11

Some Early Studies of Decompression

A. R. BEHNKE

The introduction in 1819 of flexible, closed diving dress by Siebe, and the ingenious development of the caisson by the French engineer, Triger, radically changed the course of diving and aqueous tunnel operations (Hoff 1948). In the wake of these pioneering innovations arose a malady, ostensibly a *post hoc, propter hoc* phenomenon that has challenged the resources of physiologist, physicist and physician. A brief review will be presented of earlier efforts to solve the problems inherent in decompression of divers and tunnel workers from a pressurized work environment to normal atmospheric conditions. Quantitative approaches have been limited. The extensive physiological and pathological knowledge is largely empirical, and the sophisticated mathematical overlay is unsupported in depth by basic physical data.

In biogenetics, Haeckel postulated the well-known law, 'ontogeny recapitulates phylogeny': in diving, the experience of an investigator recapitulates knowledge accumulated by his predecessors. Rediscovery is a recurring phenomenon and an investigator of a past era may find it difficult to identify his contributions. Further, his data and conclusions may be evaluated not in the light of the scientific milieu of the period but as a contemporary effort enhanced as it is by advances in technique and enriched experience. It is a tribute, therefore, to the scientific stature of Haldane and his distinguished associates, that principles formulated in 1908 as a guide to decompression practice are currently operative. In 1965 Workman could state 'The procedure for calculation of decompression schedules given in this report is not new. It is based primarily on a method developed by Boycott, Damant, and Haldane in 1908.'

These statements should in no way obscure the concomitant achievements of Leonard Hill, J. Argyll Campbell and their associates. In many respects the approach to decompression problems and the findings of the

'two Schools' were complementary not contradictory. The work of Hill and Campbell reflects quantitative endeavour. In the standard monograph (Hill 1912) is a report of the first reliable analysis of nitrogen in blood and urine at increased pressures (Hill & Greenwood 1907). Later, Campbell, and Hill (1933) were to report the direct analysis of brain, liver and bone marrow of the goat. Had such an approach been systematically developed over the decades, we would now have answers to current critical problems.

In diving, the stage method of Haldane, with progressive increase in time at the shallower stops, has taken precedence over the uniform method supported by Hill. In tunnel decompression, by contrast, it is uniform decompression following initial reduction of pressure to a first stop that is standard. One observes in current decompression following saturation diving, that slow, uniform reduction in pressure to accommodate the slowest saturating tissue advocated by Hill, is the course followed. Essentially, the difference in applicability of the two systems is whether or not exposures are short or long, relatively shallow or deep. Current practice, therefore, has drawn heavily from the contributions of both schools.

ETIOLOGY OF DECOMPRESSION SICKNESS

It is apparent from the consummate treatises of Heller, Mager and von Schrötter (1900) and Hoff (1948) that the salient features of decompression sickness were established during the period from 1870 to 1910. As early as 1670, following the invention of the vacuum pump, Boyle described with minute accuracy bubbles in the blood and body fluids of small animals subjected to low pressures. He described essentially 'intravascular decompression': 'The little Bubbles . . . by choking up some passages, vitiating the figure of others, disturbe or hinder the due circulation of blood.'

Hoppe-Seyler (1857) described blockage of pulmonary vessels by nascent bubbles and the inability of the heart to propel blood under these conditions. He proposed recompression to absorb the liberated gas and re-establish circulation. In the conclusive experiments of Paul Bert (1878), gas bubbles liberated by rapid decompression were found to consist chiefly of nitrogen. He pointed out the value of recompression therapy combined with oxygen. Bert was cognizant of the role of fat in the production of symptoms. His thin small dog survived the usually fatal decompressions from 7 to 8 ATA (198 and 251 ft), but died when fattened and subjected to the previously tolerated conditions. The recognition of the importance of fat is noteworthy in the absence of solubility values of nitrogen in fat. Later Zuntz (1897) was to overlook the role of fat in his calculation of the time required for whole body equilibration with nitrogen.

One may observe in cutaneous blood vessels of anaesthetized dogs

rapidly decompressed from high pressures, bubbles that circulate from arteries into veins prior to the onset of respiratory symptoms (Behnke, Shaw, Messer, Thompson & Motley 1936). Subsequently these bubbles become sessile as blood flow ceases. Decompression sickness may well be the result of intravascular liberation as opposed to the respiratory elimination of inert gas. The complications associated with liberated gas are many and there are incongruities difficult to explain. From the point of view of decompression procedure however the problem is straightforward; namely to regulate decompression so as to prevent bubble evolution, or if nascent bubbles form, to control their number and size so that symptoms do not supervene.

PROBLEMS IN DECOMPRESSION TO ALTITUDE

Henderson, according to Fulton (1951), was the first physiologist in modern times to consider the possibility of occurrence of decompression sickness at altitude. The extensive and systematic tests in the low-pressure chamber prior to and during World War II were a boon to medical knowledge of diving. Altitude decompression in contrast to routine surface-depth diving is preceded by a condition of equilibrium (saturation) between tissue and ambient nitrogen pressures. Thousands of decompressions from ground level to 18,000 ft (0·5 ATA) have demonstrated that a two to one ratio holds, but there is reason to believe that 'silent' bubbles may be present (Behnke 1942; Bateman 1951). Oxygen inhalation at ground level (pre-oxygenation) which clears dissolved nitrogen from tissues, protects against altitude decompression sickness. The degree of protection, which is assessed by the time of uneventful stay at altitude, is related to the duration of pre-oxygenation (Ferris and Engel 1951). However, above a level of 20,000 ft (0·46 ATA) oxygen inhalation is less effective in affording protection that ensures prolonged stay at 38,500 ft (0·20 ATA) (Behnke 1942). Such experience suggests that 'silent' bubbles may retard nitrogen clearance at the higher altitudes and proof of this will be presented subsequently in connection with studies of radioactive krypton uptake.

Men decompressed from diving depths or from 1 ATA to altitude experience similar symptoms (Behnke 1942) but the occurrence of spinal cord lesions following altitude exposure is infrequent.

Altitude decompression was employed effectively at the Experimental Diving Unit, Washington Navy Yard, during the period 1939–42 as a test for susceptibility to decompression sickness. Highly susceptible or highly resistant individuals could be selected by this test but individual variability severely restricted the usefulness of the procedure. The physiological condition of the individual has too frequently been ignored in decompression practice. One reason for giving consideration to altitude decompression is to invite attention to the analysis of Gray (1951), in which for the first time it was possible to evaluate constitutional factors in a large number of individuals. Although Gray found wide individual variation, he was able to predict effectively group susceptibility in relation to such variables as age, fatness and physical condition. Adler (1964) has reviewed all aspects of altitude decompression, and his analysis, particularly constitutional factors, is directly applicable to problems of the decompression of divers and tunnel workers.

THE PRESSURE DIFFERENCE, ΔP, AND BUBBLE FORMATION

The concept of ΔP, where ΔP is numerically equal to tissue gas tension (π) minus ambient pressure (P), is inherent in the procedure of uniform decompression advocated by Hill. A constant pressure difference (in contrast to a ratio drop in pressure) emerged as a proposal from the Harvard investigations (Behnke 1937). It was Harvey (1951a, b) who gave stature to the concept in connection with altitude decompression sickness. He stressed the importance of $\Delta P = \pi - P$, as the driving force and one of the primary reasons for bubble formation. Currently, Krasberg (1966) and Schreiner and Kelley (1967) assume a constant ΔP for the maximum allowable oversaturation. The driving force effecting inert gas elimination, however, is a combination of $\Delta P + pO_2$, where pO_2 is the partial pressure of oxygen in the lungs.

The factors which control bubble size and bubble resorption were discussed by Zuntz (1897). 'The nitrogen bubbles circulating in blood grow in size as a result of nitrogen diffusion from tissue fluids. Once formed, a gas bubble will diminish in size only gradually because the tension of nitrogen in the bubble is only slightly higher than alveolar nitrogen tension. . . . As circulation stops, the resorption of bubbles can only be effected by very slow diffusion through bloodless tissue layers to the nearest free blood vessels. It is no wonder that under such circumstances, P. Bert as late as the fourth day still found bubbles in the blood vessels of the spinal cord centres.'

Basic postulates of Haldane

Two concepts are the basis of Haldane's calculations. Bubbles of nitrogen are not liberated within the body unless the supersaturation corresponds to more than a decompression from a total pressure of 2·25 Atmospheres (Haldane & Priestley 1935). The variable time-course of nitrogen uptake for various parts of the body can be simulated by use of a

family of discrete, hypothetical half-time tissues (5, 10, 20, 40 and 75 min) to represent gas exchange in the whole body.

