more tissue types, each with a different critical volume and excess gas pressure. The sharpness of the dividing line between the two forms of decompression sickness is indicative of two distinct tissue types.

Discussion

From a practical standpoint the foregoing analysis has shown that simple linear equations quite accurately describe the relations that exist between the lowest pressure (P_2) to which a human being may safely decompress following prolonged exposure to a higher pressure, and that higher pressure (P_1). This will be most helpful to those engaged in prolonged work at raised pressures. For example, when diving from undersea habitats it should now be possible, with some confidence, to state the maximum safe depth to which the diver may descend, or ascend, in the water and then return to the habitat without requiring decompression stoppages. However, it is most important to realise that at depths greater than 300 m, or thereabouts, the limit for rapid increases in pressure will be dictated by the onset of manifestations of the high pressure nervous syndrome,

and consequently, only decreases in pressure, of the magnitude predicted by the formula, may be possible in these circumstances.

For many years the idea, first advanced by Boycott *et al.* (1908), that a constant value of P_1/P_2 was significant in determining the boundary conditions for trouble free rapid pressure changes dominated attempts to formulate practical procedures. It became apparent that such a simple approach, although a very useful concept, would not suffice, and therefore various modifications of this critical pressure ratio concept were introduced (Hawkins, Shilling & Hanson 1935) and form the bases for most current decompression procedures, Des Grange (1956), Hempleman (1969), Bühlmann (1969). Hills (1966) showed that if the goat results of Hempleman were considered as a linear plot of the form $P_1 = AP_2 + B$, where A and B are constants for a particular animal, rather than $P_1 = RP_2$, where R is the critical ratio, then this described the threshold pressure levels over a very wide range of pressure values. The goat results were obtained using air as the breathing medium and it was many years before these experiments came to be repeated on human subjects, but this time breathing a mixture of 0.22 bar oxygen with the remainder helium. As has been seen, these results served to show that the linear relation between P_1 and P_2 not only applied to human beings but also to gases other than air. If one makes a small number of reasonable assumptions then the subsequent analysis shows that the interrelationships between various breathing gas compositions are readily explicable. It is interesting to note that the solubility of the inert gas in fatty tissues is an important physical property which inter-links the calculations. If the calculations are repeated using solubility and surface tension values relating to watery tissue, then the slope and intercept of the linear relation change quite markedly, and in the case of helium breathing this leads one to suppose that it is readily possible for another tissue type to present decompression sickness of a quite different nature if an appropriate pressure profile is chosen. This supposition is entirely substantiated by Barnard (1976) and Farmer & Thomas (1973) reporting observations on the first presenting signs of decompression sickness in human subjects breathing oxy-helium gas at high pressures. It is quite clear from their observations that the majority of cases of decompression sickness developing at pressures in excess of 100 m s.w., or thereabouts, are some form of disturbance of the eighth cranial nerve, either deafness, partial or complete, in one or both ears, or perhaps vertigo, nystagmus and other indications of vestibular involvement.

Thus the end-point for defining the decompression sickness threshold has changed its character from limb pains (bends) to eighth nerve disturbance (e.n.d.). This is not meant to imply that only e.n.d. will be manifested in one set of conditions, and only 'bends' in another. What is being advanced for consideration is that e.n.d. is more easily provoked, following certain pressure profiles, than 'bends', and that when those particular pressure profiles are being followed it is the more easily provoked form of decompression sickness that should define any practical procedures.

Certain features still remain in common to both these entirely different clinical end-points. Just as marginal attacks of 'bends' can manifest on the left or right side, so can e.n.d.; and similarly, as with 'bends', there is a variable delay period with e.n.d. before the onset of signs and symptoms. This encourages one to suppose that there are also common features to the aetiology of both these conditions and it is being proposed that released gas is the common feature, in one case ('bends') in a primarily fatty 'loose' tissue and in the other case (e.n.d.) in a primarily watery 'tight' tissue.

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