Chapter 9

Thermodynamic Decompression

It is one matter to describe the many aspects of decompression sickness and their theoretical implications but quite another to rationalize the deductions into a fundamental model from which to formulate practical decompression schedules. This process of synthesis is not aided by the need to recruit the whole spectrum of scientific disciplines from theoretical and mathematical physics to clinical diagnosis and blood chemistry in covering the multitude of facets of decompression for which explanations are needed. The foregoing chapters have shown that there is no obvious model or, at least, none which would gain universal approval in the field at this time. However, while there are still a few major issues the outcome of which is not yet certain, there is an adequate volume of data on which to make qualified deductions. Having synthesized such a universal model, it then needs rigorous analysis to test its viability.

To demonstrate the type of scrutiny to which any such model must be subjected, the 'thermodynamic' approach has been selected as a convenient example. This choice is based not only upon this writer's opinion that it still offers the most fundamental set of explanations but it was presented with the most attention paid to the type of detail which needs to be covered and yet is all too often omitted in the many calculation methods produced as short cuts to diving tables.

The Model

At the start of Chapter 3 a number of fundamental questions were listed and then discussed in the light of the factual evidence (Chapter 2) to produce varying degrees of conclusion which can be synthesized into a model.

An opinion

These discussions lead this writer to the opinion that

(1) the primary event and the critical insult in producing symptoms of decompression sickness do not coincide (p. 75).

(2) The primary event is the activation of one or more of a 'reservoir of nuclei' normally present in tissue into growth and hence the inception of a stable gaseous phase. On the other hand, limb bends are determined by the local pressure differential tending to bend or otherwise distort a nerve ending beyond its pain-provoking threshold and hence their onset is more dependent upon the volume of gas separated from solution (p. 57).

(3) No more than one anatomical tissue type needs to be invoked in marginal cases of limb bends (p. 191). This is probably tendon or some other well-innervated connective tissue of minimal compliance (p. 60).

(4) For all except very rapid decompression, the gas separates from solution in extravascular sites (p. 147) and in any static blood when it can be regarded as effectively extravascular for computational purposes, deriving its gas from extravascular tissue.

(5) The much larger volumes of gas thus deposited in fatty tissues can be released into

the venous system as bubbles which remain asymptomatic unless they fail to be trapped by the lungs or are present in such numbers that they stimulate J-receptors in the pulmonary bed to invoke 'the chokes' (p. 68). Although a single decompression which deposits more gas in a fatty tissue is also more likely to deposit more gas in the critical tissue, any coincidence between the appearance of venous bubbles (e.g. as detected by Doppler) and the volume of gas 'dumped' in the critical tissue by a complex pressure history is likely to be largely fortuitous, i.e. the appearance of venous bubbles is not particularly relevant to limb bends (p. 146).

(6) Cerebral symptoms (p. 63) are caused by arterial bubbles which have permeated the lung under certain unusual conditions or can be formed by decompressions so rapid as to supersaturate arterial blood in its passage from lungs to brain, e.g. in submarine escape. However, in diving, they will not normally occur unless there is something unusual such as an extensive recompression or a pathological state in the lung impairing its bubble filtering action, e.g. after excessive exposure to oxygen (p. 68).

(7) Spinal 'hits' are less likely to be the result of arterial gas emboli (p. 64) and require rather more gas to be deposited in cortex than cause limb bends when 'dumped' in connective tissue. Hence it is probably safe to work on the basis that decompressions which avoid *marginal* 'limb bends' are unlikely to provoke any serious spinal injury and no further tissue needs to be invoked in calculations.

(8) However, another tissue *does* need to be considered to account for vestibular symptoms (category III, p. 70), particularly if diving deeper than 300–400 fsw or switching inert gases (p. 71). Symptoms are still determined by the local pressure differential analogous to limb bends (Equation 7) but any inert gas gradient between blood and the middle-ear cavity, or any counter-gradient of two gases, can contribute to this differential.

(9) This additional local differential pressure (δ_f) can be contributed by counterdiffusion, counterperfusion and gas-induced osmosis (Chapter 8) with no certainty at this time as to which, if any, predominates. This additional differential may also be a minor factor in limb bends but only from dives where there is a large amount of gas dissolved 'deep' in tissue (p. 58).

(10) Although haemoconcentration may impair the circulation, the primary cause of blood degradation in diving is intravascular bubbles. Thus blood factors need not influence decompression formulation provided the *primary event* is genuinely avoided in the critical tissue for limb bends. Preventing any gas separation in this tissue may not prevent it forming in fatty tissues but the volume should not be enough to cause the disruption of those tissues leading to lipid emboli and venous bubbles (p. 147) and hence haematological problems.

(11) The inception of the gaseous phase in any decompressed gas solution is a random process (Chapter 4) to which the critical tissue type for marginal limb bends is no exception.

(12) Limb bends are determined by the 'worst possible case'—the particular micro-region of the critical tissue where the activation of nuclei into a stable gas phase is so profuse that the tissue can only withstand minimal supersaturation before 'dumping' gas in excess of thermodynamic equilibrium into the gaseous phase (p. 100).

(13) In this densely nucleated zone, diffusion distances of any gas molecule to the nearest gas phase are so short that, when phase separation occurs, it is effectively complete within a few minutes of the decompression. Tissue regions other than this 'worst possible case' may retain their supersaturation or have a few nuclei which slowly grow into bubbles as they 'drain' the surrounding tissue of its dissolved gas in excess of saturation. Thus the behaviour of the vast majority which

determine overall gas elimination from a tissue is believed to be largely irrelevant.

(14) The onset of bends is delayed until the 'dumped' gas, which is finely dispersed, can coalesce or otherwise congregate within the extravascular tissue to produce a local pressure differential (mechanical stress) adequate to bend, or otherwise distort, a nerve ending beyond its pain-provoking threshold (p. 58).

(15) The gas dumped in the 'worst possible case' contains all gases present in tissue, the volumes contributed being proportional to their respective tissue tensions *after* separation. Thus water vapour is present at its vapour pressure, while the metabolic gases are contributed at their venous tensions or less (p. 102). Inert gas provides the difference between the total hydrostatic pressure and the total partial pressure of oxygen, carbon dioxide and water vapour needed to satisfy the thermodynamic criteria for local phase equilibration.

(16) The uptake of inert gas is limited by both blood perfusion and diffusion into the bulk of the cytoplasm (Chapter 7). The transport model envisaged is thus one of a fully stirred 'extended vascular' zone from which venous blood leaves in equilibrium with respect to all gases, while arterial blood either dilutes or replenishes the gases in that 'tank' as the case might be. From this effectively fully stirred 'extended vascular' zone the gases then diffuse into the 'cellular' zone by bulk diffusion, i.e. into a bulk of uniform permeability with no specific resistance offered by any particular membrane (fig. 65). p. 182

(17) Decompression should be optimized by preventing gas phase inception anywhere within that bulk, i.e. within the 'cellular' zone. This means applying the principle of phase equilibrium to each point in the tissue and hence involves determining the peak rather than the mean tissue tension (p. 125).

(18) If decompression proceeds to the point where the total pressure of gas in the nucleus is less than the total tension of all gases dissolved at the peak then, in the 'worst possible case', gas is dumped until they are equal and allowance must be made for the corresponding fall in the driving force for inert gas elimination to blood (p. 138).

It is doubtful whether all of these points ultimately prove to be correct but they do represent a particularly comprehensive model which differs from others in one major respect. It details not only how to optimize a decompression for a particular exposure but how to predict quantitatively the course of events if the decompression were not optimal by these criteria. This writer takes the view that whereas you may disagree with the way the other fellow formulates his decompression, it is still necessary to predict *by your model* the result of the trials of *his tables*.

General criticism

The thermodynamic approach received quite hostile comments from some quarters when it was first introduced, since its net effect was to advocate much deeper stops and an overall deeper redistribution of decompression time. Moreover, it was introduced at a time when M values had reached a popularity peak and the U.S. Navy were decompressing as far as possible on their first long 'pull' in the belief that they were obtaining the greatest driving force for inert gas elimination in so doing. That is, they were exploiting to the maximum their belief that the greatest bends-free decompression ($P\downarrow\downarrow$) resulted in the greatest driving force ($\Delta P_{N_2}\uparrow\uparrow$) in Equation 51. Thus they were placing the maximum dependence upon their assumption that the critical tissue(s) of the bends-free diver were bubble-free, so that the 'thermodynamic' approach presented one of the first comprehensive challenges to most of their axioms of decompression formulation. Their formal assessment of this 'provocative' model (Hester, 1970) produced only one real criticism; that a dual set of equations were being employed throughout the quantitative analysis. Two sets of equations were indeed developed in the original publication of the

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'thermodynamic' approach (Hills, 1966). However, what these critics had not appreciated was that this model clearly separates the *primary event* (gas phase inception) from the *critical level of insult* (local pressure differential) needed to provoke bends. Hence one set of equations refers to conditions which describes a critical volume of separated gas and hence stable bubbles (radius r_b), while the second refers to the tissue before the nucleus (radius r_n) is activated into growth. The net difference on a thermodynamic basis is minimal, actually amounting to a differential of about 2 fsw in the constant representing the 'mechanical' contribution to the total gas pressure.

To avoid any similar misunderstanding in this text, the mechanical contribution to hydrostatic pressure is designated B in the marginal bends-provoking bubble and B' in the nucleus (see fig. 29; also depicted in figs. 26 and 31). Hester (1970, 1971) also went on to point out several basic similarities in the decompression format produced by the 'thermodynamic' model and those produced essentially by empirical deduction by Bühlmann (p. 118).

Summary

The model can thus be summarized as a single tissue type (an avascular connective tissue) in which inert gas uptake is limited partly by the blood perfusion rate and partly by diffusion into the bulk of the cellular material of effectively uniform permeability (fig. 64(a)). Gaseous cavitation in this extravascular tissue is both temporally and spatially random but the imminence of limb bends is determined by events only in the 'worst possible case'. This may be only one in many million possible micro-regions but one where nuclei are activated for minimal degrees of supersaturation and where these are so profuse that any gas in supersaturated solution is rapidly 'dumped' into the gaseous phase. However, this gas separated from solution in extravascular sites will only produce limb bends after it has had time to coalesce and if, with any fluid accumulation, the total displacement can produce a local pressure differential which can bend, or otherwise distort, a nerve ending beyond its pain-provoking threshold.

On the other hand, if the gas phase is not allowed to form, let alone reach its pain-provoking volume, then the volume of gas in separated fatty tissues should not become so large as to rupture fatty tissues and so produce a blood–gas interface causing all manner of haematological disorders. Moreover, prevention of the critical insult in this one critical connective tissue should also avoid spinal symptoms, while it is only after deeper exposures or for mixed inert gases that the 'vestibular tissue' can take over as rate-controlling.

This 'thermodynamic' model is not strictly 'zero-supersaturation' (Hills *et al.*, 1976) nor 'nil-supersaturation' (Bennett and Vann, 1975), since a minimal degree overpressure or additional gas tension is needed to balance the equation for phase equilibrium (Equation 21 and fig. 26). Thus the mechanical forces contributed by the curvature of the nucleus ($2\gamma/r_n$) and tissue compliance (δ_t) can be regarded as a minimal degree of tolerable supersaturation before gas starts to separate from solution. This underscores the two vital axioms on which the whole model is based:

(a) it is really the local pressure differential and hence the volume of gas which can be 'dumped' which largely determines the imminence of limb bends;

(b) since this volume of 'dumped' gas is only a maximum when the tissue and gas phases come to equilibrium, is there really any driving force then left for eliminating gas from tissue?

Test of the basic axioms

Prior to the 'thermodynamic' approach, many proponents of models and calculation methods had discussed the volume of gas separating from solution but only Nims and Bateman (p. 126) had actually incorporated this parameter into equations for predicting the imminence of clinical symptoms of decompression sickness. However, although the 'volume' concept was contrary to popular formulation, it is really fairly obvious. The passing 'niggles'

on moving to a shallower stop of a conventional decompression table and the minimum recompression needed to relieve most bends pains bear witness to this. Moreover, relief is usually instantaneous and, for marginal cases, can be effective if applied to the site itself, such as by an inflatable cuff or by simply immersing the limb in water. Deductions made from these simple observations are fully compatible with the abundant evidence for 'silent' bubbles within the body as a whole as shown by direct detection devices (e.g. ultrasonic, radiographic and conductometric techniques). Furthermore, experiments designed specifically to indicate asymptomatic gas separating from solution *in the critical tissue* leave little doubt that this is no exception.