It is the rapid initial descent followed by progressively increased stages at 10-ft intervals that features this system (Fig. 11.1). 'The formation of gas bubbles in the living body during or shortly after decompression evidently depends on the fact that the partial pressure of gas or gases dissolved in the blood and tissues is in excess of external pressure. But it is a well-known fact that liquids, and especially albuminous liquids such as blood, will hold gas for long periods in a state of supersaturation provided the supersaturation does not exceed a certain limit. In order to decompress

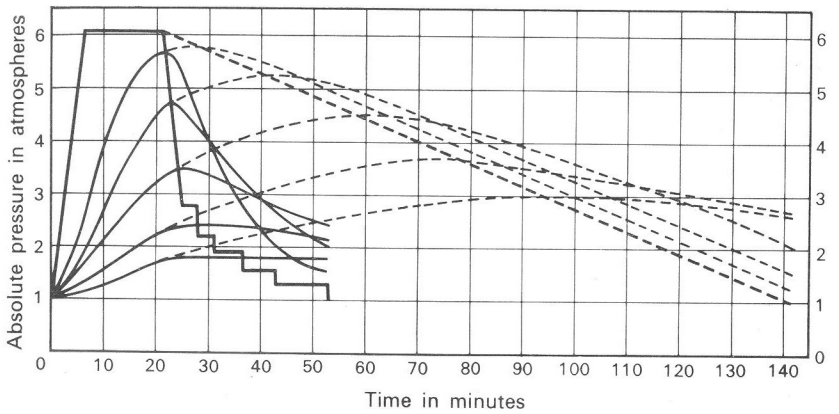

FIG. 11.1. Desaturation during stage decompression in 32 min and uniform decompression in 2 hours, after exposure for 15 min at 201 ft (6·1 ATA) with compression in 6 min

——— = stage decompression; – – – – = uniform decompression
The curves from above downward represent respectively the variations in saturation with nitrogen of parts of the body which half-saturate in 5, 10, 20, 40 and 75 min (Boycott, Damant & Haldane 1908).

safely it is evidently necessary to prevent this limit from being exceeded before the end of decompression. . . . Now the volume of nitrogen which would tend to be liberated is the same when the total pressure is halved, whether that pressure be high or low' (Boycott *et al.* 1908). Hence, Haldane thought it probable that it would be just as safe to diminish the pressure rapidly from 4 ATA to 2, or 6 ATA to 3, as from 2 to 1 ATA. 'Whether the law holds good for pressures exceeding six atmospheres is still doubtful as no experimental data exists.'

Haldane emphasized that the rate of desaturation will vary widely in different parts of the body (Fig. 11.2) even if the general circulation rate is steady. 'These fluctuations may be due to the varying vascularization and difference in composition of the tissues, especially the different proportions

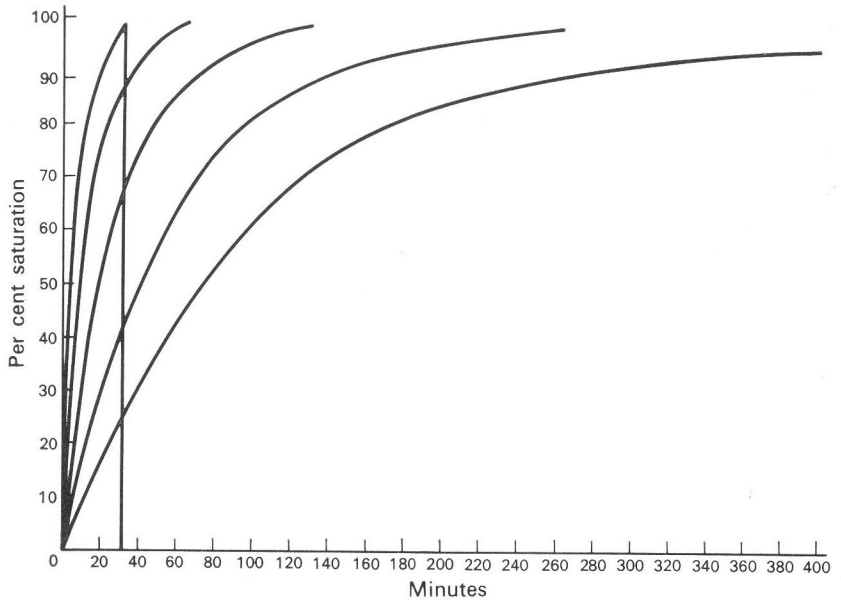

FIG. 11.2. The division of the body into 5 tissues which half saturate in 5, 10, 20, 40 and 75 min, respectively, is represented by the curves. This division forms the basis for calculating the standard decompression table for divers. For example, after exposure to any gauge pressure for 31 min the percentage saturation of each tissue is determined from the point of intersection of the perpendicular line with the curve representing the saturation of the tissue. Compare with Fig. 11.8 (adapted from Boycott *et al.* by Behnke 1937)

of water and fat. . . . Body fat, as was recently shown by Vernon who made a number of determinations at body temperature with special reference to our investigations, takes up about six times as much nitrogen as an equal weight of blood. The body weight of a well-nourished man probably contains fully 15% of its weight as fat or fatty material. . . . the whole body of a man weighing 70 Kg will take up 1 L of nitrogen for each atmosphere of excess pressure.'

It was assumed that saturation of any part of the body would follow a logarithmic curve. Conditions remaining the same, the half-time for saturation would be approximately the same as for desaturation. The estimate of the time required for saturation (98%) was derived from many tests in which goats were decompressed following exposure to high pressures. In application to man, the scaling factor applied was the ratio of surface area and of respiratory exchange of the goat compared with man. This principle generally serves well to explain the high tolerance of small animals to rapid decompression.

'Hence, if it required 3 hours' exposure to a high pressure to effect

practically complete saturation of the more slowly saturating tissues of the goat with nitrogen, about 5 hours would be required for man. A higher degree of saturation than this would scarcely be appreciable, and we have concluded that for practical purposes any slower rate of saturation than this, and correspondingly slower rate of desaturation need not be allowed for, unless the percentage of fat in the body is abnormally high. We must admit, however, that there is some evidence, both from our own experiments and from practical experience in work in compressed air, that *in the part of the body which is the seat of "bends" a still slower rate of saturation may exist.*'

Another qualifying statement is pertinent, 'Parts of the body with a rapid circulation will become very completely saturated in a comparatively short time, but the highly saturated blood which first returns from them on rapid decompression can remain but a very short time supersaturated during each round of the circulation, and on reaching the large veins will mix with the less highly saturated blood from other parts of the body. It would seem that the state of high supersaturation in any portion of the blood lasts for too short a time to enable bubbles to form' (Boycott *et al.* 1908).

In the second basic paper (Boycott & Damant 1908), the important role of fat relative to bubble nascence is discussed. 'We do not make any suggestion that the extra gas dissolved by fat produces fatal effects by its liberation *in situ*. . . . Though fat itself contains many bubbles both intravascular and among the cells, obesity doubtless favours death after long exposures because fat acts as a reservoir of nitrogen and so keeps up the nitrogen pressure in the venous blood after decompression sufficiently long for bubbles to form.'

Early diving tests

The application of the Haldane principles rendered air diving practical and safe to a depth of 204 ft (7·2 ATA), the limit of his Tables at the time. Not only was depth limited but recommended bottom time (underlined in the Tables) was of short duration, dictated in part by limitations of air supply and by equipment.

Systematic tests both in the dry chamber and in the sea were made to extend the initial diving Tables to cover a depth of 300 ft (10 ATA). Damant assisted by G. Davis (Davis, R. H. 1962) subjected 12 goats to a standard dive of 30 min at 300 ft. The goats were decompressed according to the stage method with a first stop at 120 ft and a total decompression time of 122 min. Several of the animals developed bends. The experiment was repeated and oxygen utilized from the 60-ft level to surface. Again the result was unsatisfactory. 'It had to be accepted that at 50 fathoms, or

10 Atmospheres pressure, Haldane's two to one law no longer held good, a finding for which he had prepared us.'

The ratio was then reduced to 1·75 to 1, decompression time was lengthened to 3 hours and oxygen decompression to 87 min. Decompression time was extrapolated for man and standard tests were repeated. The results were entirely satisfactory, none of the ten divers showing the slightest symptoms of decompression sickness.

