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History of Evolution of Decompression Procedures

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There are many man-made disorders for which the causative agent is easily established but the mechanism whereby the body reacts is not sufficiently understood. Decompression sickness is such a disorder. It is provoked by our ability to change the pressure and chemical composition of the gases we breathe. One procedure for complete prevention of decompression sickness, therefore, is not to venture outside the limits of air composition and pressure normally encountered on the surface of the Earth. There is, it is hoped, a second way to eliminate the harmful effects of decompression sickness, and that is to understand the underlying mechanisms and from this understanding to construct safe procedures. It is the history of man's attempt to achieve this second possibility that will now be examined.

The first successful device that could alter the ambient pressure in a controlled manner was a pump for exhausting the air from a container and was invented in the seventeenth century by Von Guericke. Using his own version of this recently invented pump, Robert Boyle became, in 1670, the first investigator in the field of decompression sickness when he decompressed a viper in his 'exhausted receiver' and described the now famous 'bubble moving to and fro in the waterish humour of one of its eyes'. From this and similar early experiments the idea arose that a rapid reduction of atmospheric pressure could lead to the release of bubbles into sensitive tissues of the body and that this could seriously impair their normal functions.

To work underwater, gases at pressures greater than atmospheric are required, and it was some years before a pump could be used to raise the ambient pressure rather than lower it, as in the Boyle experiments described above. Many attempts had been made to descend into the sea using a variety of diving apparatus, but until comparatively recent times all these devices were totally impractical for any reasonably prolonged underwater work, and offered very little advantage beyond simple breath-hold diving. The diving bell was really the first successful underwater device and it is generally agreed that Edmond Halley (1717), an Astronomer Royal, designed, built and used the first practical 'bell' system (Fig. 1.2b).

Depths as great as 18 m (60 ft) for dive durations as long as $1\frac{1}{2}$ h were attained using this particular technique.

Several important physiological points, relevant to the theoretician, should be noted. Firstly, the nature of the breathing gas must be clearly defined; variability in the concentration of such physiologically active gases as carbon dioxide and oxygen could influence the validity of any decompression observations made. Second, the diver who leaves the bell at the end of his breathing tube is being subjected to a different environment from that of his companion seated within the bell. The diver may well be surrounded by quite cold water with the well-known ensuing physiological reactions to this. In addition, he is attempting to draw fresh air for breathing purposes down a tube which is clearly giving added respiratory work and, being immersed, the hydrostatic relationships between blood pressure in the extremities and the pressure in the heart are altered and thus there are changes in the cardiovascular system. Third, some underwater workers are liable to be performing very little work, e.g. the seated attendant, but others, e.g. the diver, are required to perform quite hard physical work. Fourth, men are very different in important factors such as stature, body composition and level of physical fitness. Finally, and of extreme impor-

tance when studying the effects of exposure to raised pressures of air and other gases, it is necessary to know how long the subjects were exposed and to what pressure. With such a formidable list of variables to be brought under control in order to obtain consistent findings, it is hardly surprising that numerous and conflicting conclusions were reached from the data available in the early years of this subject.

Placing men under raised pressures of air can conveniently be considered as divisible into four separate phases, each of which has its own particular set of problems. The first phase is taking the man to pressure, and this compression phase sometimes causes pressure differentials to be established in body cavities such as the sinuses and the middle ear which can cause, in these instances, sinus pain and vertigo. The second phase comprises the sojourn at full pressure. Here the compressed air worker (diver, caisson, tunnel) encounters the effects of raised pressures of oxygen and of the inert gas nitrogen which can give rise to numerous difficulties—e.g. oxygen toxicity, nitrogen narcosis, altered thermal balance, increased respiratory work and communications problems. The third, or decompression, phase is the return to atmospheric pressure, which is followed by the fourth phase, or post-decompression period. It is these two latter phases that principally concern us in this chapter. A note of caution must be introduced, because, as will become apparent later, the events occurring in phase 2 can profoundly influence the body's responses in the decompression and post-decompression periods. For the moment, however, consider only the evidence accumulated by the turn of the century concerning decompression and its consequences. It had become apparent that decompression could be followed by harmful effects varying in severity from death to mild itching of the skin. The prevention of these ill-effects was soon seen to lie in pursuing a slow release of pressure. It would seem that everyone adopted some form of linear decompression procedure, i.e. the pressure was released at a certain number of bars (atmospheres, lb/in^2, kg/cm^2) per minute for the caisson and tunnel workers or a given number of metres (feet, fathoms) per minute for the diver. The practical problem, in those days, was to decide the most effective rate of pressure release.

An understanding of the basic processes producing the harmful effects of decompression from exposure to compressed air was lacking until the time of Paul Bert, who made numerous fundamental observations in a series of experiments between 1870 and 1890. He showed that the more serious forms of decompression sickness were provoked by the presence of large volumes of free gas, as opposed to dissolved gas. Furthermore, on careful analysis of the composition of these bubbles he concluded that nitrogen gas was the main constituent. Thus, an outline picture of the aetiology of decompression sickness could now be attempted.

It was apparently caused by the release of gas emboli from nitrogen gas dissolved at pressure, and these nitrogen gas emboli then impaired the functioning of the various tissues in which they lodged or were formed. Meantime, a clearer clinical picture was also emerging. If the decompression was grossly inadequate, then the blood literally 'frothed' and a condition descriptively termed 'the chokes' was encountered and this proved rapidly fatal unless promptly treated by recompression. If the decompression was not so provocative, then a condition known as 'the staggers' was often seen, and this, too, was a very serious manifestation of decompression sickness which could lead to permanent damage in the central nervous system, or even death. However, the most prevalent form of decompression sickness came to be termed 'the bends', so called by the workmen who constructed the bridge across the Mississippi at St Louis (Jaminet 1871), the name referring to the affected gait which was fashionable among the young ladies of the time and apparently bore a resemblance to the behaviour of those workmen who contracted the less serious, but painful, forms of decompression sickness. The term 'bends' nowadays refers only to pain in or around a joint, which can make itself felt either during the decompression or sometimes several hours post decompression.

Although much early compressed air work was performed in Western Europe, most particularly in France and England, it was the massive undertakings in the USA that provided a sound statistical basis for examining the frequency of occurrence of the various forms of decompression sickness. Table 13.1 gives the data on one such contract resulting from several years' work and involving over one million decompressions, which was reported in 1909 by Keays. As may be seen, by far the greatest number of decompression sickness incidents were attributed to the bends, and it would seem a reasonable assumption that if the decompression procedures could be arranged to avoid attacks of

TABLE 13.1 Keays (1909)

Symptom	*No of cases*	*Percentage*
'Bends' (joint pain)	3278	88.78
'Bends' with local manifestations	9	0.26
Pain with prostration	47	1.26
Central nervous system symptoms:		
1. Hemiplegia	4	0.11
2. Spinal cord symptoms	80	2.16
Vertigo ('staggers')	197	5.33
Dyspnoea ('chokes')	60	1.62
Partial or complete unconsciousness	17	0.46

bends, then decompression sickness in its various forms would become a rarity. By the turn of the century, therefore, the decompression problem had narrowed into one of understanding the physics and physiology of the initiation of the bends.

THE HALDANE CONCEPTS

The most important period in the development of decompression theory commenced at the turn of the century, when the navies of the world realized that underwater operations were about to become a necessary feature of modern warfare. Accordingly, research work either was performed by the navies themselves or was sponsored by them in external institutions, e.g. universities. The first, and, some would say, the most productive, of these navy-backed research efforts occurred in 1906, when the Royal Navy engaged the services of the renowned physiologist, Professor J. S. Haldane, for a series of investigations specifically aimed at producing regulations for the safe conduct of underwater work by divers. All serious students of decompression theory must read the original account by Haldane and his co-workers (Boycott *et al.* 1908), as it is the starting point for most modern treatments of decompression theory.

When Haldane commenced his pioneering studies, the clinical features of decompression sickness were well documented but, in order to pursue a series of experiments which would certainly have a risk of serious decompression sickness occurring from time to time, it was necessary to search for a suitable animal model. Consequently, a great variety of animals were exposed to raised pressures of air in an attempt to assess their sensitivity to attacks of decompression sickness and their general suitability as experimental material. Two principal features of the animal model were vital. In the first place, whichever animal was chosen, it must exhibit a marginal form of decompression sickness which could be clearly identified as a pain in a joint and therefore provide a realistic comparison with the principal human situation. After examining a wide spectrum of different animal species, Haldane and his co-workers decided that the goat most nearly satisfied their theoretical and practical requirements. The second essential characteristic of any suitable experimental animal was that its circulatory dynamics should be as near as possible to those of a human being. Once again, on the basis of body weight and composition (i.e. fat/water ratio) the goat was selected as the best experimental compromise. Clearly a large primate would have been more suitable, but anyone who has worked with these creatures realizes the tremendous problems they can bring and it is a tribute to Haldane's selection process that the goat is still considered quite useful even nowadays as an animal model for certain types of decompression sickness research. Relatively modern data on the relationship between body size and sensitivity to decompression sickness are shown in Fig. 13.1, and, as may be seen, the data give good support to considering the goat as a suitable animal model.

Having chosen a suitable animal, it was now necessary to discover the ground rules relating to the appearance and non-appearance of decompression sickness. If a beaker of water is exposed to a constant raised pressure of air, say P_1, and is stirred until no more gas will dissolve in the water at that pressure, then if the gas pressure above the liquid is now suddenly reduced to a new lower pressure, say P_2, it was decided that the tendency to form bubbles would be described by the magnitude of the pressure drop, i.e. $P_1 - P_2$. It was thought sensible to check whether this supposition was true in the biological situation. If an animal was exposed for a prolonged period at some raised pressure, P_1, until all the tissues of its body had equilibrated with this pressure, then when the pressure was dropped to some new value, P_2, would the animal exhibit attacks of decompression sickness if $P_1 - P_2$ remained constant? Or, perhaps, there was some other relationship between P_1 and P_2 which could be established.

With this idea in mind, Haldane and his co-workers exposed their goats for what they considered prolonged periods of time ($1\frac{1}{2}$–2 h) at raised

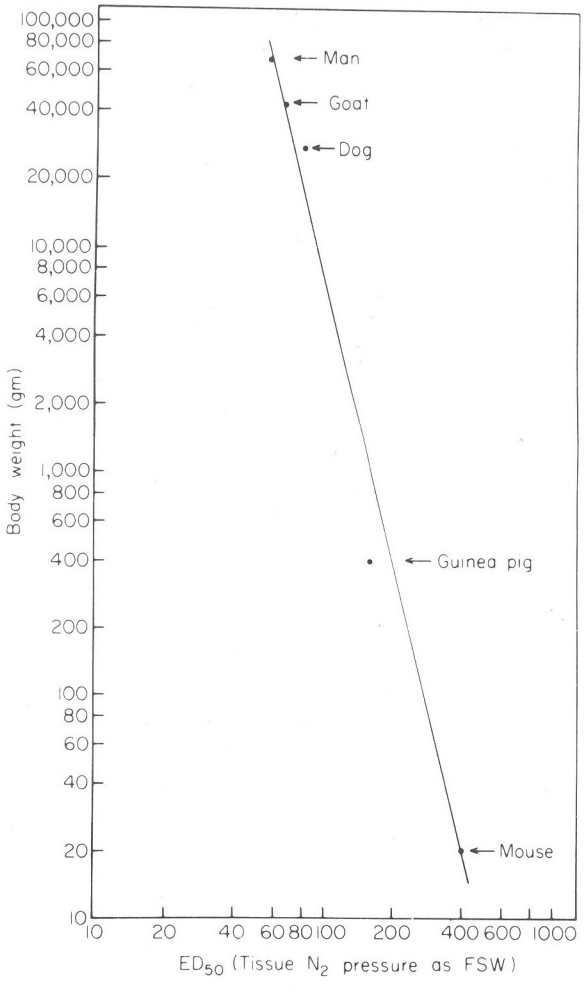

FIG. 13.1. A plot of the relationship between body mass and sensitivity to decompression sickness. The value of the goat as a suitable animal model for human work can be seen

pressures and then rapidly decompressed them to some other lower pressure, and awaited the outcome of this rapid pressure drop. They discovered that if they exposed their animals for such periods of time at a pressure equivalent to about 45 ft of sea water pressure, and then rapidly decompressed these animals back to atmospheric pressure, some of them just began to exhibit mild joint pains (i.e. presumably bends) on surfacing or shortly afterwards. It was decided therefore that a pressure difference of just over 1 ATS could be safely tolerated by all normal animals. The exposure pressure was next altered to 6 ATA and a rapid pressure drop of just over 1 ATS was indeed quite safe for all the animals but so was a rapid pressure drop of 3 ATS, and this result was clearly not consistent with the idea of a constant pressure drop defining the generation of bubbles, and, hence, the appearance of decompression sickness. From these and similar experiments Haldane reached his first working hypothesis, which could be stated as follows. After prolonged exposure on air to pressures of 8 ATA it is quite safe to decompress rapidly to 4 ATA; similarly, after long exposure at 6 ATA it is quite safe to ascend rapidly to 3 ATA, and from exposure to 2 ATA it is safe to decompress to 1 ATA. Put in simple mathematical terms, if P_1 is the exposure pressure and P_2 is the pressure to which decompression is taken rapidly, then P_1/P_2 is a constant and equal to 2. Clearly, as all tissues in the body are equilibrated following prolonged exposure to pressure, then the 2:1 ratio value is applicable in all decompression situations for all tissues of the body. This ratio concept became a cornerstone of the Haldane calculation method.

The difficulties associated with deciding the rate at which various tissues acquired and eliminated dissolved inert gas had been avoided in these early experiments. All tissues had been brought to the same state, namely equilibrated to the pressure of gas being breathed. However, not all dives have prolonged bottom times, and, in any case, having decompressed safely from P_1 to P_2 on the ratio principle, how did one now proceed from the new pressure, P_2, back to atmospheric pressure? These problems demanded a knowledge of the rates at which various tissues of the body acquired dissolved inert gas when the pressures were raised and how they eliminated their excess inert gas content when the pressure was lowered.

Consider a man breathing pure air at normal atmospheric pressure who is suddenly at some time, $t = 0$, exposed to a raised pressure of air, P_1 atmospheres. This new pressure of air will be instantly transmitted to the lung surface in the alveoli and gas will dissolve in the pulmonary tissues through which the pulmonary circulation passes. Using relatively modern knowledge, it is known that approximately 0.01 s is required for the dissolved molecules in the alveolar lining to reach the underlying capillary. In view of the fact that it takes about 1.0 s for blood to pass the length of a pulmonary capillary, it is quite certain that blood leaving the capillary is fully equilibrated with the gas pressure in the alveoli. For simplicity, therefore, it will be assumed that the arterial blood supplying the tissues is fully equilibrated with the pressure of gas being breathed, and that whenever the gas

being breathed changes, then the arterial blood instantly follows this change. Fortunately, these simplifying assumptions would not lead to more than a few per cent error, and provided that the physiology of the body does not alter markedly during the course of a dive exposure, the errors will remain reasonably constant and therefore can be discounted.

Thus, the blood is in equilibrium with the pressure of gas breathed in the lungs and this is being supplied via the arterial system to all the separate tissues of the body. The next stage of the problem is to decide how this dissolved gas in the arterial input is distributed within a particular tissue space. Histological examinations of most tissues will reveal a very large number of capillaries per unit volume. The figure will vary from several hundreds in well-vascularized tissues to perhaps only one patent capillary per cubic millimetre in a tissue such as fat. The general point to be made, however, is that the intercapillary distance in nearly all tissues of the body is measured in fractions of a millimetre, and, accepting the normal diffusion coefficients for small gas molecules such as nitrogen and helium, it would be impossible to sustain large concentration gradients within a tissue space. Again, for simplicity, and without much error, it will be assumed that the concentration of dissolved gas throughout a tissue space is uniform. Given the acceptance of these various simplifying assumptions, the physics of the situation can be represented as in Fig. 13.2.

