IV

Decompression Theory

A. *Introduction*

Following the commencement of the industrial revolution it became possible for human beings to manipulate the physics and chemistry of their environment on a grand scale. This led to the appearance of a large number of man-made disorders, for many of which the causative agent is easily established; the mechanism whereby the body reacts to these agents is, however, 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 is therefore to not venture outside the limits of air composition and partial pressures normally encountered on the surface of the earth. A second way to eliminate the harmful effects of decompression sickness is to understand the responsible mechanisms and from this understanding to construct safe procedures. It is, of course, this second possibility that is examined in this chapter.

The first successful pump for exhausting the air from a container was invented in the 17th 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.

For work underwater, gases at pressures greater than atmospheric pressure 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 by 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 Edmund Halley (of comet fame) designed, built, and used the first practical "bell' system

(Halley 1717). An artist's impression of this bell is given in Figure IV-1, and a brief description of this device serves to illustrate some of the difficulties facing the theoretician when he attempts to collect reliable data as a basis for his ideas. Halley's bell was constructed of wood and lined on the exterior surface with lead sheeting in order to give sufficient weight and stability to the bell when underwater. It had a cubic capacity of nearly 60 ft^3 (1.7 m^3) and was approximately 3 ft (0.9 m) in diameter at the top. It was realized that with two men in the bell when it was immersed in water the air contained within the bell would become foul. Drawing on more recent knowledge one can easily calculate that

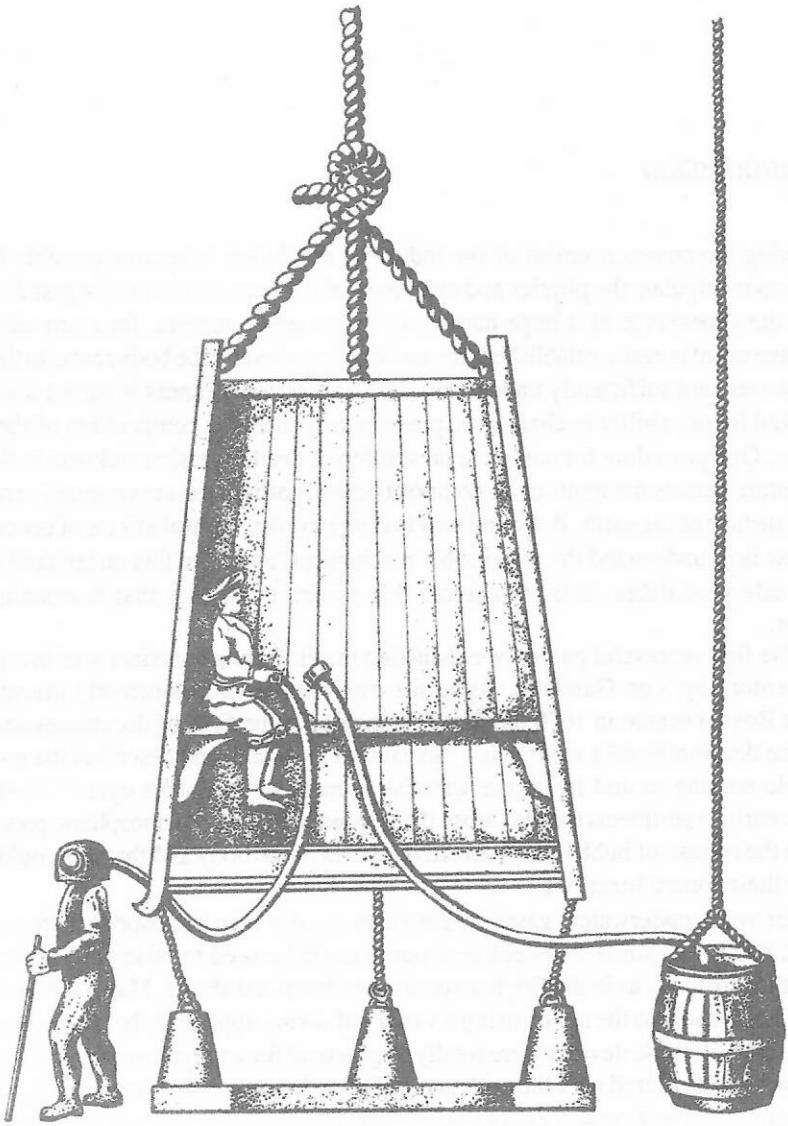

Figure IV-1. Artist's conception of Edmund Halley's diving bell (ca. 1717). [Adapted from Davis (1981).]

with two active men inside a bell the carbon dioxide concentration would reach about 3% in 1 hr, and this would be the useful duration for such a bell without some form of air replenishment. Fresh air, in this case, was supplied to the bell from lead-lined barrels having bung holes at the top and the bottom and a leather tube through which air could be forced from the barrels into the bell. After being emptied of air these barrels were hauled to the surface, where their air content was renewed, and thus the whole process was a continuous one. Depths as great as 60 ft (18 m) for dive durations as long as 1 1/2 hr were attained by using this particular technique.

