Chapter 6

Supersaturation versus Phase Equilibration

With so many approaches to prevention, many of them based on conflicting criteria, how is one to know which to use in the field or which features to select from each in formulating a method better than any advocated so far? Discussion of the aetiology of decompression sickness in Chapter 3 and the physics of gaseous cavitation in Chapter 4 leaves the impression that there is no obvious mechanism for the whole process leading to symptoms and that no one hypothesis has yet received sufficient acclaim to be recognized as a universal method for programming preventive decompression. Although many designers of diving tables have their own personal approaches, in fact, none of them may ultimately prove correct. It is cost-prohibitive to try them all over a sufficient range of exposure to draw any worthwhile conclusion. It becomes necessary, therefore, to go as far as possible making fairly safe deductions from the data described so far and then to take a hard look at the outstanding issues in the light of these data and many lesser known experiments specifically designed to answer these questions.

Theoretical summary

To delineate these vital issues, the following line of reasoning may be followed as a summary of the only hard conclusions to be drawn from discussions to this point.

(1) While there is much dissension concerning the nature of the insult required to provoke symptoms, or the level needed for it to do so, there is a wide measure of agreement that the primary event is bubble inception. Even most of the humorally mediated insults which have been proposed can be attributed to *gas separation* as the initiating factor.

(2) Prevention of any separation of gas from solution should therefore prevent decompression sickness. This is the almost universal aim underlying the many models and calculation methods described in the last chapter for designing decompression procedures whether it is actually achieved or not.

(3) Bends prevention therefore is best effected by reducing pressure gradually, the time allocated to decompression being deployed most efficiently by keeping one critical tissue, or one of several such tissues in turn, just on the brink of bubble formation without actually precipitating the gaseous phase.

This elementary line of deduction therefore comes to an abrupt end at four vital issues for which every designer of decompression tables must select answers, *or effectively assume answers*, if he invokes calculation at all.

(a) What defines the critical point for gas to separate from solution in tissue during decompression? While all methods may ostensibly prevent bubble formation, which criterion actually does so; ranging from phase equilibrium on one hand to variously described critical degrees of supersaturation on the other.

(b) Is the inert gas content of any one of these critical tissues best estimated by assuming that blood:tissue gas exchange is controlled largely by diffusion, largely by blood perfusion, or jointly by both? This determines the nature of the time function describing the total tissue gas tension to which the critical condition determined in (1) is then applied.

(c) How many actual tissues are involved, since this determines the number of separate equations to be applied?

(d) Can we assume that a model which will prevent limb bends will prevent other forms of decompression sickness and over what range of conditions might this be true?

These vital issues remain after eliminating from the initial set of questions (p. 48) those with reasonably undisputed answers and 'pruning' the remainder to leave only those with a direct bearing on preventive decompression. For instance, it does not matter whether the critical insult is mediated physically or humorally in a method which depicts a critical limit to supersaturation as the 'trigger point' for bends. By the same token, if the phase equilibrium criterion for bubble inception is found to be more relevant, then the question whether nuclei are formed *de novo* by minimal decompression or are always present in tissue is of largely academic interest.

The Vital Issue

In the controversy between the various models and calculation methods, many lose sight of the significant measure of agreement that exists to the extent that most designers are ostensibly trying to avoid bubble formation—at least until the diver reaches the point for surfacing. However, if some put the point of bubble inception as the position of thermodynamic equilibrium then, according to this most conservative criterion, other methods postulating higher degrees of permissible supersaturation would be initiating bubbles. In the past, most proponents of the 'Haldane' rationale, particularly the senior U.S.N. developers of many of the empirical modifications described earlier, have argued that their methods have not formed bubbles because they have not produced bends. However, if bubbles can be sub-symptomatic, even in the critical tissue(s), then this argument is totally invalid.

Before entering into a critical review of the evidence for these 'silent' bubbles, it is first desirable to consider why it is so important to establish whether they are formed or not. Their presence may have a cumulative chronic effect in provoking aseptic osteonecrosis or some more subtle form of diving pathology but the immediate effect of depositing a sub-symptomatic gaseous phase concerns *the driving force* for eliminating inert gas from tissue during decompression. It is felt that few designers of tables fully appreciate this serious implication of 'silent' bubbles.

Driving force for inert gas elimination

Consider some arbitrary point in time during a decompression from a simple air dive. If the nitrogen tension of a particular tissue is estimated as P_{N_2} and there has been no separation of gas from solution, then the driving force (ΔP_{N_2}) for eliminating this nitrogen via blood will be determined by the difference ($P_{N_2} - P_{AN_2}$) or gradient between tissue and arterial blood, arterial P_{N_2} being almost identical to alveolar P_{N_2}. Relating P_{AN_2} to the absolute pressure (P) by Equation 2, this gives the driving force for

$$N_2 \text{ in solution}: \Delta P_{N_2} = P_{N_2} - (P - P_w) F_{IN_2} \quad (51)$$

Thus, for nitrogen *in solution*, the rate of inert gas elimination is greatest for the *lowest* absolute pressure and hence the maximum permissible decompression ($\Delta P_{N_2}\uparrow$ as $P\downarrow$ in Equation 51). In fact, if a simple proportionality between elimination rate and driving force is assumed, as in the 'Haldane' rationale, then this expression for ΔP_{N_2} can be directly

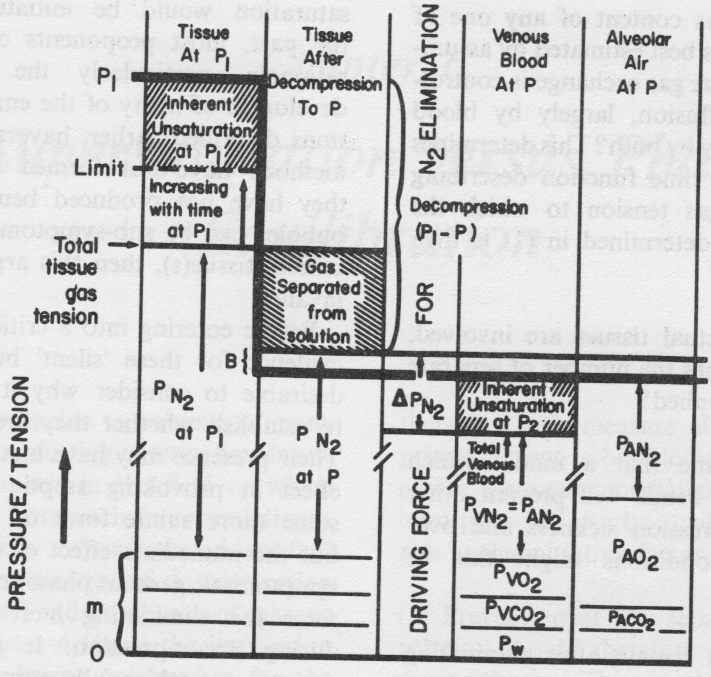

Fig. 40 Depicting the 'worst possible' case where all gas in excess of equilibrium upon decompression to P, yet in solution at P_1, is 'dumped' into the gaseous phase. Even then, there is still a driving force (ΔP_{N_2}) for its elimination which is provided by the new inherent unsaturation at P plus the mechanical factor B in accordance with Equation 52

substituted for $(P - p)$ in the rate equation (35).

However, if gas has separated from solution in tissue, then P_{N_2} no longer represents tissue nitrogen tension. In fact, if one switches from complete supersaturation, as expressed by Equation 51, to the other extreme which can be expressed quantitatively, *viz.* phase equilibrium, then P_{N_2} must be replaced by P'_{N_2} as described earlier (Equation 25). Making this substitution and eliminating m for m' for algebraic simplicity (Equation 27), the driving force $(P'_{N_2} - P_{AN_2})$ for nitrogen elimination now becomes

$$\text{with separation: } \Delta P_{N_2} = P(1 - F_{IN_2}) + B - m' \quad (52)$$

where B and m' are constants defined earlier in the text (pp. 102 and 103).

Since F_{IN_2} must be less than 1·0 (0·8 for air), $(1 - F_{IN_2})$ must be positive, so that any increase in pressure now *increases* the driving force ($\Delta P_{N_2}\uparrow$ as $P\uparrow$ in Equation 52). Thus there would be a *lower* driving force for greater decompression ($P\downarrow$; $\Delta P_{N_2}\downarrow$). Hence, if gas *has* separated from solution, further decompression can have the diametrically opposite effect to that intended when assuming a significant threshold to supersaturation (Equation 51).

Significance

In optimizing decompression, the whole object of calculation is to choose the pressure which will give the maximum rate of elimination of gas from tissue at that particular time, i.e. to maximize ΔP_{N_2} over the whole profile. Thus, by theories of critical supersaturation, such as the many variations of the 'Haldane' rationale, the diver is decompressed as far as possible on the first 'pull' towards the surface

($\Delta P_{N_2}\uparrow$ as $P\downarrow$ in Equation 51). Consequently it is U.S.N. practice for the diver completing his exposure on the bottom to 'get the hell out of it' in the belief that, by coming as close to the surface as hypothetical 'trigger points' will permit, he can then eliminate more inert gas from his critical tissue(s).

However, if these 'trigger points' did release sub-symptomatic gas from solution in these tissues, then that long first 'pull' towards the surface is the worst thing those divers could do, since there is now the lowest driving force for gas elimination at this lower pressure ($\Delta P_{N_2}\downarrow$ as $P\downarrow$ in Equation 52). (This is also illustrated in fig. 40). This leads to a total dichotomy in approaching decompression programming as seen from the methods already proposed.

It therefore becomes imperative to determine whether profiles based on critical supersaturation are really bubble-free in the critical tissue(s) and, if not, then just how little decompression *is* needed to initiate gas separation from solution?

The methods available for attacking this vital issue are of two types: those involving a direct search for bubbles or other physical evidence of the gas phase in the body; and indirect approaches which avoid the need to identify the critical tissue(s) anatomically.

Direct Detection of Sub-symptomatic Bubbles

When first confronted with the question of a limit to supersaturation *in vivo*, most people ask why a satisfactory answer has not been obtained simply by exposing animals to pressure and then searching for bubbles on decompression. The problem really revolves around knowing where to look and whether the bubbles seen are in any way related to the mechanism actually producing decompression sickness. However, before getting into the question of relevancy, it is safe to assume that if a tissue is found to contain bubbles then the pressure history leading to that point has reached the 'trigger point' or exceeded any other condition defining the onset of gas separation. Unfortunately, if a direct microscopic examination of a sacrificed animal fails to reveal any bubbles, then the converse need not hold. It could then be argued that separated gas might have been revealed if the search had been more extensive or if the experiment had been repeated more times to allow for the random nature of gaseous cavitation (Chapter 4).

However, before discussing the findings in whole animals, it is interesting to consider blood and primitive organisms.

Blood

While blood is being drawn from a subject, it frequently bubbles profusely; but this is not a fair test of its ability to maintain supersaturation because there are so many opportunities for foreign nuclei to enter the system. The proponents of the 'Haldane' rationale prefer to quote the work of Harvey (1951a, b) who was able to maintain a very high level of supersaturation in blood and in systems which had been 'denucleated' *in vitro* (see p. 84)—in fact, to a level well in excess of the metastable limit implied by any M value used in decompression formulation.

However, it would seem more relevant to look at blood *in vivo* but then there is the problem of deciding whether any bubbles observed were formed *de novo* or had extravascular origins. One factor predisposing towards bubble formation in flowing blood is the possibility of Reynold's cavitation arising from vortex formation known to occur in turbulent flow (Dean, 1944), the collapse of these cavities producing a sound (Hunsaker, 1935). Harvey (1951a) points out that this is unlikely to occur *in vivo* unless there is a restriction to flow, while recent haemodynamic studies indicate that it is even less likely that heart sounds can be attributed to this phenomenon (Patel *et al.*, 1974).

Primitive organisms

It is interesting to consider whether the body has any natural micro-cavities. The fixation process for preparing gross sections of excised tissue for microscopic examination renders

it very difficult to preserve any gaseous inclusions for histological examination, while it has been known to introduce bubbles. Hence it is almost impossible to say whether the more likely sites, such as the vacuoles, normally contain any gas. However, gas can be observed in the vacuoles of such primitive organisms as *arcella* which do not need prior fixation. Moreover, it was J.S. Haldane himself (Haldane and Priestley, 1935) who emphasized the ability of this and certain other protozoa to cavitate at will—even when unsaturated with respect to gases! These primitive organisms can secrete gas into their vacuoles when the local oxygen concentration falls in the surrounding water, thus increasing their buoyancy and so enabling them to rise into currents taking them to regions which are better oxygenated. This was a major point in Haldane's theory of active oxygen secretion by the lungs (see p. 28). This phenomenon, however, is reminiscent of the swim-bladder mechanism in fish (Scholander, 1958) and may not be relevant to mammalian tissue.

Earlier the threshold for bubble growth in shrimp was discussed (p. 140) but many of these creatures already display bubbles as they are caught live in the Gulf of Mexico; and shrimp do not have a swim bladder.