Suppose that the volume of arterial blood flowing in to the tissue is v ml/s; then the volume of venous blood flowing out must also be v ml/s, otherwise the tissue would progressively swell or shrink. Let the solubility of the inert gas in blood be s_1 ml at atmospheric pressure per ml of blood at 37 °C. If the pressure of gas being breathed is kept at a steady value of P_1 atmospheres, then the total quantity of dissolved gas entering the tissue per second is $P_1 s_2 v$ ml.

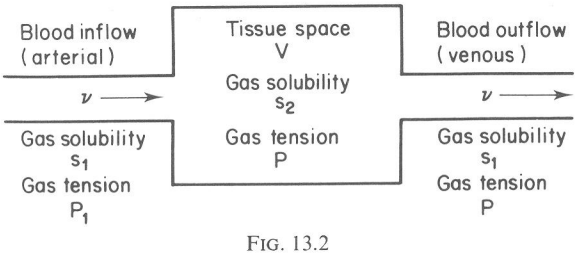

FIG. 13.2

If the tissue is considered as possessing a dissolved gas tension of P atmospheres, where P is, as noted above, uniform throughout the tissue and is therefore also the tension in the outgoing venous blood, then the total quantity of dissolved gas leaving the tissue per second is $Ps_1 v$ ml.

The quantity accumulating in the tissue per second is the difference between that entering and that leaving, i.e. $(P_1 - P)s_1 v$. Now this amount of dissolved gas is distributed per second in a tissue space of volume V and solubility s_2.

Suppose that a very small time Δt passes, then a small increase in tension ΔP will occur in the tissue, which will represent a volume of dissolved gas $\Delta P \times s_2 V$ ml. In this small time the blood has delivered $(P_1 - P)s_1 v\,\Delta t$ ml of dissolved gas. These two amounts must be equal, i.e.

$$\Delta P\, s_2 V = (P_1 - P)s_1 v\,\Delta t$$

or

$$\frac{\Delta P}{\Delta t} = (P_1 - P)\frac{s_1 v}{s_2 V}$$

s_1, v, s_2 and V are all constants for any particular tissue. Therefore, $s_1 v/s_2 V$ is constant and will be called K:

$$\frac{\Delta P}{\Delta t} = K(P_1 - P)$$

as Δt becomes smaller,

$$\frac{dP}{dt} = K(P_1 - P) \tag{1}$$

Students of elementary calculus will know that the variables are separated,

$$\frac{dP}{P_1 - P} = K\,dt$$

and then integrated,

$$-\log (P_1 - P) = Kt + c_1$$

or, expressing this differently,

$$P_1 - P = c\,e^{-Kt} \tag{2}$$

But when $t = 0$, $P = 0$. Therefore,

$$P_1 = c\,e^0 = c, \quad \text{as } e^0 = 1.$$

Rearrange equation (2) using this value of c,

$$P = P_1(1 - e^{-Kt}) \tag{3}$$

This is the fundamental equation of the original Haldane analysis, and has become the basic mathematical expression for nearly all subsequent

treatments of dissolved gas exchange in tissues. Continuous reference will be made to various aspects of this analysis and the often subtle hidden assumptions.

It is now opportune to examine how Haldane made use of this expression governing tissue inert gas exchange.

Equation (3) refers to the situation where a tissue is acquiring excess dissolved gas through sudden imposition of a constant input pressure P_1 at time $t = 0$, and as may be easily verified when $t = \infty$, $P = P_1$ and the tissue has equilibrated with the arterial gas tension. The rate at which the tissue approaches this equilibrium state is entirely dependent upon the K value chosen, and $K = s_1 v/s_2 V$ requires a substantial knowledge of the tissue physics and physiology which was certainly not available in Haldane's day. Consequently, he needed to make an informed guess, and it is worth remarking that this practice of 'informed guessing' has continued to the present day, since, as will become apparent later, no-one yet knows which tissue, or tissues, cause the bends.

In common with many situations where exponential time constants are involved (e.g. radioactive decay), it is common practice to use the half-time as a measure of the rate process. All exponential time courses have the same shape and the simple property they have in common is that if t_1 is the time taken to reach half the value at t_∞, then $2t_1$ will be three-quarters of the way to this value, $3t_1$ will be seven-eighths, and so on. Each additional half-time takes the value half-way between the previous value and the infinite value. For the construction of his decompression table calculations Haldane chose tissue half-times of 5, 10, 20, 40 and 75 min. One cannot help but notice the anomalous way he doubled up the half-time values from 5 to 40, and then called a halt at 75 instead of 80.

Let us now examine in some detail a typical dive calculation according to the Haldane method.

In these early computations air was treated as a single gas. This is obviously incorrect for, as Haldane well knew, the bubbles responsible for decompression sickness are formed from excess dissolved nitrogen, but the proportion of nitrogen in air is always constant, so taking it as a single entity is therefore permissible and avoids multiplying every air pressure by a constant factor of 0.79, i.e. the proportion of nitrogen gas in air.

The best way to reveal how these decompression procedures were obtained is to give, in its original wording, an account by Damant of the calculation of two decompression profiles for use by compressed air workers. The method for divers is exactly the same in the main features—i.e. a pressure ratio principle to determine the permissible excess dissolved gas at any time during the decompression, and the use of pressure stages for off-loading this excess gas. In the case of the original diving tables the pressure units were feet of sea water and the pressure stages, or 'stops', as they are frequently called, were placed at 10 ft (3 m) of sea water increments. When reading this account, bear in mind that 0.7 m or $2\frac{1}{4}$ ft of sea water is equivalent to one pound per square inch of pressure and therefore the two calculations which follow refer to 6 h exposure at 17.5 m (57 ft) and $1\frac{1}{2}$ h at 21 m (69 ft) of sea water. One further important fact to be noted is that although the Haldane decompression ratio of 2:1 is much discussed by everyone studying this subject, it was not used by Haldane for his calculations! These hidden 'oddities' that occur in decompression table calculations have continued to the present day, and will be pointed out at the appropriate points in the subsequent considerations.

'Haldane showed that it was safe and desirable, with working pressures up to about 80 lb/in^2 (5.6 kg/cm^2) to begin decompression by rapidly reducing the air pressure to the equivalent of half (or a little less than half) the *absolute* working pressure. From this point onwards the air pressure should be gradually reduced at a rate slow enough to ensure that no part of the body is, at any time, supersaturated to such an extent as to cause risk of bubble formation. The processes of saturation and desaturation proceed at the same rate, following a logarithmic curve, but we cannot calculate for the body as a whole because some parts or groups of tissues saturate and desaturate more rapidly than others. In calculating we must take into account parts which become half saturated in 75, 40, 20, 10 and 5 min, respectively. Table 13.2 tells us by what percentage each of these parts will become saturated or desaturated in a given number of minutes. The two last-mentioned groups of tissues, called 10T and 5T, are only of importance when the time of exposure to pressure is short, as in diving work at very high pressures.

Table 13.3 gives, for various working pressures, the pressure to which the air lock may be reduced in the first rapid stage of decompression.

TABLE 13.2

Giving the percentage saturation at 1 min intervals of tissues (75T etc.) which become half-saturated in 75, 40, 20, 10 and 5 min.

Time (min)					Saturation percentage	Time (min)					Saturation percentage	Time (min)					Saturation percentage
5T	10T	20T	40T	75T		5T	10T	20T	40T	75T		5T	10T	20T	40T	75T	
	0.14			1	1	3	5.99	12	24	45	34	8	16.0	32	64	120	67
	0.29		1	2	2		6.21		25	46	35		16.4	33	66	123	68
	0.44	1	2	3	3		6.43	13	26	48	36		16.9	34	68	127	69
	0.59			5	4		6.66		27	50	37		17.4	35	70	130	70
	0.74		3	6	5		6.90	14	28	52	38	9	17.9	36	72	134	71
	0.89			7	6		7.13			53	39		18.4	37	74	138	72
0.5	1.04	2	4	8	7		7.37		29	55	40		18.9	38	76	142	73
	1.20			9	8		7.61	15	30	57	41		19.4	39	78	146	74
	1.36		5	10	9		7.85		31	59	42	10	20.0	40	80	150	75
	1.52	3	6	11	10	4	8.11	16	32	61	43		20.6	41	82	154	76
	1.68			13	11		8.36		33	63	44		21.2	42	85	159	77
	1.84		7	14	12		8.62	17	34	65	45	11	21.8	44	87	163	78
1.0	2.01	4	8	15	13		8.89	18	36	67	46		22.5	45	90	168	79
	2.18		9	16	14		9.16		37	69	47		23.2	46	93	173	80
	2.34			18	15		9.43	19	38	71	48	12	23.9	48	96	179	81
	2.51	5	10	19	16		9.71		39	73	49		24.7	49	99	185	82
	2.68		11	20	17	5	10.0	20	40	75	50		25.5	51	102	191	83
	2.86			21	18		10.3		41	77	51	13	26.4	53	105	198	84
	3.04	6	12	23	19		10.6	21	42	79	52		27.4	54	109	205	85
	3.22		13	24	20		10.9	22	44	82	53	14	28.4	56	113	213	86
	3.40			26	21		11.2		45	84	54		29.4	59	117	221	87
	3.58	7	14	27	22	0	11.5	23	46	86	55	15	30.6	61	122	230	88
	3.77		15	29	23		11.8		47	88	56	16	31.9	63	127	239	89
2	3.96	8	16	30	24	6	12.2	24	49	91	57		33.2	66	132	249	90
	4.15			31	25		12.5	25	50	94	58	17	34.7	69	138	260	91
	4.34	9	17	32	26		12.8	26	51	96	59	18	36.4	73	145	273	92
	4.54	9	18	34	27		13.2		53	99	60	19	38.3	77	153	287	93
	4.74		19	36	28		13.6	27	54	102	61	20	40.6	81	162	304	94
	4.94			37	29	7	14.0	28	56	105	62	22	43.2	86	173	324	95
	5.14	10	20	38	30		14.3		57	107	63	23	46.4	93	185	348	96
	5.35		21	40	31		14.7	29	59	110	64	25	50.6	101	202	380	97
	5.56	11	22	42	32		15.1	30	60	113	65	28	56.4	113	225	424	98
	5.78		23	43	33		15.6	31	62	117	66	33	66.4	133	265	498	99

The following abbreviations are used. All pressures are gauge pressures (i.e. above atmospheric) unless otherwise stated.

WP: *Working pressure* to which a man has become exposed during his shift.

SP: *Saturation pressure* of a man's body or a specified part of it. The solution pressure of dissolved nitrogen will be about 80% of the SP.

LP: *Lock pressure* in a decompression lock or chamber at a specified stage of compression.

DP: *Difference of pressure* between SP and LP at a given moment during decompression. It represents the stress under which desaturation is proceeding.

p: The highest working pressure from which a man can be rapidly decompressed in, say, 2 min to atmospheric pressure without danger of resultant compressed air illness, however long the shift may have been. Decompressions are calculated so that, by the time LP reaches zero, SP will have fallen to 18 lb/in^2. Haldane considered 18 lb to be a reasonable value for *p*; this value has proved to be correct in the case of divers and is used in the following examples. Some people think it safe to shorten the decompressions of tunnel workers by using a higher value (e.g. 22 lb/in^2; 1.5 kg/cm^2) for *p*, while others believe that a lower value is necessary to ensure complete safety[1].

75T: That group of tissues in a man's body which becomes half-saturated or reaches an SP of 50% of the WP in 75 min.

40T, 20T, 10T, 5T: Those faster-saturating tissues which become half-saturated in the number of minutes indicated by the numeral.

To illustrate the method, let us begin with the case of men who have been working for 6 h or more at a WP of 25 lb/in^2 (1.8 kg/cm^2). For practical

[1] The term '*p*' (differently defined) was first used by Boulton, G. O., 'The use of air locks', *Journal of the Institute of Engineers, Australia*, **14** (1942).

TABLE 13.3

Initial rapid drops of pressure in decompression by the late Professor J. S. Haldane's system in the ratio of 2:1 (or rather more) of absolute pressure. Pressures given in lb/in^2

Working pressure	*May be reduced in 2 min to*	*Working pressure*	*May be reduced in 2 min to*
19	2	40	12
20	2	41	13
21	3	42	13
22	3	43	14
23	4	44	14
24	4	45	15
25	5	46	15
26	5	47	16
27	6	48	16
28	6	49	17
29	7	50	17
30	7	51	18
31	8	52	18
32	8	53	19
33	9	54	19
34	9	55	20
35	10	56	20
36	10	57	21
37	11	58	21
38	11	59	22
39	12	60	22

purposes the bodies can be considered as fully saturated—that is to say, the SPs of 75T, 40T, 20T, etc., have all reached 25 lb/in^2 (1.8 kg/cm^2). Our object is to reduce the LP at such a rate that by the time it has reached zero the SP in all parts of the body will have fallen to 18 lb/in^2 (1.2 kg/cm^2) ('*p*') or less. Since the process of desaturation follows the same course as that of saturation, 75T represents that part of the body which will be the last to reach the goal of SP 18 lb/in^2 (1.2 kg/cm^2), and we may disregard 40T, etc., knowing that by the time 75T has reached 18 lb/in^2 (1.2 kg/cm^2) they will have fallen to some still lower SP.

Table 13.3 shows that we can begin the men's decompression by allowing the LP to fall rapidly from 25 to 5 lb/in^2 (1.8 to 0.35 kg/cm^2) when the situation will be:

75T has SP 25 lb/in^2 (1.8 kg/cm^2), with LP 5 lb/in^2 (0.35 kg/cm^2) makes DP 20 lb/in^2 (1.5 kg/cm^2). Therefore: 75T must desaturate from SP 25 to 18 lb/in^2 (1.8 to 1.3 kg/cm^2) or by 7 lb/in^2 (0.5 kg/cm^2), which is 35% of the DP and (by Table 13.2) would require 46 min time if the LP remained steady at 5 lb/in^2 (0.35 kg/cm^2).

