MOMSEN, C. B. & WHELAND, K. R. (1939). *Report on use of Helium–oxygen Mixtures for Diving*. (Revised 1942.) U.S. Navy Experimental Diving Unit, Washington, D.C.

PICCARD, J. (1941). Aero-emphysema and the birth of gas bubbles. *Proc. Mayo Clin.* 16, 700–704.

RASHBASS, C. (1955). *Investigation into the Decompression Tables*. Report. Medical Research Council, R.N. Personnel Research Committee, U.P.S. 151, London.

REEVES, E. & BECKMAN, E. L. (1966). *The Incidence of Decompression Sickness in Dogs Following 7, 12, 18 and 24 hour Saturation Dives with 'No-stop' Decompression*, Report No. 4, Naval Medical Research Institute, Bethesda, Maryland.

ROTH, E. M. (1967). *Space Cabin Atmospheres. Part III. Physiological Factors of Inert Gases*. NASA SP-117, Washington: Office of Technology Utilization. Natl. Aeron. Space Admin.

ROUGHTON, F. J. W. (1952). Diffusion and chemical reaction velocity in cylindrical and spherical systems of physiological interest. *Proc. roy. Soc. B*, 140, 203–229.

SAYERS, R. R. & YANT, W. P. (1926). The value of helium–oxygen atmosphere in diving and caisson operations. *Anesth. Analg.* 5, 127–138.

SAYERS, R. R., YANT, W. P. & HILDEBRAND, J. (1925). *Possibilities in the Use of Helium–oxygen Mixtures as a Mitigation of Caisson Disease*. Report of Invest. No. 2670. U.S. Dept. of Interior.

SCHREINER, H. R. & KELLEY, P. L. (1967). Computation methods for decompression from deep dives. In *Proceedings 3rd Underwater Physiology Symposium*. Ed. Lambertsen, C. J., pp. 277–299. Baltimore: Williams & Wilkins.

SHAW, L. A., BEHNKE, A. R., MESSER, A. C., THOMSON, R. M. & MOTLEY, E. P. (1935). The equilibrium time of the gaseous nitrogen in the dog's body following changes of nitrogen tension in the lungs. *Am. J. Physiol.* 112, 545.

STILLSON, G. D. (1915). *Report on Deep Diving Tests*. Bureau of Construction and Repair, Navy Dept., p. 252. Washington: Gov. Printing Office.

THOMSON, E. (1927). Helium in deep diving. *Science*, 65, 36–38.

U.S. Navy Diving Manual (NAVSHIPS 250–538). (Revised 1963.) Part I. U.S. Navy Dept. U.S. Government Printing Office, Washington, D.C.

VAN DER AUE, O. E., BRINTON, E. S. & KELLAR, R. J. (1945). *Surface Decompression, Derivation and Testing of Decompression Tables with Safety Limits for Certain Depths and Exposures*. Research project X-476, Research report 1. U.S. Navy Experimental Diving Unit, Washington, D.C.

VAN DER AUE, O. E., KELLAR, R. J., BRINTON, E. S., BARRON, G., GILLIAM, H. D. & JONES, R. J. (1951). *Calculation and Testing of Decompression Tables for Air Dives Employing the Procedure of Surface Decompression and the Use of Oxygen*. Research report 1. U.S. Navy Experimental Diving Unit, Washington, D.C.

WORKMAN, R. D. (1957). *Calculation of Air Saturation Decompression Tables*. Research report 11–57. U.S. Navy Experimental Diving Unit, Washington, D.C.

WORKMAN, R. D. (1965). *Calculation of Decompression Schedules for Nitrogen–oxygen and Helium–oxygen Dives*. Research report 6–65. U.S. Navy Experimental Diving Unit, Washington, D.C.

WORKMAN, R. D. (1967). Underwater research interests of the U.S. Navy. In *Proceedings 3rd Underwater Physiology Symposium*. Ed. Lambertsen, C. J., pp. 4–15. Baltimore: Williams & Wilkins.

WORKMAN, R. D. & REYNOLDS, J. L. (1965). *Adaptation of Helium–oxygen to Mixed Gas Scuba*. Research report 1–65. U.S. Navy Experimental Diving Unit, Washington, D.C.

YARBROUGH, O. D. (1937). *Calculation of Decompression Tables*. Research report. U.S. Naval Experimental Diving Unit, Washington, D.C.

ZAL'TSMAN, G. L. & ZINOV'EVA, I. D. (1965). Comparative determinations of the permissible supersaturation value of the human body with indifferent gases under different conditions. In *The Effect of the Gas Medium and Pressure on Body Functions*. Ed. Brestkin, M. P., pp. 28–33. Collection No. 3, NASA-TT-F-358, Washington, D.C.

13

British Decompression Theory and Practice

H. V. HEMPLEMAN

The complexities of physics and physiology involved in the aetiology of decompression sickness are so great that attempts to formulate detailed quantitative analyses of the phenomena will only succeed by a happy accident. The size, shape and location of the bubbles, or bubble, in mild decompression sickness are still matters for conjecture. Even were such necessary variables clearly defined, the physics of initiation and growth of the gaseous emboli would require a knowledge of nucleation phenomena and bubble growth in biological media which certainly does not exist. Added to these difficulties is the further one of the collection of reliable data from the subject in any experimental attempt to define a gross outline of the problem.

Given these circumstances it is profitable to examine the generally accepted facts that have arisen from two separate sources, firstly from caisson and tunnel workers, and secondly from divers.

DECOMPRESSION OF COMPRESSED AIR WORKERS

In the past ten years in the United Kingdom careful records have been kept of all cases of decompression sickness occurring at any major undertakings. The decompression procedures used, which are the Regulations Relating to Work in Compressed Air 1958, have been sufficiently conservative for only 0·04% of decompressions to have been followed by severe decompression sickness. The great bulk of cases of decompression sickness, around 1% of the entries into compressed air, consists of mild or sometimes severe pains and aches in or around joints, commonly termed the 'bends'. Some of these are of sufficient severity to warrant therapeutic recompression procedure. Here arises the first difficulty. When does one consider a

case of the bends sufficiently severe to warrant therapeutic recompression, and when does one consider that the pain is so mild that recompression therapy is not appropriate? Two people participate in this decision; the man himself decides whether the pain is genuine and, if it is, whether he should report to the medical centre for treatment; secondly the doctors or medical attendants decide upon suitable treatment. Thus it is difficult to obtain reliable data on the incidence of marginal cases of decompression sickness even when both workers and medical staff are educated in these matters. It is hardly surprising therefore that data collected from time to time by various authorities have differed quite markedly in their conclusions. In the United Kingdom we have been fortunate in recent years that one medical practitioner has been largely responsible for the supervision of three major compressed air contracts. Thus one of our variables for data collection has been nearly eliminated. A vigorous campaign has been conducted to persuade the men to report all forms of unusual pains and aches that occur following their work in compressed air. Some reliance may therefore be placed upon the data which have been obtained in this way.

Threshold pressure

If men work for periods of time varying between 8 and 12 hours in compressed air at pressures less than 14 psi gauge (31 ft; 0·95 ATA), and are then decompressed back to atmospheric pressure in not less than 2 min decompression sickness requiring therapeutic recompression is rare (less than 0·2% of man-decompressions).

The Haldane decompression ratio principle

Most decompression procedures in use today consist of a fast phase followed by one or more slower phases. The pressure dividing the fast and slow phases of the decompression is based upon the Haldane ratio principle. It is certainly not true, as supposed by Haldane, that after an indefinitely prolonged stay at pressure it is just as safe to come from 6 Atmospheres absolute to 3 Atmospheres absolute as it is to come from 2 Atmospheres absolute to 1 Atmosphere absolute. Some reduction in the permissible ratio is necessary for an increase in the pressure of exposure.

Acclimatization or adaptation to compressed air working

If large numbers of men work in compressed air at pressures in excess of the threshold value many cases of decompression sickness will occur. There are two ways by which the number may be reduced: the first is to discharge from the contract those men who have regular attacks of the bends; the second is to try to avoid a large turnover in the labour force. Men of a regular work force in some, as yet unknown, way adapt themselves to com-

pressed air work and the subsequent decompression, and in such groups the incidence of the bends decreases markedly after the period of adaptation.