Bubbles in small animals

There have been a great many studies in which many workers in this field have sacrificed small animals directly before or after decompression and then searched for bubbles, their presence having been recorded in almost every major tissue type except skeletal muscle. They have also been seen in the veins draining this tissue but these vessels also receive blood from the adjacent connective tissues.

It is unfortunate from a mechanistic standpoint that in few of these studies was any attempt made to 'titrate' the decompression to a bubble point. However, this information can be deduced from one of the first and most comprehensive programmes which employed cats and has been published as a series of joint papers by Harvey *et al.* (1944a, b), McElroy *et al.* (1944a, b) and Whiteley *et al.* (1944). The incidence of bubbles relative to the extent of decompression which they found is summarized in Table 8 from which it can be seen that the occurrence of bubbles is a particularly random process, the probability tending to increase for the higher decompression ratios. No bubbles were observed when the (P_1/P_2) ratio was less than 2·5 which is within 10% of the critical value of 2·25 determined experimentally on men for the occurrence of bends (Boycott and Damant, 1908; Boycott *et al.*, 1908). This coincidence of decompression ratios for the occurrence of bubbles in cats and bends in men has been taken as support for the conventional concept of the metastable limit, or

Table 8 Bubbles appearing in cats within 5,000 seconds of rapid decompression from a pressure P_1 to a pressure P_2, data from Harvey *et al.* (1944a, b, c)

Time at pressure P_1	Initial absolute pressure P_1 (ATA)	Final absolute pressure P_2 (ATA)	Number of cats in trial	Number displaying bubbles	Resting or stimulated
∞	1	0·14	37	4	rest
∞	1	0·2	11	8 + 2?	stim
2–5 hr	2	1	5	0	stim
2–5 hr	2·5	1	6	0	stim
2–5 hr	3	1	12	3	stim
2 hr	3·5	1	18	9	rest
2 hr	3·5	1	14	12	stim
2 hr	4	1	6	6	rest
2 hr	4	1	3	3	stim
2 hr	4·75	1	3	2	rest
2 hr	4·75	1	4	4	stim

'trigger point' below which the gas phase should not be formed. However, it would not seem valid to equate absolute values in two species differing so widely in size. The extent of the error involved in extrapolating from mice to men can be appreciated from the analysis of Flynn and Lambertsen (1971). They have shown a simple relationship between the bends susceptibility of different species and their mass (see fig. 41), simultaneously demonstrating that the same basic mechanism for decompression sickness would appear to prevail in all mammals. A remarkable exception is the kangaroo rat (Hills and Butler, 1976) where tail-biting appears to correspond to limb bends in man much more closely than the death-or-recovery criterion used for other small animals.

In stimulated bullfrogs and rats, much lower ratios for bubble formation have been found than those deduced from the results quoted above. Blinks, Twitty and Whitaker (1951) summarized the work of Harris *et al.* (1945a, b)

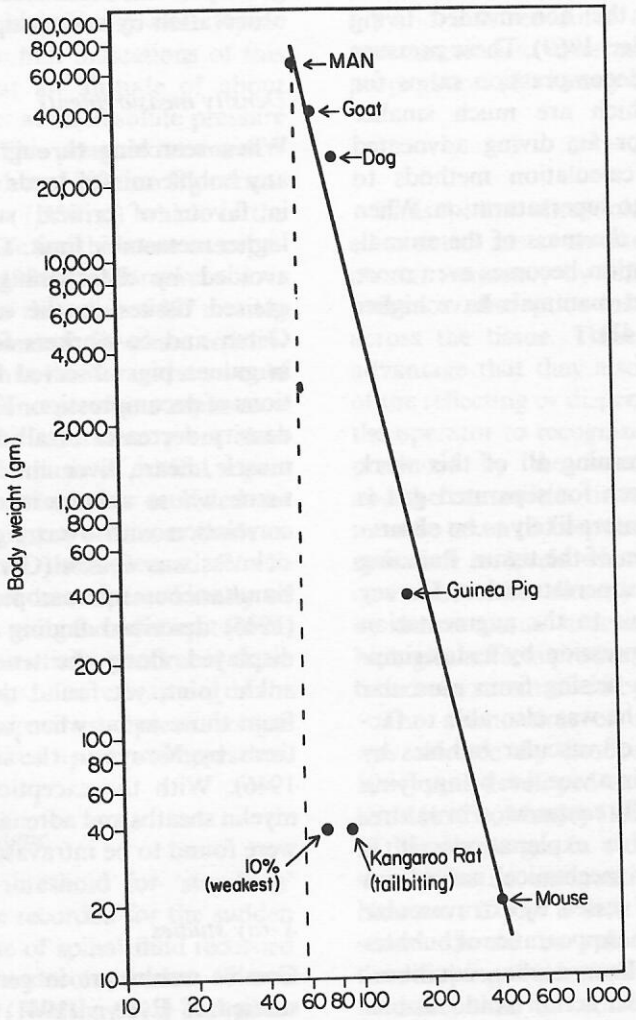

Fig. 41 Susceptibility of various mammals to decompression sickness. The minimum inspired nitrogen partial pressure which produces 50% cases after attaining steady state prior to decompression to the surface is plotted against body weight. Data from Flynn and Lambertsen (1971) with data for tail-biting in the kangaroo rat added from Hills and Butler (1976)

who have found bubbles in all 31 bullfrogs exercised on decompression to normal atmospheric pressure following one hour at pressures ranging from 5 to 60 psi (gauge). Inducing muscular activity in rats by 5 to 15 V (60 cycle/sec) AC stimuli, they record a threshold exposure of 3 psi (gauge) at which two out of five gave bubbles just large enough to be observed. Even lesser decompressions have recently been found necessary in shrimp whose translucent bodies enable bubble growth to be observed directly in the non-invaded living state (Evans and Walder, 1969). These pressure differentials indicate decompression ratios for bubble formation which are much smaller than any M value for air diving advocated in the conventional calculation methods to describe critical limits to supersaturation. When allowance is made for the mass of the animal, the lack of any correlation becomes even more apparent, since smaller mammals have higher bends thresholds (fig. 41).

Intravascular bubbles

One general fact concerning all of this work involving a direct search for separated gas is that bubbles are much more likely to be observed if there is stimulation of the tissue. Pursuing the concept of critical supersaturation, Harvey (1951a, b) attributes this to the augmentation of any external decompression by a superimposed mechanical strain arising from muscular contraction. However, he was also able to facilitate the appearance of vascular bubbles by crushing cats' hindlegs in a vice, i.e. by applying a force which is essentially *compressive* in nature.

In seeking alternative explanations, it is easy to argue that any mechanical action can facilitate the rupture of vessels by extravascular gas pockets and hence the appearance of bubbles in the monitored blood. From a 'phase equilibration' standpoint, muscular action would also be the ideal motion for coalescing into a visible form, or congregating into bubbles, gas already separated from solution in tissue (see p. 97).

Turning to larger animals, the direct observation of venous blood through a lucite calvarium has tended to indicate bubble appearance as a late rather than early sign of decompression sickness. Decompressing from 60 psi (gauge), Behnke and Shaw (1937) found that dogs were really sick, displaying rapid shallow breathing and halting circulation by the time that bubbles were seen moving through cutaneous arteries and veins; although they did see some bubbles prior to overt signs.

Before discussing the relevancy of intravascular bubbles which this raises, it is first desirable to look at techniques for detecting gas separation from solution other than direct observation by microscope.

Density measurements

When searching through a sacrificed animal, any bubble missed tends to bias the deductions in favour of critical supersaturation and a higher metastable limit. This objection is largely avoided by determining density changes of excised tissues in the method developed by Gersh and co-workers for preserving cavities in guinea pigs observed for clinical manifestations of decompression. They found appreciable density decreases in all organs except skeletal muscle, heart, liver and nerve fibres; but the tissue whose volume increase offered the best correlation with overt signs of decompression sickness was *tendon* (Gersh *et al.*, 1944). In a simultaneous microscopic examination Gersh (1945) described finding bubbles prominently displayed along the tendon sheath near the ankle joint, yet found them indistinguishable from those in fat when he attempted to locate them by X-rays in the intact animal (Gersh, 1946). With the exception of adipose tissue, myelin sheaths and adrenal cortex, such bubbles were found to be intravascular on dissection.

X-ray studies

Gersh's results are in general agreement with studies of Evelyn (1941) and Boothby *et al.* (1940) using soft X-rays to detect gas in Air Force cadets decompressed to negative gauge pressures. They showed the presence of bubbles or accumulations of gas in joint and tendon sheath spaces. Subsequently, Webb *et al.* (1944b) and Blankenhorn and Ferris (1944) have found gas as discrete round bubbles in periarticular

tissue and in joints, where there is no correlation with pain, and as 'fine longitudinal streaking' in the popliteal fossa where it appears in the X-rays as 'opaque ribbons' distributed along tendon or muscle bundles. For bends-provoking decompressions, Webb *et al.* found a very significant correlation between this 'streaking' and the occurrence of symptoms, successfully predicting 85–90% of cases on this basis. Thomas and Williams (1944) found that such changes could be observed for decompressions well below pain-provoking levels, while Ryder *et al.* (1945) found the first indications of this 'streaking' to occur at an altitude of about 10,500 ft (3,950 m), i.e. at an absolute pressure of about 483 mm Hg. This pressure is considerably higher than the value of 307 mm Hg corresponding to 23,000 ft (7,550 m) which is the minimum bends altitude for the more susceptible aviators (Gray, 1944b). Moreover, it is certainly much closer to the value of 497 mm Hg calculated as for the pressure for phase equilibration (Hills, 1966). Thus the X-ray data would certainly favour the concept of gas separation occurring closer to the saturation point than to any predicted on the basis of critical supersaturation. The calculations are included on p. 252. In view of the great advances made in X-ray technology since these Second World War studies, the writer has repeated many of them on small animals using the most up-to-date equipment and has essentially confirmed these early U.S.A.F. findings. There was no doubt that the ribbon-like opacities represented gas bacause they disappeared upon recompression.

Cerebrospinal fluid volume

The X-ray pressure threshold for 'streaking' agrees well with those recorded for the sudden increase in the volume of spinal fluid recorded manometrically on the decompression of men (Boothby *et al.*, 1940; Walsh, 1941) and goats (Armstrong, 1939). However, this could be a reflection of changes in systemic blood pressure resulting from the hypoxia accompanying decompression; although it is rather doubtful whether a vascular response would give transitions as sharply defined as those observed in cerebrospinal fluid volume. Less invasive and less potentially harmful methods were then sought to monitor man for gas continuously during decompression, ultrasonics presenting the most encouraging prospect according to feasibility studies by the Canadian Military (Huntec, 1964) and the U.S. Navy (Sutphen, 1968).

Ultrasonic methods

A gas bubble is able to scatter sound waves in tissue by virtue of its difference in both density (ρ) and bulk modulus (E), the two physical parameters also determining the velocity of propagation (U) in any medium, viz.

$$U^2 = E/\rho \tag{53}$$

Thus the first ultrasonic methods were designed to detect gas as an attenuation of the fundamental frequency by diffraction of a reflected pulse or absorption of a signal transmitted across the tissue. These techniques have the advantage that they also indicate the location of the reflecting or dispersing medium, enabling the operator to recognize any contact artefacts produced by the interfaces between tissue and the transmitter or receiver. However, care must be taken to keep the intensity of ultrasonic radiation to levels where it will not facilitate cavitation either directly or by chance focusing by denser tissue structures (Kent, 1965). The biological effects produced by ultrasound *per se* have been summarized by Hueter (1951).

The attenuation of the transmitted wave was selected by Stubbs and Kidd (Huntec, 1964) and developed as a monitor in man by Kidd (1969), Manley (1969) and Powell (1972a). If there is no gas in a uniform medium, there should be no attenuation and no reflection. However, if just a little gas is deposited, then sound should be reflected and theoretically the resulting 'back peak' should then be easier to detect than the corresponding reduction in amplitude of the transmitted wave. Thus the group in England adopted this approach (Walder, 1967; Walder *et al.*, 1968).

Both transmission and reflection techniques, however, suffer from the heterogeneous nature of tissue both in density and modulus, so that there are not only a multiplicity of surfaces

to scatter sound in non-cavitated tissue but a variation in its velocity within the medium (Equation 53). Hence both the transmitted and reflected waves have most complex profiles when displayed on an oscilloscope and what is worse, tend to change with time before decompression and even without exposure to pressure. At least, this is the impression gained by this writer and several others equally unskilled at electronics who have attempted to use it simply as a bubble-detection 'tool'.

Various modifications have since been introduced to try to circumvent this inherent instability such as the more effective use of 'filters' by selecting the second harmonic of the fundamental frequency (Welsby, 1967). Buckles and Knox (1969) have invoked the Rayleigh principle, that light and sound can interact, to produce some very impressive pictures of bubbles in the hamster cheek-pouch by acoustic-optical imaging; but the technical problems of setting up such a system are enormous.

The waning interest in the application of pulse echo techniques to bubble detection has been revived by Mackay and Rubissow in a number of papers recently reviewed by Evans (1975). In their latest publication (Rubissow and Mackay, 1973), they show a remarkable scan of bubbles in decompressed animals and have more recently used it as a direct monitor in man to programme his decompression. While this would seem most promising, it has yet to be seen what technical problems are encountered when the technique is used by the non-dedicated and whether the old instability problems of transmitted and reflected ultrasound have finally been laid asunder.