But, in practice, instead of maintaining the LP at 5 lb/in^2 (0.35 kg/cm^2), we allow it to fall at a uniform rate calculated to ensure that it will reach zero at the same time as the SP reaches 18 lb/in^2 (1.8 kg/cm^2). In the present case, while the LP is falling by 5 lb/in^2 (0.35 kg/cm^2), the SP must fall by 7 lb/in^2 (0.5 kg/cm^2). To find the time required for this amount of desaturation, using only simple arithmetic, we may suppose that the LP falls in a series of 1 lb/in^2 stages (0.007 kg/cm^2), remaining steady at each just long enough for the SP to fall by 1.4 lb/in^2 (0.1 kg/cm^2). Thus, in lb/in^2:

Stage	SP	LP	DP	SP to fall	DP (%)	Time (min)
1	25	5	20	1.4	7	8
2	23.6	4	19.6	1.4	7	8
3	22.2	3	19.2	1.4	7	8
4	20.8	2	18.8	1.4	7	8
5	19.4	1	18.4	1.4	8	9
					Total	41

Taking the case of men who have become fully saturated under a WP of 40 lb/in^2 (2.8 kg/cm^2), Table 13.3 shows that the LP may be rapidly reduced to 12 lb/in^2 (0.8 kg/cm^2). Then, while it is slowly falling from 12 lb/in^2 (0.8 kg/cm^2) to zero, the SP must fall from 40 to 18 lb/in^2 (2.8 to 1.3 kg/cm^2), or by an amount of 22 lb/in^2 (1.5 kg/cm^2), which we can conveniently divide up into eleven decrements of 1.8 lb/in^2 (0.1 kg/cm^2), and a twelfth of 2.2 lb/in^2 (0.2 kg/cm^2), and the working, briefly expressed, becomes, in lb/in^2:

SP	LP	DP	SP to fall	DP (%)	Time (min)
40	12	28	1.8	6.5	7.5
38.2	11	27.2	1.8	6.5	7.5
36.4	10	26.4	1.8	7	8
34.6	9	25.6	1.8	7	8
32.8	8	24.8	1.8	7	8
31	7	24	1.8	7.5	8.5
29.2	6	23.2	1.8	7.5	8.5
27.4	5	22.4	1.8	8	9
25.6	4	21.6	1.8	8.5	9.5
23.8	3	20.8	1.8	8.5	9.5
22	2	20	1.8	9	10
20.2	1	19.2	2.2	11.5	13
				Total	107

Short exposures

So far, we have only considered long exposures after which the desaturation of the slowest tissue, 75T, dominates the situation and the faster tissues

may be disregarded. After short exposures they must be taken into account. Thus, after a shift of 1½ h at WP 30 lb/in² (2.2 kg/cm²), Table 13.2 shows that the saturations of different parts of the body would be:

75T 57% of 30, or 17 lb/in²
40T 79% of 30, or 24 lb/in²
20T 96% of 30, or 29 lb/in²

Since 75T has not reached the danger limit p of 18 lb/in² (2.1 kg/cm²), it need not be considered at all, but 40T and 20T will both need gradual decompression. Inspection or a rough calculation shows that, although 20T is the more highly saturated of the two at the start, its faster rate of desaturation will reduce it to the lower SP of the two during the course of decompression; therefore, in this case we must base our calculation on 40T. Dropping to an LP of 7 lb/in² (0.5 kg/cm²) as shown by Table 13.3, we see that 40T has to fall from 24 to 18 lb/in² (1.8 to 1.3 kg/cm²), while LP is falling from 7 lb/in² (0.5 kg/cm²) to zero.

Dividing the decompression up into seven 1 lb stages as before, we get, in lb/in²:

SP	LP	DP	SP to fall	DP (%)	Time (min)
24	7	17	0.5	3	2
23.5	6	17.5	0.5	3	2
23	5	18	1	6	3
22	4	18	1	6	3
21	3	18	1	6	3
20	2	18	1	6	3
19	1	18	1	6	3
				Total	19

This ends the account by Damant.

Haldane issued three separate air diving tables. The first table (Fig. 13.3) was for all those dives requiring less than 30 min decompression time. The second table (Fig.13.4) was for all air dives requiring more than 30 min decompression time, and a third table was for deep air diving down to depths of 330 ft (100 m). All his decompression procedures were characterized by a rapid ascent from depth to the first one or two stages, followed by a slow staged ascent to the surface. This decompression profile represented a radical departure from previous practice, which almost invariably consisted of raising the diver at a fixed rate of a certain number of ft/min back to the surface, and it took the more conservative element in the diving world some years to recognize the value of Haldane's approach.

Haldane used some interesting auxiliary arguments to justify the rather dangerous-looking rush from depth to a first stage (or stop) and then the ever-increasingly conservative ascent back to the surface via the shallower stages. He pointed out that as it is generally believed a bubble is responsible for decompression sickness, then if we consider a small bubble forming at, say, 30 m (100 ft) i.e. 4 ATA, it is easy to see that it will double its size if the diver ascends through the water a distance of 20 m (66 ft) to a depth of 10 m (33 ft) i.e. 2 ATA. The pressure on the bubble has been halved and therefore the volume doubled. Similarly, if we imagine a small bubble at 2 ATA (33 ft, 10 m), then rapid ascent to the surface, i.e. 1 ATA, will also double the bubble's size. However, in the first case it has been necessary to ascend 20 m (66 ft) to achieve a doubling of size, whereas in the second case only an ascent of 10 m (33 ft) had been necessary.

Clearly, the nearer the surface the more the rate of expansion of any bubbles. From this it follows that we must be much more careful when decompressing near the surface. A linear decompression, as Haldane emphasized, does not take such possible bubble expansions into account, and is therefore potentially hazardous. However, the acid test of any procedure is, 'Does it work?' The answer became quite clear that for the range of depths and bottom times commonly employed in those days the Haldane tables were remarkably successful and virtually eliminated all of the various manifestations of decompression sickness, including the bends.

The Royal Navy adopted the Haldane tables in 1908, and the first tables developed for the USN, devised by French and Stillson in 1915, were based on the Haldanian concepts of a decompression ratio and also employed oxygen decompression to achieve depths between 200 and 300 ft (61 and 91 m). These tables were known as the Bureau of Construction and Repair Tables (C & R Tables). The C & R Tables were used successfully in the salvage of the sunken submarine, F4, at a depth of 306 ft (93.5 m) (French, 1916).

If the Haldane tables were so successful, why are we not using them today for air diving? Paradoxically the demise of the Haldane approach was due in large measure to its success. In providing

TABLE I.

Depth.		Pressure Pounds per Square Inch.	Time under Water, *i.e.*, from Surface to Beginning of Ascent.**	Stoppages in Minutes at different Depths.						Total Time for Ascent in Minutes.	Number of Cylinders needed. † ***	Revolutions of Pump per Minute.‡
Feet.	Fathoms.			60 ft.	50 ft.	40 ft.	30 ft.	20 ft.	10 ft.			
60–66	10–11	26½–29½	Up to 15 mins.	—	—	—	—	—	—	2	2	25
			15 to 30 mins....	—	—	—	—	—	5	7		
			30 to 48 mins....	—	—	—	—	2	8	12		
			48 to 60 mins....	—	—	—	—	3	10	15		
			1 to 1½ hrs. ...	—	—	—	—	4	13	19		
			1½ to 2 hrs. ...	—	—	—	—	5	15	22		
			2 to 2½ hrs. ...	—	—	—	—	5	20	27		
			Over 2½ hrs. ...	—	—	—	—	10	20	32		
66–72	11–12	29½–32	Up to 15 mins.	—	—	—	—	—	2	4	2	25
			15 to 25 mins....	—	—	—	—	2	4	8		
			25 to 30 mins....	—	—	—	—	3	5	10		
			30 to 45 mins....	—	—	—	—	4	9	15		
			¾ to 1 hr.... ...	—	—	—	—	5	12	19		
			1 to 1½ hrs. ...	—	—	—	—	8	16	26		
			Over 2 hrs. ...	—	—	—	—	10	20	32		
72–78	12–13	32–34½	Up to 10 mins.	—	—	—	—	—	3	5	2	25
			10 to 20 mins....	—	—	—	—	—	5	7		
			20 to 30 mins....	—	—	—	—	3	8	13		
			30 to 38 mins....	—	—	—	—	4	12	18		
			38 to 45 mins....	—	—	—	—	5	15	22		
			¾ to 1 hr.... ...	—	—	—	—	8	16	26		
			1 to 1¼ hrs. ...	—	—	—	—	9	18	29		
			1¼ to 1½ hrs. ...	—	—	—	—	10	20	32		
78–84	13–14	34½–37	Up to 10 mins.	—	—	—	—	—	3	5	2	30*
			10 to 20 mins....	—	—	—	—	—	5	7		
			20 to 30 mins....	—	—	—	—	3	8	13		
			30 to 40 mins....	—	—	—	—	4	13	19		
			40 to 45 mins....	—	—	—	—	5	15	22		
			45 to 55 mins....	—	—	—	—	8	16	26		
			55 to 65 mins....	—	—	—	—	9	18	29		
			65 to 75 mins....	—	—	—	—	10	20	32		
84–90	14–15	37–40	Up to 10 mins.	—	—	—	—	1	3	6	2	30*
			10 to 20 mins....	—	—	—	—	3	5	10		
			20 to 30 mins....	—	—	—	—	4	10	16		
			30 to 40 mins....	—	—	—	—	5	15	22		
			40 to 50 mins....	—	—	—	2	7	15	26		
			50 to 60 mins....	—	—	—	3	10	15	30		
90–96	15–16	40–42½	Up to 10 mins.	—	—	—	—	1	3	7	2	30*
			10 to 20 mins....	—	—	—	—	3	5	11		
			20 to 30 mins....	—	—	—	—	5	11	18		
			30 to 35 mins....	—	—	—	—	5	15	22		
			35 to 45 mins....	—	—	—	2	8	15	27		
			45 to 55 mins....	—	—	—	5	10	15	32		
96–108	16–18	42½–48	Up to 5 mins.	—	—	—	—	—	3	6	4	20
			5 to 10 mins. ...	—	—	—	—	—	5	8		
			10 to 15 mins....	—	—	—	—	3	5	11		
			15 to 20 mins....	—	—	—	—	4	8	15		

FIG. 13.3. A reproduction of Haldane's original Table I (see text)

procedures that almost eliminated decompression sickness as a diving hazard it became part of the advancing front in underwater technology that was liberating the diver from many of the constraints of his environment. With the development of more effective gas pumping and storage systems, and better-designed and more reliable diving suits, it became possible to descend deeper and stay longer

TABLE I.

Depth. Feet.	Depth. Fathoms.	Pressure Pounds per Square Inch.	Time under Water, *i.e.*, from Surface to Beginning of Ascent.**	Stoppages in Minutes at different Depths. 60 ft.	50 ft.	40 ft.	30 ft.	20 ft.	10 ft.	Total Time for Ascent in Minutes.	Number of Cylinders needed † ***	Revolutions of Pump per Minute.‡
96–108	16–18	42½–48	20 to 25 mins....	—	—	—	1	5	10	19	4	20
			25 to 30 mins....	—	—	—	3	7	10	23		
			30 to 35 mins....	—	—	—	4	8	13	28		
			35 to 40 mins....	—	—	—	5	10	15	33		
108–120	18–20	48–53½	Up to 5 mins....	—	—	—	—	—	4	7	4	20
			5 to 10 mins. ...	—	—	—	—	2	6	11		
			10 to 15 mins....	—	—	—	2	3	7	15		
			15 to 20 mins....	—	—	—	3	5	8	19		
			20 to 25 mins....	—	—	—	5	5	10	23		
			25 to 30 mins....	—	—	—	5	8	12	28		
			30 to 35 mins....	—	—	—	5	10	15	33		
120–132	20–22	53½–59	Up to 5 mins....	—	—	—	—	—	5	8	4	25
			5 to 10 mins. ...	—	—	—	—	3	7	13		
			10 to 15 mins....	—	—	—	2	5	7	17		
			15 to 20 mins....	—	—	—	3	7	10	23		
			20 to 25 mins....	—	—	—	4	8	13	28		
			25 to 30 mins....	—	—	—	5	10	15	33		
132–144	22–24	59–64½	Up to 6 mins....	—	—	—	—	2	5	10	4	25
			6 to 12 mins. ...	—	—	—	3	5	5	16		
			12 to 16 mins....	—	—	—	4	7	7	21		
			16 to 20 mins....	—	—	1	4	8	10	26		
			20 to 25 mins....	—	—	2	5	10	12	32		
144–156	24–26	64½–70	Up to 5 mins....	—	—	—	—	2	5	10	4	25
			5 to 10 mins. ...	—	—	—	3	5	5	16		
			10 to 15 mins....	—	—	1	4	7	8	23		
			15 to 20 mins....	—	2	3	5	8	10	31		
156–168	26–28	70–75	Up to 5 mins....	—	—	—	—	2	5	10	4	30*
			5 to 10 mins. ...	—	—	2	3	5	5	18		
			10 to 13 mins....	—	1	2	4	6	8	24		
			13 to 16 mins....	—	2	3	5	7	10	30		
168–180	28–30	75–80½	Up to 5 mins....	—	—	—	—	3	5	11	4	30*
			5 to 9 mins. ...	—	—	2	3	5	5	18		
			9 to 12 mins. ...	—	—	3	4	6	8	24		
			12 to 14 mins....	—	2	3	5	7	10	30		
180–192	30–32	80½–86	Up to 5 mins....	—	—	—	1	3	5	12	6	25
			5 to 10 mins. ...	—	1	2	3	6	8	23		
			10 to 13 mins....	—	2	3	5	7	10	30		
192–204	32–34	86–91½	Up to 7 mins....	—	2	2	3	5	5	20	6	25
			7 to 12 mins. ...	2	2	3	5	7	10	32		

* If found difficult to maintain 30 revolutions, a second cylinder may be used.
† These figures are calculated on the supposition that the pump does not leak more than 20 per cent. at pressures up to 60 lbs. Instructions as to testing of Pumps are given on pages 65 and 66.
‡ *i.e.*, using a Siebe-Gorman Two Cylinder Double-acting Pump.
** For instructions as to time for descent, see pages 89 and 90.
*** For actual quantities of air required at different depths, see page 87.

on the bottom than had previously been contemplated at the turn of the century. The urge to exploit the divers' capabilities came from both military and commercial interests. From the military standpoint the advancing importance of underwater warfare clearly demanded an investment in diving technology. The commercial interests very largely stemmed from the considerable prizes available from successfully salvaging valuable cargoes in sunken vessels. The more the Haldane tables were used, the more their inadequacies began to emerge.

Haldane's Table I is reproduced entirely in Fig.

TABLE II., showing Stoppages during Ascent after exceeding the Ordinary Limits of Time on the Bottom.

Depth.		Pressure in lbs. per sq. inch.	Time from leaving Surface to beginning of Ascent.	Stoppages at different Depths in Minutes.								Total Time for Ascent in Minutes.
Feet	Faths.			80 ft.	70 ft.	60 ft.	50 ft.	40 ft.	30 ft.	20 ft.	10 ft.	
66	11	29½	Over 3 hrs.	—	—	—	—	—	—	10	30	42
72	12	32	2 to 3 hrs....	—	—	—	—	—	—	10	30	42
			Over 3 hrs.	—	—	—	—	—	—	20	30	52
78	13	34½	1½ to 2½ hrs.	—	—	—	—	—	—	20	30	52
			Over 2½ hrs.	—	—	—	—	—	—	30	30	62
84	14	37	1¼ to 1½ hrs.	—	—	—	—	—	—	10	25	37
			1½ to 1¾ hrs.	—	—	—	—	—	—	10	30	42
			1¾ to 2 hrs.	—	—	—	—	—	—	15	30	47
			2 to 2¼ hrs.	—	—	—	—	—	—	20	30	52
			2¼ to 2½ hrs.	—	—	—	—	—	2	23	30	57
			2½ to 2¾ hrs.	—	—	—	—	—	3	27	30	62
			2¾ to 3 hrs.	—	—	—	—	—	5	30	30	67
			Over 3 hrs.	—	—	—	—	—	10	30	35	77
90	15	40	1 hr. to 1 hr. 12 mins.	—	—	—	—	—	5	10	20	37
			1 hr. 12 mins. to 1 hr. 20 mins.	—	—	—	—	—	5	15	20	42
			1 hr. 20 mins. to 1 hr. 30 mins.	—	—	—	—	—	5	15	25	47
			1 hr. 30 mins. to 1 hr. 44 mins.	—	—	—	—	—	5	20	25	52
			1 hr. 44 mins. to 2 hrs.	—	—	—	—	—	5	25	25	57
			2 hrs. to 2 hrs. 14 mins. ...	—	—	—	—	—	5	25	30	62
			2 hrs. 14 mins. to 2½ hrs. ...	—	—	—	—	—	5	30	30	67
			2½ hrs. to 2 hrs. 44 mins. ...	—	—	—	—	—	10	30	30	72
			2 hrs. 44 mins. to 3 hrs. 14 mins.	—	—	—	—	—	20	30	30	82
			Over 3 hrs. 14 mins.	—	—	—	—	—	20	35	35	92
96	16	42½	55 mins. to 1 hr. 12 mins. ...	—	—	—	—	—	5	10	25	42
			1 hr. 12 mins. to 1½ hrs. ...	—	—	—	—	—	5	15	30	52
			1½ hrs. to 1 hr. 54 mins. ...	—	—	—	—	—	5	25	30	62
			1 hr. 54 mins. to 2 hrs. 18 mins.	—	—	—	—	—	10	30	30	72
			2 hrs. 18 mins. to 2½ hrs. ...	—	—	—	—	—	10	30	35	77
			2½ hrs. to 2 hrs. 54 mins. ...	—	—	—	—	—	20	30	35	87
			Over 2 hrs. 54 mins.	—	—	—	—	—	30	35	35	102
108	18	48	40 to 50 mins.	—	—	—	—	—	8	10	20	41
			50 mins. to 1 hr.	—	—	—	—	—	10	15	20	48
			1 hr. to 1 hr. 18 mins.	—	—	—	—	—	10	20	25	58
			1 hr. 18 mins. to 1 hr. 44 mins.	—	—	—	—	—	15	20	35	73
			1 hr. 44 mins. to 2 hrs.	—	—	—	—	5	15	25	35	83
			2 hrs. to 2 hrs. 18 mins. ...	—	—	—	—	5	20	30	35	92
			2 hrs. 18 mins. to 2 hrs. 34 mins.	—	—	—	—	10	25	30	35	102
			2 hrs. 34 mins. to 2 hrs. 50 mins.	—	—	—	—	15	25	30	40	112
			Over 2 hrs. 50 mins.	—	—	—	—	15	30	35	40	122
120	20	53½	35 to 50 mins.	—	—	—	—	—	10	15	20	47
			50 mins. to 1 hr.	—	—	—	—	5	10	15	25	57
			1 hr. to 1 hr. 22 mins.	—	—	—	—	5	15	25	25	72
			1 hr. 22 mins. to 1 hr. 44 mins.	—	—	—	—	5	20	30	30	87
			1 hr. 44 mins. to 2 hrs.	—	—	—	—	10	20	30	35	97
			2 hrs. to 2 hrs. 22 mins. ...	—	—	—	—	15	25	35	35	112
			2 hrs. 22 mins. to 2 hrs. 44 mins.	—	—	—	—	20	30	35	40	127
			Over 2 hrs. 44 mins.	—	—	—	—	30	35	35	40	142