While it does not prove that gas 'dumping' goes as far as establishing phase equilibrium, this point is so close on the decompression scale to those at which X-rays and conductance measurements show a phase change (p. 142) that it is highly likely that one out of the many million possible sites in each tissue will conform to this 'worst possible case'. Moreover, measurements of the mode of volume change on decompression of excised tissue indicates that the distribution of bubbles is so varied that at least one micro-region of the critical tissue is likely to come very close to this state on most decompressions exceeding equilibrium. This is the 'worst possible' because not only does it represent the maximum number of gas molecules separating from solution but there is then the minimum driving force for their elimination from the tissue.

The Inherent Unsaturation

The real test of the 'thermodynamic' approach arises when it comes to the second axiom. How can a gas phase in apparent local equilibrium with tissue eventually dissolve and disappear without any further change of pressure or of composition of the breathing mix; what driving force can there be? Such a driving force has been derived theoretically as ΔP_{N_2} in Equation 52—a concept termed the 'inherent unsaturation' by Hills (1966) and the 'oxygen window' by Behnke (1967) in their independent approaches to prevention and treatment. The Buffalo group (Van Liew *et al.*, 1965) also deduced a similar expression from their observation of subcutaneous gas pockets which they did not name and suprisingly, perhaps, did not apply to the *prevention* of decompression sickness; although treatment was mentioned in their discussions. The significance of this driving force has recently been rediscovered by the U.S. Navy who term it the 'partial pressure vacancy' (Sass, 1976). However, the inherent unsaturation is such a vital factor in 'volume' approaches for quantifying the imminence of clinical symptoms that it really needs direct experimental verification *in vivo*. Moreover, the absence of such a driving force in his 'volume' model caused Bateman to abandon his approach (p. 126), while Nims would have been forced to do the same if he had followed Bateman's lead in attempting to formulate diving tables as opposed to predicting aerial bends.

Deduction of unsaturation

The inherent unsaturation has been largely implied from analyses of subcutaneous gas pockets in studies which have raised sporadic interest over many years. Campbell (1924) found that 500–1000 cc of air injected into rabbits changed in composition with time, the carbon dioxide rising to a steady 50 mm Hg within minutes while the oxygen fell from 150 to 50 mm Hg in 10 hours, reaching 20–30 mm Hg after $1\frac{1}{2}$–3 days. The nitrogen then provided the additional partial pressure needed to compensate for the fall in $(P_{O_2} + P_{CO_2})$. These results were largely confirmed by Coryllos and Birnbaum (1932) using dogs and were in close agreement with values of 50 mm Hg (carbon dioxide) and 20 mm Hg (oxygen) recorded by Van Liew (1962) from the analysis of injected gas 'equilibrated' with rat liver.

The manner in which the nitrogen partial pressure in the cavity increases to accommodate the difference between the fall in oxygen (150 to 20 mm Hg) and rise in carbon dioxide (0 to 50 mm Hg) has been demonstrated by

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Asknes and Rahn (1957) whose analyses show 90% nitrogen in resting dogs. This agrees well with a theoretically predicted dry-gas fraction of $100(760-20-50-47)/(760-47)=90{\cdot}2\%$ nitrogen. Moreover, for exercise, where P_{O_2} values should be lower, some of the early studies of bubbles in guinea pigs revealed 95% nitrogen (Harris *et al.*, 1945b). Van Liew *et al.* (1965) went on to point out how the nitrogen fraction could be estimated by using *venous* values for P_{O_2} and P_{CO_2} in the type of summation equation for Dalton's Law expressed by Equation 3 and depicted graphically in figs. 26 and 31. They then went on to deduce theoretically that this inferred an unsaturation in the tissue adjacent to the gas pocket.

Reaching the same conclusion quite independently of this study, the group in Adelaide had considered that the inherent unsaturation was such a vital link in the 'thermodynamic' approach that it needed to be demonstrated directly (Hills, 1966; Hills and LeMessurier, 1969). They reasoned that the inherent unsaturation is basically a pressure difference and therefore needs to be demonstrated as such. This eliminates the use of gas pockets of the type studied by Campbell and later workers since these are essentially *constant-pressure* cavities.

This reasoning led to the use of *constant-volume* cavities in which a rigid membrane permeable to all gases and water vapour is used to restrain the tissue mechanically. Thus all gases will reach a true equilibrium between the adjacent tissue and the cavity in which the partial pressure of each will attain the tension of the

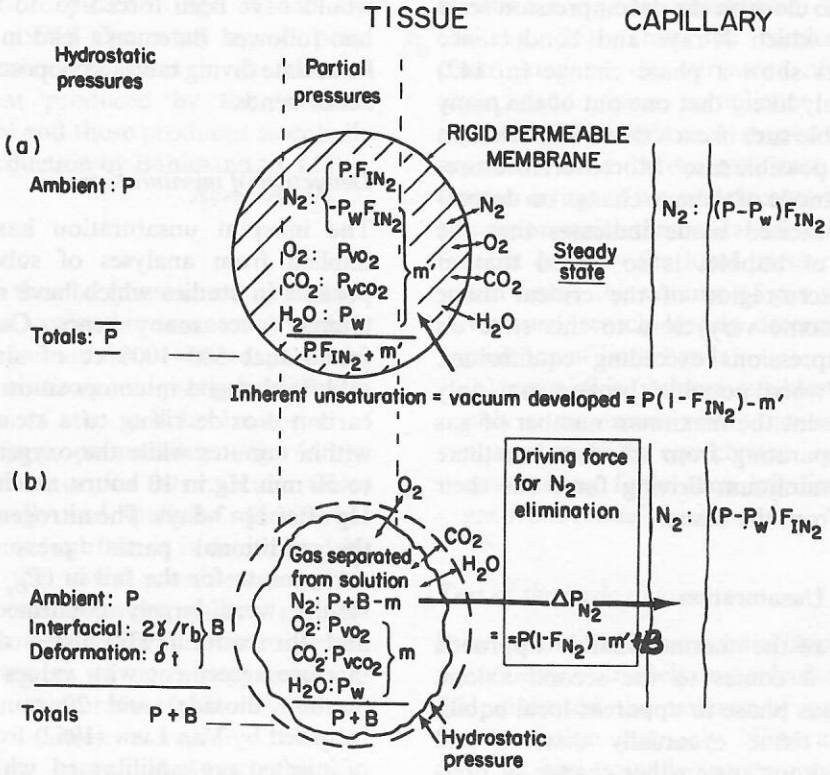

Fig. 81 (a) Depicting how a rigid cavity with a wall permeable to all gases reaches a steady state in which the total pressure of all gases ($P.F_{IN_2}+m'$) is less than the ambient absolute pressure (P), the difference (or inherent unsaturation) being manifest as a partial vacuum. (b) Showing how this unsaturation becomes a driving force for eliminating nitrogen from the cavity if the rigid wall is removed and the tissue is allowed to compress the contained gas. B is an additional yet minor contribution arising from tissue compliance and surface curvature

same gas in tissue (fig. 81a). Hence the total tissue gas tension will now equal the sum of the partial pressures in the constant-volume cavity which, by Dalton's law, must equal the absolute pressure of gas in that cavity. Thus any inherent unsaturation in the tissue is manifest as a deficit in the cavity pressure relative to ambient, i.e. as the amount of vacuum developed in the probe after equilibrium has been established (fig. 81(a)). Rigid membranes not only provide a direct measurement of the inherent unsaturation but they provide a direct gas measurement divorced from mechanical uncertainties related to tissue compliance (p. 58), any negative hydrostatic pressure in tissue (Guyton, 1963), the curvature of a direct tissue–gas interface (p. 85) and any fluid influx (p. 57), all of which complicate any deductions made from constant-pressure cavities.

While these experiments on constant-volume cavities were in progress in Adelaide, Lategola (1964) published his findings using a cylindrical capsule with silicone-rubber membranes held rigid across the ends. However, his measured unsaturation of 41–48 mm Hg is open to alternative interpretation in view of his statement that the membranes had 'relatively negligible permeability to water vapour'. This implies that he could have been simply measuring the vapour pressure of water at body temperature—47 mm Hg!

The vital experiment

The Adelaide group implanted rigid PVC tubes subcutaneously in rabbits, having first proven that the walls of these probes were permeable to oxygen, carbon dioxide, nitrogen and water vapour. A partial vacuum was found to develop in these constant-volume cavities over 12 hours and not to change significantly over the next 24 hours. Moreover, the magnitude for air breathing at normal atmospheric pressures lay within the range 80–94 mm Hg (Hills, 1966; Hills and LeMessurier, 1969). This corresponds very closely to the failure of carbon dioxide (0 → 50 mm Hg) to replace oxygen (150 → 20 mm Hg) in gas pockets as measured by Campbell and later workers, i.e. inherent unsaturation = 150 − 20 − 50 = 80 mm Hg.

This inherent unsaturation, as directly displayed by the partial vacuum, was interpreted as arising from two sources:

(1) the fact that the solubility of carbon dioxide is some 25-fold greater than that of oxygen, so that metabolism is converting a *less* soluble gas (oxygen) into a comparable number of molecules, or slightly fewer ($RQ \simeq 0{\cdot}8$), of a *more* soluble gas (carbon dioxide) so that the total tension of local tissue oxygen + carbon dioxide tends to be reduced. Note that it is total gas *tension* and not concentration which determines the imminence of phase separation;

(2) the shape of the oxyhaemoglobin dissociation curve where large elevations in arterial P_{O_2} may result in little change in venous P_{O_2} and hence in tissue oxygen levels. Compare the difference $(P'_{aO_2} - P'_{vO_2})$ for elevated oxygen with $(P_{aO_2} - P_{vO_2})$ for normal levels in fig. 10.

Varying pressure and composition

While such numerical agreement is reassuring, the most significant findings of the Adelaide group came when they varied the composition of the breathing mixture and the pressure. They then found that the inherent unsaturation

(a) increases linearly with absolute pressure for an inspired mix of constant composition (fig. 82(a));

(b) decreases linearly with mole fraction of inert gas in the inspired mix (fig. 82(b)).

This is in direct agreement with Equation 52 and, moreover, the gradients of the slopes found experimentally (figs. 82(a) and (b)) agree with those predicted in this expression. However, as it stands, the inherent unsaturation has been directly measured as a pressure difference, while Equation 52 refers to a driving force for nitrogen elimination (ΔP_{N_2}); so what is the connection?