Although much effort has been expounded upon developing the technology in adopting this approach to ultrasonic monitoring, it is disappointing that so many potentially fruitful studies have resulted in little data relevant to the supersaturation versus phase equilibration issue. However, Buckles and Knox and Mackay and Rubissow have found evidence for some gas separating from solution for decompressions rather lower than predicted by standard M values used for formulating air diving tables.

This difficulty in pinpointing the onset of a decompression-induced change in the signal returned from tissue does not apply to the most recent and most popular ultrasonic monitoring technique—that invoking the Doppler principle.

'Doppler' meters

Sound is reflected from a surface at the same frequency as the incident beam, unless the surface is approaching the observer when it is increased (or decreased when it is receding). This shift ($\Delta f'$) in frequency (f') from the transmitted to the reflected wave is related to the velocity of the surface resolved in the direction of the beam (u) as

$$\Delta f' = f'(u/U) \qquad (54)$$

If f' is of the order of megahertz then, for reasonable physiological values of blood velocity, $\Delta f'$ comes within the audible range. Thus the electronics can be arranged to produce an audible signal at the frequency difference ($\Delta f'$) whenever a sound-reflecting interface passes through the field of focus of paired piezoelectric crystals 'coupled' to the body. Suitable reflecting boundaries include many of the components of blood itself, so that they provide a good indicator of instantaneous blood flow (Light, 1972) and have been developed as a commercial instrument used for many years as a foetal blood flow detector.

When a bubble passes through this field of focus, the audible signals produced have been variously described as 'chirps', 'snaps', 'plops' and 'whistles' (Spencer *et al.*, 1969; Evans and Walder, 1970). These must be distinguished from a background of normal sounds such as 'squeaks' and 'breezy noises' produced by blood and moving tissue boundaries heard when monitoring in the recommended precordial position (Spencer *et al.*, 1973). This is an ideal location for the non-invasive probe of the instrument (see fig. 8), since the ultrasound is then beamed along the axis of the pulmonary artery—the vessel in which *venous* blood draining tissues has its highest velocity before any entrained bubbles can be filtered out by the lungs. An *in vitro* evaluation showed that bubble detection is particularly dependent

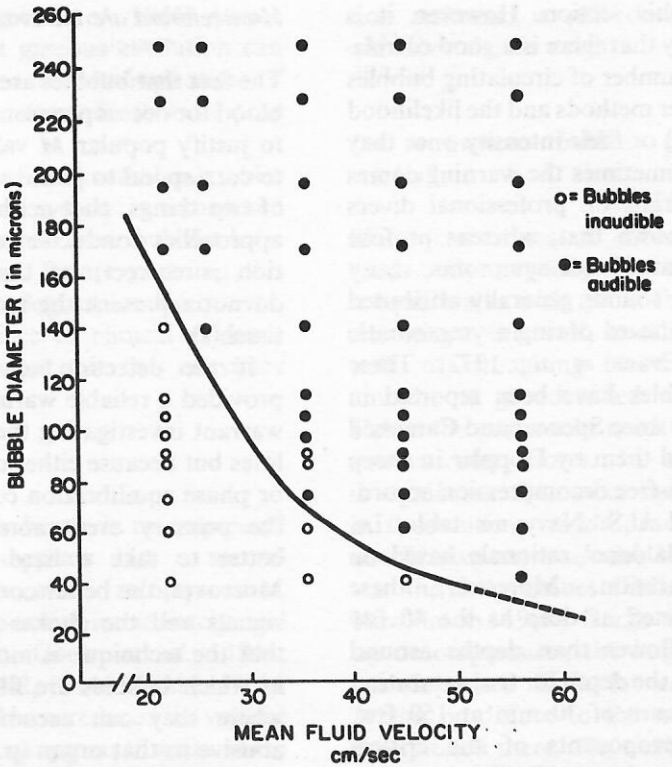

Fig. 42 Demonstrating the dependence of minimum detectable bubble diameter upon bubble velocity. o bubbles are inaudible and • bubbles are audible on a 'Sonicaid' Doppler meter. Reproduced from *Ultrasonics*, Vol. 13, No. 4, July 1975, p. 181 by permission of IPC Science and Technology Press Ltd., Guildford, Surrey

on velocity (see fig. 42) and that bubbles smaller than 40–50 μm diameter were unlikely to be distinguished unless they were so small that they would resonate at the frequency used (about 1μm diameter for 5 MHz). Bubble resonance has formed the basis of a detection method devised by Horton and Wells (1973). However, the 'black-out' zone for diameters above this resonant size could well extend up to 80 μm diameter for positive identification of single bubbles heard against a background of normal flow sounds (Hills and Grulke, 1975).

It is unlikely that the sounds heard in divers are caused by coalesced lipids, platelet aggregates or agglutinated red cells which can also be formed during decompression (p. 55 & 59) since, for the physical reasons outlined earlier, their boundaries with plasma should be much less reflective than bubbles. Doppler sounds also tend to regress with recompression (Evans and Walder, 1970). Further evidence that these sounds represent bubbles is provided by their simultaneous indication by other methods susceptible to gas such as an electromagnetic blood flow meter (Spencer and Campbell, 1968) and the pulse echo techniques described earlier (Rubissow and Mackay, 1973; Powell, 1972b). However, there is some indication from the blood-flow waveform, that aggregates of red cells and/or platelets can be detected (Nishi and Livingstone, 1973).

The question whether Doppler sounds are a good indication of impending clinical manifestations of decompression sickness becomes controversial. All manner of opinions can be found and the potential user really needs to consult such sources of reference as the proceedings of a Symposium on Ultrasonic Bubble Detection organized by Spencer and Johanson in Seattle in 1973 and most of the papers

already cited in this section. However, it is probably fair to say that there is a good correlation between the number of circulating bubbles detected by Doppler methods and the likelihood of bends occurring, or their intensity once they have arisen; but sometimes the warning comes after the event. Trials on professional divers and goats have shown that, whereas profuse bubbling is associated with symptoms, many abnormal Doppler sounds generally attributed to bubbles can be heard during asymptomatic decompressions (Evans *et al.*, 1972). These 'silent' blood bubbles have been reported in many other studies since Spencer and Campbell (1968) first detected them by Doppler in sheep undergoing a bends-free decompression according to a standard U.S. Navy air table, i.e. according to a 'Haldane' rationale based on critical supersaturation. Moreover, these bubbles were detected as deep as the 40 fsw stop but still shallower than depths around 100 fsw expected as the depth for true saturation following an exposure of 30 min at 150 fsw.

However, the proponents of the critical supersaturation theories can still argue that blood bubbles do not cause the bends and that these bubbles detected in the *venous* system by Doppler arise from an irrelevant tissue in which gas separates more easily than in any determining the imminence of clinical symptoms. By the same token, proponents of phase equilibrium can argue that gas phase separation *did* occur at the point of true saturation but in tissue beyond circulating blood and that Doppler bubbles are really a late manifestation of these changes. Unfortunately this technique which is so simple to use can only detect *moving* bubbles and therefore brings up the original question of whether intravascular bubbles are relevant to clinical symptoms. Before discussing this query in more detail, it would seem appropriate to summarize Doppler findings by stating: there is very little doubt that 'silent' bubbles do occur in the body as a whole; the circulating bubbles actually detected in the venous system are probably not responsible for decompression sickness but their propensity seems to offer some reflection of the state of the critical tissue(s)—even if late on occasions.

How relevant are intravascular bubbles?

The fact that bubbles are detected in circulating blood for decompressions which are insufficient to justify popular *M* values and yet too great to correspond to phase equilibrium implies one of two things: that neither of these theoretical approaches conducive to quantitative description is correct; or that circulating bubbles do not represent the true state of the critical tissue(s).

If the detection of Doppler signals had provided a reliable warning of bends, it would warrant investigating the first of these possibilities but because either critical supersaturation or phase equilibration could still be predicting the primary event correctly, it would seem better to take a hard look at the second. Moreover, the better correlation between these signals and the chokes is a further reminder that the technique is monitoring *venous* blood in which bubbles are filtered out by the lung where they can accumulate to reach levels abusive to that organ (p. 68).

Bubbles could easily form in extravascular sites or in blood trapped in capillaries by closure of the precapillary sphincter when they would appear as intravascular under microscopic examination. In some tissues only 5–10% of the capillary population may be patent at any moment. The enormous effort put into microscopic examination of sacrificed animals by the U.S.A.F. and U.S.N. during the Second World War tended to show more intravascular than extravascular bubbles but blood offers a much better medium in which any separated gas can coalesce to a size at which a bubble can be easily recognized under a light microscope (see p. 98). Intracellular bubbles are more difficult to detect. Even so, extravascular bubbles have been observed in synovial, amniotic and cerebrospinal fluids (Boycott *et al.*, 1908; Harvey, 1951a), liver and spinal cord (Boycott and Damant, 1908), bone marrow (Gersh, 1945) adrenal cortex and nerves (Gersh and Hawkinson, 1944) but particularly in fatty tissue (Gersh *et al.*, 1944) and intracellular fat inclusions (Gersh *et al.*, 1945). They also occur quite widely in the lymphatic system

(Blinks *et al.*, 1951; Lever *et al.*, 1966). Hence there is no doubt that gaseous cavitation can occur in extravascular sites, although some of the evidence cited above does refer to some quite severe decompressions.

Thus the relevancy of intravascular bubbles detected in the venous system is transposed to the question of whether these bubbles have an extravascular origin. Before pursuing this problem at the morphological level, however, there is one major piece of clinical evidence which is most pertinent to the relevancy issue.

Recompression

If a subject with limb bends is recompressed the pain usually disappears but a second decompression, soon after recompression, causes the pain to re-occur immediately and *in the original site* (Cook *et al.*, 1944; Blankenhorn *et al.*, 1942, 1944; Behnke, 1951). Recompression should have reduced the volume of the bubbles causing symptoms to a size where blood would carry them away from the site of pain if they were intravascular. Thus Ferris and Engel (1951) regard the re-occurrence of bends in the original sites as major evidence that the bubbles actually causing limb bends are extravascular. Their reasoning is further substantiated by more recent studies involving direct observation of the vascular bed. These confirm that short periods of recompression can clear intravascular bubbles (Waite *et al.*, 1967; Grulke, 1975; Grulke and Hills, 1976). Moreover, this writer has found that the 'ribbons of gas' observed by X-ray in the peripheral tissues of lightly anaesthetized guinea pigs re-appear virtually unchanged after a very short (two minutes) recompression to 250 fsw. Ferris and Engel (1951) go on to list other reasons for attributing limb bends to extravascular bubbles but this is their major point and one which this writer finds hard to fault—at least for the particular case of *limb bends.*

However, while this provides further evidence that bubbles detected in venous blood are probably not responsible for Type I decompression sickness, it still does not answer the question of their origin.

Do intravenous bubbles have extravascular origins?

Blood constitutes no more than 8% of the total body capacity for dissolved gas and it equilibrates with gas in the lungs at roughly one-minute intervals, so it is unlikely that the bulk of circulating blood can provide the amount of gas detected as venous bubbles. Hence the gas would need to be derived from extravascular stores. In this connection it is significant that most bubbles are observed in those micro-vessels draining the tissues in which most extravascular bubbles are formed, viz. the fatty tissues (Hill, 1912; Gersh and Hawkinson, 1944; Gersh *et al.*, 1944). Thus, on decompression, the large amount of inert gas deposited from fat could be deposited *in situ* or diffuse to the nearest blood to be precipitated there. The next time that capillary opened, it could inject the bubble into the circulation but careful observation of the micro-circulation in the hamster cheek pouch has not revealed intracapillary bubbles on decompression (Buckles, 1968).

This raises the question of whether extravascular gas can enter blood *as bubbles.* This would require rupture of the endothelial wall of capillaries and possibly the cellular membrane if the bubble originated within the extravascular cell. Haemorrhage caused by extravascular gas was postulated as early as 1879 by Leyden and its occurrence, induced by means of vascular congestion, has been actually observed by Chase (1934) and Tureen and Devine (1936). However, Gersh and Catchpole (1951) point out that intravascular gas could also be responsible for rupturing blood vessels. The most convincing evidence that circulating bubbles can have extravascular origins is provided by recent studies using the electron microscope (Bennet, 1976). These show clearly the point of rupture of the capillary wall by the bubble as it entered blood and shows the adjacent cavity which it vacated in extra-

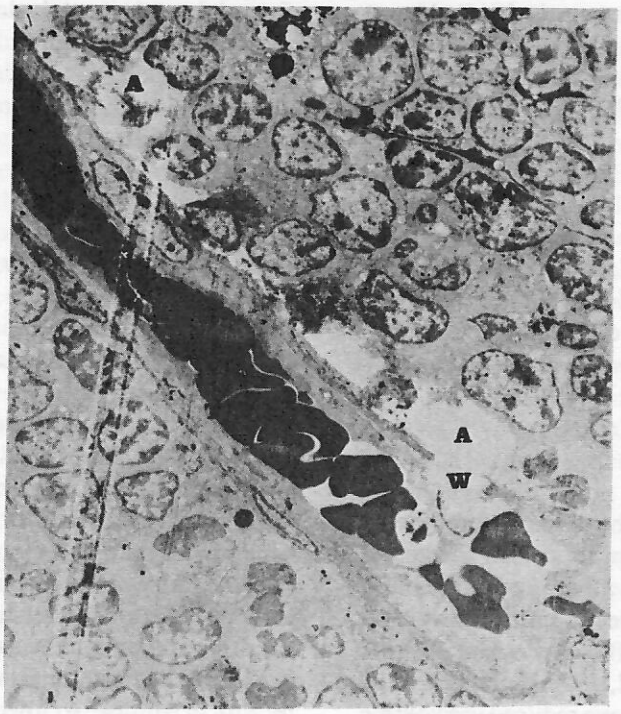

Fig. 43 An electron-micrograph of the skin of a mouse after decompression. Extravascular gas (A) has ruptured the capillary wall at W and entered the vessel, while thrombosed erythrocytes can be seen at T. (Enlargement is 2,900x). Reproduced by kind permission of Dr. R.A. Bennet

vasular tissue in doing so. An electron photomicrograph is shown in fig. 43.