FIG. 13.4. A reproduction of part of Haldane's Table II (see text)

13.3. If a dive of depth and duration such as 25 min at 100 ft (61 m) depth is planned, then it is seen that decompression stages are required at 30, 20 and 10 ft (9, 6 and 3 m), involving a total decompression time of 19 min. Reference to the current USN Standard Air Diving Tables will reveal that 25 min at 100 ft (30 m) can be safely followed by direct ascent to the surface. The knowledge that most of Haldane's Table I was indubitably safe but unfortunately grossly oversafe gradually accumulated with practical experience over the years.

If, on the other hand, one wishes to do a relatively

long dive at 100 ft (30 m) with a 2 h bottom time it would be necessary to use Table II, part of which is reproduced (Fig. 13.4), and it will be seen that the total decompression time is 92 min for this particular dive. Again using the USN Standard Air Diving Tables for comparison with modern practice, it will be seen that such a dive requires just over 143 min decompression, although most diving supervisors faced with a dive of this nature would proceed one increment further on the diving table and give a decompression time of just over 202 min. The point to be noted is that the Haldane Table II (Fig. 13.4) is clearly giving grossly inadequate decompression for the longer and deeper dives. In the years between the promulgation of these tables in 1908 and the early 1930s, it had become apparent from practical usage that Table I was mostly oversafe and Table II was in many areas very undersafe. This led to a lack of confidence in the effectiveness of the Haldane tables and, in turn, this promoted the next phase of diving research to define the nature of the problem more accurately.

Before embarking on the next stage of the development of concepts related to the aetiology of decompression sickness, which were almost entirely confined to the diving situation, it is as well to be reminded that far greater numbers of men enter compressed air for work purposes in caissons and tunnels than for diving. The authorities promulgating regulations relating to work in compressed air by tunnellers and caisson workers are, in most countries, not the same as those responsible for diving regulations. Nevertheless, the medical and physiological problems encountered by men in these different areas of work are obviously very closely related. It is worth noting that between the two World Wars, Haldane's principles for decompressing tunnel workers were adopted by several groups and, as with the diving situation, as long as the pressure was comparatively low (below 25 lb/in^2 gauge) and the time at pressure not very great (not in excess of 4 h), then Haldane's decompression profiles worked satisfactorily. When longer shifts and greater pressures became commercially desirable, the Haldane tables could not meet the challenge, and the incidence of bends, and indeed more serious forms of decompression sickness, became too great for acceptance by the contractors in the compressed air industry. It will be seen later that the problems encountered by compressed air workers began to influence the diving scene in more recent times.

THE CONCEPTS OF THE US NAVY

For the next 25 years from 1932 diving research effort was almost entirely confined to the USN. The names of Behnke, Shilling, Van Der Aue, Des Granges and Dwyer are associated with a quite remarkable series of experiments, generally involving human volunteers, which helped to define some of the boundary conditions of the decompression problem in a quantitative manner for the first time.

In order to pursue a calculation using the Haldane method it is necessary to know the time constants (K value of equation 1) for all tissues involved in the decompression problem. This, in turn, involves a knowledge of the rates of blood perfusion of these tissues and the solubilities of nitrogen in them. As remarked earlier, this knowledge was certainly not available in Haldane's day, and indeed it is still very difficult to obtain reliable, agreed, data on many tissues even today. This means that Haldane's K values are what one would term 'informed guesses'. His value for the tissue with the longest half-time was derived, in part, from a study of the behaviour of his animals, and, as he himself was well aware, it is always unsatisfactory to extrapolate from animal results and apply them to men.

Therefore, before proceeding with the human experimentation it would be helpful to examine in greater detail the fundamental animal experiments from which the whole Haldanian set of ideas are derived. As will be recalled from the brief description of his experiments given earlier, he exposed animals to a constant raised pressure of air for periods of time as long as 2 h and then decompressed them to some lower pressure in order to establish his 2:1 ratio rule. One of the first doubts that arises concerns the duration of exposure required to equilibrate all the tissues of a goat at a constant raised pressure. Haldane considered that 2 h or thereabouts was quite sufficient, but one is soon led to enquire whether this is true. Many years later this particular problem was investigated in the following manner.

Goats were exposed to an excess pressure of air, say P_1, for a time, t min, and then rapidly compressed back to atmospheric pressure, where they were carefully watched to see whether an attack of bends followed this exposure. If no bends occurred, then on the next experiment some days later the same goat was given another exposure of duration t, but the pressure was increased to some new value generally 5 ft (1.5 m) greater. If bends

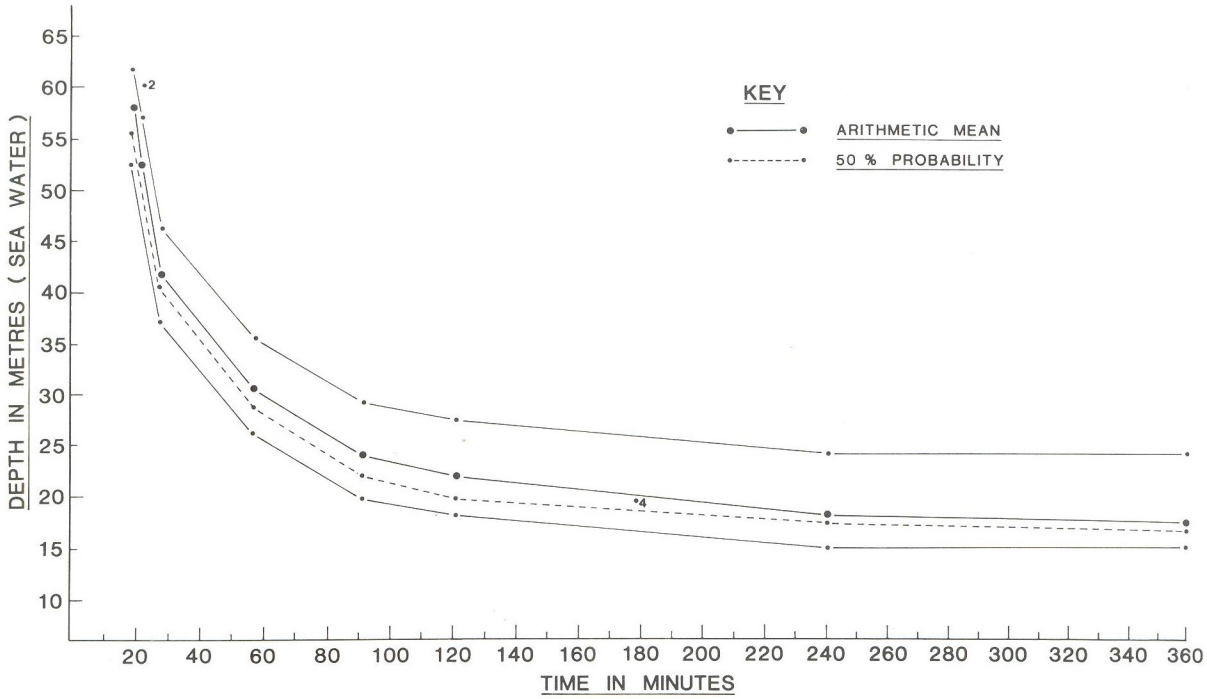

FIG. 13.5. The pressure–time relationship for the onset of mild limb bends in a population of air-breathing goats

did occur after the original exposure, then the experiment was repeated at a pressure 5 ft (1.5 m) lower in an attempt to obtain a trouble-free result.

In this way it was possible to obtain a threshold bend pressure for that particular duration of exposure in a given animal. Repeating all these observations for numbers of different animals gives a response pattern for a population of goats. Such a curve of performance is reproduced in Fig. 13.5. Several important points are immediately obvious from an examination of these results:

1. Deeper dives can only be safely performed if the duration of exposure is shortened.
2. After about 4 h exposure at pressure, and certainly after 6 h, there seems to be little or no change in the threshold bend pressure levels.
3. There is a wide variability in individual response at any particular pressure–time combination.

Such experimental results were not available to Haldane and, consequently, his idea that 2 h, or perhaps slightly more, would equilibrate all the tissues of a goat with gas at a constant raised pressure was unfortunately not correct, as can be seen by considering observation (2) above. There is clearly a difference between the threshold bend level at 3 h duration and the threshold level at 6 h. This forces recognition of the fact that longer half-times are involved in the decompression calculations than Haldane realized. In turn, this means that the decompression ratio concept was founded using animals with some of their tissues not equilibrated with gas, and renders the whole theoretical framework somewhat dubious. Thus, these later animal experiments revealed the underlying uncertainties in Haldane's concepts, and offer one possible explanation of the reasons why his tables were impractical for both short-duration and long-duration air dives. The tissue half-times had not been properly established, and the decompression ratio was probably not the value Haldane gave it.

By a different chain of reasoning these weaknesses in the Haldane approach became apparent to Shilling and his colleagues. In essence these USN experimenters undertook in the mid-1930s a set of exposures, using human volunteers, which were very similar to the much later goat experiments just described (Hawkins *et al.* 1935). Volunteers were exposed to raised pressures of air and then decompressed without decompression stages back to surface pressure (Table 13.4). As with the goats, so with the men; it can be seen that short exposures

TABLE 13.4

No. of tests	*Depth (ft)*	*Individual exposures*	*Initial exposure time (min)*	*Final time (min)*	*Decompression sickness*			
					Time to first case (min)	*No. of cases*	*No. of exposures before (first case)*	*after*
75	100	600	8.5	34.5	–	0	600	0
18	100	75	14.5	39.5	37.5	3	56	19
50	100	127	32.5	51.5	40.5	5	46	81
59	100	429	3.0	48.0	43.0	5	408	21
28	150	70	18.5	24.5	18.5	5	4	66
34	150	117	10.5	22.5	19.5	8	80	37
18	150	71	9.5	21.5	20.5	2	56	15
18	150	54	18.0	27.0	26.5	2	42	12
74	150	214	10.0	28.0	28.0	5	206	8
38	167	141	6.5	22.5	17.5	6	95	46
9	185	72	6.5	15.5	14.5	3	56	16
23	200	173	7.5	16.0	13.5	2	133	40
		2143				46	1782	361

allow a quite deep dive, whereas longer bottom times can only safely take place at shallower depths. This again suggests that perhaps there is a critical quantity of gas dissolved in the body tissues at pressure, and the longer one stays at a given pressure the more gas dissolves in body tissues and, hence, the more hazardous it becomes to lower the pressure. This, in outline, was Haldane's view, but whereas Haldane fixed every tissue half-time with a 2:1 ratio, this was clearly not tenable when actual human diving performance was analysed. Consequently, after Hawkins *et al.* analysed their findings in 1935, they concluded that each tissue half-time was associated with a particular unique decompression ratio: their theoretical analysis is given in Table 13.5. A little later Yarbrough (1937) reissued his own analysis of the data and decided that the 'fast' tissues, i.e. those with half-times of 5 or 10 min, could tolerate such large decompression ratios that in effect they could be ignored, and so he pressed ahead with the theoretical framework also given in Table 13.5. Decompression tables based upon 20, 40 and 75 min half-times, and using the lower value of the ratios for all dives to depths of 185 ft (56.5 m) or greater, were calculated by Yarbrough and issued for use by the USN in 1937. The ratios Yarbrough used for the 20, 40 and 75 min half-times were somewhat lower than those of Hawkins *et al.* (1935), because Yarbrough based them on dives involving exercise at depth which he concluded gave a greater loading of dissolved nitrogen to the tissues and this dictated a more cautious decompression. This could now be said to represent the most significant step in the progress of decompression research since the original work of Haldane. These USN tables gained world-wide acceptance alongside the original old Haldane tables, which were still in use despite their obvious deficiencies.

Let us now ask the same question of these tables as we did for the Haldane ones. Why are we not using them today? Once again, as with Haldane's tables, their downfall was a measure of their success. These new tables gave a great extension to the amount of no-stop diving, with all the consequent benefits of more useful work on the bottom and less air usage, etc. Nevertheless, when they were required for long bottom times at deeper depths, these tables suffered the same fate as Haldane's Table II, because they just did not offer sufficient protection from decompression sickness. This defectiveness was properly examined by Van der Aue in a prolonged and extensive series of experiments designed to give the USN the capabil-

TABLE 13.5

Tissue half-time	*Hawkins et al. (1935)*	*Yarbrough (1937)*
5	4.35	—
10	3.56	—
20	2.21	1.94–2.21
40	1.58	1.38–1.58
75	1.42–1.58	1.38–1.58

These decompression ratios do not treat air as a single gas, as Haldane did, but use the nitrogen pressure in the tissue. On this basis Haldane's old 2.0 ratio becomes 1.58.

ity of performing safe surface decompression diving.

It will be helpful to understand the problem facing Van Der Aue at that time. If a diver is sent down and works for a sufficient length of time, he can no longer ascend to the surface without needing decompression stages in the water. If then there should be some emergency requiring the diver to ascend immediately, he is in an extremely dangerous situation. It was decided that in this situation the diver should be rapidly decompressed through the water onto the deck of the support ship (surface decompression). Clearly, if he stayed on deck at atmospheric pressure, in these circumstances he would suffer a catastrophic attack of decompression sickness. The second part of this procedure was therefore to transfer the diver as rapidly as possible to a pressure chamber and recompress him as quickly as possible to avoid the impending attack of decompression sickness.