Implications of the inherent unsaturation

It has been established experimentally that the ambient hydrostatic pressure will exceed the absolute pressure of all gases within a constant-

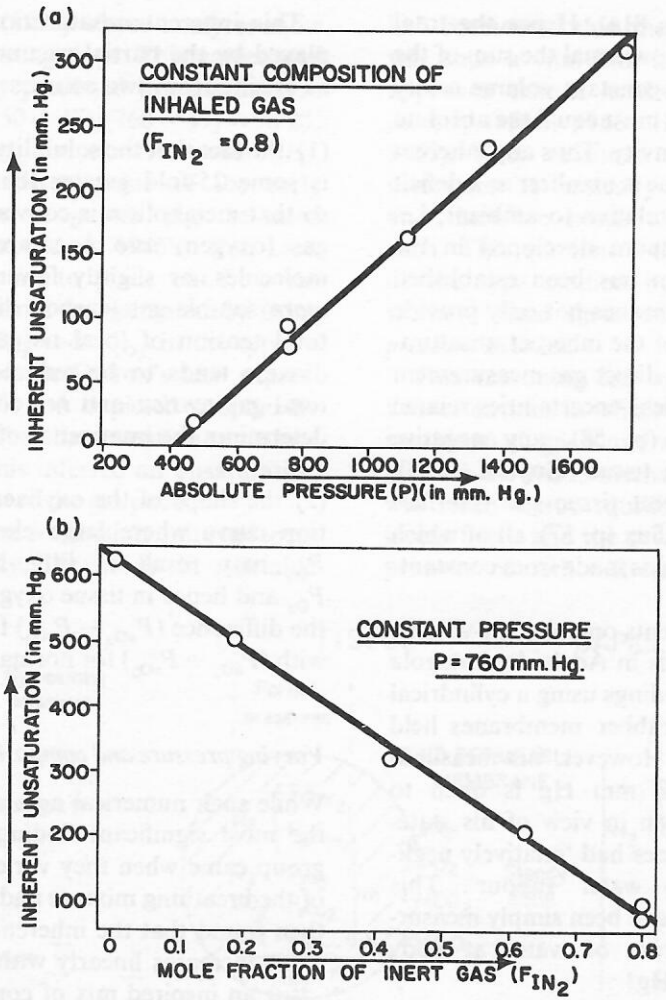

Fig. 82 Experimental measurements of the variation of the inherent unsaturation in the rabbit (a) with inert gas fraction in the breathing mix at normal pressure and (b) with pressure for a given breathing mix (air). Data from Hills and LeMessurier (1969)

volume cavity formed by a rigid membrane after the system has come to equilibrium. Thus the membrane is restraining the compliant tissue surrounding it by exerting a force equal to the developed vacuum, i.e. equal to the inherent unsaturation. If that rigid membrane is now removed, the gas is compressed by this amount. Thus the total partial pressure of all the enclosed gases will rise and exceed the total of the tensions which each strives to attain under those same steady-state conditions. Hence the equilibrium is destroyed and the degree of compression in letting the walls move (fig. 81(b)) until cavity pressure is elevated to ambient now becomes a driving force for the walls to absorb the gas in what is now a constant-pressure cavity. The inherent unsaturation thus becomes the driving force for absorbing *all* gases previously equilibrated within the permeable rigid membrane. However, since carbon dioxide, oxygen and water vapour equilibrate so much faster than nitrogen as Campbell (1924) and later workers have shown, nitrogen is the gas controlling the rate of resolution of the gas phase in tissue and hence virtually the whole of the inherent unsaturation

becomes a driving force for nitrogen resolution. There will be an additional contribution to this compression provided by tissue compliance (δ_t) and a direct tissue–gas interface ($2\gamma/r_b$) formed by removing the rigid membrane. Together these contribute a mechanical term (B) to the driving force for nitrogen elimination (ΔP_{N_2} in Equation 52 and depicted in fig. 40).

While it is convenient to start from a rigid cavity and then remove the rigid permeable walls in understanding how the driving force arises for resolving the compliant cavity remaining, the same ΔP_{N_2} applies whatever means was used to create it—including decompression. Hence it is particularly significant that there is direct experimental evidence to support Equation 52, not only from the linear relationship between ΔP_{N_2} and both P and F_{IN_2} and the value of the constant (m) but from cases of constant-pressure cavities (bubbles) induced by decompression (Chapter 6).

Significance

The inherent unsaturation is important not only for determining the driving force for inert gas elimination once a stable gas phase is established but for setting the point of phase equilibrium. In other words, it also determines both the point of inception of gas separation (fig. 29) and the volume 'dumped' if this is exceeded (fig. 31).

The concept that the driving force (ΔP_{N_2}) increases with absolute pressure when the gas phase *is* present (Equation 52) was used as major evidence in Chapter 6 that conventional diving schedules—and U.S.N. tables in particular—were really *treatment* tables for an asymptomatic gas phase produced during their first long 'pull' towards the surface. Hence direct experimental justification of Equation 52 was essential before such a serious criticism of standard practice could be made.

The concept of the inherent unsaturation implies that the term 'saturation diving' is really a misnomer, since no diver can 'saturate' with gases in the true thermodynamic sense however long he may stay at one depth. He would need to have either zero metabolism or to breathe 100% inert gas—neither state to be recommended. On the other hand, his inert gas would equilibrate at depth and so a more appropriate term might be 'steady-state' diving.

If a diver starts his ascent to the surface from a steady-state condition, then his whole decompression will be determined by the inherent unsaturation if phase equilibration dictates the state of the critical tissue. This is particularly compatible with the observation of many such exposures on men where the safe rate of ascent was found to be hyper-dependent on the inspired P_{O_2} and, at the tissue level, reflects more than simply the substitution of inert gas by oxygen (Vorosmarti *et al.*, 1975).

The final implication of the inherent unsaturation concerns oxygen breathing. Whereas it is obviously ideal for increasing ΔP_{N_2} in Equation 52 and hence superb for bends treatment (p. 232), oxygen is effective because it does not replace inert gas at the tissue level in the way that it does at the alveolar level. However, this vacancy which it leaves would not be welcome by the physician who may try to use hyperbaric oxygen to oxygenate peripheral tissues in certain disease conditions (see p. 15).

Quantitative Description

The model invokes two tissues; a relatively avascular connective tissue which determines limb bends and a 'vestibular' tissue needed for very great depths or when switching inert gases at an appreciable pressure. However, for normal diving, just one tissue and hence one equation is needed to describe the *level of insult* and hence the imminence of decompression sickness. By the same token, just one equation is needed to describe the *primary event*—the inception of a stable gas phase.

This leads to two basic equations which are needed.

(1) An expression for the condition that gas can separate from solution anywhere within the critical tissue, this event being random but possible whenever gas tensions have exceeded the point of thermodynamic equilibrium as

depicted in figs. 26 and 29. This equation for avoiding the primary event is particularly relevant to optimization when, if correctly formulated, it should also avoid haematological complications and those associated with other forms of decompression sickness—at least, unless the 'vestibular tissue' takes over as rate-determining.

(2) An expression for the imminence of symptoms and hence for the level of insult and its proximity to the pain threshold in the critical tissue. This is relevant to any decompression however it was formulated and applies where the condition for gas phase inception described by (1) has been far exceeded. Hence it is more relevant to the analysis of data and in checking the model rather than in optimizing a decompression.

If such an analysis is applied to a long history of gas phase presence, then ignoring haematological factors could introduce an error but, for the purpose of model testing, there are plenty of other cases where this should have been avoided. However, before the conditions described in (1) and (2) can be expressed comprehensively, it is necessary to know how to describe the distribution of gases *before* any inception of the gaseous phase.

Metabolic gases

Consider each of the gases likely to be present in the critical tissue and hence in any bubble. Water vapour will be present in any cavity at its vapour pressure for body temperature ($P_w = 47$ mm Hg) and will evaporate or condense on the surrounding tissue to maintain that value. Thus it will always contribute P_w towards the total tension tending to induce cavitation (fig. 29), or to grow a bubble.

The preceding discussion of the inherent unsaturation (pp. 239–243) leaves little doubt that oxygen and carbon dioxide are present at their *venous* tensions or, at least, the sum $(P_{O_2} + P_{CO_2})$ does not exceed $(P_{vO_2} + P_{vCO_2})$ and may be less. However, since this venous sum is small relative to diving pressures, little error can be introduced by taking this value which, if anything, would err on the safe side. Thus, it is safe to say that

$$m = P_{O_2} + P_{CO_2} + P_w \leqslant 117 \text{ mm Hg} = 5{\cdot}1 \text{ fsw} \quad (80)$$

Moreover, direct measurement of the inherent unsaturation indicates that this holds over wide ranges of arterial P_{O_2} (fig. 82).

Inert gas distribution

From a calculation standpoint, it is most unfortunate that the evidence on the perfusion versus diffusion controversy does not allow either to be ignored, since any hybrid model is far more difficult to describe mathematically than if one or other were to predominate. Moreover, resistance to gas transfer imparted by diffusion is not restricted to a simple membrane barrier but is imposed by the bulk of the 'cellular' phase as a whole. Thus the final model, although so simple to depict schematically (fig. 64), is most difficult to describe analytically.

This can be redrawn (fig. 83) to enable distance co-ordinates (r) to be assigned, when the effectively fully stirred 'extended vascular' zone has boundaries ($0 \leqslant r \leqslant a$) and the bulk 'cellular' phase has $a \leqslant r \leqslant b$. The 'extended vascular' zone comprises the intravascular plus that part of the interstitial space in motion or which is so permeable relative to cellular diffusivities (D_c) that it is also at the same tension as the efferent fluid (venous blood plus lymph). This is therefore effectively fully stirred and has a uniform venous tension (p_v).

The less permeable 'cellular' zone is assumed uniform but at first sight it cannot be determined whether it is spherical, cylindrical, flat, annular or even whether gas diffusion paths will converge or diverge. Thus the mode of curvature, if any, is unknown let alone the magnitude as expressed by the radius of a boundary (a or b in fig. 83). However, this has been circumvented by the use of a dimensionless 'shape factor' Hills (1969c) for the cell which must have a volume (V), surface area (A) and mean radius ($b - a$), when

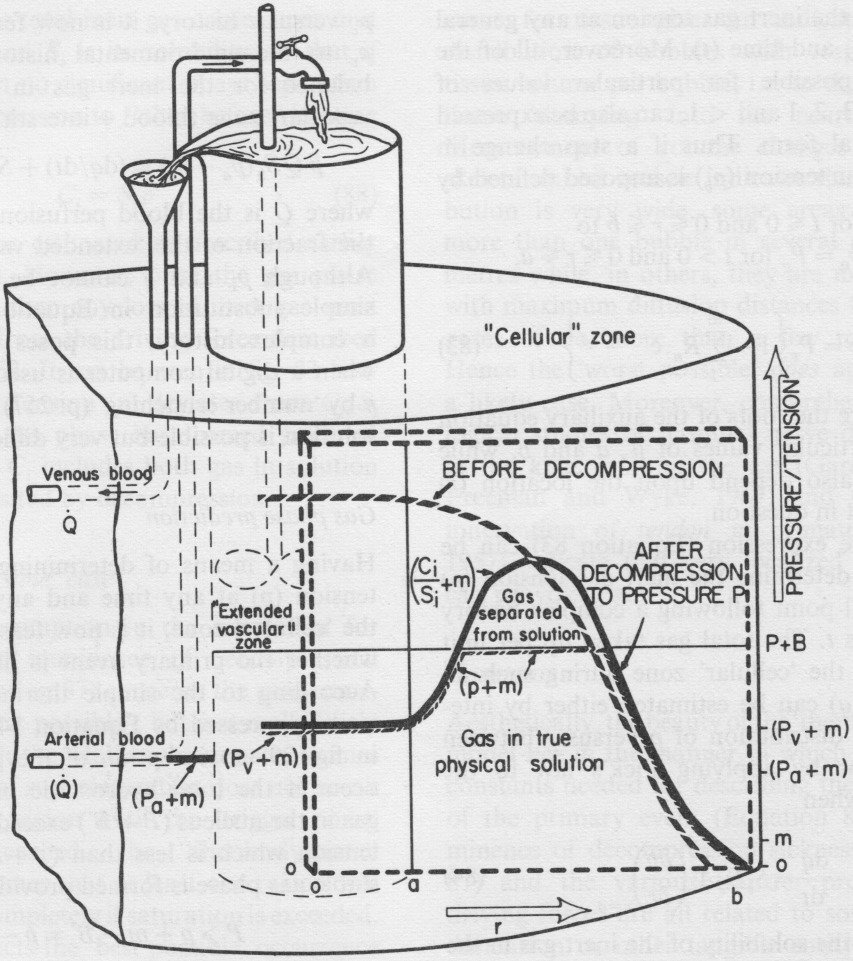

Fig. 83 The tissue model illustrated as a bulk 'cellular' zone of uniform permeability in which all transfer occurs by diffusion of gas supplied from the 'extended vascular' zone. This is depicted as the stirred tank in which arterial blood immediately mixes with the contents as it enters, while venous blood is effectively the overflow leaving in equilibrium with those contents in which there are no concentration gradients. The inset graph depicts the spatial distribution of gases through both zones for an arbitrary decompression, C_i being the inert gas content and p_i its tension. Where phase equilibrium has been exceeded, the excess gas has been 'dumped' in accordance with the 'thermodynamic' principle and the inert gas tension cut off at $P+B-m$ rather than following the contour of (C_i/S_i) as though it had remained in solution

$$\text{shape factor } (\mu) = A(b-a)/V \qquad (81)$$

where $\mu = 1{\cdot}0$ for a flat slab, $2{\cdot}0$ for a long cylinder, $3{\cdot}0$ for a sphere, etc. Taking the sphere as an example, $A = 4\pi(b-a)^2$ and $V = 4\pi(b-a)^3/3$, when substitution in Equation 81 gives $\mu = 3{\cdot}0$