When real diving took place it was found that further modifications of the Tables had to be made. The outcome was the formulation of Tables at depths from 120 to 300 ft (4·6 to 10 ATA) which incorporated the inhalation of oxygen for the lower stages of decompression. These Tables, the first of their kind, were adopted by the Admiralty. From 1931 to 1933 tests in conjunction with the Davis submersible decompression chamber were carried out in Loch Fyne from H.M.S. *Tedworth*. A remarkable series of dives were made at depths of 260 to 320 ft (8·9 to 10·6 ATA) without accident.

It was during these tests that Hill and Phillips (1932) observed a dangerous overconfidence in the divers with dulling of their mental faculties and an inability to make decisions. In 17 out of 58 dives to 200 to 350 ft (7·1 to 11·6 ATA), there were signs and symptoms of narcosis and lapses into unconsciousness (Hill, Davis, Selby, Pridham & Malone 1933).

HELIUM–OXYGEN DIVING

Early experience

In 1925 Sayers, Yant and Hildebrand proposed the use of helium–oxygen mixtures for prevention of decompression sickness. In animal experiments, they found a reduction in decompression time by a factor of six compared with air tests. In 1938 End described the use of helium in a record dive to a depth of 420 ft in Lake Michigan. The diver, Nohl, utilized self-contained gas cylinders to supply a suit of his own design. The U.S. Navy began investigating helium diving about 1929 but the results were unfavourable. In retrospect it appears that inhalation of the helium–oxygen mixtures was administered by means of a mouthpiece which induced copious salivation accompanied by swallowing considerable amounts of gas at depth. Rapid ascent to the surface induced collapse.

Tests at the U.S. Navy Experimental Diving Unit

During the period from September 1937 to 23 May 1939 (the date of the U.S.S. *Squalus* disaster) systematic experiments were conducted with helium–oxygen mixtures by Momsen, Wheland, Yarbrough, Willmon and Behnke. In a summary report (Momsen 1939) nearly 700 simulated dives

in the wet tank were reported at depths to 500 ft (16·1 ATA). Noteworthy in these divers was the feeling of well-being, comparable to an air dive at 100 ft (4 ATA), and their ability to engage in heavy work. In decompression it was necessary to bring the divers up more slowly from deep depths and to remain at a relatively deep depth for 7 min in an effort to promote helium elimination from the rapidly saturating tissues. Novel developments included a venturi recirculator in the helmet, use of carbon dioxide absorbent in a partially closed system, and the fabrication of an electrically heated fireproof garment (pure oxygen was inhaled in later stages of decompression) to circumvent the rapid conductive heat loss in the helium atmosphere.

Decompression features

Calculation of tables. The ratio principle was employed with a limitation of 1·7 to 1 as the allowable supersaturation of all gases other than oxygen relative to ambient pressure. The half-times of tissues projected in the calculations were, 5, 10, 20, 30, 40, 50, 60 and 70 min. The limitation of the half-time of the slowest desaturating tissue to 60 or 70 min was based on measurements of helium elimination which will be discussed subsequently, as well as on the consideration of helium solubility in water and fat. As in the Air Tables of Haldane so with the early Helium Tables, exposure times in the test dives were relatively short—about 20 min duration except in the special saturation tests.

Alternation of gas mixtures. Frequently in the oxygen–helium dives during a decompression a shift to air was begun at 200 ft (7·1 ATA). Air inhalation was then continued to 60 ft (2·82 ATA) level, and the remainder of the decompression completed on oxygen. In this way it was possible to reduce decompression time drastically after long exposures in an oxygen–helium atmosphere compared with air (Table 11.1).

Partial pressure vacancy, oxygen window principle. During the course of blood transport through capillaries, the oxygen is 'unloaded' in different quantities to the various tissues. The result of this transfer of oxygen from blood to tissues renders available an equivalent space for inert gas transport from tissues to lungs. This manner of gas transport is *isobaric* in contrast with the metastable conditions discussed previously. Momsen fully appreciated this principle, and he designated the fall in oxygen pressure in the capillaries, as the 'partial pressure vacancy' (PPV). Momsen employed partial pressures of oxygen with helium as high as 2·3 ATA in the effort to create a maximal PPV. This author for several years has considered that the metabolic transfer of oxygen creates a 'window' compatible with isobaric inert gas transport from tissues to lungs (Behnke 1967). Independently LeMessurier and Hills (1965) and Hills (1966) have

TABLE 11.1

Comparison of total decompression time following exposure in compressed air and exposure in an oxygen–helium atmosphere (Behnke 1942)

ATA	*Ft*	*Exposure (min)*	*Decompression (min)*	
			air	*oxygen–helium*
3·7	90	100	57	75
3·7	90	180	—	77
3·7	90	360	—	79
3·7	90	540	638	79
—	—	—	—	—
5·6	150	80	141	121
5·6	150	180	—	126
5·6	150	360	—	128
—	—	—	—	—
7·1	200	65	217	154
7·1	200	90	—	164

made an analysis of this type of approach as it applies to actual diving operations.

Measurements of helium elimination. In 1938 the subjects who participated in the helium tests were divers and medical officers. Their body fat was accurately determined by the application of Archimedes principle (Behnke 1942a). The gas mixture inhaled consisted of 73 to 76% helium, 5 to 7% nitrogen and 19 to 20% oxygen. The mixture was inhaled at normal pressure for $3\frac{1}{2}$ hours and for shorter periods at simulated depths to 300 ft (10 ATA). Recovery of helium from the tissues was brought about by having the subjects breathe air or oxygen in a closed spirometer system. The values for total helium content of the body varied less than 10% in replicate tests. Two of the lean men desaturated in 4 hours in contrast with 6 hours in two men who weighed 202 and 206 lb respectively. With the tissues of the body in equilibrium with a helium partial pressure in the lungs corrected to 760 mm Hg, the helium content per kilogram of weight for the 11 subjects was 8·0 ml ± 1·3 ml, compared with nitrogen, 18 ml/Kg ± 2·0 ml. The diffusion of inert gas through skin was also quantified. The recovery data are presented in Table 11.2. Noteworthy is the fact that the time required for helium elimination, within the accuracy of our method at the time, is approximately the same irrespective of the time of exposure to inhalation of helium.

Rescue and salvage operations: U.S.S. Squalus

In connection with these operations over 600 dives were made; oxygen–helium in 255 dives between 220 and 240 ft (7·7 and 8·3 ATA) and air in the shallower dives (Behnke & Willmon 1939). There were only two cases

TABLE 11.2
Helium recovery following varying periods of helium (73 to 76%) breathing at different pressures
(Behnke & Willmon 1941)

Subject	1	1	1	1	1	2	2	2	3	3	3
Exposure (min)	210	210	210	20	20	210	13	20	210	20	20
Depth	0	0	40	100	225	0	300	300	0	100	225
ATA	1·0	1·0	2·2	4·0	7·8	1·0	10·1	10·1	1·0	4·0	7·8
Time (min)	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(12)	(12)
3–30*	94·3	103	160	615	223	402	112	897	1041	62	177	300
30–60	32	40	127	310	40	452	66	712	469	33	47	187
60–90	22	19	68	194	19	108	32	166	185	18	14	72
90–120	10	15	32	173	14	19	23	108	94	10	8	31
120–150	9	9	34	76	6	16	17	69	53	6	3	13
150–180	5	6	12	54	5	11	11	46	34	4	—	8
180–240	7	8	10	27	6	21	14	76	40	4	—	7
240–300	4	4	9	12	4	10	9	24	16	—	—	2
300–360	1·5	—	5	10	—	5	3	5	4	—	—	—
360–420	—	—	2	3	—	—	—	2	3	—	—	—

* 0–3′ rinsing period. Subject breathes oxygen or air for alternate 30-min periods during recovery period. Helium elimination was measured only during oxygen breathing at 1 ATA, hence each test except (4), (7), (8) and (9) required 2 days.

of severe decompression sickness. This is a remarkable record for which Momsen deserves a large share of credit. Indeed, throughout the experimental period at EDU beginning in September 1937, there were no injuries from the experimental wet-chamber dives to simulated depths of 500 ft (16·1 ATA). Oxygen–helium bends were treated without complications on essentially oxygen therapy.

Surface decompression or decanting. This was used routinely in all dives. The short stops were in the water and then there was a rapid transfer to a recompression chamber. Surface decompression, when first employed, was a decision forced upon divers as a result of cold water and strong tides which rendered decompression in the open sea impractical. The procedure permits the elimination of excess gas dissolved in tissues to proceed with the diver warm, at rest and under observation. The danger of the method lies in the evolution of many nascent bubbles between leaving the last stop in the water stop and the subsequent recompression.