The immediate problems are (a) How much time can be spent between leaving the bottom to starting recompression on deck? (b) To what pressure must the diver be recompressed in the deck chamber. Back to the full pressure of his dive? Back to the pressure of what should have been his first stop if he had not made the emergency ascent, or to some intermediate value between these two extremes? (c) What decompression is necessary after settling questions (a) and (b)?

Take the last question first. Two opposed views present themselves. It could be said that because the diver had been grossly underdecompressed for part of this procedure, then he was close to decompression sickness and therefore required a decompression which ought to be very prolonged and close to a therapeutic recompression procedure. On the other hand, it could be argued that the rapid ascent to the surface had created a huge, though temporary, outflow of dissolved gas, and that if the recompression had been quick enough to prevent significant bubble formation, the diver would be in a much improved situation with far less nitrogen to remove during the subsequent decompression, which could therefore be shortened without loss of safety. An interesting dilemma which Van Der Aue solved in his own way.

The nature of his theoretical solution does not now concern us, but the practical procedure he employed to justify his approach became the starting point for the next step in decompression theory. He decided to compare the effectiveness of his proposed surface decompression procedures with the existing standard air diving procedures (Yarbrough) for the same bottom times and depths. The results of the comparison revealed that the Yarbrough tables were distinctly inadequate for dives such as 85 min at 100 ft (30 m), which gave 50% of his volunteers decompression sickness. As pointed out by Van Der Aue, this meant that the basis for the calculation of the Yarbrough tables needed urgent reappraisal. This important experimental work was completed in the period after World War II, up to 1951.

Again we must enquire why a set of procedures that are so patently inadequate managed to survive satisfactorily for so long. The answer is not far to seek when any official statistics on air diving accidents are examined. The vast majority of air dives are within the experimentally well-founded no-decompression limits and almost all the remainder of the diving involves only a few minutes' decompression. Such dives do not represent a test for the adequacy of the total theoretical framework, as they only involve short half-times. We have just seen that the great contribution of the Hawkins *et al.* group was to delineate the limits of short-duration diving, and, hence, not surprisingly, the Yarbrough tables were trouble-free with this type of diving. Unfortunately no similar experimental data had been assembled for long-duration dives, and therefore the calculations were necessarily informed guesswork. The guesses had clearly not been sufficiently near to the truth, as Van Der Aue discovered. However, these long-duration deep air dives were quite rare and therefore, although they invariably gave an unacceptable bends incidence, when tried they were submerged in any overall survey of the effectiveness of the Yarbrough tables by vast numbers of trouble-free no-stop diving results. This gave a false sense of security from just a glance at the statistics. Incidentally, this same weakness still applies to a great deal of current decompression data.

The next step, therefore, was to make some adjustment of the calculating procedures in order to keep the gains made by Yarbrough for the short-duration dives but render the decompression safer for long duration. A mathematician, J. V. Dwyer, was brought into the small team of Des Granges *et al.* (1956), who analysed the available diving data, and the Yarbrough calculations were completely revised. First, they concluded that deeper stops than previously used by Yarbrough were needed as

the depth and duration increased; in other words, the supersaturation ratio must be depth-dependent. Yarbrough had introduced the idea that the ratio was time-dependent, but this was insufficient. Second, they decided that Hawkins *et al.* had been correct in retaining the Haldane notion of tissues with half-times as short as 5 min, but of much greater historical significance is their adoption of a 120 min half-time tissue, which was considerably longer than had been considered necessary by Haldane (but see Van Der Aue *et al.* 1945).

In outline, the procedure they evolved for calculating the new tables was relatively simple but the computations involved were exceedingly tedious, and best left to a computer. The tables can be calculated as follows. The nitrogen tension in a tissue is obtained by the method given earlier in the Damant examples. Armed with this nitrogen tension and knowing the duration of the dive, one can obtain the depth of the decompression stop from a relationship which Dwyer proposed between the tension of nitrogen in a particular tissue and the permitted supersaturation ratio. In essence, then, we have various tissue half-times, each with a characteristic supersaturation ratio relationship which varies with the amount of dissolved gas in it. Note that the difference between Yarbrough and Dwyer is that Yarbrough gave a single fixed ratio to each tissue, but Dwyer gave a ratio relationship, dependent on depth, to each tissue. The calculation is therefore totally dependent on knowing the tissue half-times and a set of ratio relationships. It is now a matter of following the tensions in each tissue as the decompression proceeds and discovering which is the 'controlling' tissue at each of the stop values. This is easy to state, but if done by traditional methods, such as illustrated previously by the Damant examples, a vast amount of grinding arithmetic is involved.

Tables based on these concepts were adopted by the USN in 1956, and are still in use today. In fact, these tables have become the most widely used procedures to date.

Regrettably, the air decompression problem has still not been solved by these 1956 procedures. They represent another step forward from the Yarbrough tables but if duration is long, e.g. 1 h dives at depths in excess of about 120 ft (36 m), then too great a number of bends occur. Beckmann (1976) described a large air diving contract at depths of this order using the USN air diving tables, and it is quite clear from his experience that a re-examination of the theoretical basis to these tables must be undertaken if air diving by the USN is to be usefully extended in the future.

However, to illustrate the point made previously, it should be noted that when reporting on the 1956 tables the Naval Safety Center gave an overall incidence of bends of only 0.065% in 1976. Before leaving the present USN tables, it should be noted that there are separate calculations for exceptional exposures and that these calculations use additional half-times of 160 and 240 min, with allowable supersaturation ratios lower than the standard 120 min tissue ratios. On testing the schedule calculated for a 360 min bottom time dive to a depth of 140 ft (42.5 m) there were two serious bends cases among the six USN volunteer divers. Despite this somewhat discouraging result, the tables were issued, but only for emergency use.

THE WORKMAN '*M* VALUES'

All the previous systems of calculation used the concept of a maximum permitted supersaturation ratio. Now it is possible to regard this permitted supersaturation as a permitted excess pressure (pressure difference) rather than a pressure ratio. In view of the variation of allowable ratio values with both tissue half-time and tissue gas tension, it is not clear that a ratio concept is any better at expressing the controlling supersaturation value for a particular situation than a pressure difference. Clearly, a fixed pressure difference, as suggested by Rashbass (1958), will not suffice, any more than will a Haldanian fixed ratio, but it may be easier to vary the pressure difference concept to suit the data rather than varying the ratio. Workman (1965) investigated this possibility and evolved a calculating framework that has dominated many table calculations since he first advanced this system, and therefore this way of calculating schedules will be given in some detail. Anyone wishing to understand the theoretical basis of the available tables, commercial or military, will find that the Workman ideas are extensively used.

If an exposure to raised pressure takes place, then it is easy to calculate any tissue inert gas tension; call this P_T. Now, according to the Workman system, there is a unique value of P_T for each tissue which will allow the diver to ascend safely to a particular stop value. Suppose that we wish to ascend to a stop at a gauge depth D, then

TABLE 13.6
M values for nitrogen

Tissue half-time (min)	*Depth of decompression stop*									
	10	*20*	*30*	*40*	*50*	*60*	*70*	*80*	*90*	*100*
5	104	122	140	158	176	194	212	230	248	266
10	88	104	120	136	152	168	184	200	216	232
20	72	87	102	117	132	147	162	177	192	207
40	56	70	84	98	112	126	140	154	168	182
80	54	67	80	93	106	119	132	145	158	171
120	52	64	76	88	100	112	124	136	148	160
160	51	63	74	86	97	109	120	132	143	155
200	51	62	73	84	95	106	117	128	139	150
240	50	61	72	83	94	105	116	127	138	149

Maximum permissible tissue tensions (*M*), in feet of sea water, for ascent to next stop.

TABLE 13.7
Tissue half-time equations

Half-time (min)	*M*
5	$104+(1.8\times D)$
10	$88+(1.6\times D)$
20	$72+(1.5\times D)$
40	$56+(1.4\times D)$
80	$54+(1.3\times D)$
120	$52+(1.2\times D)$

Examples: (1) What is the *M* value for the 80 min tissue at a 20 ft stop?

$$M = 54+(1.3\times 20) = 80 \text{ ft}$$

(2) The 80 min tissue contains 80 ft of inert gas tension after completion of a dive; what is the stop to which this tissue can ascend safely?

$$\frac{80-54}{1.3} = 20 \text{ ft}$$

extensive analysis of available data reveals that in these critical values of P_T, referred to above, are called *M* values, then

$$M = M_0 + a \times D$$

where M_0 is the maximum permitted excess tissue inert gas tension allowed when $D = 0$, i.e. when the diver surfaces, and a is a constant which, like M_0, depends upon the tissue being considered.

The *M* values for various tissue half-times for any particular stop (Table 13.6) can easily be calculated from Table 13.7.

When calculating a decompression schedule for a particular dive, Workman would make an allowance for the ascent time to the first stop. This is a refinement which will be omitted in this explanation of his method.

Steps in the Calculation

1. Calculate the depth of the first stop, working to the nearest 10 ft (3 m) increment that is safe for the diver.

2. Take the absolute depth of this stop, $(D + 33)$ ft or $(D + 10)$ m, and calculate the inert gas partial pressure present at the stop, i.e. $(D + 33) \times F$, where F is the percentage of inert gas in the breathing gas, e.g. $F = 0.79$ for air.

3. Write down each tissue separately and note the value P_T, i.e. each tissue's inert gas tension, and each *M* value for the next lower stop. Find the inert gas 'extraction' pressure gradient, i.e. $P_T - (D + 33)F$, which is, of course, merely the difference between the inert gas pressure value in the tissue and that on the stop to which the tissue has been decompressed.

4. Calculate how much gas each tissue loses and discover when this allows the diver to ascend to the next shallower stop. To accomplish this aim it is first necessary to assess what fraction of the inert gas 'extraction' pressure gradient needs to be lost by each tissue, i.e.

$$f(\text{fraction to be lost}) = \frac{P_T - M}{P_T - (D+33)F}$$

When f is known, see what time this means for each tissue. For example, if $f = 0.5$ (for simplicity in explanation), this means that the pressure gradient needs to be halved, which by definition each tissue will accomplish in its half-time, i.e. 5 or 10 or 20 or 40 or 80 or 120 min.

The greatest time required to accomplish this fractional reduction is therefore the safe stop duration. In the example just given this would be 120 min.

Now the stop duration is known, use this to calculate the tissue gas tensions in each tissue. The controlling tissue will, of course, have a tension just below its *M* value, but all the other tissues will have values much less than their allowable *M* values for ascent to the next stop.

5. Ascend 10 ft (3 m) to a stop value of $D - 10$, and repeat the above calculations. Continue this procedure until the surfacing *M* value for the controlling tissue is reached at the final 10 ft (3 m) stop.

The original *M* values for using nitrogen gas or helium gas as the inert constituent of the diver's breathing gas are given in Tables 13.6 and 13.8.

Air or helium diving tables may now be calculated with relative ease, provided that sets of *M* values

TABLE 13.8
M values for helium

Tissue half-time (min)	*Depth of decompression stop*									
	10	*20*	*30*	*40*	*50*	*60*	*70*	*80*	*90*	*100*
5	86	101	116	131	146	161	176	191	206	221
10	74	88	102	116	130	144	158	172	186	200
20	66	79	92	105	118	131	144	157	170	183
40	60	72	84	96	108	120	132	144	156	168
80	56	68	80	92	104	116	128	140	152	164
120	54	66	78	90	102	114	126	138	150	162
160	54	65	76	87	98	109	120	131	142	153
200	53	63	73	83	93	103	113	123	133	143
240	53	63	73	83	93	103	113	123	133	143

Maximum allowable tissue tension, in feet of sea water (*M*), for ascent to next stop.

are available. If those who are calculating dive decompressions discover some inadequacy in the application of this system, then all they have to do is alter the table of *M* values to take account of their problem. As a calculating system this is infinitely versatile and could, with appropriate manipulations, accommodate any results established by diving trials. Consequently, this has become the most used basis for those discovering the complexities of the decompression problem and requiring quick answers to particular diving situations.

THE HEMPLEMAN CONCEPT

While the USN were engaged in revising the Yarbrough tables and preparing to introduce the 1956 tables, an interesting development was being pioneered elsewhere. In 1952 Hempleman suggested that a simple single-tissue approach could provide a satisfactory solution to the decompression problem. This idea, and some of the subsequent developments from it, will be outlined in order to illustrate several further points of importance.

It seemed to be curious that whenever a marginal case of decompression sickness occurred it resulted in a pain, in or around a joint. Furthermore, these characteristic pains (bends) could follow a short bottom time deep dive or a long-duration shallow dive. This was also true for animals, as the goat experiments described earlier reveal. This evidence strongly suggests that only one tissue type is principally involved in the bends and that there is a critical quantity of gas which this tissue can tolerate without pain.

However, if only one tissue is involved, and if the Haldane concept of tissue gas exchange described earlier is accepted, then the tissue saturates and desaturates with inert gas in a manner varying exponentially with time. If only one exponential, and therefore only one half-time, is available for consideration, it is quite impossible to fit the known data to such a model.

On examining a joint it is striking to observe that perfusion of some parts is very sparse indeed. Cartilage, for example, is attached to the bone surface and would seem to rely almost entirely for its nutriment on diffusion of molecules from the synovial membrane and the surrounding fluid. Once it is realized that diffusion is playing a dominating role in some tissues, rather than perfusion, as postulated by Haldane, then the whole concept of tissue inert gas exchange alters radically.

Let us simplify the physical factors involved. Consider cartilage as a slab of avascular tissue with one face of this slab well perfused by a network of blood vessels (synovial membrane). In essence, this is a thin layer of blood in contact with a thick layer of cartilage which permits the elementary physical laws of diffusion to be applied.

Suppose that at time $t = 0$ the diver is suddenly exposed to a raised pressure of inert gas which causes a step change in the concentration (tension) of inert gas being supplied to all organs of the body, and, in particular, to the blood flowing through the synovial membrane across the face of the slab of cartilage. Then we know from Fick's law of diffusion that at some distance x from the blood layer inside the cartilage the differential equation

$$\frac{\partial c}{\partial t} = K \times \frac{\partial^2 c}{\partial x^2}$$

where K is the diffusion coefficient, describes the movement of dissolved inert gas (c) in the slab of tissue. It would not be appropriate to discuss the various solutions to this diffusion equation for different possible boundary conditions, as they can be found in any standard mathematical textbook on differential equations. For the purposes of this model it was supposed that at time $t = 0$ the gas tension in the cartilage was some uniform value and at this moment the tension in the blood was suddenly changed and maintained at some new raised level. The fractional saturation of the slab (α) after time t can be written as:

$$\alpha = 1 - \frac{8}{\pi^2}\{e^{-kt} + \tfrac{1}{9}e^{-9kt} + \tfrac{1}{25}e^{-25kt} + \cdots\} \quad (4)$$

As can be seen, after infinite time $\alpha = 1$; and after a very short time, α is very nearly zero. Thus, this more complex diffusion function has the same basic properties as the simple single perfusion exponential function, but not the same time course. The k value is constant for a given thickness of the slab and for a diffusion coefficient that has the same value throughout the thickness of the slab. This is a rather idealized situation, but nevertheless worthy of consideration for modelling purposes.