Bends incidence in relation to pressure

There is little doubt that for a given exposure to pressure the incidence of bends increases at greater pressures. This statement is strictly true for only the British Regulations but private communications from elsewhere confirm that this is a generally observed defect of all known air decompression procedures at present in use.

DECOMPRESSION OF DIVERS

Can the three basic facts, which have been established after many years of compressed air and caisson work be extended to greater pressure ranges? The disadvantage in diving is the paucity of data available for analysis. Whereas all the caisson results are founded upon observations over many tens of thousands of decompressions, the diving data has to be established from only scores of observations. A small amount of prolonged diving followed by an immediate surfacing has been studied, and the threshold value displayed by the caisson worker is in fact very similar to that of the diver.

Most normal men would be expected to survive a very long exposure to air at a pressure under the sea equivalent to 30 ft (1·9 ATA) and to surface in a period of 2 min without any attack of bends. The problem of acclimatization in diving is entirely unanswered. It is clear that practice is profitable for a diver but it is not clear from the data available that this influences his ability to withstand attacks of the bends. The final point established in caisson work, that decompressions tend to be more hazardous from greater pressures, is certainly also true of the decompression routines at present available to divers. For instance, a 1-hour dive at 60 ft (2·8 ATA) would hardly be expected to give any incidence of bends on either U.S. or Royal Navy decompression tables, but a 1-hour dive at 160 ft would certainly cause decompression sickness in a number of men. The unsafe nature of such schedules has been confirmed by Hills (1966).

Thus, there is a great similarity in the overall picture for air diving in both compressed air workers and divers and these facts constitute the basis of calculations leading to decompression procedures. As has, however, been pointed out, there is an almost complete absence of knowledge of the underlying physics and physiology and the best that can be done is to define the problem in very general terms, observe the data, and examine whether any scheme or new hypothesis emerges which, when put to the test does not suffer from the defects of past attempts.

TESTING DECOMPRESSION TABLES

One can examine the literature of compressed air and caisson work and reach such statements as, 'only 10 cases of bends occurred from 10,000 exposures, giving an incidence of 0·1%'. Such statements, unaccompanied by a proper further analysis are valueless and misleading. Firstly, the nature of the exposures to pressure must be stated. Clearly, 10 exposures to 30 psi g (67 ft; 3ATA) followed by 10 bends and then 9990 trouble-free exposures to 12 psi g (27 ft; 1·8 ATA) would give the results quoted above, but there is very obviously something amiss with the first decompression procedure despite the very low overall figure.

The second factor of great importance concerns the men themselves. It may be, for example, that only 15 men took part in all the exposures and that initially 5 of these men shared 10 bends between them and were then eliminated as being too sensitive. The remaining exposures were then completed with the resultant group of 10 men. Such results, therefore, apply to only a proportion of the population and are no indication of general effectiveness.

There are no standards by which one can judge the effectiveness of air decompression tables and so various groups have adopted their own criteria. For compressed air work in the United Kingdom it has been tacitly accepted that a 2% incidence of decompression sickness is the maximum acceptable for work of over 4 hours' duration at pressures between 18 psi g (40 ft; 2·2 ATA) and 40 psi g (90 ft; 3·7 ATA). Such an incidence is established for a specific contract from the wide variety of reasonably fit men, aged mainly between 18 and 40 years, some of whom work every day for months and others who work only once or twice. It is therefore a figure derived from a mixed group of acclimatized and unacclimatized men, and is subject to fluctuations largely caused by the inflow of new labour. Any standards of bends incidence are consequently only valid when bearing these variables in mind.

For divers using compressed air the vast majority of work takes place at depth and pressure combinations where either direct surfacing or very little decompression is necessary. In these circumstances the bends incidence is very low. As pointed out earlier, it is only unusual dives such as 1 hour at 160 ft that begin to test the adequacy of the decompression procedures.

THE BASIS OF THE PRESENT BRITISH NAVAL DIVING TABLES

The assumptions

In 1952, Hempleman suggested that a simple, single-tissue approach

should provide a satisfactory solution to the decompression problem. Calculations made using this simple approach were initially so successful (Rashbass 1956) that the underlying concepts have influenced all the subsequent concepts. As with the Haldane approach, a number of modifications have since had to be introduced to cope with the new knowledge obtained from diving to greater depths and for longer durations. This has unfortunately removed some of the simplicity and elegance of the original scheme. Nevertheless, the possibility that tissue diffusion is a dominating factor in the tissue inert gas exchange relevant to the decompression problem, raises interesting physiological problems. Some of these problems have been examined by Hempleman (1967) and Hills (1966).

The first assumption is that there is a direct relationship between the quantity of nitrogen and the risk of decompression sickness. The oxygen partial pressure is considered not to play any part in the aetiology of decompression sickness. This assumption is now known to be incorrect, and oxygen may cause marked circulatory changes at certain partial pressures. It is not however the immediate purpose of this chapter to discuss the validity of these underlying assumptions but to record how the British Tables were first calculated. Recent improvements will be discussed elsewhere.

The second assumption is that the arterial blood immediately suffers all the nitrogen partial-pressure changes in the breathing medium. This implies that if the pressure of the breathing medium is changed the tissues are at once supplied with blood, the nitrogen pressure of which corresponds exactly to that of the new pressure.

The third assumption is that only one tissue or type of tissue participates in mild decompression sickness such as joint pain, and that this tissue saturates with dissolved gas by diffusion.

The fourth assumption is that the rate at which gas enters the relevant tissue during the exposure to raised pressure is less than the rate at which the gas leaves during decompression. This is related to the idea that 'silent' bubbles form, even during trouble-free dives, and that they interfere with gas elimination from the tissue.

The fifth assumption is that a certain excess quantity of gas can be tolerated by the tissue and that bends are only possible if this critical excess quantity is exceeded.

These qualitative assumptions define the outline of the method, but three quantitative statements must be made in order to proceed. The rate at which the gas diffuses into the tissue is given by the equation for linear diffusion into a slab of tissue, one face of which is exposed to the arterial dissolved nitrogen tension. For all diffusion-like processes the quantity of gas diffusing into the slab, cylinder or sphere is proportional to the square

root of the time for small values of time. For example, for a homogeneous slab of tissue at constant temperature it is estimated that the square root relationship would be correct to within 5% accuracy for approximately 100 min. Thus if, after exposure to pressure P_1 for time t_1, a quantity of gas Q_1 has diffused into the tissue, then

$$Q_1 = P_1\sqrt{t_1}$$

and after exposure to P_2 for t_2

$$Q_2 = P_2\sqrt{t_2}$$

Now according to the fifth assumption a certain critical excess quantity of gas can be tolerated without causing decompression sickness. If no stops are made (i.e. minimal decompression) it follows that all such safe exposures must not exceed some safe excess quantity on surfacing, say Q_c. So, for two different exposures, where the man reaches atmospheric pressure and just avoids decompression sickness

$$Q_c = Q_1 = Q_2 = P_1\sqrt{t_1} = P_2\sqrt{t_2}$$

thus

$$\frac{P_1}{P_2} = \sqrt{\frac{t_2}{t_1}}$$

Now when $P_1 = 100$ ft sea water pressure, t_1 is taken as 22 min, therefore

$$\frac{100}{P_2} = \sqrt{\frac{t_2}{22}}$$

or

$$P_2\sqrt{t_2} = 475$$

This relationship may now be tested for all times of exposure less than 100 min. Taking the extremes first, let $t_2 = 100$ min, then P_2 is 47·5 ft, i.e. the vast majority of men should be able to stay 100 min at 47·5 ft under the sea and then surface without decompression. This would be generally accepted as quite safe.