Bubbles breaking into capillary blood might be expected to carry along portions of the vessel wall and indeed segments of endothelial cells have been found in circulating blood (Philp *et al.*, 1972). It is conceivable that haemorrhage is initiated by the initial rupture of the cell membrane by bubbles (Hill, 1912; Gersh and Hawkinson, 1944) which might also deposit cell contents into the circulation but examination of several tissues has failed to reveal the change in histamine and serotonin levels which this should cause (Kindwall, 1974). However, bubble-induced haemorrhage in adipose tissue would be expected to dump fat into blood (Haymaker, 1957; Rait, 1959) and indeed the appearance of circulating lipid emboli does appear to coincide with that of intravascular gas emboli (Cockett *et al.*, 1972a, b).

This evidence, together with the observations described earlier that most bubbles are observed in veins draining fatty tissues containing most extravascular bubbles, would strongly suggest that these tissues would be the first to deposit extravascular gas into the blood. Just on a simple mechanical basis, the roughly five-fold greater volume of nitrogen deposited from fat compared with water by the same decompression should cause a fatty tissue to rupture before an aqueous tissue. Thus the venous bubbles first detected in the circulation are most likely to arise from within lipid tissues. Moreover, these tissues have very few nerve endings, if any, so there is no difficulty in explaining why the extravascular gas deposited in them need not give rise to Type I decompression sickness. The question of what type of tissue *is* likely to give rise to clinical symptoms is discussed in more detail elsewhere (p. 59). Moreover, it is even harder to conceive the gas causing any insult worse than bursting

an extravascular cell so, if this fails to precipitate symptoms in lipid tissues, it is doubtful whether anything can—at least, by physical mediation.

Thus, if an aqueous tissue is responsible for marginal bends, then gas deposited from lipid tissues is irrelevant. It is therefore necessary to look beyond Doppler ultrasonic methods to try to pinpoint the onset of gaseous cavitation during decompression.

Electrical conductance

Gas has a very much higher electrical resistivity than tissue, which is a fairly good conductor by physico-chemical standards. Any separation of gas from solution could therefore cause extensive disruption of electrical pathways, particularly if the gas is deposited in a finely dispersed state as indicated by X-rays (p. 142). Furthermore, this would occur whether the gas was formed in extravascular or intravascular sites and, since it should cause an appreciable reduction in conductance for minimal growth, it should therefore provide an early indication of gas phase inception.

This line of reasoning led to direct measurements of electrical resistance by placing platinum electrodes across tissues of animals killed just before decompression and maintained at body temperature to keep gas solubilities constant (Hills, 1971d). The results showed clear transition points in electrical resistance upon starting from normal atmospheric pressure and decompressing rats' tails to negative ambient pressures at a uniform rate. This tissue was considered most relevant to the bends mechanism in view of the preponderance of nerve endings and the implications of this tissue in limb bends (see p. 59).

Transition points, i.e. sudden 'jumps' in the electrical resistance occurred for decompressions of 96–145 mm Hg starting from steady-state at atmospheric pressure breathing 20% oxygen mixed with 80% of various inert gases of widely differing solubility (helium, nitrogen or nitrous oxide)—see fig. 44. There is little doubt that the device was monitoring the gas phase since resistance changes were reversed with recompression; but it could be argued that the early onset of gaseous cavitation could have occurred at the unnatural interface

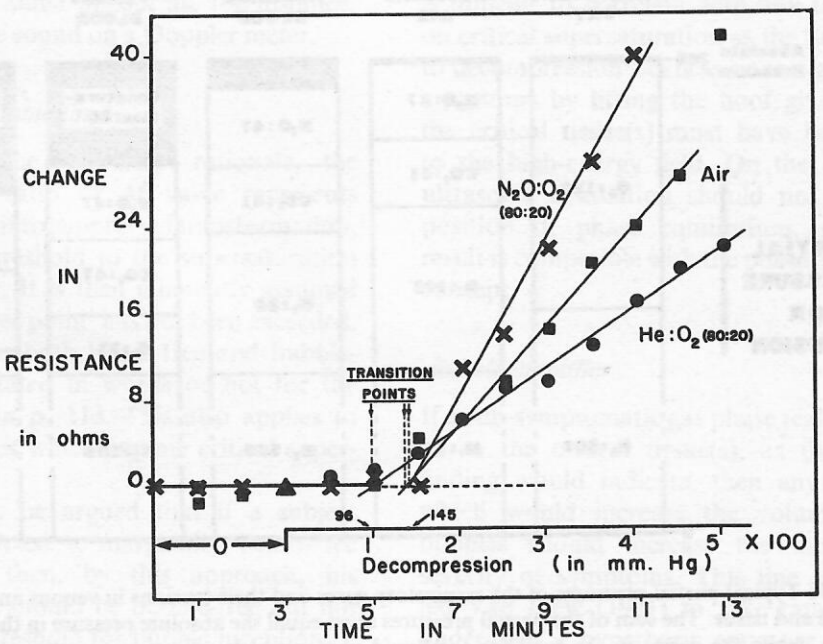

Fig. 44 The change in electrical resistance across the tails of rats sacrificed immediately after decompression from normal pressure where they had been breathing air, 80:20 $He:O_2$ or 80:20 $N_2O:O_2$ for six hours. Data from Hills (1971d)

created by placing platinum against the tissue. However, the early findings were essentially confirmed using a device without electrodes (Searle and Hills, 1976; Searle, 1975) which invoked the principle of inductive conductivity to avoid any possible artefacts attributable to the tissue: instrument interface. The simultaneous use of electrode and inductive methods permits simultaneous conduction measurements in both transverse and longitudinal directions in decompressed tissue. Results indicate the tendency for gas to separate along fascial planes, as indicated by X-rays. This tendency not to disrupt electrical pathways in the axial direction might explain why more peripheral nervous disorders are not found in simple bends cases.

The transition points in the resistance plots (fig. 44) are regarded as highly significant since they indicate that gas has already started to separate from solution by the time there is only 96 mm Hg of decompression from normal atmospheric pressure, i.e. bubble inception has already occurred by the time the absolute pressure reaches about 664 mm Hg. This is just about the total tension (665) of nitrogen (569) plus carbon dioxide (49) plus water vapour (47) after allowing for complete metabolic consumption of any oxygen after death (see also fig. 45). The fact that this point of true saturation of tissue lies so close to the point of bubble inception has convinced this writer, at least, that some tissue regions can withstand negligible degrees of supersaturation.

Perturbations in conduction induced by gases could also explain some of the changes in the ECG of divers observed for more extensive decompression (Wilton-Davies, 1970).

Summary

To summarize the direct search for subsymptomatic gas, there is now very little doubt that 'silent' bubbles can occur within the body as a whole. Moreover, gas would appear to start separating from solution in some tissue regions, however few, for negligible degrees of supersaturation and certainly for much less

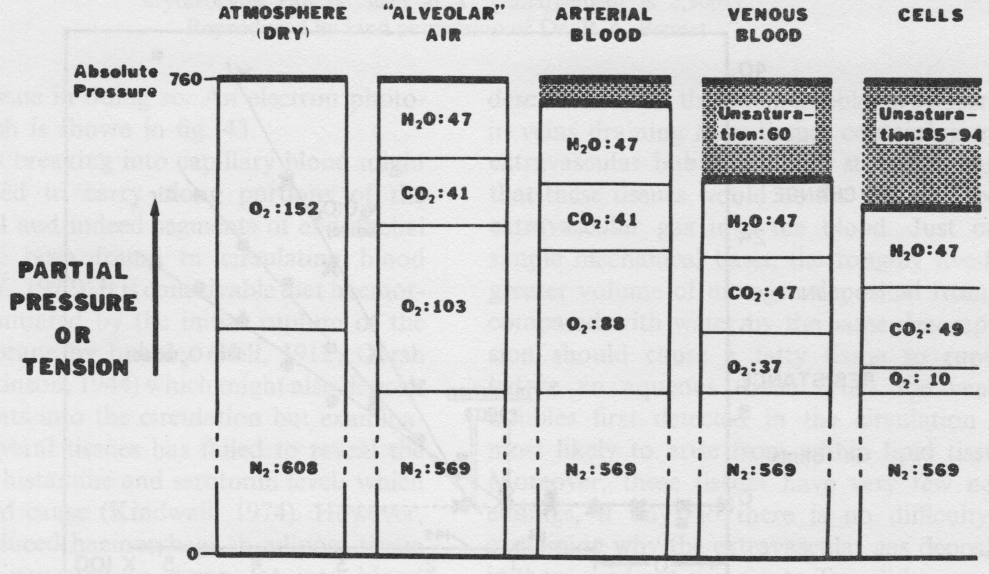

Fig. 45 Typical partial pressures of the respiratory gases and their tensions in venous and arterial blood and tissue. The sum of the partial pressures must equal the absolute pressure in the gaseous phase, while the deficit in the total for the liquid phases which occurs even under steady-state conditions represents the inherent unsaturation depicted by the shaded areas. From Hills (1974a) reproduced by permission of Cambridge University Press

decompression than implied by any M value for air diving. Even so, since intravascular bubbles do not appear to be responsible for marginal symptoms, it could still be argued that the direct evidence for negligible supersaturation refers to an irrelevant tissue. The bends-provoking tissue is unlikely to differ too greatly from others in its physical properties. However, until it can be identified positively, the site(s) of the relevant bubbles remain unknown and therefore the question remains whether 'silent' bubbles can occur *in the critical tissue(s)*.

Indirect Methods

The foregoing objections to the direct methods can be largely avoided by using indirect methods which use the occurrence or non-occurrence of symptoms of decompression sickness as the only indicator. This circumvents the question of anatomical identity of the critical tissue(s) very neatly. Unfortunately, the results often need theoretical deduction to be applied in answering the vital issue concerning supersaturation versus phase equilibration and this seldom has the same impact as, for instance, hearing a bubble sound on a Doppler meter.

Tests of a metastable limit

According to the 'Haldane' rationale, the decompression ratio or M value represents a metastable limit to suppressed transformation, i.e. a critical threshold to the supersaturation of tissue by gas. It is then inherently assumed that if this 'trigger point' has not been exceeded, then the diver is both bends-free and bubble-free, whether stated in words or not for the reasons given on p. 113. This also applies to other approaches which assume critical supersaturation.

Hence it can be argued that if a subject has just completed a marginally bends-free decompression then, by this approach, his critical tissue(s) should be bubble-free but not far below the threshold for bubble inception.

In Chapter 4 it was seen how, thermodynamically, a metastable limit to supersaturation is really an energy threshold. Consequently, if a large amount of energy is supplied to a marginally subcritical solution, then the energy threshold should be exceeded, gas should be deposited from solution and the bends should become manifest sometime later. At least, this is the argument by the 'Haldane' rationale and any other supersaturation hypothesis.

This line of reasoning led Hills (1970a) to determine the minimum bends depth of goats by exposing them to successively higher pressures of air for eight hours each time and returning them to normal atmospheric pressure within two minutes. They were then exposed for the same time to a pressure 10 fsw *less* than their minimum bends depth but, on decompression, the lower half of each leg was placed in a high-intensity ultrasonic cleaning bath to supply their critical tissues with large amounts of energy. This irradiation did not precipitate bends as one would have expected if the critical tissues were supersaturated. Moreover, they did not occur even if the intensity was increased almost to the point where ultrasound produced pain without any previous hyperbaric exposure. This finding is difficult to correlate with any theory based on critical supersaturation as the 'trigger point' to decompression sickness since a goat displays symptoms by lifting the hoof giving pain, so the critical tissue(s) must have been exposed to the high-energy field. On the other hand, ultrasonic irradiation should not change the position of phase equilibrium, so that the result is compatible with the phase equilibration concept.