QUANTITATIVE STUDIES OF INERT GAS UPTAKE AND ELIMINATION

Measurements of nitrogen transport

In man the first measurements of nitrogen elimination when oxygen is inhaled, were reported in 1913 by Bornstein in studies of cardiac output. It was not until 1931 that Campbell and Hill, using a modification of the Bornstein method, found that approximately 200 to 300 ml of nitrogen, about 25 to 33% of total content for a lean man, were eliminated during the first 9 min of oxygen breathing. Subsequent experiments have confirmed and extended these findings in some degree (Shaw *et al.* 1935; Behnke *et al.* 1935; Willmon & Behnke 1941; Stevens *et al.* 1947; Jones 1950; Boothby *et al.* 1951; Kety 1951; Lundin 1960).

Nitrogen uptake and elimination in the dog. During the period 1932 to 1935, Shaw *et al.* designed a closed system of 100 L volume to measure exchange of nitrogen in the anaesthetized dog placed in 99% oxygen. Rinsing of the system and time required for decompression from higher pressures precluded recovery of the first 7 to 8 min of nitrogen output. A high degree of accuracy was achieved in the analyses so that with few exceptions, the range of variation in control tests was within 3 ml. Typical desaturation curves (Fig. 11.3) show individual variation due to size, age and fatness. In lean dogs nitrogen recovery reached an endpoint after 3 to 4 hours, in contrast with elimination from the fat dog. These experiments served to confirm data in another species and conclusions drawn from the goat experiments (Boycott *et al.* 1908) in regard to desaturation time, and the importance of fat as a nitrogen reservoir (Boycott & Damant 1908).

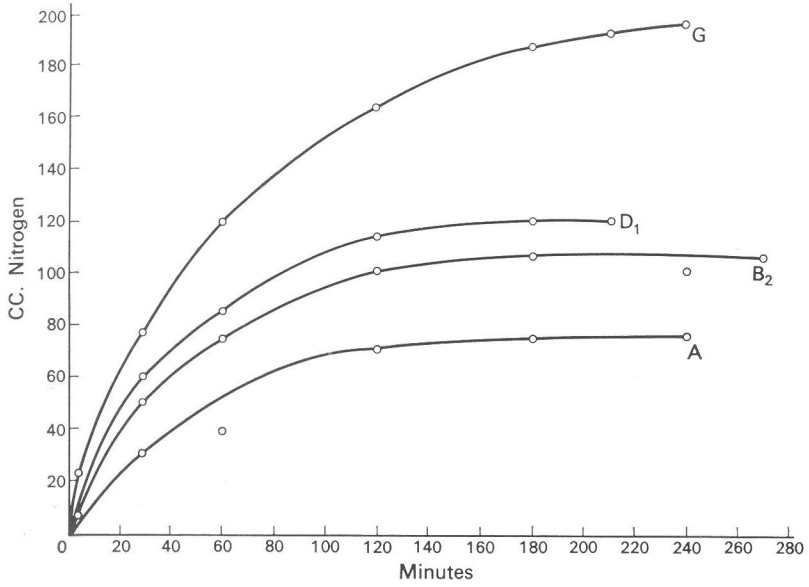

FIG. 11.3. Nitrogen recovery from four anaesthetized dogs placed in a closed oxygen (99%) system for periods up to 280 min at 1 ATA. At the end of this period, nitrogen elimination is not complete in dog (G) which was old and fat (from Shaw *et al.* 1935)

Other curves (Fig. 11.4), however, provided new and essential data. The cumulative nitrogen elimination with the exception of the first 7 min was measured in the same dog (D) on different days after equilibration at 1, 2 and 4 ATA (Shaw *et al.* 1935).

Two findings are of fundamental importance: the absence of a 'break' or departure from normal in the nitrogen curve despite the abrupt drop in pressure from 4 to 1 ATA, and that the desaturation curve following partial equilibration is not the reverse of the saturation curve. The time required for nitrogen elimination following partial saturation is the same, within our error of measurement, as the time required following complete equilibration. Expressed as elimination rate (percentage saturation), it is possible to superimpose the curve depicting partial equilibration, on the complete equilibration curve (Shaw *et al.* 1935).

In these tests it was not possible to decompress from higher pressures after long exposures in an effort to change the shape of the nitrogen recovery curve. Either the dog lived and the curve was 'normal' or death from fulminating embolization occurred within minutes.

Nitrogen elimination in man. The amount of nitrogen recovered per minute diminishes during the course of oxygen inhalation from an initial 50 ml/min (P_{N_2} 573 mm Hg) to less than 0·1 ml/min at the end of 9 hours.

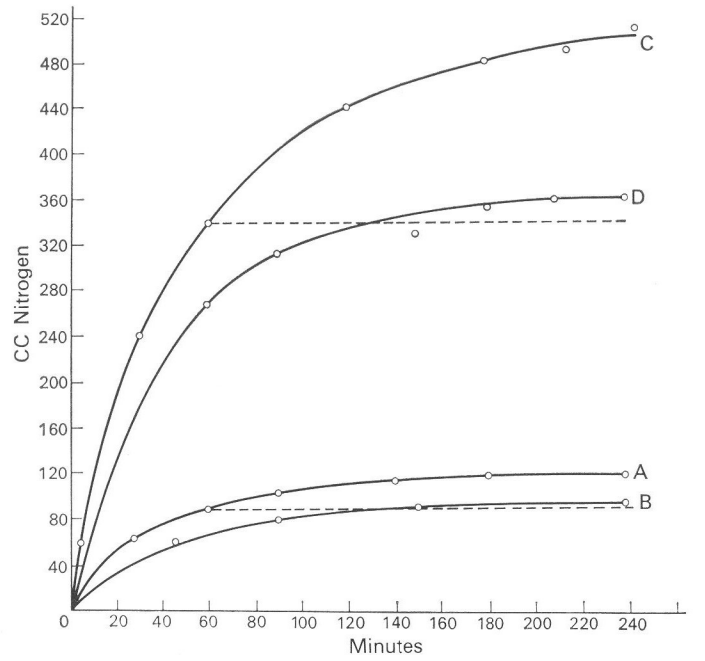

FIG. 11.4. Saturation time compared with desaturation time for anaesthetized dog (D). Nitrogen eliminated during the first 7 min (lung rinsing) was not measured. Curve A follows equilibration at 1 ATA; curve B follows equilibration for 67 min at 1 ATA; curve C follows complete saturation at 4 ATA; and curve D follows 67 min equilibration at 4 ATA (Shaw *et al.* 1935)

At this time diffusion of nitrogen through the skin (body, except head, surrounded by air) accounts for the greater part of the nitrogen recovered (Behnke & Willmon 1941). The curve representing cumulative nitrogen recovery from the body (Fig. 11.5) has been analysed in terms of exponential components, each represented by

$$Y = A(1 - e^{-kt})$$

$$A = \frac{(Y_1)^2}{2Y_1 - Y_2}$$

and

$$k = \log_e \frac{A}{A - Y} \cdot \frac{1}{t}$$

where Y is the quantity of nitrogen recovered at time (t), and A is total nitrogen for the segment of the whole body curve represented by the single exponential member in the first equation; A can be calculated from Y_1 at

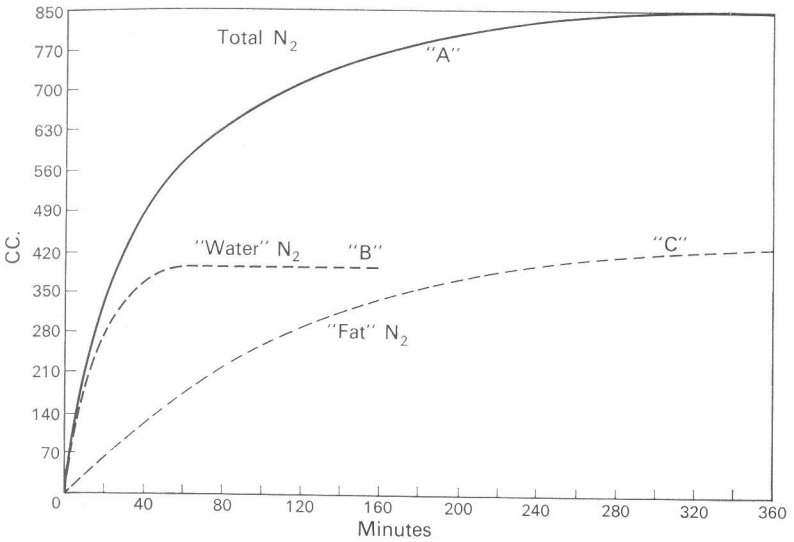

FIG. 11.5. Nitrogen elimination. Curve A represents the average values for cumulative nitrogen from three lean men (average weight 64 Kg) who breathed oxygen at atmospheric pressure in a helmet system. 'Water' N_2 (B) and 'fat' N_2 (C) are hypothetical curves which represent nitrogen elimination from the chief body solvents. B and C are exponential curves with half-times of 7 and 82 min respectively. Nitrogen recovered during the first 5 min (rinsing period) would add a third (faster) half-time component, while increased fat would add a fourth or even fifth half-time component (Behnke 1937)

time t_1 and from Y_2 at time t_2, provided that the time interval of t_2 is in each case, twice that of t_1 (Behnke, Thomson & Shaw 1935; Behnke 1937).