Now suppose $P_2 = 200$ ft, then

$$t_2 = (475/200)^2 = (2\cdot375)^2 = 5\cdot6 \text{ min.}$$

Again, a dive of $5\frac{1}{2}$ min to 200 ft followed by direct surfacing would be considered very unlikely to cause bends. The effectiveness of this approach in this type of diving is therefore beyond doubt, and it becomes clear that certain simple relationships can give quite satisfactory answers. Unfortunately, for times longer than 100 min, it is necessary to abandon the square

root relationship and use the accurate diffusion equation. The principles used for finding the quantity of dissolved gas in the tissue are also relatively simple but require a greater exemplification.

The graphical method of obtaining an answer to the exact solution of the diffusion situation is due to Rashbass, and is as follows. In order to calculate decompression tables, it is necessary to be able to calculate the quantity of nitrogen in the tissue at all stages during the decompression. In general this means being able to calculate the quantity of nitrogen in the tissue following changes in blood concentration from conditions of non-equilibrium. The rate of nitrogen transfer not only depends on how much nitrogen there is in the tissue but also on how it is distributed. An example may help to make this more clear. Consider the tissue when the diver has been at 30 ft (1·9 ATA) for a long time, for instance 3 days. Equilibrium will have then been reached and the nitrogen tensions in the blood and tissue will be uniform, and a certain quantity (30 ft worth) of nitrogen will be in the tissue. If the diver stays where he is nitrogen will neither enter nor leave the tissue (Fig. 13.1).

Now consider the tissue when the diver has been at 300 ft (10 ATA) for a short time (about $2\frac{1}{2}$ min). In this time the same number of molecules, i.e. 30 ft worth of nitrogen, may have entered the tissue but will be crowded near the capillary wall at high tension and be spreading only slowly deeper into the tissue. If the diver now ascends to 30 ft (1·9 ATA) the nitrogen, due to its higher tension, will immediately begin to leave the tissue and the total quantity of nitrogen in the tissue will therefore fall to less than 30 ft worth. Ultimately, if the diver remains at 30 ft, nitrogen will again start entering the tissue from the blood as it soaks into the remote recesses of the tissue (Fig. 13.1).

The problem to be solved can now be stated. Given that the time-course of the concentration of nitrogen in the blood is known (and it is justifiable to consider that the blood is always in equilibrium with the air which is being breathed and therefore the blood nitrogen concentration is proportional to the absolute depth of the diver), then what will be the time-course of the quantity of nitrogen in the tissue?

The solution

Except in very special cases the solution in algebraic terms is unmanageable. The solution proposed here is graphical.

The time-course of the quantity of nitrogen in the tissue following a step-function in the blood concentration when the blood and tissue were initially in equilibrium is taken to approximate closely enough to the solution of Hill (1928). This solution includes the thickness and permeability of the slab of tissue as constants which in this case are unknown. However,
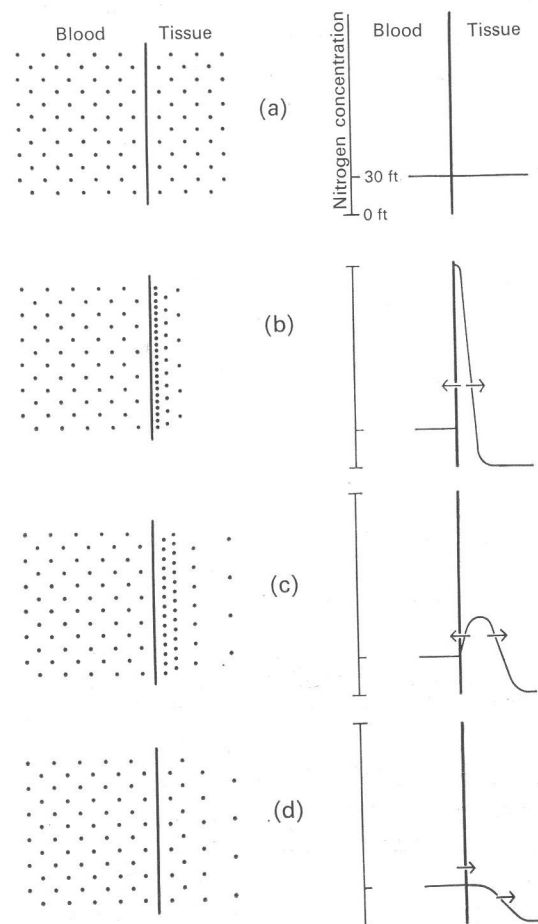
FIG. 13.1. Diagrammatic representation of the quantities of inert gas present in a sample of capillary blood and adjacent tissue

(a) When saturated at 30 ft; (b) on arrival at 30 ft after a short excursion to 300 ft from the surface; (c) shortly after this arrival at 30 ft; (d) having been at 30 ft for a longer time.

this expression can be reduced to a form involving only one arbitrary constant. If the quantity of nitrogen in the tissue be expressed in 'foot's worth' and the diver at time zero rapidly descends 1 ft, the blood concentration will perform a unit step function. The quantity of extra nitrogen which will enter the tissue at any time t will be

$$1 - \frac{8}{\Pi^2}\left[e^{-Kt} + \frac{1}{9}e^{-9Kt} + \frac{1}{25}e^{-25Kt} + \cdots\right]$$

The value of K has been adjusted to suit the known safe direct ascent durations and has a value of 0·007928 when t is measured in minutes. This function will be referred to henceforth as $\phi(t)$. In Fig. 13.2 are diagrammatically represented the concentrations of nitrogen in the blood and tissue in such a case and this explains the terms used. It will be seen that as $t \to \infty$, $\phi(t) \to 1$.
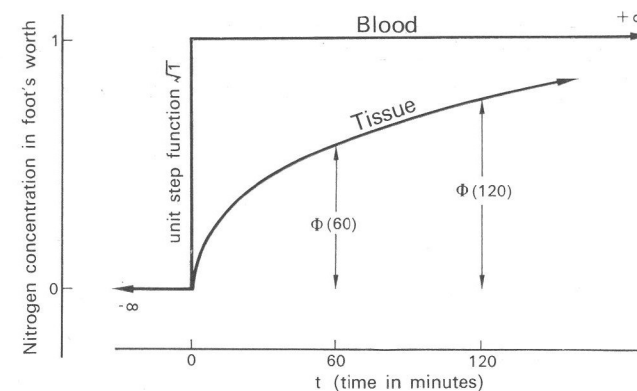

FIG. 13.2. Graphical representation of the change of nitrogen concentration in the blood and tissues following a descent of 1 ft

In general, diving consists of a series of step-functions because the time spent during ascent and descent is small compared with the time spent at each depth. This is the easiest case to consider and so the solution for that will be given first. From that solution it will be possible to deduce the effect of linear rates of ascent and descent and hence to investigate the lack of precision that has been introduced by assuming the changes of depth to be instantaneous. Finally, for completeness, it will be shown how the quantity of nitrogen in the tissue could be calculated for any type of diving procedure.

The graphical solution depends entirely on the principle of superposition. This can be stated formally as follows. If a certain dive which has the time-course $D_1(t)$ produces in the tissue a quantity of nitrogen with the time-course $F_1(t)$ and another dive having the time-course $D_2(t)$ produces in the tissue a quantity of nitrogen with the time-course $F_2(t)$, then the dive which is at all times at a depth equal to the algebraic sum of the depths of the two dives thereby having a time-course $D_1(t) + D_2(t)$ will produce in the tissue a quantity of nitrogen which is at all times the algebraic sum of the quantities of nitrogen produced by the separate dives, i.e. $F_1(t) + F_2(t)$. This follows mathematically from the formulation of Fick's law because

superposition applies to all first order differential equations. It is physically readily understandable because in diffusion processes the movement of any molecule of the diffusing substance is independent of any other molecule of the same sort. Hence the effect of the two dives D_1 and D_2 can be visualized as being superimposed on one another.

Solutions of special cases. In all these cases it is assumed that the tissue is in equilibrium at the outset.

(1) Step-function dive of depth N feet, $\underline{}N$.