'Bubble amplifier'

If a sub-symptomatic gas phase really *has* formed in the critical tissue(s), as the foregoing finding would indicate, then any manoeuvre which would increase the volume of those bubbles should increase the incidence and severity of symptoms. This line of reasoning led Van Liew (1971) to take rats which had undergone a hyperbaric exposure on air and switch them to 80 : 20 nitrous oxide : oxygen on return to normal pressure, thus using nitrous

oxide as a 'bubble amplifier'. He points out that nitrous oxide can be expected to diffuse into a nitrogen bubble 11 times faster than nitrogen will diffuse out (Piiper *et al.*, 1962). His finding that the switch to nitrous oxide : oxygen on 'surfacing' increased the incidence of decompression sickness from 30% to 70% is then interpreted as favouring the concept of 'silent' bubbles in the sensitive tissue(s). However, the proponents of limited supersaturation might still argue, that the more soluble gas (nitrous oxide) could have a lower tension threshold (metastable limit) than nitrogen, so that substitution of nitrous oxide for nitrogen on a tension basis might still potentiate breakdown of the supersaturated state to produce bubbles and hence symptoms.

Exposure to various inert gases

In reaching his conclusion, Van Liew assumes that it is the volume of gas separating from solution which will give rise to symptoms. Experimental evidence favouring this as the bends criterion in small animals has been provided by the Oxford group (Lever *et al.*, 1971a) who have exposed mice to pressure while breathing mixtures including various inert gases: nitrogen, helium, argon, nitrous oxide, carbon tetrafluoride and sulphur hexafluoride. However, they also use death or recovery as the end-point, so this may not be relevant to marginal bends in man. Moreover, proponents of the critical supersaturation concept can always argue that the potential volume also determines the 'trigger point' for gas to separate from solution in the first place. This follows Haldane's own volume-based explanation of the decompression ratio and can be shown to be compatible with a metastable limit to true supersaturation (see p. 114).

Rather than switching breathing mixes, several groups have studied the incidence of bends in subjects inspiring two or more inert gases simultaneously. There is some evidence to indicate a slight reduction in the imminence of symptoms after decompression from steady-state on nitrogen:helium mixes compared with either inert gas alone at the same inspired P_{O_2} (Zal'tsman and Zinov'eva, 1965; Lambertsen, 1967; Bennett and Hayward, 1968). Thus some proponents of the 'Haldane' rationale argue that if a tissue is saturated with helium, then adding 10 fsw of supersaturation of nitrogen is less effective in inducing bubble formation than adding 10 fsw of the same inert gas (helium) even though the addition of nitrogen would lead to a larger bubble on the basis of phase equilibration (see Equation 31). In fact one study (Bennett *et al.*, 1975) even goes so far as to regard the replacement of one inert gas by another as comparable to the substitution of oxygen in the breathing mixture in avoiding decompression sickness (p. 37). Their evidence is based upon their finding that nitrogen elimination after decompression is about the same at the same pressure whether the subject breathes oxygen or a helium : oxygen mix. However, they apparently discount the helium taken up in the second case in assisting the nitrogen to activate nuclei upon decompression, implying a 'helium window' in the example cited above. This appears contrary to Van Liew's experience with nitrous oxide as a bubble amplifier (p. 151) and would mean that there is maximum in the hypothetical M value with variation in the ratio of the two inert gases present. This is totally incompatible with reasoning based upon the volume of separated gas (Equation 31) and therefore inconsistent with phase equilibration yet is still a little difficult to believe on thermodynamic grounds even if supersaturation prevails in determining the imminence of decompression sickness. Even so, this apparent partial lack of synergism of two inert gases in inducing bends is sometimes quoted in favour of critical supersaturation. However, in addition to theoretical doubts, there is considerable uncertainty about the experimental validity of the evidence in view of the comprehensive study by the Oxford group. They found that, using various pairs selected from six inert gases, the incidence of bends on any mixture lay intermediate between those recorded using either inert gas alone (Lever *et al.*, 1971a). Although their results refer to

death or recovery of small animals, they cast serious doubt upon an effect which would favour critical supersaturation, if substantiated experimentally.

This decompression analysis of subjects which have already attained a steady state should not be confused with transient situations where it is claimed that the decompression time of men can sometimes be halved using 50 : 50 nitrogen : helium (mixed with oxygen) compared with either inert gas alone (Workman, 1963b). This case is greatly complicated by the kinetic differences between nitrogen and helium—a subject discussed in more detail later in connection with the perfusion : diffusion controversy (Chapter 7).

Uptake : elimination asymmetry

Occasionally proponents of critical supersaturation approaches will concede that their decompression profiles are forming 'silent' bubbles but then show reluctance in changing their calculation method to comply with this fact. For those not acquainted with the argument expressed on p. 137 that mathematical symmetry is based upon true supersaturation, i.e. no bends implies no bubbles in the critical tissue(s), then a more direct demonstration of the asymmetry between uptake and elimination during decompression is, perhaps, more convincing.

The group at R.N.P.L. devised an ingenious experiment for determining the number of tissue types involved in limb bends but one which also provides an insight into the question of symmetry of gas exchange in those tissues. Earlier (p. 35) it was described how a bounce dive, i.e. one with no decompression stops, can be 'titrated' on men or goats simply by increasing 'bottom time' (t) with successive exposures to a given depth (H) until the subject displays marginal symptoms on return to the surface. The same result can be obtained by fixing the time and titrating the depth. Moreover, this fixed 'bottom' time can now be spent at two depths rather than one—as t_1 at depth H_1 immediately followed by t_2 at depth H_2 (fig. 46). This is effectively two consecutive exposures. Keeping t_1 and t_2 constant,

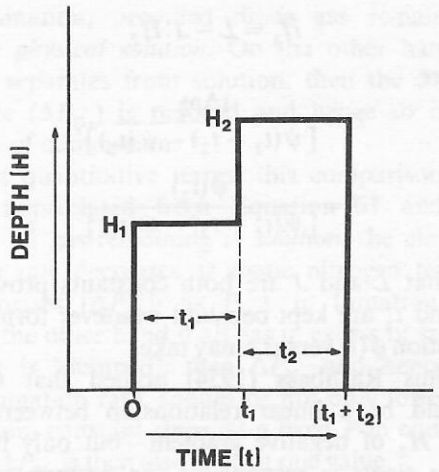

Fig. 46 Depicting a double exposure consisting of time t_1 spent at a depth H_1 immediately followed by time t_2 at H_2 and then rapid (no-stop) return to the surface. H_2 can be greater or less than H_1

H_2 can be titrated for each value of H_1. Thus, if the first exposure is deeper, the second must be shallower and vice versa.

It was shown earlier how the imminence of bends occurring after a bounce dive is determined by the amount of gas taken up at depth irrespective of the mechanism. If we now consider just one tissue whose uptake of nitrogen for air diving follows any function of time (t), simply designated $\psi(t)$, then the increase in mean tissue tension ($\Delta\bar{p}$) for the two-stage exposure is given by

$$\Delta\bar{p} = H_1 \cdot \psi(t_1 + t_2) + (H_2 - H_1)\psi(t_2) \quad (55)$$

This is simply derived using the principle of superposition (p. 226) in that the two consecutive exposures (fig. 46) are equivalent to an exposure of H_1 for time $(t_1 + t_2)$ plus one of $(H_2 - H_1)$ for time t_2. $(H_2 - H_1)$ can be positive or negative, depending upon whether $H_1 \rightarrow H_2$ requires decompression or further compression.

That particular tissue should incur bends if $\Delta\bar{p}$ exceeds a critical value $(\Delta p)_c$, when Equation 55 can be rearranged to give the relationship between values of H_1 and H_2 for this marginal condition as

$$H_1 = L - J.H_2 \qquad (56)$$

where

$$L = \frac{(\Delta p)_c}{[\psi(t_1 + t_2) - \psi(t_2)]}; \qquad J = \frac{\psi(t_2)}{[\psi(t_1 + t_2) - \psi(t_2)]} \qquad (57)$$

so that L and J are both constants provided t_1 and t_2 are kept constant whatever form the function $\psi(t)$ versus t may take.

Thus Rashbass (1954) argued that there should be a linear relationship between H_1 and H_2 of negative gradient—but only if *the same tissue* is responsible for decompression sickness over the whole range of conditions covered. Hence it is particularly significant that his experiments on goats did show a linear relationship between H_1 and H_2, so he concluded that this result indicates the involvement of no more than *one tissue type*. This is one of the 'cleanest' pieces of scientific evidence which can be cited in connection with that issue; but proponents of multi-tissue approaches might still argue that, since t_1 and t_2 were kept constant, the same 'Haldane tissue' would still be invoked by their reasoning, so that they could also predict a linear relationship between H_1 and H_2.

However, to return to the supersaturation issue, this experiment was repeated by Hempleman (1957) who redistributed total dive time between t_1 and t_2 to emphasize that part of the H_1 *versus* H_2 relationship concerned with significant decompression, i.e. the second phase of the exposure. The results essentially confirmed the findings of Rashbass but showed a significant deviation from linearity where $H_1 \gg H_2$, i.e. only where there had been appreciable decompression in going from H_1 to H_2 (see fig. 47). Thus the basic time function, $\psi(t)$, and not just the gas exchange rate as determined by the pressure gradient, must have been reduced by the decompression. Thus the subject could not tolerate such a high first exposure (H_1) when it was followed by a large decompression to the second (H_2) even though bends were never likely to occur until the final decompression. Moreover, the deviation from

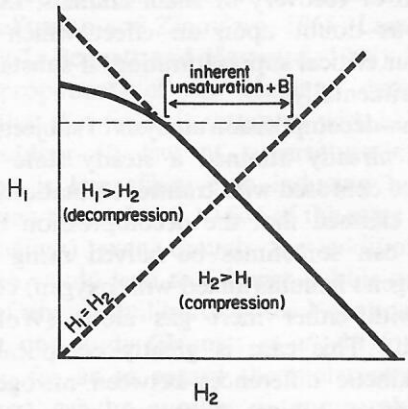

Fig. 47 Combinations of the two depths for consecutive exposures (H_1 and H_2 in fig. 46) which give marginal limb bends in goats on return to the surface. It can be seen that the experimental plots deviate from Equation 56 when there is decompression in going from H_1 to H_2 and only when this decompression exceeds the net unsaturation predicted at H_1. Redrawn from Hempleman (1957)

linearity was greater for greater decompression (see fig. 47). Hence this classical experiment leaves little doubt that there were 'silent' bubbles present *in the critical tissue(s)* during the second phase of the exposure whenever $H_1 \gg H_2$. Furthermore, Hempleman goes on to point out the mathematical asymmetry which this implies between gas uptake at pressure and gas elimination following excessive decompression. It is also interesting to note that the deviation from linearity (fig. 47) starts at just about the point where $(H_1 - H_2)$ is not far in excess of the inherent unsaturation at H_1 plus mechanical factors (B) (see p. 86), i.e. just about the permissible limit for bubble-free decompression based upon thermodynamic equilibrium. Much the same conclusion can be drawn from the very extensive series of heliox exposures of men performed by Barnard (1975).

Inert gas elimination during decompression

One might well ask that, if it is so important

to establish whether there is mathematical asymmetry of gas exchange from the standpoint of calculating tables, then why not make a direct measurement of gas elimination during decompression and compare it with the rate without decompression? The simple answer is that, whereas it is easy to measure elimination from the whole body, any result is no more relevant to the critical tissue(s) than the direct methods of bubble detection described in a previous section. Even if the gas in whole tissues can be estimated employing such sophisticated techniques as the mass spectrometer, or using radioactive tracers, then which tissue should be monitored? If a connective tissue is chosen as a likely candidate for provoking marginal symptoms (p. 59), then these tend to be too small or too thin for the direct implant of a probe. Moreover, the veins draining these tissues tend to merge with those from other organs before they reach a size at which it is feasible to monitor efferent blood. However, although these arguments may detract from reaching any firm conclusions relevant to the supersaturation issue, it is still interesting to see how decompression *per se* affects inert gas elimination from the whole body.

If a subject has been breathing air at 100 fsw, and is switched to a nitrogen-free breathing mix *at the same pressure*, then the nitrogen in his body will be released at a continuously reducing rate. After allowing for the greater total quantity, the profile of the nitrogen wash-out will closely follow that observed for switching breathing mixes at normal pressure—a profile which is an exact mirror image of uptake and whose form will be analysed in detail later in connection with the perfusion: diffusion controversy. The plot of cumulative expired nitrogen versus time is asymptotic but not exponential; although it is popular practice to equate it to a number of exponential terms (see p. 175).

If the subject is now *decompressed* on switching to the nitrogen-free mix, there is the same step change in inspired nitrogen partial pressure as imposed without decompression. There would then be the same driving force and hence the same time course for nitrogen elimination, provided tissue gas remains *in true physical solution*. On the other hand, if gas separates from solution, then the driving force (ΔP_{N_2}) is reduced and hence so is the rate of elimination.

In quantitative terms, this comparison can be appreciated from Equation 51 and 52. For all gas remaining *in solution*, the elimination rate decreases as tissue nitrogen tension decreases ($\Delta P_{N_2}\downarrow$ as $P_{N_2}\downarrow$ in Equation 51). On the other hand if all gas in excess of saturation is 'dumped', then ΔP_{N_2} and hence the elimination rate, should be not only lower but remain constant since, at a fixed P in equation 52, ΔP_{N_2} is then also fixed at one value.