Several important physiological points relevant to the theoretician should be noted. First, 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 that is clearly giving added respiratory airflow "drag." He is rebreathing expired air. His head, neck, shoulders, and particularly his respiratory passages are at risk of squeeze effects if his helmet is attached to his suit. If his helmet is open ended and not equipped with a nonreturn valve he is in danger of drowning upon any lowering of his helmet below the water level of the bell. (In contrast, any raising of his helmet above the bell's water level will produce a free flow of air into the helmet, with relief from the foul air and possible squeeze effects.) And with immersion 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 likely to be performing very little work (e.g., the seated attendant, who may or may not be pumping air into the diver's own hose by means of bellows), but others (e.g., the diver) are required to perform quite hard physical work. Fourth, human beings are very different in important factors such as stature, body composition, and level of physical fitness. Finally, and of extreme importance in 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 discipline.

Placing human beings under raised pressures of air can conveniently be considered divisible into four separate phases, each of which has its own particular set of problems. The first phase is taking the person to pressure, and this compression phase sometimes causes the establishment of pressure differentials in body cavities such as the sinuses and the middle ear, producing in these instances sinus and ear 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, carbon dioxide, toxic gas contaminants such as carbon monoxide, and the inert gas nitrogen, any or all of which can give rise to numerous difficulties (e.g., oxygen toxicity, nitrogen narcosis, carbon dioxide intoxication, carbon monoxide toxicity), 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. A note of caution must

be introduced because, as becomes 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, let us 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 bar (psi, kg/cm^2) per minute for the caisson and tunnel workers or a given number of meters (ft, fathoms) of ascent per minute for the diver. The practical problem in those days was to decide the most effective rate of pressure release.

B. *Defining the Problem*

An understanding of the basic processes producing the harmful effects of decompression was lacking until the time of Paul Bert (1878), 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, within body tissue. Furthermore, after careful analysis of the composition of these bubbles he concluded that nitrogen gas was the main constituent. Thus an outline picture of the etiology of decompression sickness could now be attempted: It is apparently caused by the release of gas emboli from nitrogen gas dissolved at pressure, and these nitrogen gas emboli then impair the functioning of the various tissues in which they lodge or are 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; this too was a very serious manifestation of decompression sickness that 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 River at St. Louis (1869–1874), the name referring to the affected gait that was fashionable among the young ladies of the time and apparently bore a resemblance to the behavior of those workmen who contracted the less serious, but painful, forms of decompression sickness (Jaminet 1871). The term nowadays refers to pain in or around a joint that 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 France and England, it was the massive undertakings in the United States that provided a sound statistical basis for examining the frequency of occurrence of the various forms of decompression sickness. Table IV-1 gives the data on one such contract; these data, resulting from several years' work and involving more than one million decompressions, were reported in 1912 by Keays (see Keays 1912). As may be seen, by far the greatest number of decompression sickness incidents were attributed to the bends, and it would

Table IV-1
Frequency of Symptoms of Decompression Sickness[a]

Symptom	Number of incidents	Percent
Bends (joint pain)	3278	88.78
Bends with local manifestations	9	0.26
Pain with prostration	47	1.26
Central nervous system symptoms		
Hemiplegia	4	0.11
Spinal cord symptoms	80	2.16
Vertigo (staggers)	197	5.33
Dyspnea (chokes)	60	1.62
Partial or complete unconsciousness	17	0.46

[a]From data by Keays (1912).

seem a reasonable assumption that if the decompression procedures could be arranged to avoid attacks of 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 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 was either 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 J. S. Haldane for a series of investigations specifically aimed at reaching 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 that would certainly have a risk of the occurrence of serious decompression sickness from time to time, it was necessary to search for a suitable animal model. Consequently a great variety of animals were exposed to raised pressure 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 must exhibit a marginal form of decompression sickness that could be clearly identified as a pain in a joint and therefore could 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 the 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-to-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; it is a tribute to Haldane's selection process that even nowadays the goat is still considered useful 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 Figure IV-2, and as may be seen, the data give good support to considering the goat to be a suitable animal model.