Moreover, it was pointed out (Hills, 1969c) that, for all but cylindrical systems ($\mu = 2{\cdot}0$), the Fick–Fourier equation (19) for bulk diffusion of an inert gas ($M = 0$) in a static phase ($\dot{Q} = 0$) can be expressed in the general form

$$\frac{1}{D_c}\cdot\frac{\partial p}{\partial t} = r^{(1-\mu)}\frac{\partial}{\partial r}\left[r^{(\mu-1)}\left(\frac{\partial p}{\partial r}\right)\right] \qquad (82)$$

where p is the inert gas tension at any general location (r) and time (t). Moreover, all of the solutions possible for particular values of μ, e.g. $\mu = 3, 2, 1$ and < 1, can also be expressed in a general form. Thus if a step change in extracellular tension (p_v) is imposed defined by

$$p = 0 \text{ for } t \leqslant 0 \text{ and } 0 \leqslant r \leqslant b \text{ to}$$
$$p_v = P_v \text{ for } t > 0 \text{ and } 0 \leqslant r \leqslant a,$$

then

$$p = P_v\left\{1 - \sum_1^{\infty} R_n . e^{-\alpha^2 n D_c t}\right\} \quad (83)$$

where α_n are the roots of the auxiliary equation for the particular values of μ, a and b, while R_n values also depend upon the location (r) of the point in question.

The same expression (Equation 83) can be applied to determine the inert gas tension (p) at a general point following a complex history of p_v versus t. The total gas taken up per unit volume by the 'cellular' zone during such an operation (q) can be estimated either by integrating the distribution of p versus r between a and b or by applying Fick's law to the boundary when

$$\frac{dq}{dt} = S_c D_c A' \left(\frac{\partial p}{\partial r}\right)_{r=a} \quad (84)$$

where S_c is the solubility of the inert gas in the 'cellular' phase and A' is now the surface area per unit volume.

For a single step in p_v, the solution for q is exactly similar to Equation 83, where the roots (α_n) are the same but coefficients (R'_n) are different.

The great advantage of the shape factor lies in the fact that α_n and R_n values lie on smooth curves when plotted against the values of μ, i.e. 3·0, 2·0, 1·0 and $< 1{\cdot}0$, for which mathematical solutions to Equation 82 are feasible (Hills, 1967b, 1974a). The shape factor is by no means comprehensive, nor offers a unique value for a particular cell, but it does give some 'handle' on the particularly difficult mathematics posed by the irregular nature of tissue 'geometry' and avoids the need to postulate ideal shapes.

Having related both p and (dq/dt) to the p_v versus t history, it is now feasible to relate p_v to the environmental history by a mass balance for the inert gas in the 'extended vascular' zone (blood + interstitium), when

$$\beta \dot{Q} S_b (p_a - p_v) = (dq/dt) + S_b (dp_v/dt) \quad (85)$$

where $\dot{Q}$ is the blood perfusion rate and β is the fraction of the 'extended vascular' region. Although p_v and q cannot be eliminated by simple substitution in Equations 83–85, for a complex history, this poses little difficulty when a digital computer is used to determine p by 'number crunching' (p. 257). An analytical solution is possible but very difficult to handle.

Gas phase prediction

Having a means of determining the inert gas tension (p) at any time and any point within the 'cellular' zone, it is now feasible to predict whether the primary event is likely to occur. According to the simple thermodynamic criterion expressed by Equation 14 and depicted in fig. 29, no inception of the gas phase can occur if the total hydrostatic pressure of the gas in the nucleus ($P + B'$) exceeds the total gas tension which is less than ($p + m$). Hence no stable gas phase is formed provided

$$P > p + m - B' = p - c' \quad (86)$$

at all points within the tissue, where c is $(B' - m)$.

Imminence of bends

While Equation 86 describes the primary event, the gas phase must grow considerably before it can cause the level of insult to reach its threshold for symptoms. The imminence of limb bends depends upon the local pressure differential to bend a nerve ending which, in turn, is largely determined by the maximum volume of gas (v) which can separate from solution in unit volume of tissue ($V = 1$), i.e. the 'worst possible' case. Substituting for v according to Equation 9, Equation 31 gives the condition that bends can occur if for one gas (i)

$$\frac{C_i - S_i(P + c)}{(P + c)} > \frac{S_c - \delta_f}{K} \quad (87)$$

where C_i is the total inert gas content per unit unit volume and S_i is its solubility, so that if there had been no significant phase separation before decompression to P then before violating Equation 86

$$C_i = S_i p \qquad (88)$$

where p can be estimated as described earlier (pp. 244–246). However, once the gas phase *is* established, then any loss of inert gas must be estimated from the driving force described by Equation 52. It can be seen in fig. 83 how the actual gas tension (p) deviates from C_i/S_i wherever the gas phase has formed since, in those regions, C_i includes both gas in solution and that deposited by decompression.

The 'worst possible' case

The major assumption in the expression for estimating the imminence of decompression sickness (Equation 87) concerns the case considered the 'worst possible'; but is it really likely to occur? While the 'Haldane' rationale assumes that the state of supersaturation does not break down in any region until the 'trigger point' has been reached, the 'thermodynamic' approach assumes that at least one region will break down completely if saturation is exceeded. Thus one depicts the 'best possible' occurrence and the other the 'worst possible' while something in between may be more appropriate but much more difficult to describe mathematically.

To test the probability that the 'worst possible' is likely to occur, this writer has performed an experiment based on the following argument. It can be measured or predicted mathematically how a single bubble will grow as it drains the dissolved gas from a large volume of tissue which has been decompressed. Two bubbles far apart will give twice the volume change but otherwise follow the same profile. However, when two bubbles are near to each other, they will start by following the same profile but the rate of growth will reach a plateau sooner as they both start to drain gas dissolved in the same tissue zone. Thus the shape of the volume-versus-time curve for tissue gives an indication of the distribution of bubbles with respect to the tissue volume per bubble, i.e. to the volume of tissue 'dumping' into the nearest bubble. Measurement of volume changes in excised skeletal rabbit muscle indicates that the distribution is very wide, some areas having no more than one bubble in several cubic millimetres while, in others, they are most profuse with maximum diffusion distances for dumped gases of no more than a few micrometers. Hence the 'worst possible' *does* appear to be a likely case. Moreover, comprehensive histological studies of terminal nerve distributions in the knee joint of the cat (Gardner, 1944; Freeman and Wyke, 1967) and the general innervation of *tendon* in primates (Stilwell, 1957) indicate that very localized stimulation can provoke pain.

Constants

Aesthetically, the beauty of the 'thermodynamic' model lies in the manner in which each of the constants needed for describing the occurrence of the primary event (Equation 86), the imminence of decompression sickness (Equation 87) and the various transfer processes and driving forces are all related to some physical dimension or other tangible entity. Surveying Equations 80–88, these include D_c, S_c, S_b, A, A', μ, B, B', m, a, b, δ_t, δ_f and K. Some of these are known without a doubt, such as $S_b = S_c =$ solubility of the inert gas in water at body temperature, while other constants which cannot be assigned values without positive identification of the critical tissue can be grouped. Thus the final list of constants reduces to

(1) (D_c/a^2) with dimensions of $(\text{time})^{-1}$.
(2) μ—the dimensionless shape factor.
(3) b/a—dimensionless (unnecessary for $\mu = 1$).
(4) $\dot{Q}$—with dimensions of $(\text{time})^{-1}$.
(5) $(\delta_t - \delta_f)/K$—index of pain sensitivity (Z)—(volume) $(\text{pressure})^{-1}$ (unless δ_f varies).
(6) c or c' related to mechanical factors (B or B') and the total venous tensions (m), while the other constants can be related to each other as

$$A = \beta A'/V = \beta\mu/(b-a) \quad (89)$$

and

$$\beta = (a/b)^{\mu} \quad (90)$$

thus reducing the effective number of constants to only six. Moreover, if a particular anatomical tissue is selected, then the geometric and perfusion terms are fixed to reduce the number of degrees of freedom in selecting constants to only two.

Having described the model quantitatively it is now feasible to assess its viability, not only in qualitative terms but to ensure that the equations are compatible with the words since *only the equations* are ultimately used to optimize a decompression.

Qualitative Assessment of the Model

Before any model is used to optimize the decompression procedure, it should first be tested for compatibility with the many general qualitative facts and then with basic quantitative data. Moreover, the derivation of certain fundamental constants from these quantitative analyses and any agreement with known physiological values offers the final test of fundamental adequacy.

From a quantitative standpoint, the comprehensive expression describing the imminence of decompression sickness (Equation 87) is essentially a comparison of two terms, the left-hand side describing the level of insult to the tissue, while the right-hand side relates the critical threshold to individual factors.

Level of insult

It can be seen that the condition for bends is more likely to occur if the total inert gas content of the tissue (C_i) is higher. This is compatible with the fact that longer and deeper exposures (p. 36) or those with a greater fraction of inert gas in the breathing mixture (p. 37) are more likely to produce bends—all for the same decompression.

A more soluble inert gas ($S_i\uparrow$) is more likely to violate the condition expressed by Equation 87 such as nitrogen tending to produce lower minimum bends depths than helium for the same decompression and fraction of the breathing mix (p. 38). On the other hand, a faster diffusing gas ($D_c\uparrow$ in Equations 82–84) will increase the content of inert gas (C_i) before it asymptotes, so that air can take over from heliox as superior for deep 'bounce' dives (p. 188).

The comprehensive expression (Equation 87) shows that the critical condition for bends is more likely to be violated if there is greater decompression *per se* ($P\downarrow$). Moreover, the equation expresses the concept of a threshold, where reversal of the insult by elevating P (i.e. recompression) can reverse the development of symptoms (p. 41).

So far only the factors related to the degree of insult as expressed by the left-hand side of Equation 87 have been discussed—all equally well explained by other theories.

Individual factors

The right-hand side of Equation 87 refers more to the threshold itself and is therefore more closely related to individual factors. The distribution in susceptibility is determined largely by individual differences in threshold pressure differential (δ_t) for bending or otherwise distorting a nerve ending. Osmosis would act by increasing δ_f while potentiating factors such as serotonin could decrease δ_t where the threshold term is then more likely to be exceeded by the insult—i.e. by the left-hand term in Equation 86.

Obesity is reflected by an elevation of lipid inclusion in the critical tissue, if it is tendon (as observed in that tissue in guinea pigs—p. 60) which, in turn, elevates the solubility (S_i) and hence the likelihood of symptoms (p. 41).

Adaptation to decompression (p. 60) is expressed by the decrease in the modulus ($K\downarrow$) with successive exposures, thus elevating the threshold so that the same insult is less likely to exceed it. Similarly the increased susceptibility with age (p. 60) can be explained by the critical tissue becoming less compliant ($K\uparrow$), thus increasing the likelihood of exceeding the threshold.

Fluid shifts

Individuals with a higher natural fluid turnover rate are likely to have a lower tissue fluid pressure ($\delta_f\downarrow$) and hence higher ($\delta_t - \delta_f$) to explain their marginally lower susceptibility (p. 43). By the same token, the reduction of extravascular fluid pressure ($\delta_f\downarrow$) which should result from administering low molecular weight dextran is likely to decrease the threshold and even reverse a marginal bend—as observed clinically (p. 232).

Haematological disorders leading to trauma or oedema and a general exodus of fluid from the vascular system will elevate extravascular fluid pressure ($\delta_f\uparrow$), thus reducing the level of insult needed to exceed the bends condition (Equation 87). However, no claim is made in its presentation that the model can accommodate blood disorders, since this writer, at least, can find no simple means of quantifying them in terms of environmental history and hence relating them to δ_f.