Reasoning somewhat along these lines, Willmon and Behnke (1948) exposed men to 100 fsw for 75 min and measured expired nitrogen for the first 30 min and then the next hour after switching them to pure oxygen with and without decompression. They found that whatever the decompression, the nitrogen elimination rate was not constant as they would have predicted on a 'silent' bubble theory but continued to fall. However, when repeating the experiment for an exposure of 30 min at 100 fsw, the nitrogen elimination rate was appreciably less at the surface than for lesser

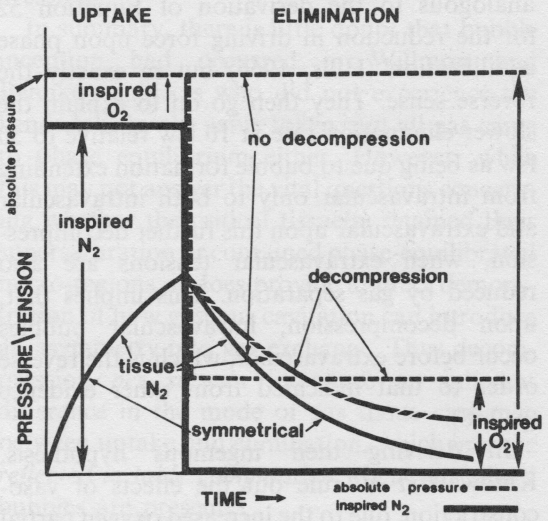

Fig. 48 A graphical presentation of the classical experiment of Willmon and Behnke (1948) depicting the relatively lower rate of nitrogen elimination at lower hydrostatic pressure despite the same step change in inspired nitrogen partial pressure

decompression, i.e. for taking the diver only as far as 44–66 fsw. Unfortunately, the fact that the elimination rate was neither constant nor the same as without decompression (fig. 48) resulted in Willmon and Behnke drawing no conclusions, although their line of reasoning was far ahead of its time. Their experimental findings have been essentially confirmed in later studies (D'Aoust *et al.*, 1976), while similar reductions in nitrogen wash-out rates have been recorded with switching to pure oxygen upon decompression to altitude (Jones *et al.*, 1942). Their results are also consistent with those of Kindwall *et al.* (1975) to the extent that these workers found that, upon breathing 80 : 20 helium : oxygen after decompression from 100 fsw on air, the wash-out of nitrogen was about 10–20% lower in men at 10 fsw than at 50 fsw. However, upon wash-out *without decompression* (i.e. remaining at 100 fsw), the nitrogen elimination was found to be lower than at either of the lesser depths. To explain this unexpected result Kindwall *et al.* postulate intravascular bubble formation at 50 fsw facilitating nitrogen elimination by dropping the blood inert gas tension relative to tissue and so increasing the driving force for extravascular tissue desaturation. This is analogous to the derivation of Equation 52 for the reduction in driving force upon phase equilibration (Hills, 1966) but in exactly the reverse sense. They then go on to explain the slower elimination rate at 10 fsw relative to 50 fsw as being due to bubble formation extending from intravascular only to both intravascular and extravascular upon this further decompression, when extravascular tensions are also reduced by gas separation. This implies that, upon decompression, intravascular bubbles occur before extravascular, which is the reverse order to that indicated from other evidence (p. 148).

In deriving their ingenious hypothesis, Kindwall *et al.* rule out the effects of vasoconstriction, due to the increased oxygen partial pressure at the higher pressures, on the grounds that the rate of wash-out of helium at 100 fsw was roughly the same whether the man breathed air or pure oxygen (Kindwall, 1975). However, this assumption does not take account of cutaneous gas transfer, which can be quite substantial according to the evidence collected by Behnke (1975) and much emphasized by this worker. Vasoconstriction can be as extensive in cutaneous tissue as in most others. Hence it could be significant that Kindwall's men used as controls in helium elimination (without decompression) were exposed to air while, in the decompression studies of Kindwall *et al.*, the men were also exposed to air but, in this case, nitrogen was the gas monitored. Thus, in one case, the monitored gas (helium) is being eliminated across the skin *in competition* with the gas collected for analysis while, in the other case, the gas collected from body tissues is being *augmented* by transcutaneous transfer. Thus, switching to oxygen during helium wash-out without decompression would tend to reduce loss across the skin and therefore increase respiratory collection, so masking the effect of vasoconstriction. On the other hand, the increased oxygen partial pressure at 100 fsw on 80 : 20 helium : oxygen by comparison with the P_{IO_2} at 50 fsw and 10 fsw would reduce both elimination of body nitrogen and its augmentation by transcutaneous uptake, so emphasizing vasoconstriction. Hence the results of Kindwall *et al.* could still conform to the simple view that decompression reduces inert gas elimination in accordance with equation 52 yet, at 100 fsw on 20% oxygen, there is extensive vasoconstriction reducing both nitrogen elimination from tissues and its transcutaneous uptake.

To try to settle this very serious question, this writer has essentially repeated the classical experiment of Willmon and Behnke but has tried to avoid the possible objections to the experiments of Kindwall (1975) and Kindwall *et al.* (1975) by monitoring nitrogen elimination at different pressures but at the *same inspired* P_{O_2}. This was performed on large guinea pigs using 0·2 AT of oxygen but 'topping up' to the desired absolute pressure with helium (see fig. 49). Moreover, the nitrogen monitored was not simply that exhaled, but that vented from the whole chamber to include transcutaneous elimination in all cases. By allowing the

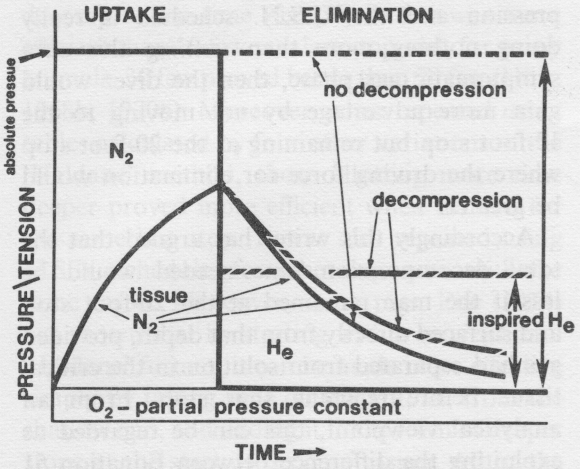

Fig. 49 Showing the nitrogen elimination rate from guinea pigs on switching to a nitrogen-free breathing mixture at the same partial pressure of oxygen. The rate decreases at reduced hydrostatic pressure. Unlike the experiment shown in fig. 48, the oxygen partial pressure during nitrogen wash-out is 'topped up' to the desired absolute pressure with helium

animals to breathe freely in the chamber, the switch to helium : oxygen mixes applied to the skin as much as the breathing mixture, temperature being kept constant to minimize its effect on elimination (Balldin, 1973). It was found that the rate of nitrogen elimination still fell continuously, even with the greatest decompression, but the overall fall in the rate was rather more pronounced than in Willmon and Behnke's results. The smaller differential recorded in their runs can be attributed to hyperoxia-induced vasoconstriction, Willmon and Behnke's non-decompressed subjects eliminating nitrogen while at an inspired P_{O_2} of 133 fsw! Thus, if their P_{IO_2} values had not been changed in a manner tending to oppose the effect they were seeking, they would probably have recorded an even larger decrease in nitrogen elimination rate with decompression *per se*.

This writer has taken the view (Hills, 1966) that gaseous cavitation is a random process (Chapter 4) so that, after decompression, some tissue regions will remain supersaturated while others will 'dump' their gas and approach phase equilibrium. Thus some essentially follow Equation 51 and eliminate gas as though there had been no decompression, while others follow Equation 52 and transfer nitrogen to blood at a uniform rate until these 'dumps' are exhausted. Now the nitrogen expired at the lung comes from all organs; and within each organ, from both supersaturated and phase equilibrated regions. All manner of intermediate states may also exist but these do not affect the basic argument which is easier to follow if it is viewed solely as a simple combination of these two limiting cases. Hence, for greater decompression, the probability of bubble inception in each micro-region increases, so that the number of gas 'dumps' increases relative to the number of regions retaining their true supersaturation. Thus the rate of nitrogen elimination still continues to fall gradually with time on account of the supersaturated regions but the overall rate is lower due to more nitrogen being derived from gas 'dumps'.

This approach therefore offers a convenient method for estimating the extent of gas phase separation within the whole body as the relative volumes of nitrogen contributed to overall elimination by supersaturated and by phase equilibrated regions for a particular decompression.

In summary, there is little doubt that bubble inception had occurred in Willmon and Behnke's subjects who did not experience the bends but, by the same token, not all gas came to phase equilibrium either. However, while this may not answer the vital questions concerning whether the critical tissue(s) retained their supersaturation or contained phase-equilibrated micro-regions, it does provide a direct demonstration of how gaseous cavitation can introduce an asymmetry into gas exchange. Thus decompression *per se* can introduce an inherent difference in the mode of gas tissue exchange between uptake and elimination, which *must be reflected in table computation* whenever 'silent' bubbles are present.

Titration of the last stop

Returning to methods specifically designed to

answer the vital issue *in the critical tissue(s)*, the difference in nitrogen elimination rates between supersaturated and phase equilibrated regions (gas dumps) has been exploited in a very simple method devised by Hills (1968b, 1970a).

Consider a diver who has come up to the 20-foot stop by following a decompression schedule calculated by conventional methods based on critical supersaturation, e.g. a U.S.N. table for an exposure of 60 min at 160 fsw computed using standard M values (Workman, 1969). Now, if these M values really represent 'trigger points' to supersaturation, that diver would increase his gas elimination rate if he continues to follow the table and moves from the 20-foot to the 10-foot stop as soon as those M values (and the estimates of tissue gas tensions) indicate that it is safe to do so. In this way he should decompress right to the surface in the minimum time if gas has remained *in true physical solution* as use of the 'Haldane' rationale implies. On the other hand, if gas has *separated from solution* early in the decompression and the U.S.N. schedule is really doing nothing more than *treating* this subsymptomatic gas phase, then the diver would gain more advantage by not moving to the 10-foot stop but remaining at the 20-foot stop where the driving force for elimination would be greater.

Accordingly this writer has argued that the total decompression time needed would be less if the man remained at the 20-foot stop and surfaced directly from that depth, provided gas had separated from solution in the critical tissue before reaching that stop. From an analytical viewpoint, this can be regarded as exploiting the difference between Equation 51 for true supersaturation and Equation 52 for phase equilibration. Decreasing P by 10 fsw in moving from the 20- to the 10-foot stop therefore, increases ΔP_{N_2} by 8 fsw in Equation 51 ($F_{IN_2} = 0{\cdot}8$ for air) but *decreases* ΔP_{N_2} by 2 fsw in Equation 52.

In a very large series of trials on goats at

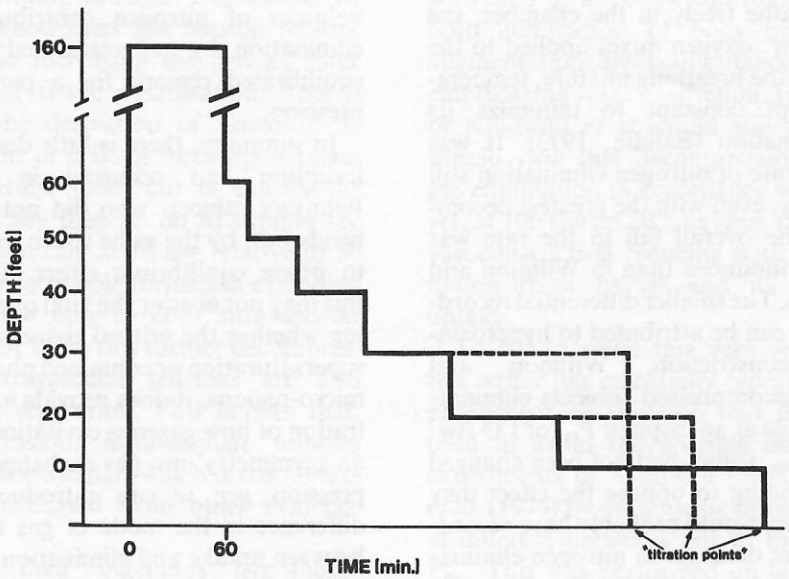

Fig. 50 Depicting a U.S. Navy profile for 60 min at 160 feet on air in which the subject surfaces from 10 feet, as advocated, or remains at 20 feet and surfaces directly from that depth or does the same from 30 feet. The total decompression times are depicted for equal bends incidence in goats after titrating each variation of the Navy table on the same animals. Data from Hills (1968b, 1970a)

R.N.P.L. it was found that by titrating total decompression time (fig. 50) 20 fsw was more efficient than 10 fsw as a last stop and, for many animals, 30 fsw proved better than 20 fsw (Hills, 1968b, 1970a). Moreover, when repeated using tables calculated by four other variations of the 'Haldane' method, those allotting more time deeper proved more efficient when titrated for the same last stop. It was also repeated using U.S.N. schedules for other exposures with the same result. Moreover, using pigs, Fife (1974) has shown that by starting with a conventional 'Haldane' format, it can be made more effective simply by taking time off the tail, i.e. from the shallow end, and adding it in the form of deeper stops at the start of decompression. All of the above findings leave little doubt that conventional Naval tables are not preventing bubble inception but are really treating a sub-symptomatic gas phase probably formed during the first long 'pull' towards the surface so characteristic of U.S.N. profiles.