It follows immediately from the principle of superposition that since a $\underline{1}$ dive produces $\phi(t)$ foot's worth of nitrogen in the tissue, a $\underline{2}$ dive will produce $2\phi(t)$ foot's worth of nitrogen (Fig. 13.3). It follows that a
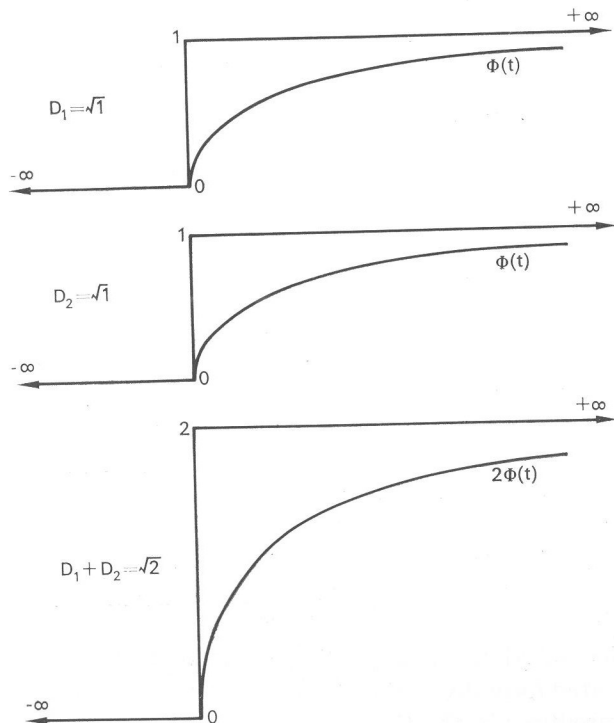

FIG. 13.3. Graphical representation of superposition

step-function dive of depth N feet will produce $N\phi(t)$ foot's worth of nitrogen in the tissue for any value of N. As $t \to \infty$, $N\phi(t) \to N$ which is merely a restatement of Henry's law of gaseous solubilities.

(2) A step-function dive of depth $-N$ feet, $\underline{}-N$.

Again it is obvious that the tissue of a diver coming instantaneously a

distance N feet upwards will lose nitrogen at the same rate as it would have gained nitrogen had he gone N feet downwards.

(3) A step-function dive of depth N feet followed after an interval of T minutes by a step-function dive of $-N$ feet.

The solution of this case is seen in Fig. 13.4, which shows how to calculate the time course of the quantity of nitrogen in the tissue during and following a dive to N feet and returning straight to surface after a stay of T minutes. During the time that the diver is on the bottom the quantity of nitrogen in the tissue will rise along the curve AB which is the beginning of the curve $N\phi(t)$. After surfacing, the quantity of nitrogen will fall along
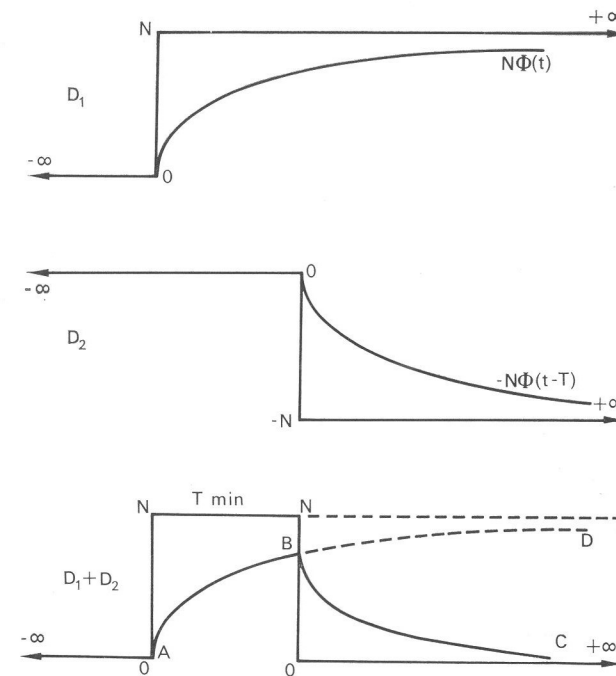

FIG. 13.4. Graphical representation of gas elimination

the curve BC which is the algebraic sum of the two curves $N\phi(t)$ and $-N\phi(t - T)$. This would be obtained in practice by plotting downwards from the dashed curve BD the ordinates of the curve $N\phi(t - T)$ which is the same curve as $N\phi(t)$ shifted T minutes along to the right. It will help to clarify and justify this procedure if consideration be given to an example. Suppose that at time $t = 0$ the diver be compressed to 100 ft (4 ATA), then the quantity of nitrogen in the tissue will be represented at subsequent times by the curve ABD eventually reaching saturation with

100 ft worth of nitrogen. Suppose now that 30 min later all the extra air which was put into the pressure chamber to raise the pressure to 100 ft (4 ATA) be replaced instantaneously by different air, the nitrogen molecules of which were identifiable (as some hypothetical radio-isotope) and called N_2^*. Now, the total quantity of nitrogen in the tissue will continue to accumulate along the curve ABD because no change has been made in the external total nitrogen concentration. If however, the N_2^* only is considered, then the diver has in effect dived to 100 ft of N_2^*, at the instant of the change over, and N_2^* will accumulate in the tissue along the same curve as the total N_2 ($N_2 + N_2^*$) but starting 30 min later. From that time onwards the quantity of N_2 in the tissue will be the total nitrogen content ($N_2 + N_2^*$) less that part of it which is N_2^*, i.e. the curve BC. However, from the point of view of the N_2 the diver surfaced at the moment of change over. Therefore the curve BC is that which represents the quantity of nitrogen which would be in the tissue following a dive of 30 min at 100 ft.

(4) A step-function of depth N feet followed after an interval of T minutes by a step-function dive of $-M$ feet.

This represents the case of a diver having spent T minutes at a bottom depth of N feet and having come up to his first stop M feet from the bottom. The solution is seen in Fig. 13.5.
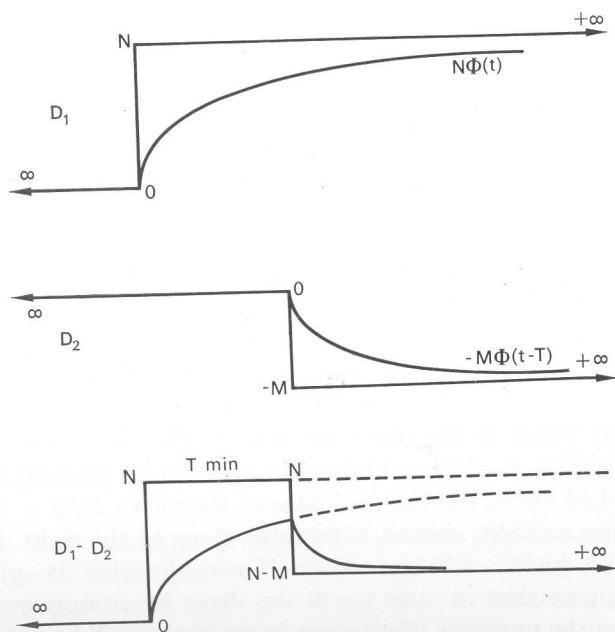

FIG. 13.5. Graphical representation of gas elimination following incomplete ascent, i.e. to first stop

A special case in this situation arises when the quantity of nitrogen in the tissue, at the moment of leaving the bottom, is precisely that quantity which would be in the tissue if saturated at the depth of the first stop. Fig. 13.6 shows the resulting construction.
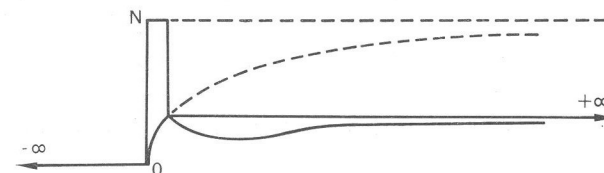

FIG. 13.6. Graphical representation of gas elimination following incomplete ascent in the special case where the quantity of nitrogen in the tissue, at the moment of leaving bottom, is precisely that quantity which would be in the tissue if saturated at the depth of the first stop

This is the solution of the situation illustrated in Fig. 13.1 and shows that in such a case nitrogen first leaves and later re-enters the tissue as was to be expected.