When dissolved gas molecules diffuse uniformly from one face into a slab of tissue, they behave as if they were in a semi-infinite space until they begin to reach the opposite face, at which time the diffusion gradients become influenced by the fact that the gas molecules cannot diffuse any further. Up to this time, and indeed for an appreciable time afterwards, the quantity of gas diffusing into the tissue is proportional to the square root of the time. If this model is near enough correct for most practical purposes, and if there is a fixed critical excess quantity of gas which can be tolerated on decompression, then for a dive to a depth P for a time t there will be some critical fixed quantity Q of dissolved gas such that

$$Q = P\sqrt{t} \tag{5}$$

So if a diver goes to P_1 for t_1, or P_2 for t_2, then to remain marginally safe the following will be true:

$$P_1\sqrt{t_1} = P_2\sqrt{t_2} = Q \tag{6}$$

In fact, this relationship has proven to give a remarkably good fit of the USN no-stop dive data for all dives with bottom times less than 100 min. Table 13.9 compares the prediction of the formula with the data given in the manual. This is very encouraging, especially when one looks back on the rather complex calculations that generated the data in the USN Diving Manual. Consequently, Hempleman decided to use this model as the basis for calculating diving tables. However, it is much less versatile than the Workman M value treatment just described but it has a certain appealing simplicity. Once the k value is fixed in equation (4) above, the fractional saturation is also fixed and is solely a function of time, whereas with the M value scheme there are seven tissues available to fit the data!

One other important feature of the Hempleman system of calculating is also worth attention.

TABLE 13.9

Depth	*USN table*	*Q = 500 prediction*
50	100	100
60	60	69
70	50	51
80	40	39
90	30	31
100	25	25
110	20	21
120	15	17
190	5	7

Anyone who has taken part in experimental diving observes that some dives can cause a persistent low-level pain in a joint, which is variously called a 'niggle', 'inkle', etc. Such 'niggles' can sometimes remain with the diver for two or three days. An extreme example was provided by an Ocean Systems diver who Hamilton *et al.* (1966) reported as sensitive to flight in an unpressurized aircraft 10 days after completion of his saturation helium dive. On conventional Haldane ideas it would be necessary to assign a half-time to the tissue involved, which would be quite absurd. The dilemma is resolved 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 a bubble is not occluding the circulation.

Now once this concept of a bubble–tissue complex is accepted, it gives rise to doubts about whether such complexes are not also, to a lesser degree, influencing the whole decompression process. In effect, this would imply that the uptake and elimination of inert gases were not reversible processes. Therefore, when acquiring dissolved gas during the compression and bottom time Hempleman used one k value, but when decompression started, it was supposed that there was a change in the physics of the situation and the elimination became much reduced and the k value was altered to account for this.

Another argument also was used to support the idea of elimination being considered slower than uptake, and this was as follows. In any population there will obviously be those who acquire gas at pressure more rapidly than others, either because of their cardiovascular mechanisms and general physiology, or because they are exercising or some similar external factor. Equally, during decompres-

sion there will be those who eliminate dissolved gas rather more slowly than others. Now as decompression tables are being designed for a large population of different people, it is essential to take some account of this obvious truth. The 'safest' path is therefore to assume that the tables are dealing with the most rapid acquirers of gas and the slowest eliminators. Of necessity, therefore, an asymmetry must be introduced into the calculations.

Both the idea of a single-diffusion-limited tissue and the view that the rate of uptake of dissolved gas at pressure and its release on decompression are not the same were radical departures from the then current thinking. It might very reasonably be expected that such a very different theoretical basis would yield a vastly different set of decompression procedures, but, as we have seen already, this model generates a set of no-stop dives almost identical with that of the USN tables. The only noticeable differences arise when deeper, longer dives, e.g. 1 h at 100 ft (30 m), are being undertaken. For such dives the diffusion-limited model gives a more conservative decompression, and indeed the 1968 Air Diving Tables issued for general use by the Underwater Engineering Group (UK) have become very popular with contractors doing such arduous dives, as they undoubtedly lead to less decompression sickness than the USN tables, especially if the oxygen-stops version is used.

The important point for the impartial observer to notice is that there are two philosophies possible in approaching the decompression problem. The first philosophy can be summed up in the words: 'What does the model matter as long as it works?' The second philosophy is: 'If you don't understand what you're doing it's potentially dangerous, and the model must therefore reflect the major features of the physiology during decompression.' The first leads to a calculating system, e.g. M values; the second, to attempting to identify a tissue (or tissues) responsible, e.g. the cartilage diffusion concept.

THE HILLS CONCEPT

In order to satisfy the requirement that the tissue (or tissues) responsible for decompression sickness take several hours to equilibrate with gas at raised pressure, it was necessary, with the diffusion-limited model, to assume that we were dealing with a relatively thick avascular piece of tissue, e.g. cartilage, tendon, bone, etc. However, Hills (1966) challenged the accepted values of the diffusion coefficient for dissolved inert gases in tissues and used values 10^3 times smaller than the generally accepted Krogh values. This drastically alters the k values in the solution to the differential equation given above. Such very low diffusion coefficients imply very slow inert gas exchange between the blood in the capillaries and the intercapillary tissue, and consequently the time scale of the decompression process can now be accommodated by relatively well-vascularized tissue. Accordingly, Hills assumed for his model a central capillary with a surrounding cylinder of tissue. At time $t = 0$, when a dive commences, the blood in the capillary experiences a sustained step change in gas concentration which then diffuses outwards into the surrounding cylinder of tissue. The mathematical solution to this situation is rather more complex than for the tissue slab, but the square root relationship will still hold, as noted previously, for small t values.

It would seem that more recent measurements of the diffusion coefficient for dissolved inert gases in tissues do not support the very low values Hills used in his analysis, and consequently one is thrown back on the original idea of a 2 or 3 mm thick slice of avascular tissue in order to satisfy the time scales involved. However, in the course of his analysis Hills also drew attention to several important points, and some of these have become part of the current thinking.

When the gas tensions of the various dissolved gases in blood and tissue at atmospheric pressure are examined, an interesting feature emerges, as seen in Table 13.10.

The partial pressure of nitrogen in the alveoli must be in equilibrium with the dissolved nitrogen gas tensions throughout the body, but oxygen is being used metabolically and its tension therefore drops noticeably. Some extra carbon dioxide is, of course, generated from the metabolic usage of this oxygen but does not replace the used oxygen tension. Consequently, when the gas tensions are added together, it is found that this total does not equal the hydrostatic pressure (760 mmHg, in this case) on the body. Any small bubble introduced into a tissue would rapidly equilibrate with the gas tensions in the surrounding tissue and the total bubble pressure, being less than the outside pressure on the body, would mean that such a bubble would start to shrink and continue shrinking until it disappeared completely under the influence of this excess hydrostatic pressure.

TABLE 13.10
Gas tensions in blood and tissue at 1 ATM

	Oxygen pressure (mmHg)	*Nitrogen pressure (mmHg)*	*Carbon dioxide pressure (mmHg)*	*Water vapour pressure (mmHg)*
Air (dry)	152	608	—	—
Alveolar gas	103	569	41	47
	Oxygen tension	*Nitrogen tension*	*Carbon dioxide tension*	*Water vapour tension*
Arterial blood	88	569	41	47
Venous blood	37	569	47	47
Tissue cells	10	569	49	47

For simplicity the effects of surface tension have been ignored, but they would exert an extra pressure also tending to shrink the bubble.

The deficit in gas tensions in the various physiological situations, first noticed by Rahn (1961), was termed 'the inherent unsaturation' by Hills. Quite independently, Behnke (1951) also drew attention to the fact that oxygen usage created a disequilibrium in gas tensions and he called the deficit 'the oxygen window'. This idea of an 'oxygen window' or 'inherent unsaturation' has become a cornerstone in some of the attempts to quantify the decompression process. As has been pointed out above, it should lead to bubble shrinkage and is therefore porentially very useful in opposing the bubble growth that leads to decompression sickness. At raised pressures of air, for example, the inherent unsaturation can be extremely large. Consider a dive to 66 ft (20 m) using air. The total pressure is near enough 3 ATA and the oxygen pressure is very nearly 0.6 ATS. Owing to the fact that nearly all oxygen is carried in blood as oxyhaemoglobin, and very little is dissolved in the plasma, a simple calculation will show that although there has been a massive increase from the surface value of 0.2 to a level of 0.6 ATS in the oxygen pressure of the breathing gas, very little extra oxygen is carried to the tissues.

The situation from the tissue oxygen tension viewpoint is hardly affected, and consequently the 'inherent unsaturation' or 'oxygen window' value almost equals the oxygen pressure in the diver's breathing gas, i.e. 0.6 ATS.

If this concept is applicable to the decompression problem, then a factor of primary importance is available for consideration.

Before leaving this diffusion-dominated treatment of the tissue inert gas exchange, it is worth seeing how a fusion between perfusion- and diffusion-limited ideas is now occurring. As was pointed out by Hempleman (1963) and independently by Perl *et al.* (1965), it is misleading, given generally accepted values for the tissue diffusion coefficients of dissolved inert gases, to think of a tissue as an isolated unit with a particular response dependent solely upon its own characteristics. If the whole body is sectioned, one sometimes sees very well-vascularized organs placed next to quite poorly vascularized ones. It is a matter of elementary truth that if the well-vascularized tissue rapidly acquires dissolved inert gas and the poorly vascularized tissue only slowly acquires dissolved gas, then the 'fast' tissue will begin to act as a source of dissolved gas for the 'slow' tissue at the interface where the two tissues meet. One way in which molecules can transfer from the 'fast' to the 'slow' tissue is by diffusion.

Two points are apparent from this consideration. First, there are very few purely perfusion or purely diffusion-limited processes in the body; and second, every 'slow' tissue will have a 'fast' component and every 'fast' tissue will have a 'slow' component. From a computational viewpoint this means that very few, if any, tissues of the body can be regarded as saturating exponentially with time and having a simple single characteristic exponential half-time. It was the lack of understanding on this point that led to the rejection of the single-tissue concept. If a single tissue has only one half-time, then indeed the diving data cannot be explained by using such a model; but if a single tissue is liberated from this constraint by intertissue diffusion, then a single-

tissue perfusion-limited model is available for consideration.

CAISSON AND TUNNEL DECOMPRESSION PROCEDURES

So far the discussion on theoretical concepts underlying decompression procedures has been largely restricted to those used by divers. However, as mentioned earlier, there have been far more men breathing compressed air for caisson and tunnel work than for diving purposes. It is necessary to pay attention to their problems because they are closely interrelated to those of the diver. Furthermore, because of the large number of exposures to compressed air, the data tend to be more capable of statistical evaluation. A typical compressed air contract would have in excess of 250 000 entries into compressed air.

In 1954 Paton and Walder reported their findings from a compressed air contract concerned with the construction of a tunnel under the River Tyne, in England. The decompression tables used were those originally devised by Haldane, and the diving community would not be surprised to learn that large numbers of bends were encountered when shifts of a duration greater than 4 h were undertaken at pressures exceeding 22 lb/in^2 gauge (50 ft, 15 m). Accordingly, steps were taken to reduce the incidence of decompression sickness—for example, by lowering the threshold of 22 lb/in^2 gauge (50 ft, 15 m) stated by Haldane as requiring only 2 min decompression, i.e. virtually a no-stop exposure, to 18 lb/in^2 gauge (40 ft, 12 m). This is typical of the history of compressed air working in tunnels. A decompression procedure is adapted, it is tried and found inadequate in certain respects, and 'on the spot' alterations are made to attempt to remove the difficulties. This contract would, therefore, have been one of a series of entirely unremarkable undertakings had it not been for the observation that the incidence of bends varied quite markedly with time even when the working conditions (pressure, temperature, etc.) were quite stable. Various possibilities for this variation were examined.

For example, it was thought that perhaps some decompressions terminated with the atmospheric pressure at a relatively low value and this was expanding bubbles beyond their size at normal atmospheric pressure. Quite a reasonable supposition, but barometric pressure showed no correlation with the fluctuations in the numbers of bends. After investigation of several such hypotheses the only one that could be strongly supported by analysis was the relationship between the influx of new labour and the bends incidence. It became apparent that 'new starters' in compressed air were at a greater risk than those who had been regularly working in compressed air. They decided that this increased resistance to decompression sickness was due to 'acclimatization'. Since then other, perhaps more appropriate, words, e.g. 'adaptation', have been used to describe this phenomenon, but the fact is well established that regular exposure of the workforce can lead to a noticeable decrease in decompression incidence. Nevertheless, despite the unassailable evidence from this form of working, it would be highly speculative to assume that all forms of exposure to compressed gases will endow the same increased resistance.

In 1956 these same workers were joined by a small additional team, and attention was turned to examining the evidence from the Dartford (River Thames) Tunnel (Golding *et al.* 1960). Once again the 'acclimatization' factor as influencing the bends incidence was confirmed, and it was discovered that whenever there were long absences from regular work, the men had lost their acclimatization. Opportunity arose to analyse more closely the time scale for this process of deacclimatization and it was found that a 7 to 11 day half-time was appropriate. That is, if a group of workers were absent from regular work for about a week, then the bends incidence on return to work was about half-way between that of 'new starters' and that of fully acclimatized men (Fig. 13.6).

This was a very interesting finding, and although it added complications to understanding the aetiology of decompression sickness, it was an important practical point for consideration when assessing the success of decompression schedules.

At first the ability to 'acclimatize' men to working in raised pressures of air was seen as a highly desirable solution to the practical problem of keeping the bends incidence as low as possible. However, doubts began to arise about the wisdom of such procedures. It is by no means certain that avoiding an attack of acute decompression sickness also avoids all tissue damage. Perhaps these 'acclimatized' men should really be termed 'desensitized' men who are suffering gentle attacks of decompression sickness without feeling the pains they would have experienced if unacclimatized.

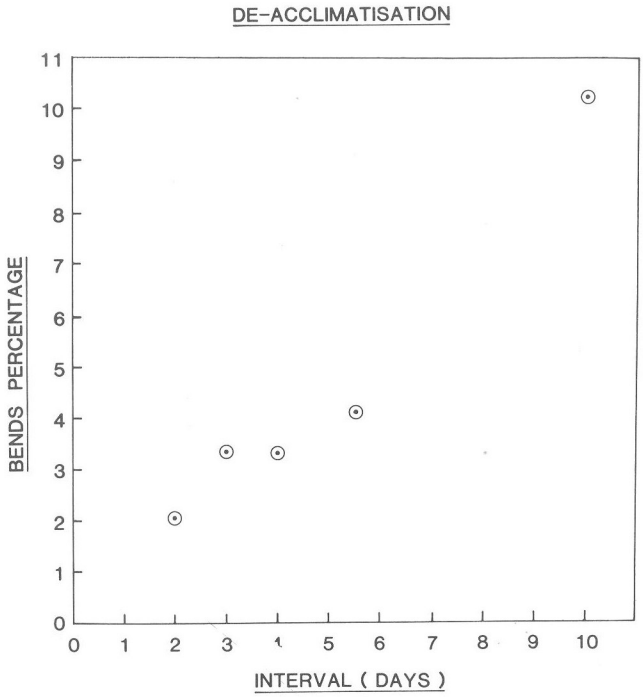

FIG. 13.6. Data showing the change in bends risk with time away from regular tunnel working. The trend is obvious but only the 2 day and 10 day points are firmly established statistically

Such thoughts receive considerable support from evidence, both indirect and direct (from ultrasonic bubble monitoring), that, even when decompression does not lead to an attack of bends, there are often 'silent bubbles' circulating in the blood or lodged in tissues. Consequently, many workers turned their attention to examining the chronic effects of decompression to uncover any adverse effects that had not presented during, or shortly after, the completion of decompression. There were two principal sources of concern. The first was the relationship between osteonecrosis and work in compressed air, and the second was the suggestion that the central nervous system was being irreversibly affected as a result of hyperbaric work.

Cases of painful and sometimes disabling osteonecrosis had been occasionally reported in caisson and tunnel workers from the turn of the century, but it was only the advent in 1965 of large-scale radiographic surveys that established that this disorder is quite widespread in these workers. The establishment of a link between hyperbaric exposure and subsequent osteonecrosis was one of the turning points in the history of hyperbaric medicine and, in particular, of decompression procedures. Osteonecrosis became far more worrying to both employers and employees than attacks of acute decompression sickness. The latter were relatively easily treated, and although a nuisance because they temporarily diverted the employers' resources on to nonproductive activities, they did not lead to the large claims for compensation as was the case when an employee developed osteonecrosis.