The curve representing total nitrogen recovery (Fig. 11.5) was divided into 'fluid' and 'fat' components or fast and slow tissues, which now can be measured accurately. The extensive measurements of Jones (1950) have been resolved into some five components referable to tissues with different rates of blood flow. A representative breakdown of tissues and perfusion rates based in part on the analysis of Jones has been fitted into the framework of entities we can measure, namely, fluid and fat (Table 11.3). However, diffusion plays an important role (Hills, 1966) and further, Haber (1951) states that it is hazardous to infer that the exponential components derived from an accurate fit of data are representative of such entities as blood, muscle and fat. Furthermore an exponential equation is so flexible that a variable number of constants can be found to fit the data without being representative of the actual physical mechanisms involved. Haber pointed out the value of measurements of minute to minute quantities of eliminated nitrogen in addition to accumulated nitrogen. This objective has been realized in part by Jones (1950) and advanced by Lundin (1960).

TABLE 11.3
Atmospheric nitrogen content of tissues and organs in relation to rates of blood perfusion and nitrogen elimination at rest during the inhalation of oxygen by young men

Unit	*I* *Blood brain heart kidney*	*II* *Muscle skin spinal cord nerves*	*III* *Bone (mineral and fat-free)*	*IV* *Bone marrow*	*V** *Adipose tissue*	*VI†* *Adipose tissue*
Weight (gm)	15,000	37,000	3,500	1,500	9,500	15,200
Water (gm)	12,000	30,000	2,000	240	2,000	2,000
Fat (gm)	350	100	—	1,200	7,000	12,600
Nitrogen (ml)	126	275	18	63	368	647
Blood perfusion (ml/min)	4,000	1,200	80	50	375	375
Nitrogen transport (1st min)	40	12	0·8	0·5	3·75	3·75
k‡	0·39	0·044	0·044	0·008	0·01	0·058
Nitrogen elimination (half-time min)	1·8	16	16	85	69	120

* Lean man, fat (lipid) = 10% body weight.
† Average man, fat (lipid) = 18% body weight.
‡ k, exponential decay constant, half-time rate (min) = $0{\cdot}693/k$.

A prime consideration is that the nitrogen elimination curve for the body as whole provides for each individual an experimentally determined set of time constants (k values) for each exponential segment of the curve, as well as a measure of the quantities of nitrogen involved in uptake and elimination. These entities would appear to constitute 'essential' data for the calculation of rational decompression tables.

Optimal pressure level commensurate with nitrogen elimination during inhalation of oxygen. Since the toxic effects of oxygen on the nervous system are pressure-related, it was an early objective to measure nitrogen elimination at various pressure levels between 1 and 4 ATA (0 and 100 ft). Following a standard exposure of 75 min at 4 ATA, oxygen was inhaled at various stops in separate tests for 30-min periods. The pressure was then reduced to 1 ATA and measurements of nitrogen recovery continued. There was some indication that nitrogen elimination was less rapid at 4 ATA or following abrupt reduction in pressure from 4 to 1 ATA. From the preliminary tests (Tables 11.4 and 5) nitrogen recovery was independent of the level at which oxygen was breathed.

UPTAKE OF RADIOACTIVE KRYPTON

Regional studies—uptake in the hand

In 1941, with the advice of Hildebrand, who had earlier pioneered the application of helium in diving, Lawrence, Jones and Hamilton employed

TABLE 11.4

Nitrogen recovery from four subjects during the inhalation of oxygen at levels of 100, 50, 20 and 0 ft (4, 2·52, 1·43 and 1·0 ATA) following exposure in air for a period of 75 min at 100 ft (4 ATA) (from Willmon & Behnke 1941)

Subject	*Stop ft*	*Tests*	*Nitrogen recovery (ml)*		
			3 to 30 min at stop	*33 to 90 min 0 ft (1 ATA)*	*3 to 90 min total*
1	20	1	1478	834	2312
	50	2	1533	957	2590
	100	2	1415	739	2154
2	20	2	1127	777	1904
	50	3	1220	809	2029
	100	1	849	785	1634
3	20	—	—	—	—
	50	1	1587	982	2569
	100	1	1486	949	2435
4	20	1	1081	687	1768
	50	1	1079	674	1753
	100	2	1010	754	1764

TABLE 11.5

Nitrogen recovery from a diver exposed to a simulated depth of 100 ft for 75 min compared with nitrogen recovery following an exposure of 30 min at 100 ft. Pure oxygen was inhaled throughout the recovery period

Exposure		*Stop*		*Tests*	*Nitrogen recovery (ml)*		
min	*ft*	*ft*	*ATA*		*3 to 30 min at stop*	*33 to 90 min 0 ft (1 ATA)*	*3 to 90 min total*
75	100	20	1·43	1	1478	834	2312
75	100	50	2·52	2	1533	957	2590
75	100	100	4·0	2	1415	739	2154
30	100	44	2·33	1	1343	548	1891
30	100	50	2·52	1	1312	565	1877
30	100	66	3·0	1	1341	522	1863
30	100	0*	1·0	1	626	401	1027
30	100	0*	1·0	1	1191	499	1690
30	100	0*	1·0	1	892	856	1748
30	100	0*	1·0	1	1147	511	1658

* 2 minutes decompression from 100 to 0 ft (4 to 1 ATA) (from Willmon & Behnke 1941).

radioactive krypton (half-life 34 hours) and body surface scanning in a joint effort with the Experimental Diving Unit to locate post-decompression bubbles. Although bubbles were not located, it was possible to follow radioactive uptake and elimination of krypton not only in the body as a

whole but also in regional areas such as the hand (Jones 1951). Greatly accelerated uptake followed release of pressure in a sphygmomanometer cuff which had occluded blood supply to the hand for a period of 10 min (Fig. 11.6) (Behnke 1945). The area between the normal curves and upper

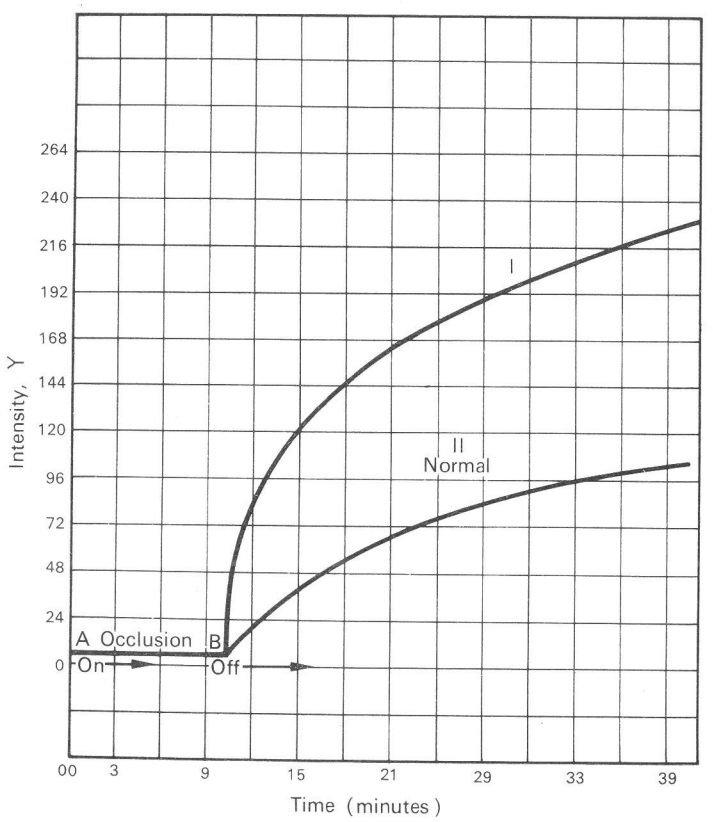

FIG. 11.6. Absorption of inhaled radioactive krypton tracer by the tissues of the shielded hand and forearm. The lower curve represents uptake in a man, sitting and relaxed. A second experiment the following day was identical except that blood flow to the hand was occluded for 10 min. The upper curve represents the greatly accelerated krypton uptake in response to reactive hyperaemia and the circulatory adjustments following release of pressure. Technique and application by Jones, Hamilton and Lawrence in 1942 (Behnke 1945)

curves presumably reflects the dilated capillary bed as a result of reactive hyperaemia. It is possible that a similar mechanism may be operative in acclimatization which would enable the capillary bed to accommodate a larger quantity of gas in bubble form.