(5) A step-function of depth N feet followed after an interval of T minutes by a step-function dive of $-M$ feet and after another S minutes a further step-function dive of $-L$ feet.

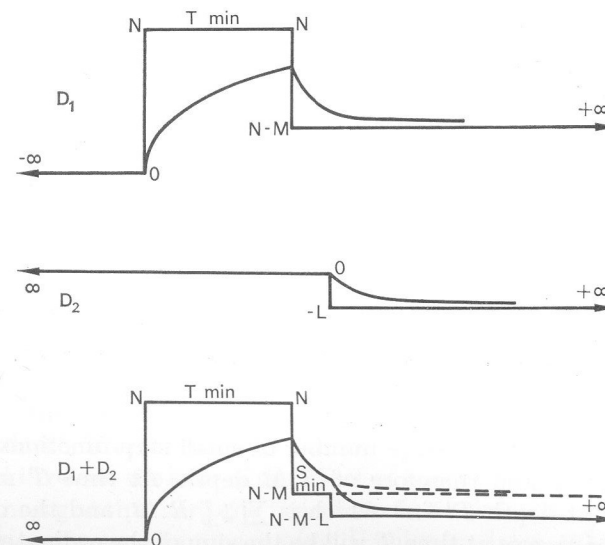This represents the time-course of the quantity of nitrogen in the tissue

FIG. 13.7. Graphical representation of the time-course of nitrogen elimination including the second stop

after the diver has left his first stop and is at his second stop. D_1 is now the curve representing events at the first stop and is derived by the process of Fig. 13.5. D_2 is $-L[t - (T + S)]$ and the solution is the algebraic sum of these two curves (Fig. 13.7).

These examples have shown how to manipulate the curves for dives which can be considered as step-functions, i.e. for which rates of ascent and descent are infinitely rapid. It is now intended to investigate the effect of a finite rate of descent. Consider a diver, initially at equilibrium on the surface descending at a uniform rate of R ft/min (Fig. 13.8). This can be con-
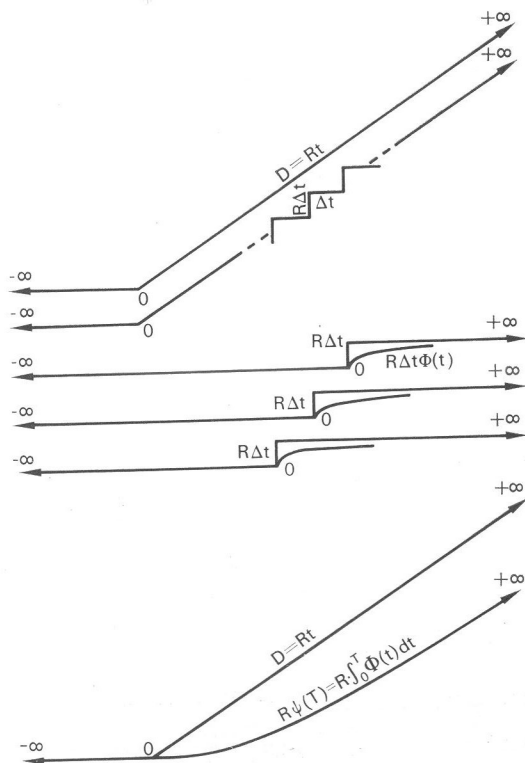

FIG. 13.8. Graphical representation of a finite rate of descent

sidered as consisting of a large number of small step-functions each of Δt minutes duration and therefore of $R.\Delta t$ depth. At time T minutes the diver will be at depth Rt feet which is $\sum_{t=0}^{t=T} R.\Delta t$, and the quantity of nitrogen in the tissues at time T will be the sum of the ordinates at time T of all the 'tissue' curves corresponding to all the steps which have so far been taken up. The ordinate at time T of the step taken at time t will be

$[R.\Delta t].\phi(T - t)$ and the sum of all such steps will be $\sum_{t=0}^{t=T} R.\Delta t.\phi(T - t)$. As $\Delta t \to 0$ this sum approaches the integral $R \int_0^T \phi(t).dt$ which we can call $R.\psi(T)$. The required tissue nitrogen uptake curve in the case of a uniform rate of descent is therefore in the case of a step-function the integral of the uptake curve. This is to be expected, because a linear descent $D = Rt$ is obtained from the step-function $D = R$ by integration with respect to time and since the processes involved in integration are ones of addition
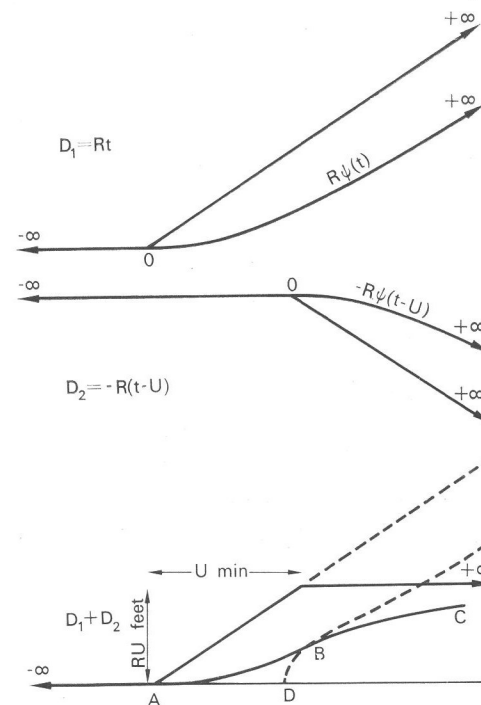

FIG. 13.9. Graphical representation of the effect of a linear descent which terminates when the desired depth has been reached

only, the same processes should be applied to $\phi(t)$ to obtain the tissue nitrogen content. Thus there is now a curve of nitrogen uptake by the tissue for a linear rate of infinitely prolonged descent.

In practice there is interest in a linear descent which terminates when the desired depth is reached. This can be obtained by the procedure of Fig. 13.9 and involves the sum of the effect of an infinitely prolonged linear descent beginning at time zero and the effect of an infinitely prolonged linear ascent at the same rate but beginning U minutes later, where U is the duration of the descent. The curve AB is therefore the beginning of the integral $R.\psi(t)$ and the curve BC is the difference between $R.\psi(t)$ and

$R.\psi(t - U)$; the depth of the dive is $N = RU$ feet therefore $R = N/U$. As $U \to 0$ the dive becomes a step-function of depth N feet and the curve BC becomes $(N/U)[\psi(t) - \psi(t = U)]$ which when $U \to 0$ is $N.(d/dt).\psi(t)$ or $N\phi(t)$ as is already known.

In practice, graphical integration is performed by the summation of ordinates of the integrand at small abscissa intervals. In this present problem 1 min would be considered sufficiently short compared with the times with which one is dealing and hence, for ascents and descents lasting less than 1 min, the curve BC is identical with $\phi(t)$. The effect of ascents and descents lasting as long as 3 min have been investigated and the resulting curve BC differs negligibly from the curve $\phi(t)$. For all practical diving, nitrogen uptake can therefore be considered in terms of step-function dives. The instant of the effective step-function will be at the point D (Fig. 13.9) which is obtained by extrapolating the curve BC backwards as though it were the curve $\phi(t)$. This instant occurs at some time during the descent and can be ascertained. In the calculation of the tables it has been taken to be either at the beginning or the end of the ascent or descent according to which gives the error on the safe side.