The discovery of large numbers of men with osteonecrosis in the tunnel and caisson industries naturally led to investigations of a similar nature in the diving industry. A rather different, and less worrying, picture emerged here. It was apparent that vast numbers of ordinary divers never suffered either acute attacks of decompression sickness or osteonecrosis. The main diving source of osteonecrosis was from either professional divers who indulged in repetitive air diving over many years, e.g. Japanese pearl fishers, or deep oxyhelium divers using inadequately established procedures. Several surveys of some such professional groups are given in Table 13.11 (see Chapter 20).

At first glance these results are clearly alarming, but two factors have since emerged which now render the problem less severe than might be thought from examining these gross data. First, there are various manifestations of osteonecrosis, and only juxta-articular involvement is the real hazard to the worker, and even these juxta-articular lesions will not always lead to breakdown of the articular surface and thus the necessity for surgical intervention. The second feature that began to be noticed was that the incidence of the serious forms of this disorder is certainly not as great nowadays in either divers or tunnel workers. For example, there were no recorded cases of osteonecrosis from the Seattle, San Francisco and Milwaukee tunnel projects, in the 1970–80 decade. The osteonecrosis problem drew attention to the way that decompression schedules had been constructed with only the

TABLE 13.11

Survey	*Divers*	*Divers with lesions*	*% Incidence*
Herget (1948)	47	13	34
Alnar (1963)	131	72	55
Ohta and Matsunaga (1974)	301	152	50
Beckman (1976)	30	8	27
	509	245	48

avoidance of bends as the objective. To avoid bend attacks is obviously highly desirable, and, as mentioned previously, if one can avoid the bends, then almost all forms of acute decompression sickness are reduced to negligible proportions, but it has become clear from the identification of osteonecrosis as a serious threat to hyperbaric work that a closer look must be given to the possibility of delayed effects.

A further example of delayed effects from apparently 'innocent' dives was afforded by the haematological investigations of Martin and Nichols (1972). Prior to their experiments it had been established by the Canadian workers Philp *et al.* (1971) that there can be haematological changes during the course of exposure to diving pressures and sometimes shortly afterwards.

However, Martin and Nichols took these observations a step further. They selected a standard dive which had always been free from decompression sickness, and which nevertheless was a quite substantial exposure to pressure, namely 1 h at 30 m (100 ft). They used the RNPL 1968 Air Diving Tables for the decompression procedure. No volunteers gave any indication of acute decompression sickness in the course of the experimentation and therefore this schedule was considered adequately safe by normal standards. Nevertheless, the platelet count did exhibit changes, not during or shortly after the dive but about 1 or 2 days later, and several days were needed for a complete return to the predive levels. The time course is illustrated in Fig. 13.7. These observations are discussed further in Chapter 17. It must now be realized that asymptomatic changes are taking place in the body despite apparently adequate decompression. More recently Nichols (1979) has been showing quite gross changes in the erythrocyte sedimentation rate (ESR) during the course of prolonged decompressions from both oxyhelium and oxynitrogen saturation diving. Some of these changes take as long as 3 weeks after completion of decompression before

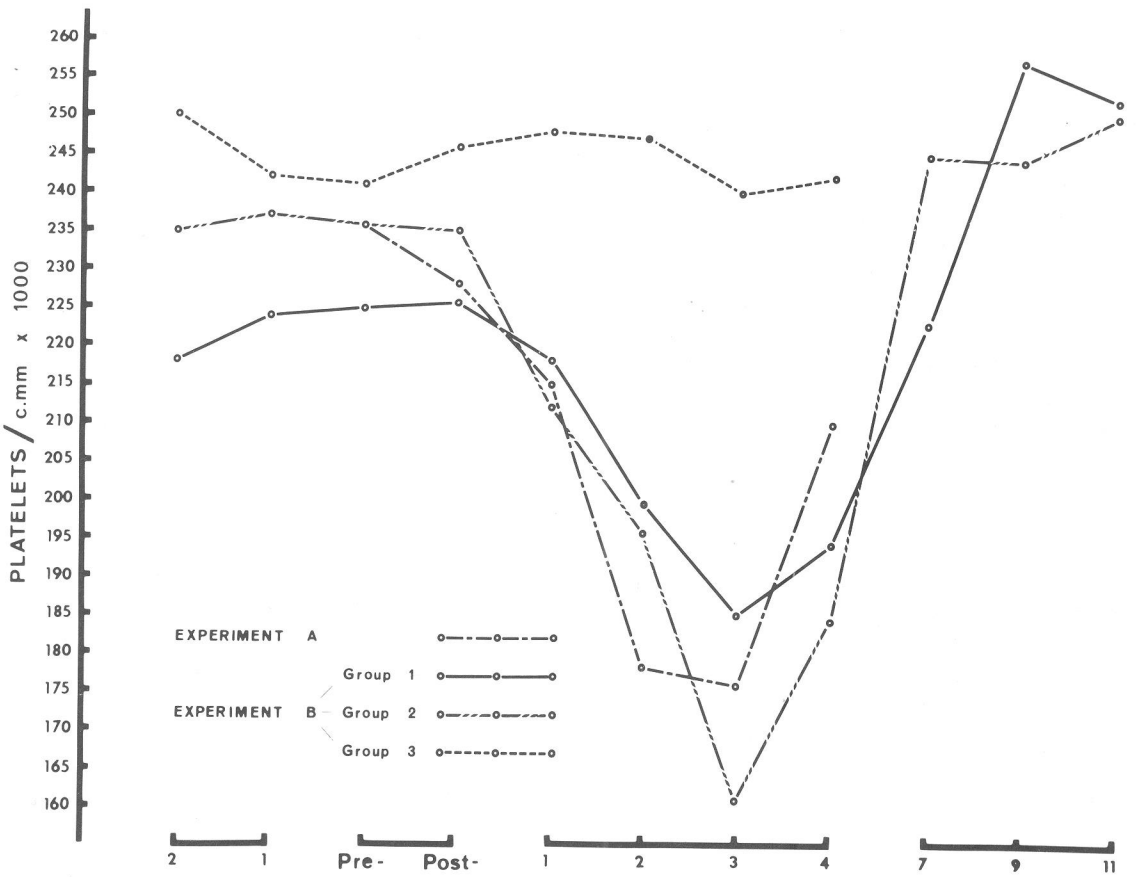

FIG. 13.7. The platelet count changes in groups of men given A, a fictitious control exposure, and B, a trouble-free chamber dive of 1 h duration at 30 m (100 ft). Each group involved six volunteers, except Group 2, with only five men

they return to pre-dive values. It is now a question of deciding whether such platelet and ESR changes have any real significance from the viewpoint of assessing the adequacy of decompression procedures.

In addition to the changes taking place in the bones and blood, there have been changes noted by Rozsahegyi (1959) attributable to central nervous system damage. However, it should be mentioned that the old Hungarian decompression procedures which led to these findings are very inadequate when judged by any of the theoretical considerations outlined earlier in this chapter. Nevertheless, it is clear from the work of Palmer (1979) that goats given bend-producing schedules do exhibit spinal cord damage which can be seen histologically in animals sacrificed several days, or weeks, post dive. Thus, there is further support for the view that hidden damage is taking place in a variety of body tissues and that the adaptability of the body is masking these problems except in those tissues where a pain results.

Detection of bubbles

To some extent these features of the decompression problem could have been anticipated, because it had been concluded as early as 1951 by Bateman and Behnke that asymptomatic bubbles must be produced from many diving procedures, and they termed such bubbles 'silent'. For many years the evidence for silent bubbles remained circumstantial, but the advent of ultrasonic monitoring techniques showed that asymptomatic bubbles could be detected in the bloodstream using Doppler ultrasonic probes.

Spencer and Campbell (1968) were foremost in establishing this technique as a major new contribution to the study of decompression procedures. Indeed, the efficacy of various diving tables has been compared by obtaining the numbers of bubbles generated per unit time, or the total number of bubbles as a measure. The classification of types of bubbles 'heard' has become more refined, and only certain types are considered to be significant by some workers. However, it does seem that although these ultrasonic bubble analysis techniques are useful with relatively short exposures to raised pressure, they are not very helpful in assessing the likely outcome of saturation schedules. The summary statement by Kenyon at the May 1974 Undersea Medical Society meeting effectively states the conclusion of most with experience in this field: 'Doppler ultrasound monitoring of the divers was conducted. At no time were bubbles detected during either the saturation excursions or during ascent to the surface. This was found also in the case of decompression sickness.'

Before leaving the use of ultrasound it is worth noting that the Doppler technique requires moving bubbles for detection, but that other techniques are available. In particular, there is ultrasonic imaging, pioneered by Rubissow and Mackay (1973), which can detect stationary bubbles and, as later adapted, is available for both moving and stationary bubbles. However, these techniques require extremely careful experimental measurements, and such factors as very small movements by the subject can disturb the interpretation of the findings. The conclusion would seem to be that ultrasound is a promising but not yet proven tool for investigating decompression procedures (see Chapter 16).

One principal point of interest from the above account is to note that Doppler ultrasound seems to give good prognoses for nonsaturation dives, but not for saturation exposures, whereas the ESR is in the reverse position, i.e. good indication of impending trouble for saturation diving but unsuitable for short bottom time dives. The significance of these facts is not yet apparent.

Isobaric counterdiffusion

In 1971 Blenkarn and co-workers observed a curious skin rash developing as a result of what they termed 'sequential breathing of various inert gases at 7 ATA'. The explanation they offered for this urticaria was that each inert gas had its own characteristic diffusion and tissue solubility properties, and, consequently, switching breathing mixtures at raised pressure gave enhanced possibilities for generating quite large gas concentration gradients inside the body. Large concentration gradients imply large osmotic pressure effects, with consequent movements of tissue fluid, and this was probably causing the skin effects.

The matter might well have rested on this explanation had it not been for some rather more dramatic experiments by Graves *et al.* (1974), who also subjected their volunteers to gas switching, but this time the pressure was much greater and equivalent to 1200 ft (366 m), and the gas being breathed within the chamber was oxyhelium but switching to oxyneon or oxynitrogen breathing took

place via a built-in breathing system. Thus, men were breathing one inert gas mixture while surrounded by another—in this case, helium. After a quite long delay period, sometimes 45 min, the men developed distressing symptoms and signs of vestibular disturbance. This was thought to be due to bubble formation caused by the presence of two inert gases diffusing in opposite directions and leading to a tissue supersaturation. The hypothesis is discussed in Chapter 15.

As may be seen, it is possible to reach a steady state with gas diffusing through a lipid layer with different solubility and diffusion properties from the adjacent aqueous layer and to envisage a total gas tension at the interface which could exceed the hydrostatic pressure by as much as 30% according to these calculations. Following this initial explanation, there have been several other ingenious attempts to account for isobaric bubble formation, and it has been well established that isobaric gas switching can lead to extensive and continuous bubble formation. For example, using ultrasonic Doppler probes to detect circulating bubbles, volunteers were exposed for several hours to compressed air at 132 ft (40 m) and then transferred with no change of pressure into a second chamber with an entirely new environment of oxyhelium gas. After about 30 min in this second chamber, circulating bubbles were readily detectable in the blood of all volunteers tested, and, indeed, one man developed bends from this procedure. Although various explanations of this phenomenon, which has come to be called 'counterdiffusion supersaturation' are available, the important practical point to note is that if the body is surrounded by helium gas at pressure, then it is very inadvisable to ask the diver, or patient, to breathe another inert gas mixture from a mouthpiece or helmet.

One matter that became unquestionably clearer from the isobaric counterdiffusion experimentation was that the release of free gas is the cause of decompression sickness and that not very large pressure differentials are needed to lead to large-scale bubble formation, provided that a sufficient supply of dissolved inert gas is made available in the tissues. Obviously, if bubble formation could be prevented or rendered more difficult in some way, then decompression sickness would vanish or become a very rare disorder. A search for understanding the origins of bubble formation has therefore been one of the main lines of investigation in decompression research. Unfortunately, the matter has not been resolved, but a good deal of evidence has accumulated and some of this is relevant to increasing awareness of the nature of the problem.

Micronuclei and bubble formation

If a beaker of pure water at constant temperature is exposed to a raised pressure of a gas such as nitrogen or helium, and sufficient time is allowed to elapse so that equilibrium is reached, at which point no more gas dissolves, then decompression to a much lower pressure will have no visible effect, i.e. no bubbles will be formed. For a bubble to form in these circumstances sufficient molecules have to come together in one small volume and the chances of this happening can be shown to be unworthy of consideration. However, if a foreign surface is introduced into the beaker upon decompression or the liquid is stirred, then violent gas release often takes place. All this type of experimentation leads to the conclusion that a 'nucleus' is necessary for promoting the formation of free gas from dissolved gas.

In 1944 Newton-Harvey demonstrated the same phenomenon in blood—namely, that it is impossible to provoke bubble formation in supersaturated blood, but when stirred, it can be made to effervesce profusely. He then went on to note that although he had demonstrated the virtual impossibility of producing bubbles in blood, nevertheless it was a matter of common observation that animals decompressed after exposure to only quite modest pressures of air often had bubbles visibly circulating in their vascular systems. This must mean, he decided, that the walls of the blood vessels have some property whereby they promote bubble formation, and he introduced the idea of crevices of gas within the vessel walls which remain permanently stable unless subjected to enormous pressures, and these 'gas nuclei' act as sources for formation of bubbles when dissolved gas diffuses into them. Furthermore, from experimentation with excised lengths of blood vessel it was decided that arterial vessels are most liable to be responsible.

At first thought it might seem dubious to implicate the arterial system as the source of bubbles in blood, because when an animal is decompressed, the arterial blood coming through the left side of the heart is in equilibrium, ignoring shunts with the alveolar gases, and therefore there is little or no supersaturation in such blood to promote bubble formation; and this assumption, as

we saw earlier, is the basis of all the current decompression calculating systems. However, it must be remembered that some arterial blood on its passage to those tissues which it supplies may pass through, or alongside, a tissue that contains a large concentration of dissolved inert gas. This dissolved gas will diffuse into the blood vessel and the arterial blood in that particular vessel may reach a far from negligible level of supersaturation. Thus, it is not impossible to conceive that arterial blood could be a source of bubbles, in conformity with Newton-Harvey's suggestion.

If bubbles are generated in water supersaturated with dissolved gas, there is quite convincing evidence that when the bubble redissolves, by reapplication of pressure or because the water becomes undersaturated, a small deposit of impurity is left behind. Should there be a further decompression, then a new bubble will form using this small deposit as its nucleus. From observations with a low-powered microscope Liebermann (1957) deduced that the deposit had a volume between 10^{-4} and 10^{-5} mm^3, and when the bubble was very small provided a 'skin' around the free gas. This 'skin' slows the diffusion rate from small (less than 0.1 mm diameter) bubbles and this slowing down can be readily demonstrated. Since this early work, and the theoretical analysis of bubble growth and decay by Epstein and Plesset (1950), there have been numerous refinements made in the observations, but basically there have been no major changes in the general conclusions. More recently the experiments of Yount (1978), using specially prepared sections of gelatine, have supported the organic skin concept.