Desaturation time following inhalation of radioactive krypton.

A paramount consideration in decompression is the length of time required for complete desaturation following fractional periods of uptake of inert gas. In the studies of Tobias, Jones, Lawrence and Hamilton (1949), it was found that an uptake of krypton during a 20-min period of inhalation required a period of more than 100 min for elimination, whereas an uptake of krypton over a much longer period (117 min) required only a slightly longer time (about 120 min) for krypton desaturation. However, the shape of the desaturation curves is different for the relatively short and long periods of uptake.

Tobias *et al.* (1949) were able to distinguish between 'three distinctly different reservoirs' containing inert gas. The filling of these three reservoirs appeared to be somewhat independent, each characterized by a time-rate constant within a definite range. It is noteworthy that the three half-time values for each of the three components is nearly identical, irrespective of the period of preliminary krypton inhalation. Thus, following a 20-min period of uptake, the half-time values are 6, 39 and 310 min respectively for each component. Following an uptake period of 117 min, half-time desaturation values are 6, 42 and 320 min, and following 'saturation' uptake the half-times are 6, 41 and 310 min for the respective components. These findings are in accord with nitrogen elimination data from the dog and helium recovery data from man, namely that the tension of inert gas in various parts of the body (excluding perhaps bone and spinal cord) tends towards equality during desaturation, irrespective of the previous degree of partial saturation.

'Bends' retardation of krypton elimination at simulated altitude

In the dog experiments (Shaw *et al.* 1935) it was not possible to demonstrate any real change in the nitrogen elimination curve following abrupt reduction of pressure from several elevated pressure levels which was presumably sufficient to induce 'silent' bubbles in the blood stream. Reference was made to the less effective nitrogen elimination during oxygen inhalation at simulated altitudes above 20,000 ft (0·46 ATA), possibly due to impairment of blood flow by 'silent' bubbles. Evidence that the bends is tantamount to the presence of intravascular bubbles is again found in the experiments of Tobias *et al.* (1949) as shown in Fig. 11.7. It is observed that there was a slowing of rate of krypton desaturation at chamber altitude during the time when the subject had incapacitating pain in his knee, shoulder, elbow and wrist. After recompression to ground level the rate of krypton exchange rapidly reached normal. Three out of five subjects who remained free from bends at 35,000 ft (0·24 ATA) showed no altitude-induced change in their krypton curves. One subject, susceptible to bends on previous exposures to altitude, showed a definite slowing of krypton elimination during an altitude test in which he had no pain. This was possibly due to the effect of 'silent' bubbles.

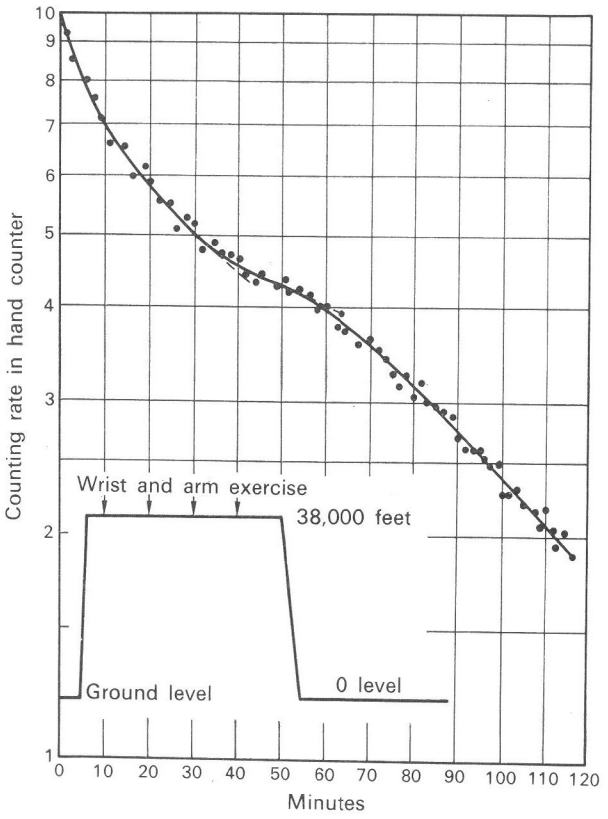

FIG. 11.7. Demonstration of retardation of krypton elimination in the hand during a 1-hour stay at simulated altitude of 38,000 ft (0·204 ATA) associated with incapacitating bends. On recompression to ground level (1·0 ATA) pre-recovery rate was restored (from Tobias, Jones, Lawrence & Hamilton 1949)

APPLICATION OF NITROGEN ELIMINATION DATA TO DECOMPRESSION PRACTICE

In the years 1932 to 1935, bends following the Haldane stage decompression method complicated long exposures at 4 ATA (100 ft) in the Harvard chamber. By contrast, uniform decompression (12 to 24 hours) was reported to be highly effective following saturation exposures for several days at 30 psi (3 ATA). This led to a re-evaluation of decompression

practice for chamber exposures of long duration. Several postulates (Behnke 1937) that appeared reasonable at the time were:

(1) A metastable condition of gas transport probably did not extend through successive decrements of pressure.

(2) What appeared to be a condition of supersaturation was a reflection of tolerance of the vascular system to bubble accumulation.

(3) During decompression following partial equilibration, the pressure of nitrogen tended to equalize in various parts of the body as a result of diffusion of nitrogen into unsaturated fat. (In retrospect, since diffusion may be too slow a process, one might consider as did Haldane that blood from rapidly and slowly saturating tissues was equilibrated in passage to the lungs. Further, on rapid decompression, gas nuclei or 'seed' bubbles not eliminated in the lungs, would be disseminated throughout the body.)

(4) Essentially for the purpose of decompression the experimental nitrogen curve for the body as a whole, specific for each individual, would serve to define 'equivalent depth' and the decompression rate.

In Fig. 11.8, one can compute equivalent depth following a 31-min dive to a depth of 100 ft (4 ATA) as 53 ft. In the proposed decompression, one would ascend to equivalent depth plus an additional 22 to 33 ft equivalent to a ΔP of 0·67 to 1·0 ATS. Decompression would then proceed to surface at a uniform rate. The rate selected is derived from the total nitrogen curve as the average rate of elimination of nitrogen over a 31-min period.

Following a saturation exposure, one would apply a decompression rate corresponding to the time required for the body as a whole. At the present time, one would adjust ΔP to conform with the isobaric principle and 'oxygen window' concept of inert gas elimination.

In concluding this discussion, it is the conviction of the author that any resolution of the intricacies of decompression practice must be coupled with measurements of gas uptake and elimination during the entire period of pressurization and decompression. So far our effort in realization of the unlimited potential of such quantitative analysis has been meagre over the last 35 years and much remains to be done.