All the above special cases lead to the solution of the general case of a diver, initially in equilibrium on the surface at time zero, who then dives to any depth by any time-course. Let the depth of the diver at time t be represented by the function $f(t)$ (Fig. 13.10) and it is required to find the quantity of nitrogen in the tissue at time $t = T$ min.
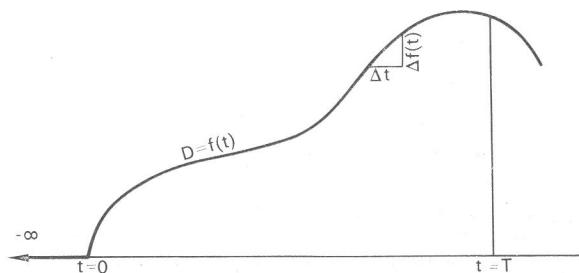

FIG. 13.10. Graphical representation of the solution of the general case whereby the diver dives to any depth by any time-course

The dive can be considered as consisting of elemental step-functions each of duration Δt and depth $\Delta f(t)$. The quantity of nitrogen in the tissue at $t = T$ is the sum of the ordinates of the 'tissue' curves for each of these elemental step-functions. These will have the value $\Delta f(t).\phi(T - t)$ and this sum will be $\sum_{t=0}^{t=T} \Delta f(t).(T - t)$. As $\Delta t \to 0$ this approaches $\int_0^T f^1(t).\phi(T - t).dt$ where $f^1(t) = [d.f(t)]/dt$. This can be evaluated either graphically or numerically.

The calculation of the tables

In order to calculate tables one other piece of information is required and that is how much nitrogen is permitted in the tissue at any ambient pressure without symptoms arising? Haldane (Boycott et al. 1908) introduced the concept of a ratio of nitrogen pressure in the tissues to nitrogen pressure in the lungs and this idea has dominated most investigations into the problem. The safe ratios for various tissues have been readjusted (Hawkins, Shilling & Hansen 1935; Van der Aue, Kellar, Brinton, Barron, Gillian & Jones 1951) to suit the observed occurrence of decompression sickness in divers. Investigations by Newton Harvey (1951) on the occurrence of detectable bubbles would indicate that in his experiments the excess tissue pressure necessary to cause bubbles was not independent of the ambient pressure. Behnke (1951) writes 'Efforts to use a difference in pressure ΔP have not been translated into working out successful decompression procedures. . . . A fixed ratio does not appear to hold, and, on the other hand, the concept of a constant value for ΔP may require the inclusion of some correction factor for pressure level.'

An experiment to investigate this point was performed with rats in which the depth of a half-hour minimal-decompression dive which produced a 50% death rate was determined. The experiment was then repeated at an ambient pressure of twice normal atmospheric pressure by taking the rats, the rat pressure chambers and the experimenters down to the equivalent of 33 ft of sea water. On returning to the surface (1 ATA) 50% of rats died if they had been subjected for $\frac{1}{2}$ hour to a pressure of 9·5 ATA, whereas returning to 2 ATA about 50% died only after they had been subjected to a pressure of 16·3 ATA. This result is certainly not consistent with a constant pressure difference although it suggests that even a ratio tends to diminish with increasing depth.

Accordingly it was decided arbitrarily to assume that it would be safe to be at a depth of D feet if the tissue nitrogen did not exceed $(30 + 1\cdot6D)$ ft worth. A few preliminary decompression schedules were calculated and tested and it soon became clear that they were unsafe for rather severe dives such as 180 ft (6·4 ATA) for 45 min. These tables were therefore abandoned and new ones calculated on the basis of a constant pressure difference (i.e. that it would be safe to be at depth D feet if the tissue nitrogen did not exceed $(30 + D)$ ft worth). Although having simplicity in its favour, this assumption needs some justification in the face of the evidence to the contrary. It is tentatively suggested that this justification can be found by considering decompression sickness as occurring in either 'tight' or 'loose' tissues as suggested by Nims (1951).

Most of the evidence for a ratio derives from observations in which

'loose' tissue bubbles are responsible for the signs. In a 'loose' tissue a bubble grows relatively unhampered and its effect is determined by its maximum volume. This bubble volume is a function of the ambient pressure, higher ambient pressures requiring a higher tissue nitrogen tension surplus than lower ambient pressures in order to produce equal sized, and therefore equally effective, bubbles. The pain of 'bends' on the other hand, probably occurs in a 'tight' tissue and pain is felt when the pressure in the bubble reaches some constant value in excess of the ambient pressure. In a 'tight' tissue the internal bubble pressure tends to rise until it is equal to the tissue nitrogen tension and therefore pain might be expected to occur when the tissue nitrogen exceeds the ambient pressure by a constant amount.

The tables which are being proposed here were calculated on this assumption. Before presenting them a detailed description will be given of the calculations involved in a single example, 90 ft (3·7 ATA) for 70 min (references are to Fig. 13.11 which is drawn to scale).

Depth of the first stop. $90\phi(t)$ is plotted in foot's worth against minutes and would ultimately become horizontal at 90 foot's worth. This is curve
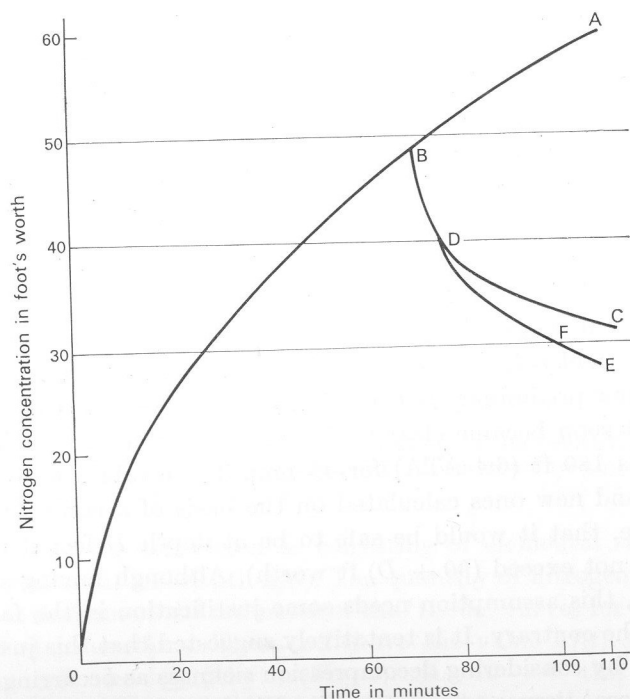

FIG. 13.11. The graphical solution of a 90-ft dive for 70 min

OA. The curve is continued for a duration which is estimated to be the total time for the dive from leaving surface to return to surface. In this case 70 min are to be spent on the bottom and 30 to 40 min are expected to be occupied in returning to surface, the curve being plotted up to 110 min. It is seen that after 70 min on the bottom some 48 ft worth of nitrogen will have been acquired. The first stop will therefore be at 20 ft because it is not considered safe to come up to 10 ft if the tissue nitrogen exceeds 40 ft worth whereas it is considered safe to come up to 20 ft so long as tissue nitrogen does not exceed 50 ft worth.

Time of ascent to first stop. It was not possible to lay down in practice a rigid rule about rate of ascent for air divers who control their own ascents by 'spindling' up a shot rope. These divers will always come upwards as quickly as possible and it was decided that the tables should be calculated and tested on the assumption that the rate of ascent would be 2 ft/sec. This time for ascent to the first stop is added to the time on the bottom for purposes of calculating the tissue nitrogen concentration in accordance with the principle that the ascent and descents are considered, according to which errs towards safety, as having occurred at the beginning or end of the actual ascent or descent.

Duration at the first stop. A step-function dive of −70 ft (3·1 ATA) is therefore said to begin at time 71 min and the tissue nitrogen is plotted as in Fig. 13.5. At 73 min a point is plotted downwards from OA a distance $\frac{7}{9}$ of the ordinate of curve OA at time 2 min ($\frac{7}{9}$ is the factor because curve OA is $90\phi(t)$ and now $70\phi(t)$ is required). At 75 min a point is plotted downward from OA a distance $\frac{7}{9}$ of the ordinate of curve OA at 4 min. This process is continued and the points joined to give the curve BC which is the tissue nitrogen concentration so long as the diver remains at 20 ft (1·6 ATA). This curve is found to cross the 40 ft worth level at $76\frac{1}{2}$ min. The first stop will therefore be 7 min at 20 ft. (For estimating the duration of the stop the ascent is assumed to have taken place at 70 min, again erring on the side of safety, and the time at the stop is taken to the next larger whole minute.) This can now be entered into the appropriate column of the diving table.