On the other hand, it is an easier task to relate the fluid shift caused by any gas-induced osmosis to environmental parameters. Particularly during a 'saturation' decompression, where there is a large reservoir of inert gas deep in tissue, this gas could exert a significant osmotic pressure tending to 'pull' water out of blood and so increase δ_f. Hyperoxia would tend to augment this process (p. 222), as would a switch of inert gas, quite apart from any effect which this might have on the volume of any bubbles present. Since the shifting of fluid is a slow process, it is possible to spend too long decompressing when δ_f reduces the threshold (right-hand) term in Equation 87 to a level at which a minimal gas-mediated insult (left-hand term) is all that is needed to provoke symptoms. This could explain the low bends incidence (Bühlmann, 1969) in some short (8-day) decompressions from 1000 fsw compared with 21 days used by the U.S. Navy (Workman, 1969). However, these are exceptional exposures during which any one of a host of physiological factors could become critical.

The 'vestibular' tissue

For such long deep exposures, a second tissue with a high inherent threshold (δ_t) could take over as symptom-presenting if the combination of distensibility ($1/K$) and fluid shift (δ_f) were to reduce the threshold level for the critical insult ($\delta_t - \delta_f)/K$ to a value lower than needed to provoke marginal limb bends in the aqueous connective tissue. Moreover, δ_f is likely to be greatest if shifts due to a switch from helium to nitrogen as the inert gas are superimposed upon pre-existing pressure differentials. Thus the rate of fluid shift would be directly proportional to the osmotic driving force $\sigma S_c(p_i - P_{ai})$ i.e. $\delta_f \propto \sigma S_i(p_i - P_{ai})$ where P_{ai} is the arterial inert gas partial pressure. If, therefore, the proportionality constant is (K'), then for decompression from a steady state at an absolute pressure at P_1 to P_2 breathing the same mix (inert gas fraction F_i):

$$P_1 = P_2 + (\delta_f/K'\sigma S_c F_i) \qquad (91)$$

Thus, on a plot of P_1 versus P_2, such a tissue would have a lower gradient (1·0) yet an appreciably higher intercept ($\delta_f/K'\sigma S_c F_i$) than the connective tissue for limb bends. A physiological system particularly sensitive to internal fluid shift is the vestibular apparatus. Hence it is very interesting that Equation 91 gives a straight line which would intersect the limb bends line (Equation 27) in just about the same manner as found for P_1 versus P_2 plots for 'vestibular' and 'limb' bends in practice (fig. 20). Moreover, the gradient of unity is particularly compatible with the critical insult arising from an incompressible fluid—as seen in Equation 91.

If this explanation is correct, then it is tempting to speculate on the use of osmotic agents and mild diuretics during decompression. In fact, this could be a factor contributing to the successful prescription of alcohol to his divers by Krasberg (1976), particularly when given at the start of decompression from 1000 fsw and therefore most effective over the deeper stages where vestibular problems are more likely.

Kinetic factors

To return to normal exposure pressures, there are a number of features of the model not expressed mathematically yet which need appraisal. Most of these involve time.

The first is the random occurrence of symptoms (p. 32). For this to occur there must be a random physical or physiological mechanism—and what could be more appropriate than the random nature of nucleation (Chapter 4), particularly of the gas phase by decompression (p. 84). This is expressed quantitatively in Equations 86 and 87 that no gas phase or no bends will occur if the respective thresholds for nucleation or pain are not exceeded but this does not state that the converse need apply. If the right-hand sides of Equations 86 and 87 exceed the respective left-hand sides, then there are only certain probabilities that a nucleus will be activated into growth or that nucleation will be so profuse that the 'worst possible case' will actually occur on that occasion.

The random onset times of symptoms (p. 33) are highly compatible with the concept that the induction period represents the time needed for separated gas to coalesce until the combined mechanical stress of the congregated gas exceeds the local pain threshold. Moreover, exercise hastens onset (p. 45) and can be envisaged as the ideal motion for coalescing gas deposited in the sheaths of any connective tissue. Also, the more that the decompression exceeds marginal limits, the more gas separates and hence less coalescence is needed for the increasing level of insult to exceed the threshold. Thus the model can explain why severe cases have shorter onset times (p. 34).

Another action likely to promote coalescence is alternate expansion and contraction of bubbles. Hence the lower bends altitude found on repetitive aerial exposures (p. 39) can be explained by the need for less gas to separate from solution in the first instance, when the congregating mechanism is more effective. Similar arguments can explain apparent anomalies in decompression needed for repetitive dives (p. 162).

Coalescence can also explain why a delay in recompressing a bends case can unduly prolong the treatment needed, while it also offers a simple explanation for the change in the X-ray of the subject's soft tissues. Postulated as the process delaying the onset of symptoms, coalescence could also be the factor rendering surface decompression/decanting feasible. The earlier appearance of symptoms on helium diving can be attributed to congregation by the faster rate at which the larger of two adjacent bubbles grows at the expense of the smaller (p. 96). A minor criticism of the model concerns the virtually instant 'dumping' of gas on decompression assumed for the sake of mathematical simplicity. It is quite likely that growth will prove to be a rate-contributing factor but probably not as important as coalescence (p. 96). However, the introduction of growth terms immensely complicates Equation 87 and has yet to be justified, since it is much more important to predict *whether* bends will occur rather than *when*.

Exercise and temperature

Exercise 'on the bottom' must increase the blood perfusion rate ($\dot{Q}$) and hence the gas taken up by the 'cellular' zone (q) and, in turn, C_i in Equation 87; so that bends become more likely on subsequent decompression.

Moreover, the increased gas uptake reflected by the decrease from 35 to 25 min in safe 'bottom' time at 150 fsw for no-stop air decompression (p. 46) is just about what would be expected from the increased perfusion and vasodilation of the *tendon* to which the exercising muscle is connected. The effect is much less than any anticipated for muscle itself. When it occurs, the bends pain is felt around the joint selectively exercised (p. 33) and in sites where there are tendons, among other connective tissues. Similarly, exercise during any pre-oxygenation will tend to accelerate nitrogen wash-out, reducing C_i and so tending to protect aviators against decompression sickness (p. 46).

By conventional reasoning, one might expect exercise during decompression to hasten inert gas wash-out and so reduce the likelihood of symptoms but the reverse is found to hold

in practice (p. 46)—at least using U.S. Navy tables. By the 'thermodynamic' approach exercise *during* decompression would act in two opposing ways: firstly to coalesce gas already separated from solution into a pain-provoking bubble; secondly, to increase blood flow ($\dot{Q}\uparrow$ in Equation 85) and vasodilation ($A'\uparrow$ in Equation 84) tending to eliminate what has remained in solution. This writer therefore contends that the outcome of the race between these processes will depend upon how much gas is 'dumped' and how much remains in solution. Thus a U.S. Navy profile with its characteristic long first 'pull' is likely to precipitate most of the tissue gas allowing coalescence to predominate in hastening eventual symptoms. On the other hand, during a decompression with more time spent deeper and hence more gas remaining in solution, increased wash-out of this gas should win and exercise should prove advantageous—as observed in caisson workers before the 'Haldane' rationale was introduced (p. 46).

Increasing the metabolic rate without the mechanical action of coalescence should decrease tissue P_{O_2} more than it elevates the P_{CO_2}, thus decreasing the value for m (Equation 86) and hence c in Equation 87. The small change in c with time of day is only likely to be significant relative to low values of P, when it can explain the small diurnal effect noticed in aerial decompressions (p. 44).

Temperature is as well explained by the 'thermodynamic' as by any other model. Apart from the obvious thermal effects on vasoconstriction and hence gas exchange in the peripheral tissues, there is the increased solubility of gases at lower temperatures. Thus at high pressures, where the thermal capacity of the inspired gas is significant relative to the pulmonary circulation, there could be slight cooling of blood in the lung resulting in a higher gas uptake. This could lead to a small amount of supersaturation as arterial blood returns to core temperature. This, in turn, would have the net effect of increasing the tissue inert gas content (C_i in Equation 87) and hence the likelihood of decompression sickness. This line of reasoning adds further justification for warming the breathing mix at greater depths.

Critical features

So far, the 'thermodynamic' approach has been assessed in the light of practical experience (Chapter 2) rather than on the results of experiments specifically designed to test certain facets of the model. These have already been described in detail in Chapters 3, 6 and 7 and their compatibility requires no further discussion, since the same conclusions were used in deriving the model originally.

Many of these critical tests centred around the inherent unsaturation and its ability to provide a driving force for inert gas elimination. One of the more practical cases which the model can interpret is the lesser advantage to be gained by undertaking pre-oxygenation at altitudes which can precipitate sub-symptomatic bubbles (p. 37). It also explains the great advantage gained by the use of oxygen and high pressure ($P\uparrow$) in treatments ($\Delta P_{N_2}\uparrow\uparrow$ as $F_i \rightarrow 0$ or $P\uparrow$ in Equation 52). p. 138

Quantitative Assessment

In addition to offering explanations for some basic facts, the qualitative assessment has indicated how the analytical description of the model is also compatible. In other words, the equations predict changes in the right direction. However, the next step is to determine whether those correctly predicted changes are of the right magnitude. Perhaps the simplest order for undertaking this more rigorous appraisal is to look first at the relationships between the key variables, then to see what agreement there is between various values derived for the constants and, finally, to compare the values which the decompression data would indicate for basic tissue parameters with known values.

Apparent decompression ratio

The first characteristic to explain is why a constant decompression ratio (P_1/P_2) appears to hold for simple air diving up to 300 fsw or,

rather, a linear relationship between P_1 and P_2 (fig. 35). Just such a linear relationship was derived on p. 120 (Equation 43) on the basis that a constant volume fraction of separated gas (v in Equation 28) is responsible for marginal limb bends. This same equation (28) forms the basis for the comprehensive expression for the imminence of bends (Equation 87) after relating v to the critical pain threshold (δ_c). Moreover, the constants in these equations are compatible if the tissue is predominantly aqueous or, at least, contains up to 3% lipid (p. 194).

The derivation of these constants leads to the second set of data needing correlation by any comprehensive expression for the imminence of limb bends; the comparison of the minimum bends depth for air, the same for helium : oxygen and the minimum bends altitude for aerial decompression. It was shown on p. 193 how these can be correlated exactly if the volume fraction of separated gas ($v = 0{\cdot}0045$) and the constant (c) has a value of 5·1 fsw for the marginal onset of bends. Thus, from Equations 64 and 80, a value for the combined mechanical contribution (B) of interfacial tension and tissue compliance can be derived:

$$B \leqslant 10{\cdot}2 \text{ fsw} \qquad (92)$$

This value will be related to more fundamental constants later (p. 253).

The third basic feature of decompression data concerns the remarkable adherence of bounce dives to the $\sqrt{(t)}$ relationship (p. 122). Thus, for a single exposure, $C_i \propto \sqrt{(t)}$ in Equation 87. It was discussed earlier (p. 189) how uptake by diffusion into a bulk of almost any reasonable shape follows the $\sqrt{(t)}$ relationship for small values of t. This is compatible with the predominantly bulk diffusion aspect of the 'thermodynamic' model.

Constants

Another check of the analytical description of the model is provided by the point of inception of the gas phase in tissue. Mathematically, this is described by Equation 86 which gives the absolute pressure for aerial phase separation as $0{\cdot}8(760 - 47) - B' + m$. When compared with altitudes of 10,000–12,500 feet ($P = 474$–533 mm Hg) for the first changes in the X-rays or in cerebrospinal fluid volumes, this gives $B' > 202$ mm Hg (8·8 fsw), a result compatible with the earlier estimate of B from minimum bends depths as 234 mm Hg (10·2 fsw).