MINIMAL DECOMPRESSION, 'NO STOP', DATA FOLLOWING DIVING EXPOSURES

In 1934 data was available (Kagiyama 1934) to show that divers could ascend progressively from deeper depths without decompression, provided that exposure time was shortened. Thus a dive could be made to 82 ft (3·5 ATA) for 30 min followed by minimal time of ascent to the surface without decompression stops. Similarly a dive could be made to a depth of

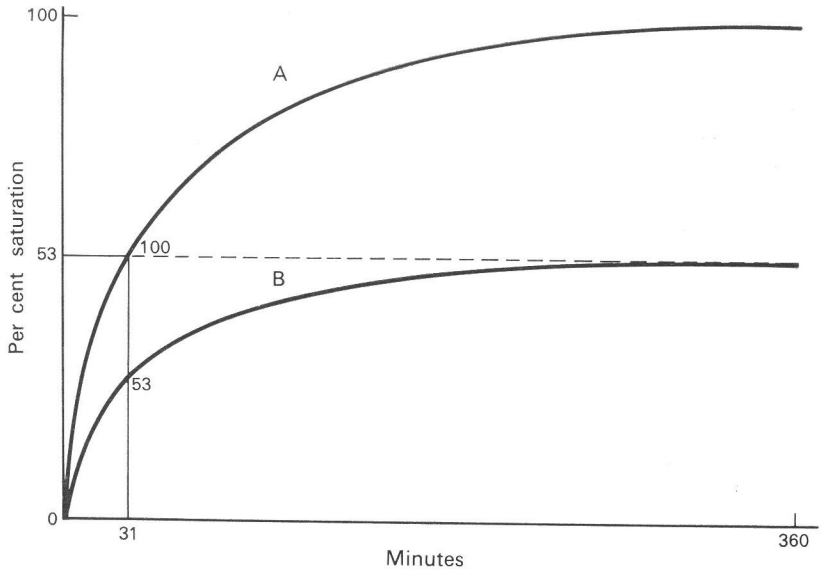

FIG. 11.8. Proposed method of calculating decompression of a diver based on his experimentally determined nitrogen elimination curve. After a dive of 31 min, for example, the percentage saturation in the body of this lean diver is 53 as shown on curve A. Equivalent depth is 53 ft and gauge pressure could be reduced to this level. Curve B represents probable course of N_2 elimination if oxygen were inhaled at the 40-ft level. After a saturation exposure, decompression would begin from the plateau of curve A. Rate constants for each exposure are taken from the nitrogen elimination curve and are calculated from percentage saturation relative to time for the body as a whole (Behnke 1937)

164 ft (6·0 ATA) for a stay of 15 min, followed by rapid ascent to the surface. In submarine escape drills Shilling and Hawkins (1936) compiled a remarkable series of rapid ascents in the pressurized wet chamber. Two thousand one hundred and forty simulated escapes were made with a Momsen lung using air or oxygen from depths of 100 to 200 ft (4 to 7 ATA) with a graded exposure time which was increased about 1 min per test until bends supervened. It was found safe under these conditions to remain 37 min at 100 ft, 18 min at 150 ft and 14 min at 185 ft (6·6 ATA), followed by minimal decompression. One may observe 'acclimatization' in the divers who were able to extend greatly their initial depth exposure. Subsequently acclimatization was also demonstrated in tunnel workers (Paton & Walder 1954).

The safe exposure time at depth followed by minimal decompression may be computed from the nitrogen elimination curve for the body as a whole (Behnke 1937). The body appears to tolerate a constant volume of inert gas uptake prior to minimal decompression. The uptake may be at

relatively high pressures for short periods or at lower pressures for longer periods.

HISTORICAL ASPECTS OF SATURATION AIR DIVES AND TUNNEL DECOMPRESSION PRACTICE

Prolonged exposure in compressed air was tested clinically many years ago. Patients have lived in pressure chambers (3 ATA or less) for periods of a week or longer. At the Experimental Diving Unit from 1940 to 1945 saturation exposures served as an effective test challenge to methods of decompression. The results are directly applicable to the problem of decompressing tunnel workers safely. In saturation exposures followed by minimal decompression, a few feet separate good physical condition from symptoms. The critical level following 12-hour (saturation) exposure attended by minimal decompression is about 35 ft (2·1 ATA). In tunnel compressed air exposures, the critical level is higher (2·2 ATA, 40 ft) but work time is 8 hours or less. Although the quantity of nitrogen absorbed after 8 hours is small, it appears to be critical. The difficulty in freeing tissues of excess nitrogen is evident from the data in Table 11.6. Even oxygen is not as fully effective. It should be possible, for example, to

TABLE 11.6
Saturation air dives in the dry or wet tank (Behnke 1940; Van Der Aue 1945)

No. tests	*ATA*	*ft*	*Exposure time (hr)*	*Decompression*		*Outcome**
				Depth stop (ft)	*Time (min)*	
4 Resting	2·00	33	12	—	—	NS
4 Work	2·00	33	12	—	—	NS
4 Resting	2·00	33	24	—	—	NS
8 ,,	2·00	33	36	—	—	2 (X) 6 NS
4 Resting	2·06	35	12	—	—	1 (O) 3 NS
4 Work	2·06	35	12	—	—	NS
14 Resting	2·21	40	12	—	—	4 (O) 9 NS
14 Work	2·21	40	12	—	—	5 (O) 4 NS 5 (X)
1 Resting	2·82	60	12	60 oxygen	63	(X)
1 ,,	2·82	60	12	60 ,,	69	(X)
1 ,,	2·82	60	12	60 ,,	80	(X)
1 ,,	2·82	60	12	60 ,,	92	(X)
1 Resting	3·82	90	6	40 oxygen	111	NS
1 ,,	3·82	90	6	40 ,,	111	(X)
4 Resting	4·00	99	6	33 air	12 hr	1 (X) 3 NS
2 ,,	4·00	99	9	33 ,,	12 ,,	1 (X) 1 (O)
2 ,,	4·00	99	9	33 ,,	18 ,,	NS
2 ,,	4·00	99	12	33 ,,	24 ,,	1 (X) 1 (O)

* NS = No symptoms; (X) = Bends, recompression; (O) = mild bends, no recompression.

decompress from 2·82 ATA (60 ft) after 12 hours' exposure in about 64 min if oxygen is breathed throughout the decompression period. This premise is based on the assumption that it is safe to surface with 1·82 ATA (27 ft) in the slowest desaturating tissues (bone marrow and adipose tissue fat). This is not, however, the case. Divers were adversely affected even though oxygen decompression was extended to 92 min. It would appear that it is not safe to maintain a ΔP of 27 ft as the driving pressure head of nitrogen in decompression from higher pressures.

Experience in tunnel decompression practice

In 1909 Keays, medical director at the East River Tunnels for the Pennsylvania Railroad reported on 3692 cases of decompression sickness out of 557,000 decompressions. There were 20 deaths. Men worked 8 hours out of 24 at pressures up to 32 psi gauge (72 ft; 3·2 ATA). Some remarkable feats of work in compressed air were accomplished in these tunnel operations. In 23,000 decompressions from 40 to 42 psi g (90 to 95 ft; 3·7 to 3·9 ATA), there were no serious or fatal cases. Three hundred and thirty men were employed for 36 days in a two-shift daily schedule that called for 3 hours on shift with a 3-hour rest interval at normal pressure. The total time for decompression for each shift was 48 min. Reporting on 8510 of these decompressions, Keays recorded 1·6% of minor cases. Noteworthy is his comment that 'only seasoned men were employed'. Contrast the decompression time of 48 min for a 3-hour shift at 40 psi g (90 ft; 3·7 ATA) with 160 min (U.S. Navy), and 98 min (British Regulations).

These data serve to emphasize one of the paradoxes in tunnel decompression practice, namely, that there can be a wide discrepancy in decompression time for a given work shift which is not commensurate with the incidence of bends. It is the probability of greater bone involvement, not bends that dictates on empirical grounds the requirement for longer decompression.

The importance of acclimatization is clearly evident from Keays's data. In 1947 Van Der Aue (unpublished data) subjected 12 divers in good condition to a dry-chamber exposure that simulated the work time and decompression schedule followed in New York State from 1912 to 1922. The divers were *at rest* throughout the period of chamber residence. The debilitating outcome of these tests was impressive: of the 12 healthy divers at rest 8 developed symptoms and 2 required recompression therapy.

The procedure followed in the decompression of tunnel workers is 'half-and-half'; that is, the initial stage is an abrupt drop in pressure (Haldane), usually the gauge pressure is halved, followed by uniform decompression (Hill). The striking feature of the practice is the short time for decompression allowed when compared with diving. However, as working pressures

are increased, time of exposure is decreased such that the quantity of excess nitrogen absorbed by the slowly saturating tissues is about the same at each increased pressure level (Behnke 1937). Despite the sharp curtailment in work time, decompression is inadequate as shown by the high incidence of bone necrosis.

REFERENCES

Adler, H. F. (1964). *Dysbarism.* Aeromedical Reviews, USAF School of Aerospace Medicine, Brooks Air Force Base, Texas.

Bateman, J. B. (1951). Review of data on value of preoxygenation in prevention of decompression sickness. In *Decompression Sickness.* Ed. Fulton, J. F. Philadelphia & London: Saunders.

Behnke, A. R. (1937). The application of measurements of nitrogen elimination to the problem of decompressing divers. *U.S. nav. med. Bull.* **35**, 219–240.

Behnke, A. R. (1942a). Investigations concerned with problems of high altitude flying and deep diving; application of certain findings pertaining to physical fitness to the general military service. *Milit. Surg.* **90**, 9–28.