Subsequent ascents. At the point D the diver will come up from 20 to 10 ft (1·6 to 1·3 ATA) making a step-function dive of −10 ft. The duration of the ascent is a matter of seconds and no adjustment is required for this. Two minutes to the right of point D plot downwards from BDC a point $\frac{1}{9}$ the ordinate of curve OA at time 2 min. Four minutes to the right of point D plot downwards from BDC a point $\frac{1}{9}$ the ordinate of the curve OA at time 4 min. The factor, $\frac{1}{9}$, is because we are now considering an ascent of 10 ft (1·3 ATA) compared with the original dive of 90 ft (3·7 ATA). On joining these points the curve DE is obtained. This curve is found to

cross the 30 ft worth level at F at time 99 min and the diver is then safe to surface. The duration of the 10-ft stop is therefore 99 less $76\frac{1}{2}$ which (to the next larger whole minute) is 23 min which is entered in the table.

With experience the work can be abbreviated by plotting only those parts of the curves which are relevant.

By using this particular method of calculation, all the complexities of deciding which tissues participate in the manifestations of decompression sickness are ignored. Only one mathematical equation is needed, and this can be reduced to a simple square root relationship for all except prolonged exposures.

Over 1200 dives in dry chambers were performed using this method of calculation. Most dives were reasonable tests of the adequacy of the procedure, for instance, 45 min at 180 ft (6·4 ATA), 80 min at 100 ft (4 ATA). Only one minor bend occurred. However, on being tested in the sea, the first 56 dives resulted in 7 cases of decompression sickness. This was unacceptable and some re-appraisal became necessary. The difficulty of transferring schedules from the dry chamber into sea-going conditions are not always fully appreciated. Success in the dry chamber is encouraging but for some reason, this by no means ensures subsequent success in the sea. No decompression procedures are convincing until successfully performed in the practical circumstances required, in the tunnel or caisson for compressed air workers or in the sea for divers.

Irreversibility

Anyone who has taken part in experimental diving realizes that a dive can result in a persistent low-level pain ('niggle') in a joint, and that such a niggle can cause intermittent trouble over a period of two or three days. An exaggerated example is provided by the Ocean Systems diver who was still sensitive to flight in an unpressurized aircraft several days after completion of his saturation helium dive (Hamilton et al. 1966). On conventional Haldane principles it would be necessary to assign a half-time to the tissue involved and this would clearly result in some quite absurd value. This dilemma is solved by inferring that in such cases there is a bubble-tissue complex and that the half-time of such a situation is not representative of the half-time of the tissue when the bubble is not occluding the circulation. Once this concept of a bubble-tissue complex is accepted grave doubts arise about whether or not such bubble-tissue complexes are not also, to a lesser degree, influencing the whole decompression problem.

In a double approach to this problem, the author considered that he had succeeded in showing that in normal diving procedures the rate of acquisition of gas was not the same as the rate of loss of gas. It is necessary to emphasize the phrase 'in normal diving procedures' where large initial

pressure changes are involved. It may be however possible by such techniques as are being advocated by Behnke (1967) and Hills (1966) to avoid the formation of a gas phase.

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

(1) Expose for 6 hours to pressure P_1.

(2) Decompress rapidly to P_2, such that no decompression sickness occurs. A trouble-free decompression ratio $r = P_1/P_2$ is thus established.

(3) Wait 6 hours at P_2 to equilibrate all tissues to the new pressure level.

(4) Decompress to P_3, such that

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

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

Proof that a physical change in the body has occurred as a result of decompression is not given by the above experiments. The results could have been obtained from a change in physiological responses caused by, for instance, breathing raised pressures of air for such prolonged periods. In order to attempt to investigate the physics and physiology of this situation a second entirely different series of experiments was performed. In outline these experiments were as follows

(1) The decompression sickness threshold pressures were obtained for several animals. They were rapidly compressed to pressure P for a time t, at the end of time t they were decompressed back to atmospheric pressure in 150 sec. Certain fixed values of t were selected and the P values were obtained which gave mild threshold 'bends' on return to atmospheric

pressure. Assume now that there exists a set of tissues with half-times 5, 10, 20, 40, 80 min. The choice of these particular tissues is purely arbitrary but this does not affect the validity of the subsequent reasoning. It is now possible, as a result of performing a range of exposure times, to assign permitted ratios to each of these hypothetical tissues for each of the animals. As the threshold 'bends' are all obtained at atmospheric pressure the possibility of altered physiological factors interfering with the experimental results is eliminated.

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

The second exposure consisted, as before, of a rapid pressurization to some pressure P_2 and a stay at this pressure for 25 min, followed by decompression back to atmospheric pressure. P_2 is the pressure at which a threshold 'bend' just occurs when the animal returns to atmospheric pressure for the second time. It is now quite easy to calculate the excess gas present in the various tissues at the time of reaching atmospheric pressure following this double pressure excursion. The 'bend' threshold values for the various tissues for the single and double dives can now be compared as in Table 13.1 where goat 34 is given as a typical example of this experimental series. Here the 5-min tissue had shown in the dive (A) of 25 min to 130 ft (4·9 ATA) that it was capable of performing a decompression ratio of 4·81 on return to atmospheric pressure. Anything less than a 4·81 ratio should not affect this tissue, as these values represent its threshold performance. Examination of the double dive data reveals that this particular tissue only performed a 4·08 ratio drop. This difference is very great and must mean that the 5-min tissue played no part whatsoever in the bend produced by the double dive.

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

TABLE 13.1

Examples of bend threshold experiments

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

ated. Six other goats were used in these experiments. Each tissue half-time on every goat showed disparities similar to the example given above. Thus it is quite impossible to use the same reasoning for the uptake of the gas as for the elimination. It is proposed that a tissue-bubble complex is formed during decompression. The sole question remaining is whether such a complex situation forms from every decompression, or whether there are ways of avoiding this. It is considered that the approach of Hills (1966), where one considers the worst possible case in which there is always gas separation, represents the safest practical solution unless other experimental evidence can be produced.

The graphical solution to the uptake and elimination of gas in a strictly reversible tissue gas exchange situation has been given earlier. The accuracy of solution given by this method is of course related to the accuracy with which the relevant graphs are drawn. This has led to difficulties and has long since been abandoned in favour of using accurate numerical solutions to the diffusion equation but employing the same reasoning of the superposition of positive and negative pressure changes. A further change has also been instituted since it became clear that the rate of uptake of gas is not the same as its elimination and when it also became clear from sea trials that this simple approach would not guarantee diving without decompression sickness.

At the moment of decompression it is assumed there is a change in the rate of gas exchange and a slower rate is employed for the removal of gas than for the uptake. The curve used has the same shape, i.e. a solution to the diffusion equation, but the time required to reach a particular percentage saturation is increased by one-half when compared with the uptake curve. Using these methods of numerical addition and subtraction of diffusion-like gas exchange curves combined with the concept of a 30 ft worth permissible tissue gas content in excess of ambient pressure the present Royal Navy Diving Tables were calculated. The times of the stoppages were given to the nearest 5-min increment to further protect the diver.

The supposition that a fixed ΔP value could be used as a sensible limiting condition for all forms of diving was clearly not correct. To take the Rashbass idea of $\Delta P = 30$ ft, this would imply that at atmospheric pressure it should be possible to decompress to 33 less 30, i.e. 3 ft pressure absolute (1·0 less 0·9, i.e. 0·1 ATA). This would be a rapidly fatal experiment for human subjects. Again, after a prolonged dive to 100 ft (4 ATA) it is certainly permissible to decompress quickly and safely to a value much nearer atmospheric pressure than 100 less 30, i.e. 70 ft (4 less 0·9, i.e. 3·1 ATA). Thus the fixed ΔP concept is dangerous with low ambient pressures, and grossly oversafe with high exposure pressures. A return to

the Haldane ratio principle is necessary to rationalize the calculations, and with the most recent decompression procedures used by the caisson and tunnel workers this has been done. All quantities of gas in the hypothetical tissue are calculated using the principles given earlier for the R.N. diving tables, but instead of using a fixed ΔP value for the limiting condition a variable decompression ratio is employed, calculated as follows:

$$r \text{ (ratio)} = \frac{400}{P + 180}$$

P = absolute pressure in pounds per square inch at which a saturation exposure has occurred.