Vasoconstriction due to changes in inspired P_{O_2} cannot be offered as an explanation since it would be greater at 20 fsw than at 10 fsw and would therefore tend to cause a greater drop in nitrogen elimination for the 20-foot last stop; but this still proved to be the more efficient anyway.

Hence the results of this extensive series of trials must be regarded as strong evidence that 'silent' bubbles can be formed *within the critical tissue(s)*. It is very difficult to find any other explanation.

Single-stop titration

About the only argument which could be brought to the defence of the critical supersaturation concept in the light of those last results is to say that neither the U.S. Navy nor the designers of the other four 'supersaturation' tables used were employing the correct M values or tissue half-times in their computations. However, even this argument can be overcome if the titrated *last* stop is the *only* stop; so that it cannot be claimed that the preceding stages of the decompression predisposed the subject to

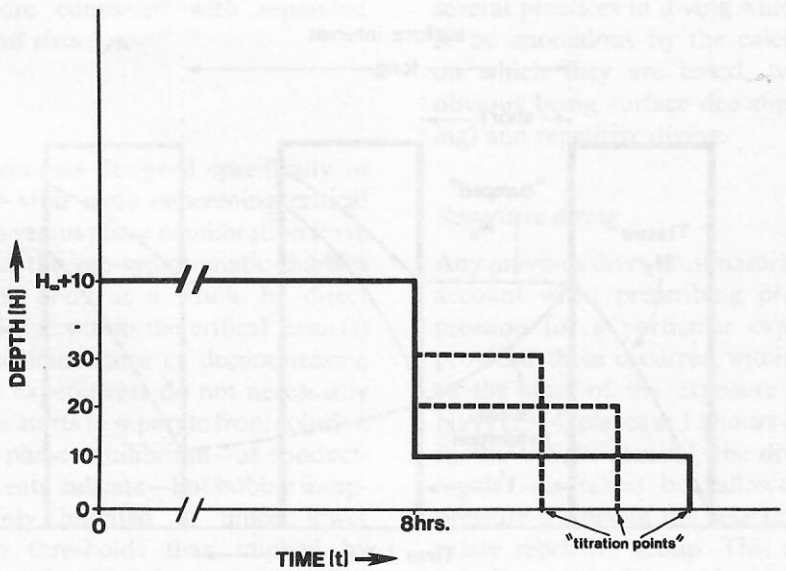

Fig. 51 Comparison of the duration of a single-stop decompression needed just to avoid marginal limb bends after an exposure of 8 hours at $H_\infty + 10$ on air when that one stop is placed at 10, 20 or 30 feet. H_∞ is the minimum bends depth for air. Data from Hills (1968b)

bubble formation by virtue of their incorrect computation.

Consider a subject whose maximum bends-free depth is H_∞ for an eight-hour exposure on air followed by no-stop decompression. To reiterate the 'Haldane' rationale, decompression from H_∞ to the surface gives no bends and hence should give no bubbles. If he is now exposed for the same time to a depth $(H_\infty + 10)$ and then decompressed to 10 fsw, this should not precipitate gas either since, by this critical supersaturation criterion, this decompression, $(H_\infty + 10) \rightarrow 10$, represents a lower ratio than $H_\infty \rightarrow$ surface. Hence, if the 'Haldane' principles are correct and gas remains in true physical solution in the sensitive tissue(s) for a bends-free decompression, 10 fsw would be the ideal stop providing the maximum driving force for eliminating nitrogen until it is safe to surface.

On the other hand, if the decompression from $(H_\infty + 10)$ to 10 fsw has deposited a sub-symptomatic volume of gas, as predicted by the phase equilibration concept, then the subject would eliminate more gas prior to surfacing by selecting a greater depth for his only stop—say 20 or even 30 fsw.

This line of reasoning led to a further series of trials in which goats were exposed to pressures 10 fsw greater than their experimentally determined maximum safe depth for no-stop decompression (H_∞)—Hills (1970a). Total decompression time is now only that spent at the one stop needed and can be titrated as described earlier (see fig. 51). Three titrations were completed on each animal, one each for stopping at the 10, 20 or 30 fsw level. As in the previous experiment, it was found that a stop at 30 fsw required less time than at 20 fsw which, in turn, required less than at 10 fsw; although the differences were not as pronounced as before. This result is totally contrary to any reasoning based on limited supersaturation for the reasons given earlier and yet again provides strong evidence that 'silent' bubbles can occur *in the critical tissue(s)*.

Repetitive exposures

The foregoing conclusion has been confirmed in an ingenious experiment designed by the Oxford group with the only reservation that its experimental execution used death-or-recovery

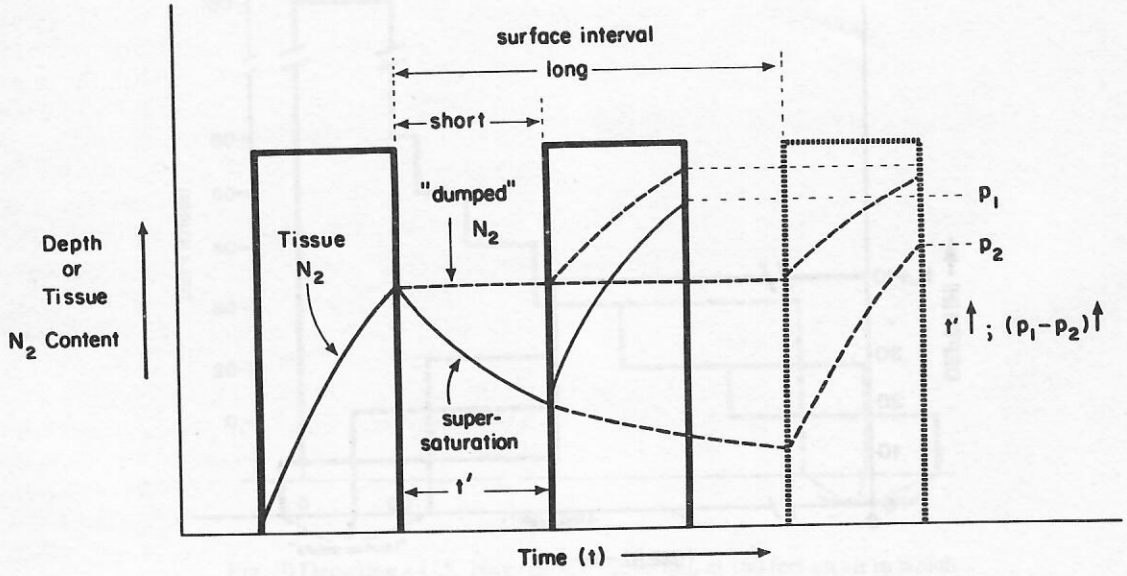

Fig. 52 Two equal exposures on air separated by a surface interval (t'). By conventional reasoning, there should be more nitrogen eliminated during a long bends-free surface interval than during a short one to give a smaller tissue nitrogen tension upon the final decompression ($p_2 < p_1$) and yet, in practice, the bends incidence is found to increase with the surface interval (Griffiths *et al.*, 1971)

of small animals as the end-point (Griffiths *et al.*, 1971). They argued that if a subject has returned to normal pressure after a safe air exposure not requiring gradual decompression, then he will proceed to eliminate nitrogen. The elimination rate should be appreciable according to the 'Haldane' rationale, since the gas is remaining in solution because he is bends-free. If he is then returned to pressure for a second exposure sometime later and subsequently decompressed, he should stand less chance of getting the bends if the two exposures are separated by a longer interval, since this should then allow more nitrogen to be eliminated before starting the second (see fig. 52). However Griffiths *et al.* found precisely the reverse, *i.e.* the incidence of decompression sickness increased as the interval separating exposures was increased. They concluded that this was contrary to the 'Haldane' rationale and confirmed sub-symptomatic gas separating from solution in the sensitive tissue(s) between exposures. They reasoned that a longer interval between exposures allowed more time for bubbles to grow before recompression but it would also allow more time for coalescence of 'dumped' gas to occur so, either way, their results are more consistent with separated gas *in the critical tissue(s)*.

Conclusion

Thus the experiments designed specifically to investigate this vital issue concerning critical supersaturation versus phase equilibration leave little doubt that the sub-symptomatic bubbles observed in the body as a whole by direct methods also occur within the critical tissue(s) determining the imminence of decompression sickness. These experiments do not necessarily confirm that gas starts to separate from solution at the point of phase equilibrium—as conductance measurements indicate—but bubble inception is certainly initiated at much lower supersaturation thresholds than implied by M values, at least by those representing maximum bends-free decompression on air.

However, before proceeding to the next vital issue, it is interesting to see whether this conclusion is compatible with practical diving experience.

Practical Experience

The vital issue concerning whether limited supersaturation or phase equilibration determines the occurrence of bends is sometimes referred to in the diving industry as 'beat the bubble versus treat the bubble'. Although most companies devise tables intended to 'beat the bubble', many divers have the 'feeling' of bubbles during decompression, even though they may not hurt. Upon moving from the 40- to the 30-foot stop, many subjects may feel a 'niggle' around a joint which soon goes away sometimes to return just after making another move between stops, say from 20 to 10 fsw. If this awareness of potential discomfort does not go away by the end of the stop, then decompression to the next stop usually precipitates a bend. This feeling of 'treat the bubble' is too subjective to record as a hard scientific datum but it is an impression which many divers describe after an ostensibly bends-free decompression using conventional schedules.

To turn from impressions to facts, there are several practices in diving which would appear to be anomalous by the calculation methods on which they are based, two of the most obvious being surface decompression (decanting) and repetitive diving.

Repetitive diving

Any previous dives must naturally be taken into account when prescribing preventive decompression for a particular exposure; at least, provided these occurred within a period prior to the start of the exposure which the U.S. Navy (1974) places at 12 hours for air breathing. In the U.S.N. manual, the diver still uses the regular air tables but allows for the recent pressure history in the selection of the appropriate repetitive group. This selection is now based upon an 'equivalent single dive time' rather than the actual dive time from which it is derived by adding a 'residual nitrogen time'. Values for the latter are then taken from

tables relating the duration of the previous exposure to the surface interval between dives and, as their name suggests, are prescribed to allow for the diminishing nitrogen retained by the critical tissue(s) at the surface (Workman, 1969). Although appearing somewhat 'cook-book' at first sight, this repetitive group selection procedure has the advantage that it is simple to follow and yet enables some allowance to be made for the large number of parameters which renders repetitive diving so difficult to tabulate. Like the single exposure tables, the residual nitrogen times also give the appearance of empirical modification to reflect practical reality.

It is therefore particularly interesting that, according to the U.S.N. manual, a continuous exposure of 40 min is equally as safe as spending the same time at the same depth (80 fsw) but divided into two exposures of 20 min each, despite being separated by a surface interval of 30 min during which nitrogen must be lost from all tissues. A truly supersaturated tissue must have an appreciable driving force for losing nitrogen during the surface interval ($\Delta P_{N_2}\uparrow\uparrow$ as $P\downarrow\downarrow$ in Equation 51) while, if gas is 'dumped', the driving force is minimal ($\Delta P_{N_2}\downarrow\downarrow$ as $P\downarrow\downarrow$ in Equation 52). Whatever the theoretical implications, the practical reality embodied in the U.S.N. table of 'residual nitrogen times' is consistent with experience in aviation medicine and the animal results of the Oxford group described earlier.

Repetitive aerial decompression has been found to reduce an aviator's minimum bends altitude, despite breathing pure oxygen during the whole sequence of exposures, in some cases from at least 23,000 feet down to 17,000–18,000 feet (Houston, 1947). Hence practical experience of repetitive decompression in both diving and aviation is fully consistent with the results of the repetitive exposures of small animals undertaken by the Oxford group, so it must add further confirmation to their conclusion that 'silent' bubbles *are* present during the surface interval.

One might then ask how this would affect the final decompression, particularly if following a U.S.N. table calculated on a critical supersaturation basis. Repetitive tables tend to give much trouble and this is one area where the answer to the critical supersaturation versus phase equilibration issue makes a great deal of difference to the method of prevention recommended.

Preoxygenation at altitude

Two of the experiments specifically designed to settle the issue concerning supersaturation versus phase equilibration compared the relative decompression times needed for stops at 10, 20 or 30 fsw. The results obtained on large animals were particularly difficult to interpret by any critical supersaturation approach.