Yet another crude estimate of B can be obtained from the analysis of bubbles formed by decompression (Harris *et al.*, 1945b). In exercising animals ($P_{O_2} \simeq 0$; $P_{CO_2} \simeq 50$ mm Hg) these contained 95% nitrogen on a dry gas basis at normal pressure:

$$\frac{\text{(nitrogen)}}{\text{(total gas)}} = \frac{(760 + B - 50 - 47)}{(760 + B - 47)} = 0{\cdot}95 \qquad (93)$$

which gives the mechanical 'overpressure' of the bubble (B) as 287 mm Hg. This estimate is crude, since a resting value of $P_{CO_2} = 46$ mm Hg gives $B = 207$ mm Hg but the range encompasses the earlier estimate of B.

Relation to fundamental parameters

It could be argued that B (or B' before a nucleus is activated) is simply part of a 'fiddle factor', c, needed to make the data fit the comprehensive expression for the imminence of symptoms (Equation 87) and its forerunners (Equations 26 and 28). Of the two terms m and B contributing to c, there can be little argument that 5·1 fsw (117 mm Hg) is a reasonable maximum value for total gas tensions of the metabolic gases (m in Equation 80). However, this leaves B for which the most comprehensive analysis (p. 194) gives $c = 5{\cdot}1$ fsw (117 mm Hg) and hence $B = 234$ mm Hg. If B has the fundamental significance claimed, then it should give reasonable values for the fundamental physiological parameters δ_g and $2\gamma/r_b$ as defined in Equation 14. Taking δ_g as 11–26 mm Hg for no fluid shift ($\delta_f = 0$ in Equation 7), this gives a value of 208–223 mm Hg for $2\gamma/r_b$. Taking 50 dyne cm^{-1} for plasma as the value for γ, this gives a bubble radius (r_b) of 3·5 μm, i.e. a diameter of 7·0 μm, which would seem eminently reasonable by comparison with capillary diameters of 8·0 μm and intercapillary distances of the order of 20–40 μm.

Other mechanical factors

The next question in assessing whether the numbers are fundamentally consistent with the model concerns the stress which such a bubble would create. For no fluid shift ($\delta_f = 0$) and a pain-provoking stress of 11–26 mm Hg (Equation 7), together with a critical bends volume fraction of separated gas of $v = 0{\cdot}0048$, Equation 9 gives a bulk modulus for tissue distension (K) of $3{\cdot}7 \times 10^4$ dyne cm^{-2}. This should not be compared with Young's modulus for unidirectional stress in tendon, nor with values for compression which is largely measuring the compressibility of water but with those for bulk displacement. This is compatible with values of $2\text{–}11 \times 10^4$ dyne cm^{-2} found for connective tissue (Harkness and Harkness, 1965).

Diving data

So far the quantitative analysis has not involved time. Hence, in analysing data for practical dives which have actually been performed, it is necessary to select values for (D/a^2) and (b/a). The computations are too lengthy to repeat here but thirteen sets of naval dives have been analysed by the 'thermodynamic' approach (Hills, 1966), to predict the bends cases out of hundreds of exposures involving at least fifty different decompression profiles. Most were formulated by conventional calculation methods (e.g. the data of Crocker, 1957). The constants which enabled such a successful analysis to be made were $(D_c/a^2) = 0{\cdot}129$ min^{-1} and $b/a = 5{\cdot}29$ for rest to 4·73 for exercise—all for an annular model ($\mu = 0{\cdot}47$). The value of (D_c/a^2) agrees well with values for other 'transient' determinations (Table 12) while, taking 8·0 μm as the capillary diameter ($2a$), the (b/a) ratios give a fibre diameter $2(b-a)$ of the order 32 μm, well within the range for tendon of 10–40 μm in skin (Bear, 1952; Gustavson, 1956) and sometimes up to 300 μm (Bear, 1952; Verzar, 1957).

Allowing the ratio (b/a) to vary provides a convenient mathematical means of accounting for vasodilatation and hence exercise.

As for shape factors, the geometric configuration used in the foregoing analyses gave $\mu = 0{\cdot}47$ but the first bulk diffusion model of Hempleman (p. 122) was planar ($\mu = 1{\cdot}0$), while wash-out analysis from a different tissue (skeletal muscle) gave $\mu = 1{\cdot}55$ (Hills, 1967b). This corresponds to roughly cylindrical fibres in which about 22% of their surface is in contact with their neighbours. Unfortunately this is about the only study which has paid much attention to the *mode* of curvature, if any, of the boundary between the 'cellular' and vascular or 'extended vascular' zones. However, if this value of 1·55 is applied to the resting and working ratios (5·29 and 4·73) in accordance with Equation 90, it gives a volume fraction for the 'extended vascular' zone (β) of 7–9%. This value coincides with 8·0% for the blood volume of man as a whole but because tendon is a relatively avascular tissue, it is greater than blood volume and indicates that 'extended vascular' is an appropriate term for the effectively fully-stirred zone.

Derivations such as these may be dull and arduous but the sensible values which they produce for the various physical and physiological parameters enhances confidence in the fundamental adequacy of the model. It is now feasible to contemplate using this model to *optimize* a decompression and to 'graduate' from testing its viability on data which often included decompressions whose formulation would appear far from optimal by 'thermodynamic' reasoning. However, although tedious, this is much more cost-effective than jumping straight into formulating a new book of tables only to discover any major errors at great expense in the field—a practice all too common.

Decompression Optimization

Perspective

Whatever model or calculation method one may adopt for optimizing decompression, the designer attempts to select the depth at which the rate of inert gas elimination from the critical area is a maximum at each particular time. It is inefficient to proceed towards the surface

too slowly and yet if one attempts to 'beat the system', then there is a penalty to be paid later. By conventional 'supersaturation' approaches, this penalty is simply that bends will occur if one violates a 'trigger point' for bubble formation. However, if the 'thermodynamic' model is correct and the primary event does not coincide with the critical level of insult then, simply by waiting for bends to occur, the designer can be fooled into paying the penalty of over-decompression without realizing it. Hence the U.S. Navy, among others, (Chapter 5) probably far exceed the conditions for initiating the primary event (Equation 86) and yet do not appreciate the fall in gas elimination rate from the critical area unless they exceed it by such a margin that the volume of separated gas reaches the pain threshold (Equation 87). Once again, there is no point in measuring overall gas elimination rates, even from one tissue, since these reflect the behaviour of the statistical mean and not the 'worst possible' case.

It is all too easy to criticize other approaches but how can the expressions for predicting the primary event (Equation 86) and for estimating the level of insult (Equation 87) be used for optimization, assuming them to be correct?

Sequence of gas elimination and 'dumping'

The no-stop decompression limits leave no doubt that, on return to the surface, man can tolerate a gas content of his critical tissue well in excess of normal and yet not experience symptoms of decompression sickness. Moreover, much of the gas taken up at depth can be 'dumped' into the gaseous phase and yet remain asymptomatic under normobaric conditions. Hence the gas assimilated at depth which exceeds the quantity which can be tolerated at the surface needs to be removed over the depth range affording the greatest driving force for its elimination. In this way part of the total pressure change in returning to the surface is used for 'dumping' gas and part for eliminating it by gradual decompression. This leads to three alternatives:

(a) 'dump' and then eliminate;
(b) eliminate and then 'dump' the rest;
(c) do both simultaneously.

The last of these is simply a compromise between the first two and will not be considered in the basic argument. Taking the first approach, a long first 'pull' towards the surface will 'dump' much gas initially and then bring about the position of needing to use Equation 52 (appropriate to a gaseous phase) to estimate the subsequent elimination over the remainder of the decompression at the graded rate. However, this equation (52) indicates that the driving force is greater at greater pressure ($\Delta P_{N_2}\uparrow$ as $P\uparrow$). Therefore it has always been the contention of this writer (Hills, 1966) that it is better to reverse the conventional procedure (1) and eliminate the excess gas at the greater depths, where there is more driving force, followed by a rapid decompression over the last 20–30 fsw to 'dump' the remainder. This procedure (2) still forms a gaseous phase but to an extent just below the dimensions predicted to give pain (Equation 87). According to 'thermodynamic' reasoning, the U.S.N. tables represent an extreme form of sequence (1) since, in molecular terms, they initially 'dump' more gas than could be tolerated at the surface. Hence much of the gas to be eliminated must be derived from the gas 'dump' and therefore needs to be first resorbed from the gaseous phase, thus adding a further potential resistance to the kinetics in addition to reducing the driving force. This becomes more acute if coalescence has occurred between 'dumping' and resorption.

Hence, by 'thermodynamic' criteria, it is better to follow sequence (2). However, in advocating deeper stops for the pressure range for gradual decompression, just how deep should these start?

Criterion for optimum

Consider a subject making an arbitrary first 'pull' towards the surface following completion of his exposure on the bottom. If he is decompressed so far as to form the gaseous phase, then the appropriate expression (Equation 52) reveals that he would have achieved a better elimination

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rate at the first stop if he had made that stop rather deeper ($\Delta P_{N_2}\uparrow$ as $P\uparrow$). On the other hand, if he decompresses only a short distance and does not form the gaseous phase, then the expression appropriate to this case (Equation 51) reveals that a better driving force would have been achieved by coming up further ($\Delta P_{N_2}\uparrow$ as $P\downarrow$).

By repeating this exercise for successively deeper first stops *with* the gas phase forming and successively shallower first stops *without* it forming, the two come together to give a maximum ΔP_{N_2} for the unique point where the gaseous phase is just on the brink of inception. Thus the optimal condition is expressed by Equation 86 describing the primary event. Hence the diver rapidly decompresses by the inherent unsaturation plus the mechanical factors (B'), compressing gas in the unactivated nucleus (fig. 31). He then continues to follow the critical condition for initiating the primary event (i.e. gas inception) as this gradient continues to provide the maximum driving force for eliminating the excess gas, all of it just remaining in solution if the calculations are correct.

Since the particular transport model considered relevant to the kinetics of the critical tissue involves a bulk 'cellular' phase, this optimum condition (Equation 86) needs to be applied at all points. Thus it is first necessary to determine the point of maximum gas content and then ensure that this peak does not exceed the critical condition for initiating the primary event. For a single exposure other than steady state, the highest total tension of all will be located in the 'extended vascular' pool and the immediately adjacent wall. Thus the first stop is based on venous gas tensions only. However, as gradual decompression proceeds, the peak will gradually recede from this wall deeper into the tissue. If the overall gas content has not been reduced to a level at which it is safe to jump to the surface sooner, then the peak will recede to the 'back wall' and stay there. This is the point at which the decompressions for short exposures ('bounce' dives) join the universal profile found for starting with a steady-state condition (the 'saturation' curve).

Procedure

This line of reasoning has led to the following procedure for decompression optimization according to the 'thermodynamic' approach.

(1) Calculate the pressure (P_1) to which the diver could be rapidly decompressed from his bottom pressure (P_b) without precipitating the primary event in venous blood and hence in the 'extended vascular' zone. Applying Equation 86 to this case:

$$P_1 > p_{vi} + m - B' = F_i P_b - c \qquad (94)$$

where $c = B' - m' = B' - m + F_i P_w$ (Equations 27 and 64).

(2) Start the decompression at a rate of 20 fsw min^{-1} from P_b to the value of P_1 calculated in Equation 93 or to the next deepest 10 fsw depth interval if $(P_1 - 33)$ does not prove to be a whole number of tens of feet. Call the pressure corresponding to this depth P'_1.

(3) At one-minute intervals from the start of decompression, calculate the total gas tension $(p + m)$ in venous blood and at each of twelve points in the bulk 'cellular' phase equally spaced between the interface $(r = a)$ and the back-wall $(r = b)$, using the kinetic equations (81–85) for the model (fig. 84). The input is the arterial inert gas tension (p_a in Equation 85) which will vary with the continuously decreasing absolute pressure according to Equation 2.

(4) At each of these one-minute intervals, determine which of these points has the highest inert gas tension (p_{max}).

(5) Apply the critical gas inception equation (86) to this peak value, i.e. putting $p = p_{max}$ to determine the new absolute pressure P to which it is safe to decompress (fig. 84).