Thus at atmospheric pressure, approximately 15 psi absolute pressure, the permitted ratio is 400/195 = 2·05.

At 100 ft (4 ATA), approximately 60 psi absolute, the ratio is 400/240 = 1·67.

The formula is obviously only applicable over a certain pressure range, because at 220 psi absolute (460 ft; 14·9 ATA) the ratio becomes 1·0 and no decompression would be possible. A significant exposure at such pressures using air is not possible in the present state of knowledge, and therefore this limitation is purely theoretical.

Decompression procedures for compressed air workers have been calculated using these principles (i.e. a relatively rapid uptake of gas, a decompression ratio which varies with the amount of gas present in the hypothetical tissue, and a relatively slow elimination of gas) with the calculation of tissue gas quantities based upon the superposition of positive and negative pressure excursions and using the iterative, non-graphical, accurate solution for the diffusion into a tissue slab. These calculations have also been fitted into the 'stage' method of decompression. Some 40,000 exposures using these schedules at pressures ranging between 35 psi g (79 ft; 3·4 ATA) and 42 psi g (95 ft; 3·8 ATA), for periods at pressure varying between 4 hours and 12 hours have resulted in nearly 1·0% therapeutic recompressions. Unfortunately, outside the United Kingdom comparable reliable data does not appear to exist for such long periods at such relatively high pressures. With this experience in the United Kingdom, it may be said that from the view of 'bends' incidence these results are very encouraging. The really important point is whether the incidence of necrosis of bone will be reduced to negligible proportions by these procedures but this will not be established until the bone radiographic survey has continued for some time.

HELIUM DIVING

As with air diving, the knowledge necessary for a complete understanding of the problems concerned with helium diving is not available. One must

resort to crudely established data and examine whether a theoretical frame-work can be constructed which will permit safe deep diving, even though it may be realized that the predicted procedures are probably not the most economical of decompression time.

The first unknown concerns the rate at which helium saturates the relevant tissue or tissues of the body. If one supposes that the tissues concerned are perfusion limited then, when comparing the rate of helium saturation with the better known rate of nitrogen saturation, the following becomes clear. The fatty tissues are mainly responsible for the slow components of the whole body nitrogen saturation curve (Behnke et al. 1941). Helium is much less soluble in fatty tissue than nitrogen and therefore the 'slow' component of the whole body nitrogen curve will be absent. On these ideas only a very poorly perfused tissue with a blood/tissue solubility ratio which did not alter with a change of gases could be responsible for any slow helium component to body uptake. If one introduces ideas that diffusion plays a role in the uptake and distribution of inert gases, then the predictions become even more defined.

Thus, it is expected that helium will saturate all body tissues very much more rapidly than nitrogen. The sole exception being the possibility of a small contribution from a poorly perfused tissue which has solubility properties as stated above. In order to resolve this situation and also to render the data more quantitative, a series of goat experiments have been performed which show that, for the tissues concerned in decompression sickness, helium effectively saturates approximately three times more rapidly than nitrogen (Hempleman 1967). This finding does not seem to agree with some human work (Duffner 1960) and therefore recently the experiments have been repeated on eight human volunteers.

Following an exposure of 24 hours' duration breathing 20% oxygen, 80% helium it was noted that a very transient 'niggle' occured at 45 ft (2·4 ATA) in only one man. At 50 ft (2·5 ATA) for 24 hours a minor 'bend' occurred and it was decided that 50 ft was the depth for onset of decompression sickness following a 24-hour exposure. The time of exposure was then reduced to 6 hours and again mild bends were obtained at 50 ft (2·5 ATA). There is therefore no detectable difference, on this mixture and at these depths, between 6-hour and 24-hour exposures. Recent animal experiments have revealed the important part that the oxygen partial pressure can play in experiments designed to discover tissue half-times. Breathing 20% oxygen, 80% helium at 132 ft (5 ATA) gives an oxygen partial pressure of 1 Atmosphere which certainly causes some vasoconstriction and hence a probable delay in inert gas uptake. This will alter the conclusions on the rapidity with which helium or nitrogen saturates or desaturates the tissues.

It is possible, using the principles enunciated previously, to calculate successful schedules for helium diving. In fact at this laboratory simulated dives to such depths as 600 ft (19 ATA) and 800 ft (25 ATA) are commonplace and quite trouble-free except for occasional transient 'niggles'. This does not mean that any pretence is made that the problem is even partially understood.

A great deal of this work has been of a trial and error nature in order to establish decompression ratios. There is a growing conviction that the Haldane 'stage' method of decompression is in practice more desirable and just as economical in decompression time as the continuous 'bleed' procedures. Finally it must be stated that although the simulated dives are quite trouble-free, the same is not true for the corresponding sea dives. At sea the physical, physiological and psychological aspects of the experiments change unavoidably. However, because only helium mixtures are used throughout the decompression any therapeutic procedures needed are relatively simple and very successful (Barnard 1967). Thus failures in the decompression procedures are now only regarded as annoying, and not as major stumbling-blocks to further work.

REFERENCES

Barnard, E. E. P. (1967). The treatment of decompression sickness developing at extreme pressures. In *Proceedings 3rd Underwater Physiology Symposium*. Ed. Lambertsen, C. J. Baltimore: Williams & Wilkins.

Behnke, A. R. (1951). In *Decompression Sickness*. Ed. Fulton, J. F., p. 80. London and Philadephia: Saunders.

Behnke, A. R. (1967). The isobaric (oxygen window) principle of decompression. *In The New Thrust Seaward*. Trans. 3rd Ann. Conf. & Exib. Washington: Marine Technology Society.

Behnke, A. R. & Willmon, T. L. (1941). Gaseous nitrogen and helium elimination from the body during rest and exercise. *Am. J. Physiol.* **131**, 619–638.

Boycott, A. E., Damant, G. C. C. & Haldane, J. S. (1908). The prevention of compressed air illness. *J. Hyg., London.* **8**, 342–443.

Duffner, C. J. & Spider, H. H. (1958). Effects of exposing men to compressed air and helium-oxygen mixtures for 12 hours at pressures of 2–2·6 atmospheres. *Research report. 1–59*. U.S. Navy Experimental Diving Unit, Washington, D.C.

Golding, F. C., Griffiths, P. D., Hempleman, H. V., Paton, W. D. M. & Walder D. N. (1960). Decompression sickness during construction of the Dartford Tunnel. *Br. J. indust. Med.* **17**, 167–180.

Hamilton, R. W., MacInnis, J. B., Noble, A. D. & Schreiner, H. R. (1966). *Saturation Diving at 650 feet*. Ocean Systems Technical Memorandum B-411. Tonawanda, New York.

Harvey, Newton E. (1951). In *Decompression Sickness*. Ed. Fulton, J. F., pp. 90–144. London and Philadephia: Saunders.

Hawkins, J. A., Shilling, C. W. & Hansen, R. A. (1935). A suggested change in calculating decompression tables for diving. *U.S. nav. med. Bull.* **33**, 327–338.

Hempleman, H. V. (1952). *Investigation into the diving tables. Report 111*. Medical Research Council, R.N. Personnel Research Committee, U.P.S. 131.

Hempleman, H. V. (1962). In *Proceedings 2nd World Congress of Underwater Activities*, pp. 60–63: British Subaqua Club, London.

Hempleman, H. V. (1967). Decompression procedures for deep, open sea operations. In *Proceedings 3rd Underwater Physiology Symposium*. Ed. Lambertsen, C. J. Baltimore: Williams & Wilkins.

Hill, A. V. (1928). The diffusion of oxygen and lactic acid through tissues. *Proc. roy. Soc. B.* **104**, 39–96.