Perhaps some of the most significant experiments were those by Evans and Walder (1969 and personal communication), who took shrimps (*Crangon crangon*) at atmospheric pressure and decompressed them to approximately 0.1 bar, at which pressure all the shrimps could be seen through their translucent shells to have bubbles. The shrimps were then sealed in a polythene bag full of sea water and compressed hydrostatically to about 100 bar. On decompression from this pressure back to atmospheric pressure very few of the shrimps could be made to exhibit bubbles on further decompression to 60 mmHg. It was concluded that the few minutes at high pressure had crushed most of the bubble nuclei and therefore prevented bubble formation. However, a very important further observation was made when it was found that if the shrimps were compressed and then decompressed hydrostatically but left about 4 h to recover, they bubbled profusely, when taken to the subatmospheric 60 mmHg pressure level. This shows that gas nuclei can be crushed out of existence but that either they re-form or new nuclei are born. This means that the whole process is, or can become, dynamic and that the body has a number of gas nuclei always present which it can replace or reconstitute every few hours. If these observations are confirmed, then it opens interesting possibilities for the aetiology of decompression sickness.

Also considered by the above investigators was the possibility that fissile material (uranium) in the diet was providing the particle energy necessary to create a small bubble. This is an interesting suggestion, and doubtless some bubbles could be generated in this way, but is not tenable as representing the principal aetiological mechanism.

Finally, there are those who believe that mechanical factors lead to bubble formation in supersaturated tissues. Two principal concepts dominate current thinking. First, it is possible that joint movement, which can involve very large shearing forces, or tribonucleation, i.e. movement of surfaces over one another, could bring gas out of solution. Second, it may be possible that vortical motion in the heart or at arterial bifurcations could cavitate the blood and cause regular injections of bubbles into the tissues. Both these suggestions are very reasonable.

It will be necessary to complete a number of quite subtle experiments to establish which of the various explanations really satisfies the evidence.

Once a bubble is formed, there is the further difficult problem of knowing how it grows or shrinks. For a given pressure gradient a helium bubble will probably grow more rapidly than a nitrogen bubble in aqueous tissue, but in fatty tissue the situation may be reversed. The problem with even attempting semiquantitative statements is that the diffusion coefficients and solubilities of the inert gases in the various tissues of the body are not known with sufficient accuracy. It is therefore possible to hold several views about bubble growth without any possibility of knowing which is nearest the truth. One of the main problems is that no-one yet knows where the offending bubble is located. Given such major uncertainties, it would not be sensible to enter into the detailed analysis. A few simple physical points can be made. If one doubles the pressure on a bubble of diameter greater than

10 μm, then Boyle's law will hold true and the volume will be halved, but if the bubble is spherical, then the diameter will decrease by only about one-fifth. Should the gas be released as a long cylindrical embolus in a blood vessel, then of course the length will almost halve. On the other hand, if the gas is a combination of both these possibilities, there could be considerable change within the tissue (see Fig. 13.8).

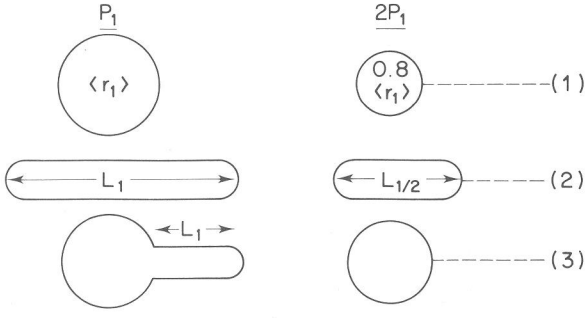

FIG. 13.8

One other point that must be mentioned is that if bubbles decrease to a diameter less than that of a capillary, i.e. about 10 μm, the surface tension inside such small bubbles starts to increase markedly unless surfactant substances are present, and this causes rapid dissolution of the bubble. Further, if the bubble is trapped in a blood vessel, then reducing its diameter to below 10 μm will cause it to move through the capillary bed.

In addition to these biophysical considerations, which have been discussed at some length, there are other approaches being used to achieve knowledge of the decompression process and, hence, to control it. Most noteworthy, of recent years, is the work of Chrysanthou (1973), which indicates that smooth muscle stimulating factors are implicated in the pathogenesis of decompression sickness. At present these studies have been restricted to experimenting with the more serious forms of decompression sickness and to working with small animals, but it will be interesting to see these concepts tested on man or large animals using mild decompression sickness as the end-point.

The complexities of the problem can be appreciated, even from these simple considerations. As seen earlier, the simple ratio principle of Haldane did not survive the test of usage and was replaced by mathematical manipulations designed to account for the undoubted fact that the permitted ratio was pressure-dependent. Although 2:1 was satisfactory for low-pressure values, around one atmosphere, it was far from safe to use this same ratio value at several atmospheres pressure. No controlled experimentation to discover the exact relationship between the ratio change and pressure was attempted until comparatively recently. In 1957 Hempleman exposed goats to raised pressures of air (P_1) for 6 h and then decompressed them rapidly to a new lower value (P_2), and waited at P_2 to see whether a bend occurred. In essence this was repeating Haldane's old experiments but using a much longer time of exposure in order to ensure that all tissues of the goat were equilibrated (saturated) to the raised pressure before decompression took place. From these experiments Hempleman concluded that Haldane was, near enough for practical purposes, correct and that P_1/P_2 was constant over a wide range of pressure values (Hempleman 1957). However, Hills (1966) examined the data critically and showed that a relationship of the form

$$P_1 = a\,P_2 + b \qquad (7)$$

much more accurately described the results. There have now been similar experiments using human volunteers and there is little doubt that a similar relationship describes these results also. For dives on oxyhelium to pressures as great as 300 ft (91 m) the equation

$$P_1 = 1.397\,P_2 + 5.7 \qquad (8)$$

satisfactorily describes the pressure P_2 in m to which one can rapidly, and safely, decompress after exposure to pressure P_1 m for at least 24 h with an oxygen partial pressure of 0.22 bar (i.e. normoxic). For deep helium diving the expression changes to

$$P_1 = 1.113\,P_2 + 24.1$$

This different equation is necessary because the form of decompression sickness presenting at deep depths from sudden drops in pressure changes from limb bends to mainly vestibular involvement. For air the relationship is

$$P_1 = 1.361\,P_2 + 3.4 \qquad (9)$$

This was deduced by Hennessy and Hempleman (1977) from the experimentally observed helium equation (8). In all these equations the P_1 and P_2 values are expressed in absolute terms—i.e. when $P_1 = 10$, the pressure is 1 bar, or near enough 1 ATS pressure. The various predictions for P_1 and P_2 using formula (9), and previous estimations by

others, are shown in Fig 13.9. This illustrates the way the decompression data are being refined and given more security as a basis for calculation and to help understand the aetiology.

Table safety

There are, as can be seen, several quite successful ways of constructing decompression tables despite the basic underlying physiological facts not being available. No-one yet knows the history of the origin of the bubble or bubbles that give rise to acute decompression sickness (Type I), nor is the site of action of these gaseous foreign bodies established. Added to these fundamental areas of ignorance are lesser, but important, ones such as whether an attack of the bends can lead, or be an indicator of, impending bone necrosis or CNS damage. From the viewpoint of truly understanding the nature of the decompression problem the present level of knowledge is grossly inadequate.

Of the various tables available for use the following account should be taken as guidance.

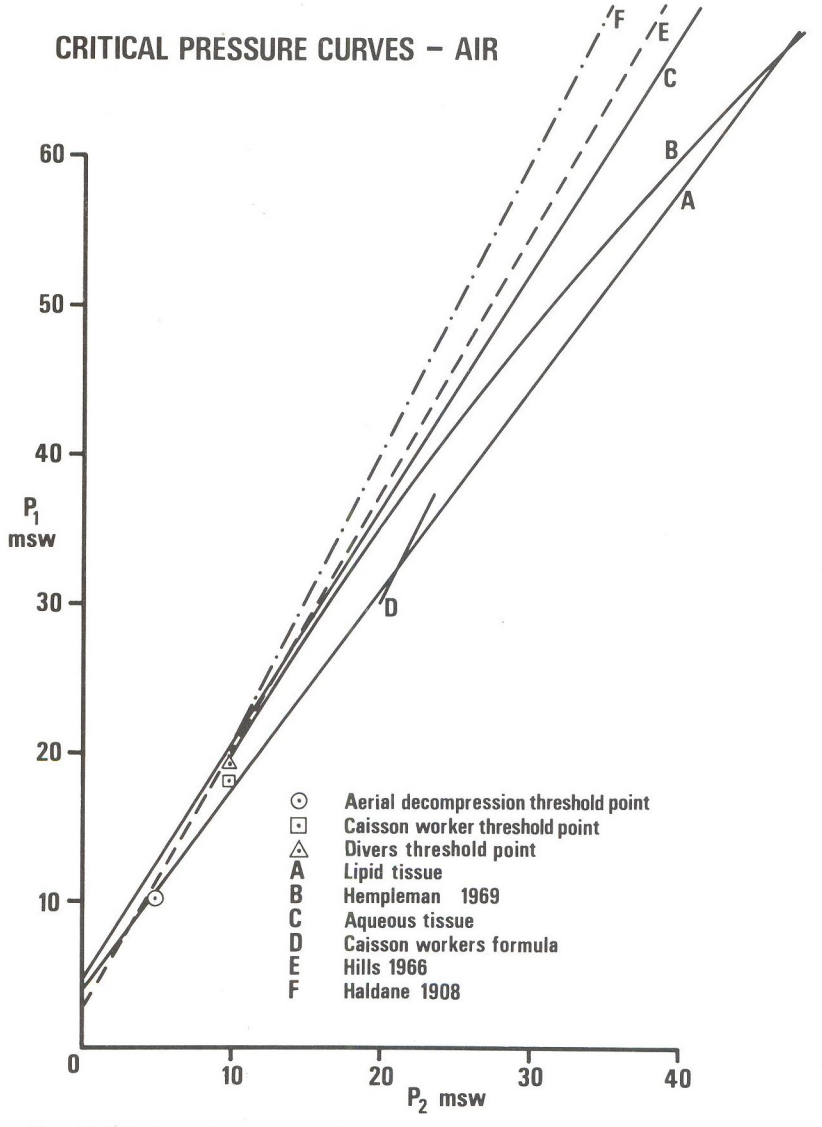

FIG. 13.9. Prolonged exposure to air at pressure P_1, followed by rapid ascent to a new lower pressure, P_2, is best described by a simple straight line. Various interpretations of this linear relationship are plotted above

Most of the air diving tables from the major navies of the world (USN, French navy, RN) are very satisfactory for the vast majority of air diving work (or sport). Indeed, comparing the bottom times, depths and decompression times, the similarity in international decompression requirements is obvious. However, if the decompression requirement exceeds about 1 h, then all of these tables will begin to approach, or even greatly exceed, 1% bends incidence. At this point it has been found by Beckman (1976) that resort to extra decompression is mandatory and he recommends following the 1968 Air Diving Tables issued in the UK by the Construction Industry's Research and Information Association, and using their oxygen breathing Table II. Even these tables fail for really prolonged exposures to air pressures and it is then necessary to consult the *NOAA Diving Manual*.

Oxyhelium diving is now largely a commercial offshore activity and the various major diving firms have evolved their own decompression procedures, generally designed around particular forms of diving practice. Their basic theories are most frequently some form of Workman's M value concepts which were described earlier in this chapter. Experience has dictated which M values give least trouble in a given set of circumstances. The USN have had a set of 'bounce' (i.e. short bottom time) helium diving tables for many years, but experience shows that using these helium partial pressure tables is not successful at depths greater than about 300 ft (90 m) for bottom times in excess of 20 min. The commercial 'bounce' tables appear better able to cope with this form of diving but are normally labelled 'commercial-in-confidence' and it is difficult to obtain any objective appraisal of their success. However, it does seem that no groups perform regular 'bounce' diving to depths in excess of 150 m (500 ft) and that bottom times exceeding 1 h are not yet considered sufficiently trouble-free at depths of 80 m (250 ft) or greater.

Saturation and excursion decompression

For prolonged diving in excess of 50 m (165 ft) it is not worth considering any technique other than 'saturation diving'. There are two basic forms of saturation diving. In the first form the diver is placed in his submersible chamber at the pressure of the work site and he stays at this nearly constant pressure for as many days as it is necessary for him to complete the job. His tissues are completely equilibrated (saturated) at this pressure and he now only requires a single prolonged decompression back to the surface. Clearly, the longer the bottom working period the more worthwhile this technique becomes. The second form of saturation diving occurs when the diver is placed in his submersible chamber at a pressure lower than that of the work site, and as he goes out to work on the job, he descends to this greater pressure for a working period of up to 8 h, after which he returns, without need for decompression, back into the submersible chamber.

These descents to and from the work site are termed 'excursions', and excursion diving is very popular when there are difficulties in placing the chamber alongside the work site. This 'excursion' diving requires knowledge of the no-stop dive relationships for all the various 'holding' or 'storage' depths to which the diver returns after his work at the site. For example, if the diver is being held, or 'stored', at a depth of 60 m (200 ft) and he descends to work for 3 h, what is the greatest depth at which he can safely work and return without stops back to 60 m (200 ft)? Suppose now we imagine such a 3 h work shift being required four times a day. How does this alter the depth to which the diver can be expected to work safely? The variations in possible bottom times, rest periods at the holding pressure and the holding pressure itself are infinite and could never be issued as tables for general use.

This becomes even more apparent if during the excursion the diver breathes a gas of different composition from that in the chamber at the storage depth. The complexities are so enormous that only guidance tables for certain particular situations have been issued. For nitrogen–oxygen diving NOAA have evolved various suitable procedures and for oxyhelium diving the USN, RN and various large commercial groupings (e.g. COMEX, Taylor Diving, Oceaneering) have also suitable techniques. The student of this scene soon realizes that a suitable computer program is the only satisfactory solution to the very diverse set of possibilities. However, the real difficulty is basing the computer program on a reliable mathematical model, which has been one of the principal topics of consideration in this chapter, and, as can be seen, there is, as yet, no sufficiently versatile theory available.

Furthermore, it is not good enough just to reach a satisfactory calculating system that has no physiological basis. This may be illustrated by the

shoulder joint, which was considered earlier in this chapter to show that the synovial membrane/slab of cartilage was a reasonable model. It requires very little extra speculation to suppose that inert gas would dissolve in other areas of the joint and, in particular, inside the bony capsule at the head of the joint, which is largely composed of fatty tissue and is relatively avascular. Such a tissue would take a very long time to saturate and desaturate with gas and a large pressure could be generated inside the bone which would indubitably cause pain. Indeed, bubbles inside a bony capsule with long half-times involved would explain why ultrasound does not detect the occurrence of decompression sickness from saturation diving. It is impossible to 'see' inside a bone by using ultrasound, and the aetiological agent would therefore be undetectable by this technique. On the other hand, the presence of bubbles inside the bone, with the consequent generation of pressure, would interfere with bone marrow function and lead to the haematological changes that have been observed. Also it could be supposed that repeated insults of this type must lead to permanent bone damage, i.e. osteonecrosis. The whole picture can be seen to accord with most, if not all, of the relevant available data, and it would be a trivial mathematical problem to generate several calculating systems from this basic model, one of which would doubtless be as good as any available at present!

SUMMARY

The day is not far distant when the theoretician will be able to offer divers or tunnel workers a set of versatile concepts which will render all forms of decompression sickness very rare events. It will be many years, however, before it is known whether these theoretical concepts are optimized for the great bulk of mankind and thus give the least time-consuming and safest possible pressure–time courses for returning to atmospheric pressure. As may readily be appreciated, it is a monumental task to achieve optimized safe procedures for males, females, fat or thin, fit or unfit, working hard or resting, in cold or warm water, breathing various partial pressures of inert gases and oxygen, with short or long bottom times, during single or complex repetitive dive routines. Perhaps the practical answer will arrive with some device which can detect the very first indications of impending trouble. The optimal pressure–time course could then be monitored on an individual basis, and avoid the necessity for understanding detailed physiological mechanisms.

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