Behnke, A. R. (1942b). Effects of high pressures; prevention and treatment of compressed air illness. *Med. Clin. N. Am.* July 1942, 1213–1236.

Behnke, A. R. (1945). The absorption and elimination of gases of the body in relation to its fat and water content. *Medicine*, **24**, 359–379.

Behnke, A. R. (1967). The isobaric (oxygen window) principle of decompression. In *The New Thrust Seaward.* Trans. Third Marine Tech. Soc. Conf. 5–7 June, San Diego. Washington, D.C.: Marine Tech. Soc.

Behnke, A. R., Thomson, R.M. & Shaw, L. A. (1935). The rate of elimination of dissolved nitrogen in man in relation to the fat and water content of the body. *Am. J. Physiol.* **114**, 137–146.

Behnke, A. R., Shaw, L. A., Messer, A. C., Thomson, R. M. & Motley, E. P. (1936). The circulatory and respiratory disturbances of acute compressed-air illness and the administration of oxygen as a therapeutic measure. *Am. J. Physiol.* **114**, 526–533.

Behnke, A. R. & Willmon, T. L. (1939). U.S.S. *Squalus:* Medical aspects of the rescue and salvage operations and the use of oxygen in deep-sea diving. *U.S. nav. med. Bull.* **37**, 629–640.

Behnke, A. R. & Willmon, T. L. (1941). Gaseous nitrogen and helium elimination from the body during rest and exercise. *Am. J. Physiol.* **131**, 619–626.

Bert, P. (1878). *Barometric Pressure. Researches in Experimental Physiology.* Trans Hitchchock, M. A. & Hitchcock, F. A. 1943. Columbus, Ohio: College Book Co.

Boothby, W. M., Luft, U. C. & Benson, O. O. (1951). *Gaseous nitrogen elimination.* Proj. no. 21-53-003, Rpt. 1, USAF School of Aviation Medicine, Randolph Field, Texas.

Bornstein, A. (1913). Weitere untersuchungen uber Herzschlagvolumen. *Z. exp. Path Ther.* **14**, 135–150.

Boycott, A. E. & Damant, G. C. C. (1908). Experiments on the influence of fatness on susceptibility to caisson disease. *J. Hyg. Lond.* **8**, 445–456.

Boycott, A. E., Damant, G. C. C. & Haldane, J. B. (1908). The prevention of compressed air illness. *J. Hyg. Lond.* **8**, 342–343.

Boyle, R. (1670). New pneumatical experiments about respiration. *Philos. Trans.* **5**, 2011–2058.

Campbell, J. A. & Hill, L. (1931). Concerning the amount of nitrogen gas in the tissues and its removal by breathing almost pure oxygen. *J. Physiol.* **71**, 309–322.

Campbell, J. A. & Hill, L. (1933). Studies in the saturation of the tissues with gaseous nitrogen. *Q. J. exp. Physiol.* **23**, 197–210.

Davis, R. H. (1962). *Deep Diving and Submarine Operations*, 7th edn. pp. 7–9. London: St Catherine Press.

Ferris, E. B., Jr. & Engel, G. L. (1951). The clinical nature of high-altitude decompression sickness. In *Decompression Sickness.* Ed. Fulton, J. F. Philadelphia & London: Saunders.

Fulton, J. F. (1951). *Decompression Sickness*, p. 1. Philadelphia & London: Saunders.

Gray, J. S. (1951). Constitutional factors affecting susceptibility to decompression sickness. In *Decompression Sickness.* Ed. Fulton, J. F. Philadelphia & London: Saunders.

Haldane, J. S. & Priestley (1935). *Respiration*, p. 337. London: Oxford University Press; New York: Longmans, Green.

Haber, H. (1951). Appendix in USAF Report by Boothby, Luft & Benson, cited above.

Harvey, E. N. (1951a). Physical factors in bubble formation. (1951b) Bubble formation in cats. In *Decompression Sickness.* Ed. Fulton, J. F. Philadelphia & London: Saunders.

Heller, R., Mager, H. & von Schrötter, H. (1900). *Luftdruckerkrankungen*, 2 Vols. Vienna: Hölder.

Hill, L. (1912). *Caisson Sickness and the Physiology of Work in Compressed Air.* London: Edward Arnold.

Hill, L. & Greenwood, M., Jr. (1907). The influence of increased barometric pressure on man. *Proc. roy. Soc.* **79**, 21–27.

Hill, L. & Phillips, A. E. (1932). Deep-sea diving. *J. R. nav. med. Serv.* **18**, 155–173.

Hill, L., Davis, R. H., Selby, R. P., Pridham, A. & Malone, A. E. (1933). *Deep Diving and Ordinary Diving.* Report of Committee appointed by the Admiralty.

Hills, B. A. (1966). *A Thermodynamic and Kinetic Approach to Decompression Sickness.* Thesis. Adelaide: Libraries Board South Australia.

Hoff, E. C. (1948). *A Bibliographical Sourcebook of Compressed Air Diving & Submarine Medicine.* U.S. Navy Dept. Bur. Med. Surg., Washington, D.C., p. 7, 11. U.S. Gov. Print. Office.

Hoppe-Seyler, F. (1857). Ueber den Einfluss welchen der Wechsel des Luftdruckes aus das Blut ausübt. *Arch. Anat. Physiol., Lpz.* **24**, 63–73.

Jones, H. F. (1950). Respiratory system: Nitrogen elimination. In *Medical Physics.* Vol. 2. Ed. Glasser, O. Chicago: Year Book Publ.

Jones, H. F. (1951). Molecular exchange and blood perfusion through tissue regions. In *Advances in Biology & Medical Physics.* Ed. Lawrence, J. New York: Academic Press.

Kagiyama, S. (1934). Studies on prevention of caisson disease. *J. Kumamoto med. Soc.* **10**, 562–564.

Kety, S. (1951). The theory and applications of the exchange of inert gas at the lungs and tissues. *Pharmacol. Rev.* **3**, 1–41.

Krasberg, A. R. (1966). Saturation diving techniques. *4th Intern. Congr. Biometeorology.* New Brunswick, New Jersey: Rutgers University.

LeMessurier, D. H. & Hills, B. A. (1965). Decompression sickness. A thermodynamic approach arising from a study of Torres Strait diving techniques. *Hvalrådets Skrifter*, Nr. **48**, 54–84.

Lundin, G. (1960). Nitrogen elimination from the tissues during oxygen breathing and its relationship to fat: muscle ration and the localization of bends. *J. Physiol.* **152**, 167–175.

Momsen, C. B. (1939). *The Use of Helium–Oxygen Mixtures for Diving* (revised Oct. 1942). Rpt. Exp. Div. Unit, Navy Yard, Washington, D.C.

Paton, W. D. M. & Walder, D. N. (1954). *Compressed Air Illness.* Medical Research Council Spec. Rpt. Series 281, London: Stationery Office.

Sayers, R. R., Yant, N. P. & Hildebrand, J. H. (1925). Possibilities in the use of helium–oxygen mixtures as a mitigation of caisson disease. *Rep. Invest. U.S. Bur. Mines*, No. 2670, 1–15.

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

Shaw, L. A., Behnke, A. R. Messer, A. C., Thompson, R. M. & Motley, E. P. (1935). The equilibrium time of the gaseous nitrogen in the dog's body following changes in nitrogen tension in the lungs. *Am. J. Physiol.* **112**, 545–553.

Shilling, C. W. & Hawkins, J. A. (1936). The hazard of caisson disease in individual submarine escape. *Nav. med. Bull. Wash.* **34**, 47–52.

Stevens, F. D., Ryder, H. W., Ferris, E. B., Jr. & Inatome, M. (1947). The rate of nitrogen elimination from the body through the lungs. *J. Aviat. Med.* **18**, 111–133.

Tobias, C. A., Jones, H. B., Lawrence, J. H. & Hamilton, J. G. (1949). The uptake and elimination of krypton and other inert gases by the human body. *J. clin. Invest.* **28**, 1375–1385.

Willmon, T. L. & Behnke, A. R. (1941). Nitrogen elimination and oxygen absorption at high barometric pressures. *Am. J. Physiol.* **131**, 633–638.

Workman, R. D. (1965). *Calculation of Decompression Schedules for Nitrogen–Oxygen and Helium–Oxygen Dives.* Research Report 6–65. Exp. Div. Unit, Washington Navy Yard, Wash. D.C.

Zuntz, N. (1897). Zur Pathogenese und Therapie der durch rasche Luftdruckänderungen erzeugten Krankheiten. *Fortschr. Med.* **15**, 632–639.