The argument was basically that, for any sub-symptomatic decompression likely to cause phase separation (according to equilibrium criteria), the elimination of inert gas was greater at higher pressure—the exact converse of what would be expected if that gas had remained in solution in a supersaturated state. This is borne out in practice in aviation medicine. It is well known that preoxygenation, or inspiring pure oxygen before a marginal decompression, is more effective in preventing bends if undertaken at lower altitude (Fraser, 1943; Gray, 1944a; Fryer, 1962), i.e. more effective in removing nitrogen at higher pressure ($\Delta P_{N_2}\uparrow$ as $P\uparrow$ in Equation 52 and not 51). This is also consistent with the reduced bends rate found by the Royal Navy's experience when adding the time spent at the 10-foot stop of a conventional naval decompression onto the 20-foot stop and surfacing directly from 20 fsw (p. 260).

Thus there are at least two pieces of practical experience which are consistent with the experiments involving 10–20–30 foot titrations whose results are so difficult to interpret by the concept of critical supersaturation.

Surface decompression/decanting

Long before the industry became so sophisticated, surface decompression referred to a very specific procedure corresponding to decanting in tunnel construction. This is a method

first instigated at sea by the U.S. Navy whereby a diver who has had an exposure not far in excess of no-stop decompression limits can be returned directly to the surface, transferred to a deck pressure chamber, recompressed and then continue his decompression away from the natural hazards of the ocean. Surface intervals as long as 13 min can be symptomless (Gouze, 1944) but the fear of generating 'nascent' bubbles (Behnke, 1969) has caused the U.S. Navy to put the limit at 5 min. If the air exposure does exceed the bounce-dive curve by more than the tolerable margin, the diver can decompress in the normal way on air to 20 feet in the ocean and then transfer to the deck pressure chamber in which he is recompressed to 20 fsw. He then undergoes a prescribed decompression which includes a 10-foot stop, the overall time being greater than if all decompression had been performed in the water (see fig. 53).

On the other hand, if oxygen is available in the recompression chamber, then the U.S.N. (1974) manual prescribes a different decompression involving less time to the point where the diver can surface directly from 30 feet. He is then transferred to a deck recompression chamber, compressed to 40 fsw and breathes pure oxygen for the time prescribed in the manual (see fig. 53).

Inconsistency

The U.S.N. approach to surface decompression shows at least two inconsistencies.

(a) It is difficult to conceive two optimal methods of eliminating nitrogen from the same air-breathing diver completing the same exposure, depending upon an event which has not yet occurred—whether or not he will inspire pure oxygen on recompression. The two paths to 30 feet are depicted in fig. 53.

(b) The fact that a man can 'drop out' from an incomplete decompression means that he must have violated the '*M*' value for at least one 'tissue' on the 'Haldane' rationale used to compute U.S.N. schedules. Hence he has 'triggered' bubble inception and bends must be the inevitable consequence according to that method (p. 110).

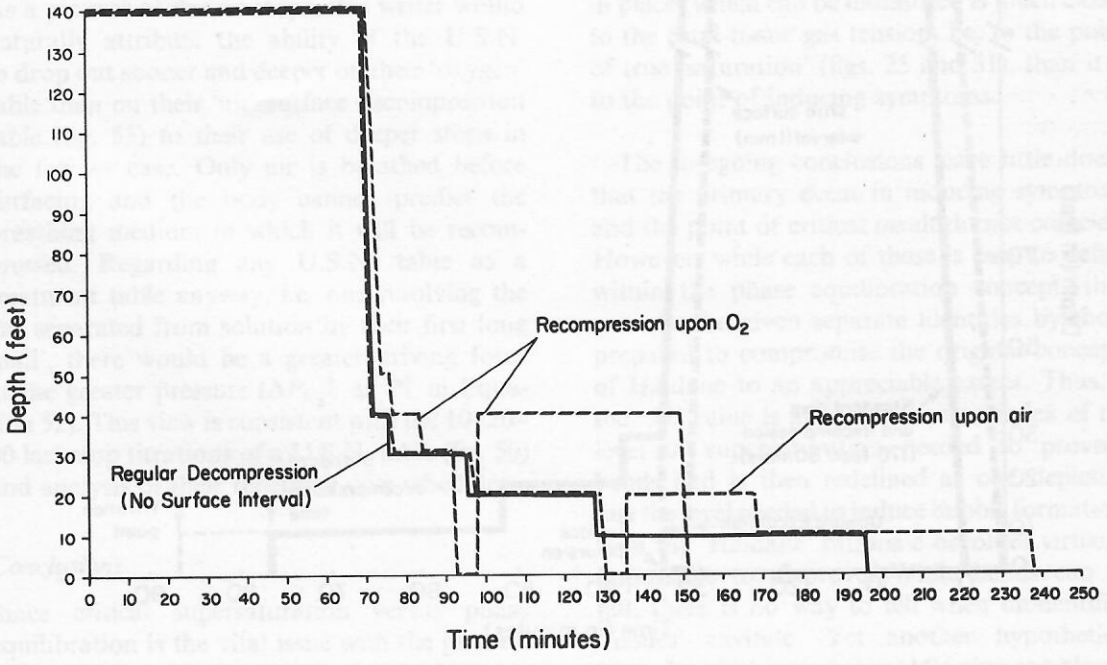

Fig. 53 Comparison fo the U.S. Navy (1974) table for a regular air dive of 70 min at 140 feet with their surface decompression schedules for recompression on either air or oxygen

Thus the fact that surface decompression is possible—and indeed works—must be regarded as evidence incompatible with the concept of critical supersaturation. However, proponents of the conventional view could argue that the U.S.N. surface decompression tables *do* 'trigger' bubbles but treat them before the onset of symptoms. The fact that the prescribed recompression exceeds the unfinished portion of the initial decompression cut off by surfacing is virtual admission that this is a treatment. Otherwise, all of their hypothetical 'tissues' would have been eliminating nitrogen during the surface interval, thus *reducing* and not increasing any further time needed at pressure.

Upward excursion

This line of argument has led to a critical test of the 'Haldane' rationale. If a subject completes an exposure such as 30 min at 170 fsw, then the U.S.N. tables advocate 30 fsw as the first stop, presumably on the basis of the '*M*' value of a particular 'tissue'. Thus, if the subject overshoots this stop and goes to 10 fsw or even to the surface for one to two minutes, before returning to the 30-foot stop, then he has violated at least one 'trigger point' and probably those for many other hypothetical 'tissues'. Nuclei activated into growth *in vitro* produce bubbles within one minute of decompression (Hills, 1967a; Strauss, 1974). Hence, according to the 'Haldane' rationale, the subject should therefore develop symptoms soon after returning to the first stop of his normal decompression. Nuclei activated by the overshoot, i.e. the upward excursion, must grow at this stop since that depth was determined in the first place by the maximum degree of supersaturation which that 'tissue' was supposed to tolerate—as expressed by the appropriate '*M*' value.

However, in an extensive series of trials on goats (Hills, 1971a), it was found that interposing a one to two minute upward excursion, even a short surface interval between the

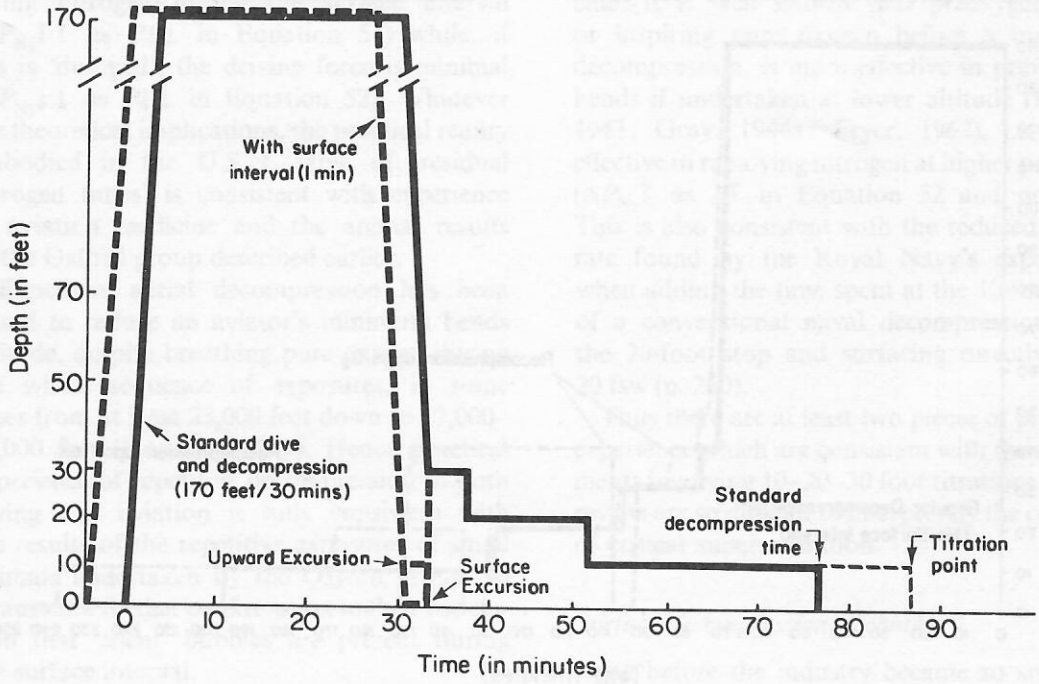

Fig. 54 'Titration' of the total decompression times for a standard U.S. Navy table with and without interposing an upward excursion or surface interval between the exposure and the same decompression. Data from Hills (1971a)

exposure and the decompression (fig. 54), had no detectable effect when total decompression time was titrated. While this is totally incompatible with critical supersaturation, it can be explained on the basis of local phase equilibration. If thermodynamic equilibration is the relevant state, then the critical micro-region will be in phase equilibrium at the start of the first stop *whatever path* the diver has taken to reach it from his 'bottom' depth—provided, of course, he has not taken so long that it has influenced the total gas content of the critical tissue. Accordingly, the same marginally subsymptomatic volume of gas should have separated from solution soon after the start of the first stop of a typical U.S.N. air table irrespective of whether there was an intervening upward excursion or not. Hence the feasibility of surface decompression—and this experiment in particular—must be regarded as strong evidence in favour of the concept of micro-phase equilibration.

Comment on U.S.N. surface decompression

As a pioneer of deeper stops, this writer would naturally attribute the ability of the U.S.N. to drop out sooner and deeper on their 'oxygen' table than on their 'air' surface decompression table (fig. 53) to their use of deeper stops in the former case. Only air is breathed before surfacing and the body cannot predict the breathing medium in which it will be recompressed. Regarding any U.S.N. table as a treatment table anyway, i.e. one resolving the gas separated from solution by their first long 'pull', there would be a greater driving force at the greater pressure ($\Delta P_{N_2}\uparrow$ as $P\uparrow$ in Equation 52). This view is consistent with the 10–20–30 last-stop titrations of a U.S.N. table (fig. 50) and analysis of their repetitive dive schedules.

Conclusions

Since critical supersaturation versus phase equilibration is the vital issue with the greatest influence on table formulation, it is desirable to apportion the likelihood of one or other being correct even if no firm conclusion can be reached. From all of the evidence collected in this chapter, it would seem fair to make the following statements.

(1) There is no doubt that, during decompression, bubbles can be formed within the body *as a whole* which do not produce overt symptoms of decompression sickness, these bubbles certainly occurring intravascularly and most likely extravascularly.

(2) There is good reason to doubt that bubbles form *de novo* in blood; while there is substantial evidence to support the view that intravascular bubbles may have extravascular origins.

(3) Gas can also separate from solution and remain asymptomatic *in the critical tissue(s)* responsible for marginal limb bends, all indirect methods of detection giving this impression.

(4) It is very difficult to pinpoint the initial onset of phase separation in the critical tissue(s) during decompression but direct methods show that the pressure needed to initiate the gas phase in places which can be monitored is much closer to the total tissue gas tension, i.e. to the point of true 'saturation' (figs. 25 and 31), than it is to the point of inducing symptoms.

The foregoing conclusions leave little doubt that the primary event in inducing symptoms and the point of critical insult do not coincide. However, while each of these is easy to define within the phase equilibration concept, they can also be given separate identities by those prepared to compromise the original concepts of Haldane to an appreciable extent. Thus, if the '*M*' value is abandoned as an index of the level of supersaturation needed to provoke bends and is then redefined as one depicting just the level needed to induce bubble formation, then the 'Haldane' rationale becomes virtually impossible to disprove. While bends can be felt, there is no way to tell when unidentified 'tissues' cavitate. Yet another hypothetical 'tissue' with the necessary '*M*' value can always be produced by the computer to explain the results which did not fit beforehand and all

that the critics of that method can do is to show how unlikely it all is and to what absurd limits the calculation method needs to be stretched.

It is therefore rather futile to proceed any further with this vital issue. Howard and Bradner (1976) have aptly compared this controversy to the difficulty which Copernicus experienced in introducing his concept of planetary motions in the sixteenth century, at which time the established views of astronomers were based on the geocentric model of Ptolemy. They point out that, just as additional 'tissues' are added to the 'Haldane' rationale (with their appropriate half-times and '*M*' values) to account for any distinct unexpected decompression situation, so additional epicycles (with suitable amplitudes) were incorporated into the Ptolemaic model to account for any unpredicted planetary motions. Hence the basis for our present understanding of planetary motion was not accepted until the model of Ptolemy had divagated into incredible detail. Is the same thing happening in conventional procedures for formulating decompression tables?