(6) If this new value of P is greater than $(P'_1 - 10)$ by the time the diver reaches P'_1, then he must stop at P'_1 and steps (3) to (5) must be repeated at one-minute intervals until the peak (p_{max}) has subsided to the extent that

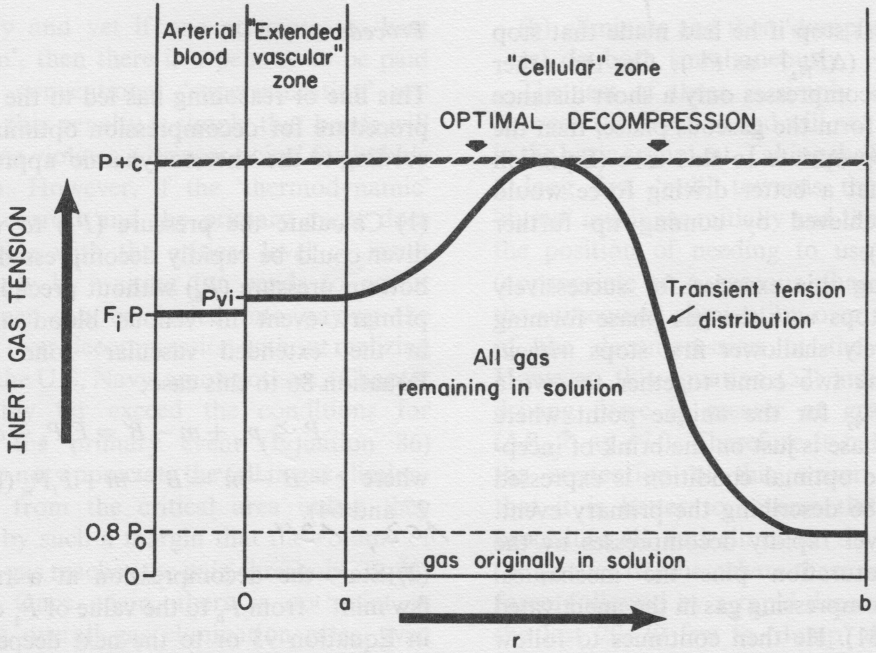

Fig. 84 Depicting the condition for optimal decompression according to the 'thermodynamic' method. At any instant the absolute pressure is selected which causes the peak gas tension to coincide with the point of phase equilibrium, i.e. just keeping all gas in true physical solution. It is convenient to work in terms of inert gas when other gases and mechanical factors can be 'lumped' into one minor constant (c), where $c = B - m' = \delta_g + (2\gamma/r_n) - P_{v_{O_2}} - P_{v_{CO_2}} - (1 - F_{IN_2})P_w$. Redrawn from Hills (1966)

this value enables Equation 94 to be satisfied for $P = P'_1 - 10$. The diver is then moved 10 fsw closer to the surface—or a greater number of 10-foot intervals, provided the peak has subsided sufficiently for the new absolute pressure (P) not to violate Equation 94.

(7) The change in arterial inert gas tension (p_a) resulting from this change in absolute pressure is fed into the 'kinetic' component of the computation via Equation 85.

(8) If P'_1 is greater than the ($p_{max} - 10$) value calculated from the peak by the time the diver reaches P'_1, then he need not stop at P'_1 but can continue decompressing at the same rate until recalculations of the peak, in the light of his continuing change in arterial gas tension, show that he would violate Equation 94.

(9) When his depth becomes 40 fsw or less, then the same distribution data computed on the whole dive history to estimate the peak is also used to calculate the imminence of decompression sickness as though that diver had been decompressed from that depth to the surface, i.e. Equation 87 is used for $P = 33$ fsw.

(10) Continue the gradual decompression by repeating steps (3) to (7) using Equation 94 as the criterion until an absolute pressure (P_s) is reached at which (9) shows that Equation 87 would not be violated if the diver decompressed from P_s to 33 fsw.

(11) When this occurs, decompress from this surfacing pressure (P_s) to the surface at 20 fsw min^{-1}.

Variations

Many variations on this theme are possible. One-minute intervals are used for updating

the computation because this is approximately the circulation time of blood in the body. The decompression rate of 20 fsw min^{-1} is selected for the two rapid phases of decompression ((2) and (11)) to avoid supersaturating arterial blood (see p. 65).

Since P_s usually lies in the region of 53–63 fsw (20–30 feet depth), it is convenient to put in stops at 35 feet and 25 feet to improve efficiency. This does not add any inconvenience because the very shallow stops at 10 and 20 feet found in conventional tables are now omitted.

Computer programs

The expressions describing gas transfer in the 'thermodynamic' model (Equation 82), together with the difficulty of eliminating such variables as q by simple substitution, must leave the initial impression of great complexity in any calculation involving them. Their usage would certainly be tedious if it were not for the ease with which they can be programmed for the digital computers so readily available today. Hence much of the old fear of the mathematics in invoking any transport system more complex than a simple 'Haldane tissue' is no longer justified.

However, it must still be remembered that no profile is better than the program fed into the computer and extreme care must be taken to ensure that the program is completely 'debugged'. In preparing this, the sequence of calculation steps ((1) to (11)) listed in the preceding section provides a convenient outline for the underlying logic diagram. There are basically two approaches to programming this model: the analytical approach and numerical methods involving finite difference techniques commonly used in solving many engineering problems concerned with heat transfer.

This technique has enabled the thermodynamic model to be simulated by a number of compartments in series, the first representing the 'extended vascular' zone and the remainder depicting segments of the bulk 'cellular' phase. Thus the first of the 'nodes', or boundaries between these 'tanks', is at arterial gas tension, while the last represents the 'back wall'. By the finite difference method, the rate of transfer between any two compartments is directly proportional to the tension difference between them. The proportionality constant of the first is chosen to reflect the blood perfusion rate while the rest are determined by the shape of the 'cellular' zone. Each compartment is also assigned a capacity proportional to its ability to store dissolved gas. This program is therefore somewhat flexible in allowing the designer to select his blood perfusion rate ($\dot{Q}$), fraction of the 'extended vascular' zone (β), the mode of curvature (μ) and relative dimensions (b/a) of the bulk 'cellular' phase.

Confidence in the technique was gained from the agreement between the profiles produced by this program and one devised by an analytical method using Legendre quadrature to describe the particular case of a purely diffusion-limited radial system (Hills *et al.*, 1976). Although the finite-difference approach is particularly versatile, great care must be exercised in selecting the coarseness of the elements to prevent the programme from 'going critical'. This also applies to time intervals. However, the agreement between the profiles generated by this technique and those produced by a thermal analogue (fig. 85) and another by the analytical approach certainly allayed the inherent suspicions which this writer, at least, had of computers and their programmers.

Profiles

The type of profile which the 'thermodynamic' procedure produces is shown in fig. 85, where it is compared with a U.S. Navy air table in which total decompression time has been similarly titrated on both to give an equal bends incidence on large goats (Hills, 1966). The two major differences are the 'drop out' from 25 feet and the much deeper initial stops, together with an overall shift of time towards the deeper end of the decompression. Hence the calculation procedure is actually producing the type of format anticipated from the thermodynamic model, viz.

(a) more time spent deeper to avoid gas

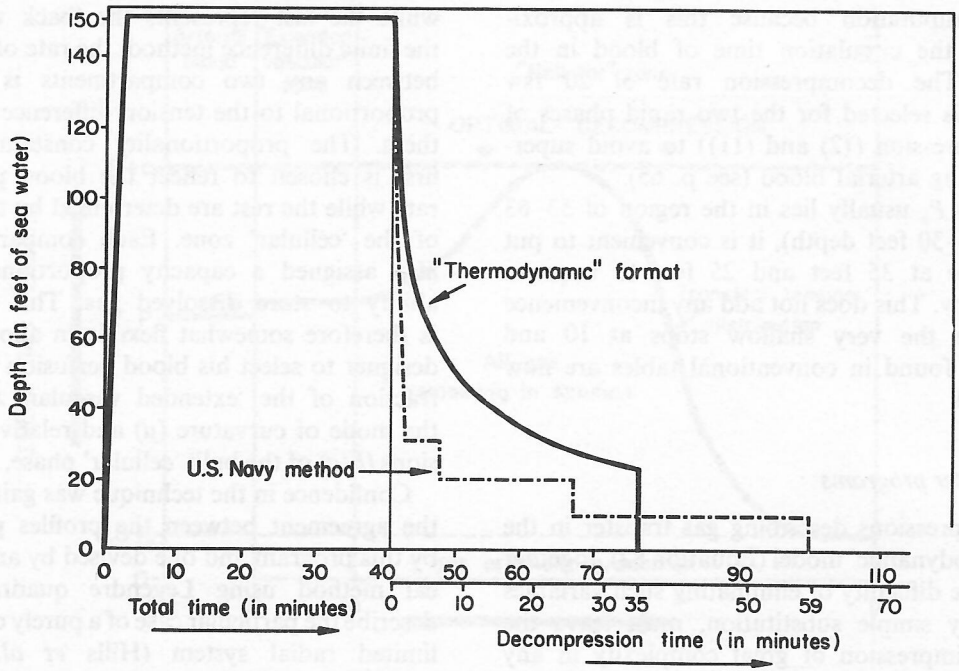

Fig. 85 A typical profile produced by the 'thermodynamic' approach and compared with a U.S. Navy profile for the same air exposure, both schedules being cut off at the total decompression time for equal bends incidence on the same goats. Redrawn from Hills (1966)

phase inception and hence giving a greater driving force to remove the excess gas, followed by

(b) a 'drop out' from 20–30 fsw to 'dump' the remainder of the gas as a sub-symptomatic gas phase.

It is over this last stage that the diver on a 'thermodynamic' profile overtakes those following more conventional calculation methods to result in a table more economical in overall decompression time for the same apparent safety. However, before discussing the success or otherwise found by using this method in practice, nothing has been said of the breathing mix to use.

Dual optimization

At each time in a decompression, there are two parameters for which the designer is free to chose values, i.e. there are two degrees of freedom. These are the depth and the composition of the breathing mix—the fraction of oxygen in particular. Consequently, to perform a simultaneous optimization of both depth and composition against time, two constraints are needed and these are provided by decompression sickness and oxygen toxicity. Thus the diver returning from the optimal decompression would be within known, yet safe, margins of both developing the bends and displaying the effects of oxygen poisoning.

If there is the facility to maintain a constant inspired P_{O_2} during the dive, then the highest value should be selected from the oxygen dose–time curve consistent with a reasonable margin of safety, e.g. using the U.S. Navy limits as shown in fig. 76. Normally this is not possible and it is seldom feasible to use more than four gas mixes during the entire exposure. However, it is quite practicable to alternate between any two mixes by filling the chamber with one and telling the diver when to breathe the other on BIBS. Thus in a dive to 500 fsw, the diver could breathe 7% oxygen mixed with predominantly helium on the

bottom, 14% oxygen from 300–350 fsw to 120–140 fsw, air to 40–60 fsw and then alternate between air and pure oxygen up to the drop-out depth.

In a particularly long dive, any sign of chronic oxygen toxicity should be used to switch to a subtoxic mixture. However, in shorter dives, the advantages to be gained from a significantly higher inspired P_{O_2} could result in convulsions arising too suddenly to prevent by evasive action. Hence the cumulative oxygen toxicity index (COTi on p. 226) has been programmed for continuous updating along with the total UPTDs on a separate computer program (Hills *et al.*, 1976).

This programme is used to compute the COTi for the thermodynamic program and at the same one-minute intervals. It is also used to compute another index: CO(T + 10)i. This is the index for the same history but ahead by 10 min, as though the diver were breathing the next more toxic mix for the whole of that period at that pressure. This continually updated CO(T + 10)i is used as a 'trigger' for the decompression program. It has no effect until its value falls below 1·0, which is the signal that it would be safe for the subject to switch to the mix with the next highest oxygen fraction for the next 10 min. In doing so, the new fraction of inert gas is fed into the decompression program. Upon return to the less toxic mix, the process is repeated so that the two programs, one for bends and the other acute oxygen toxicity, are effectively integrated. The interval 'on' the more toxic mix can be varied, but an arbitrary value of 10 min has been selected as a convenient time for the diver to be on BIBS without the annoyance of switching too often.

However plausible these optimizations may sound academically, are they doing what is intended?

Animal trials

Many trials on goats and kangaroo rats of the

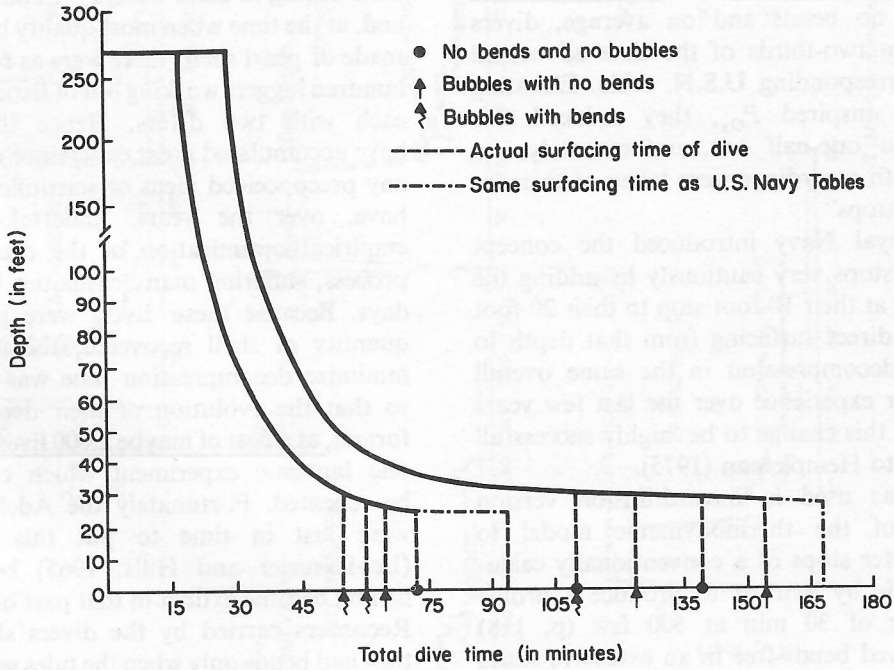

Fig. 86 Results of using the Doppler meter to monitor goats during decompressions following two thermodynamic profiles. Bubbles were never detected before surfacing while, on the U.S. Navy schedules for the same air exposures, asymptomatic bubbles were detected as early as the 40-foot stop. Data from Vann *et al.* (1973)

form shown in fig. 86 have shown that the 'thermodynamic' approach offers shorter decompression times; but is the body really conforming to the model? It is particularly difficult to detect bubbles *in vivo* for the reasons already outlined but if venous bubbles detected by the Doppler technique are at all meaningful, then it is interesting that none were detected in goats using a profile computed by Vann and Hills (Vann *et al.*, 1973) until after the drop-out (fig. 86). Moreover, those detected on the surface were asymptomatic, while U.S. Navy tables tested on the same animals *did* produce bubbles at depth.

Human experience

The greatest difference between thermodynamically and conventionally computed tables is seen in short, deep dives. Hence the first trials to test this method used tables computed on this model by Dr. Walter Starck and used by him and his crew for short deep air dives of up to 300 fsw off the Bahamas. In almost 200 dives they had no bends and on average, divers surfaced in two-thirds of the time advocated by the corresponding U.S.N. table. Elevating the mean inspired P_{O_2}, they reduced this fraction to one-half but, unfortunately, no actual depth recordings were taken of their in-the-water stops.

The Royal Navy introduced the concept of deeper stops very cautiously by adding the time spent at their 10-foot stop to their 20-foot stop with direct surfacing from that depth to complete decompression in the same overall time. Their experience over the last few years has shown this change to be 'highly successful' according to Hempleman (1975).

Vann has used a linear-diffusion version ($\mu = 1{\cdot}0$) of the thermodynamic model to improve later stops of a conventionally calculated profile by Bennett to produce a profile for a dive of 30 min at 500 fsw (p. 118) which proved bends-free in an extensive series of human chamber trials at Duke University (Bennett and Vann, 1975). However, by far the most extensive series *in the ocean* must be those of Krasberg (comment at 6th Symposium, Underwater Physiology) who, in over 800 dives for up to one hour and down to 600 feet, recorded only four bends (three Type I) of which several could be attributed to diver error. Moreover, using a linear-diffusion version of the model, his outstanding record of success has now been extended to exposures of up to 800 feet (Krasberg, 1976). Unfortunately, his final tables remain proprietary information.

Pearl divers

Returning to lesser depths and air diving, which is still the major part of the industry, by far the greatest experience of human exposure over the years must have been accumulated by the pearling fleets operating in the deep tidal waters off the northern coast of Australia. They employ many Okinawans who regularly dive to depths of up to 300 feet on air for as long as one hour, usually making two such dives per day, working six days per week and ten months per year. They have been fishing in these waters for about a century and, at the time when most quality buttons were made of pearl shell, there were as many as nine hundred luggers working out of Broome alone—each with two divers. Hence these people have accumulated great experience and without any preconceived ideas or scientific knowledge have, over the years, undertaken a truly empirical optimization of the decompression process, suffering many casualties in the early days. Because these divers were paid by the quantity of shell recovered, the incentive to minimize decompression time was very great, so that the evolution of their decompression format, at a cost of maybe 2,000 lives, represents one fantastic experiment which could never be repeated. Fortunately the Adelaide group were just in time to put this on record (LeMessurier and Hills, 1965) before pearl fishing became extinct in that part of the world. Recorders carried by the divers showed that they had bends only when the tides were running strongly and their simple use of the length of lifeline as their sole depth indication was badly in error. This brought the diver appreciably closer to the surface than intended

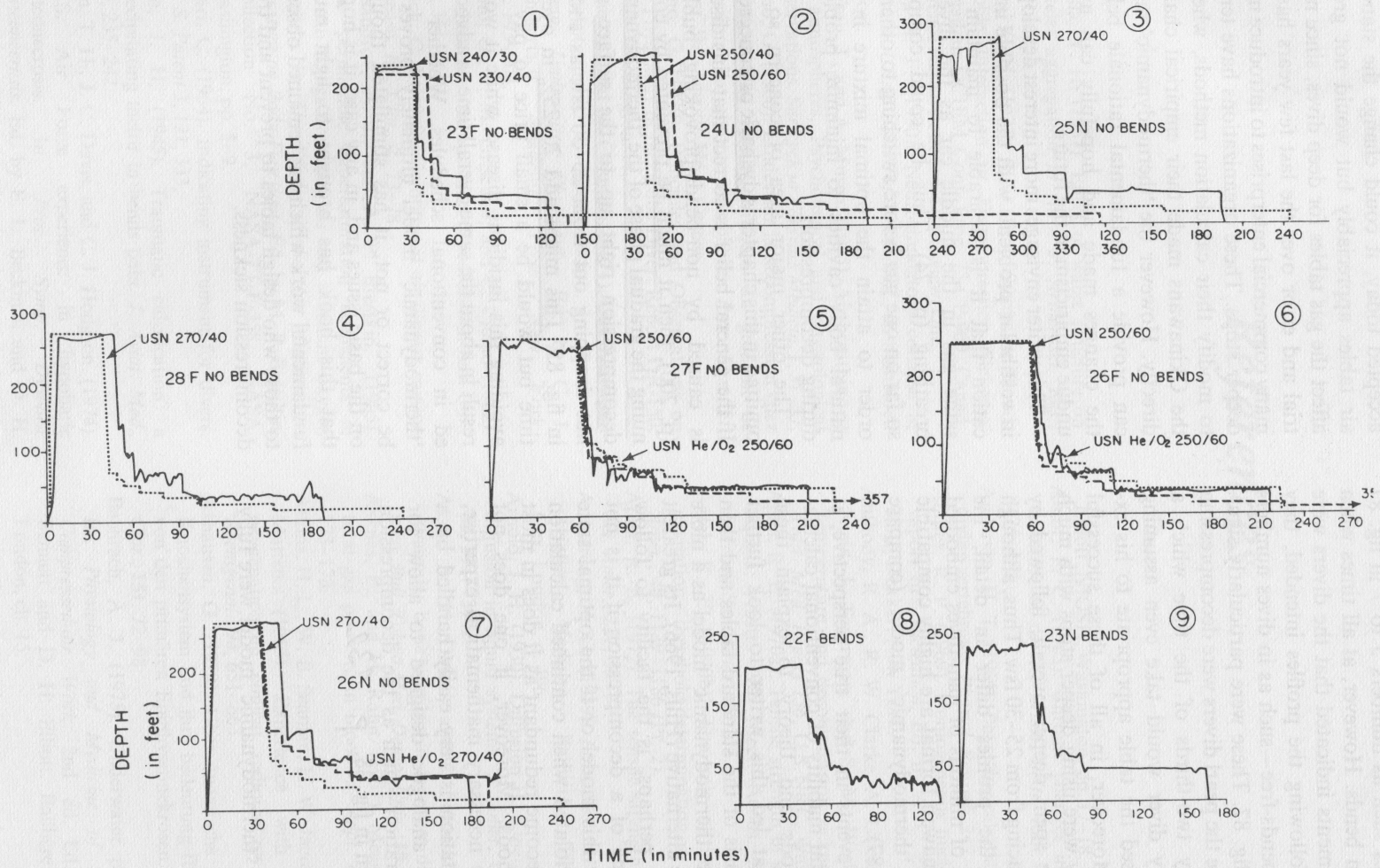

Fig. 87 Actual recordings of decompressions performed by Okinawan pearl divers operating in Australian coastal waters. They have devised these methods purely by trial and error over almost a century with the economic incentive of minimizing total ascent time. Data from LeMessurier and Hills (1965)

when dives such as numbers 5 to 7 in fig. 87 resulted in bends. However, at all times when the instruments indicated that the divers were actually following the profiles intended, they surfaced bends-free—such as in dives numbers 1 to 4 in fig. 87. These were particularly significant since the pearl divers were decompressing in roughly two-thirds of the time which a U.S. Navy diver would take even assuming that he used the table appropriate to his exposure. Moreover, in all of these successful dives, they were using deeper stops with much more time spent deeper overall, followed by direct surfacing from 25–30 fsw. Thus, although many of the profiles differ in detail, the experience of millions of man-dives embodied in the Okinawan format are highly compatible with the 'thermodynamic' model (compare figs. 85 to 87).

To put events in their true perspective, it was the total inability of conventional calculation methods and theory to explain these records that led this writer to look further into the basis of the standard tables and then to offer the 'thermodynamic' model as a more compatible alternative (Hills, 1966). Its greatest advantage, perhaps, is the facility to follow the outcome of a decompression if it is not optimal by this model or if the optimal state *has* been violated when continued calculation does not become redundant as it does in most other methods. Moreover, if one does not possess the necessary mathematical expertise, the computation is very easily handled by a mechanical analogue designed to allow for phase separation—such as the decompression meter shown in fig. 38. p. 132

The future

Even if the 'thermodynamic' model were fully accepted today, it could change the standard air tables appreciably but would not greatly affect the gas tables for deep dives, since much trial and error over the last few years has led many commercial enterprises to introduce much deeper stops. These organizations have tended to modify their calculation methods, whereas the Okinawans made their empirical changes directly. However the 'thermodynamic' model can provide a fundamental rationale behind the changes made and hopefully can avoid undue empiricism in the future.

This writer envisages more interest developing in vestibular problems with the possible implication that it is desirable to maintain the same gas in the middle ear as the diver is breathing (p. 74). Thus he could compress so far on one gas before switching to others in order to attain the optimal mixture in the natural body cavities to minimize problems during decompression.

The other major area of concern so far omitted in this chapter is dysbaric osteonecrosis. If the general belief is correct that this disease is caused by non-bends-provoking bubbles (p. 200), then it might be prevented by continuing the gradual phase of the 'thermodynamic' decompression right up to the surface and not dropping out from 25–30 fsw as shown in fig. 85. This might add 20–25% in overall time but would be a small price to pay for avoiding this insidious disease, while it would result in about the same overall time as advocated in conventional schedules. Whether the 'thermodynamic' model ultimately proves to be correct or not, it has stimulated thought on the basic issues and, in any case, it is hoped that this book has brought to light much fundamental work which has remained obscure to those who design tables to prevent and treat decompression sickness.

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