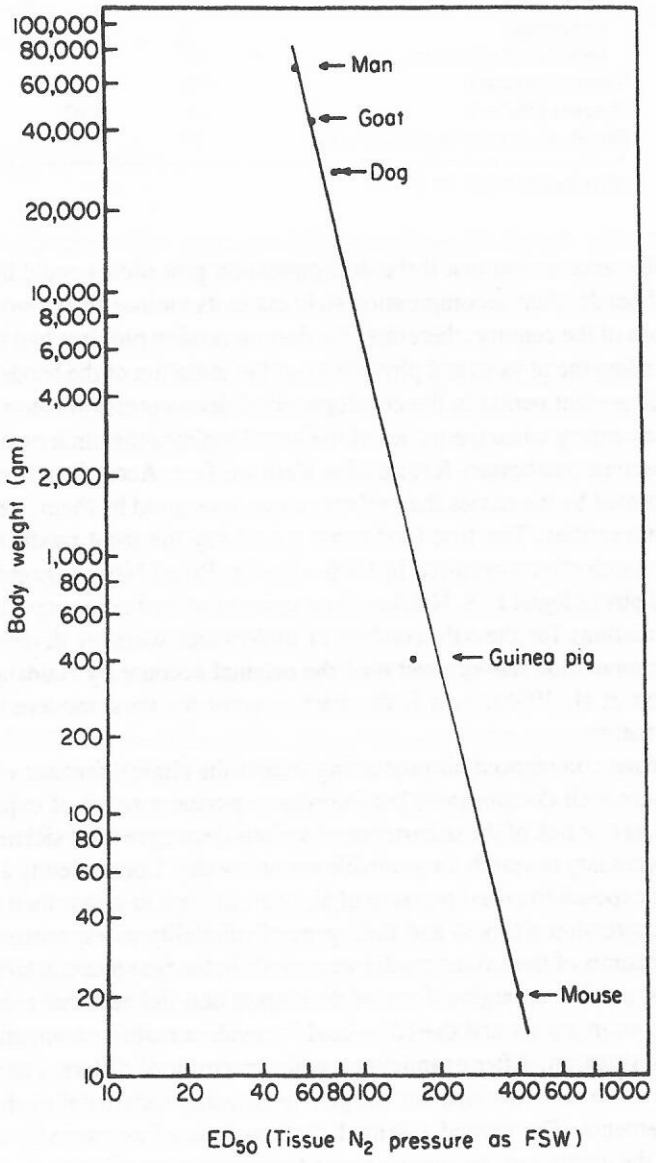

Figure IV-2. Relation of body weight (size) to sensitivity to decompression sickness. [From Flynn and Lambertsen (1971).]

C. *The Haldane Concepts*

Having chosen a suitable animal it was now necessary to discover the ground rules relating to the appearance and nonappearance 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, the tendency to form bubbles could 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 rapidly dropped to some new value, P_2, would the animal exhibit attacks of decompression sickness if $P_1 - P_2$ remained constant? Or perhaps some other relationship between P_1 and P_2 could be established. With this idea in mind Haldane and his co-workers exposed their goats for what they considered prolonged periods of time (1.5–2 hr) at raised pressures, then rapidly decompressed them to some 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 gauge pressure equivalent to pressure of about 45 fsw (2.36 ATA) 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 afterward. It was decided therefore that a pressure difference of just over 1 atm pressure could be safely tolerated by all normal animals. The exposure pressure was next altered to 6 ATA (pressure of 165 fsw) and a rapid pressure drop of just over 1 atm (from 6 to 5) was indeed quite safe for all the animals, but so was a rapid pressure drop of 3 atm (from 6 to 3), and this result was clearly not consistent with the idea that a constant pressure drop defines 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 atm it is quite safe to ascend rapidly to 3 atm, and from exposure to 2 atm it is safe to decompress to 1 atm pressure. Put in simple mathematical terms, if P_1 is the exposure pressure and P_2 is the pressure to which decompression is taken rapidly, them 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 ratio value of 2.0 is applicable to 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 pressures were lowered.

Consider a man breathing pure air at normal atmospheric pressure who is suddenly at some time, $t = 0$, exposed to a raised air pressure of P_1 atm. 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. From relatively modern knowledge it is known that approximately 0.01 sec is required for the dissolved molecules in the alveolar lining to reach the underlying capillary bed. In view of the fact that it takes about 1.0 sec for blood to pass the length of a pulmonary capillary, it is quite certain that blood leaving the capillary bed is fully equilibrated with the gas pressure in the alveoli. For simplicity, therefore, let us assume 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 an error of more than a few percent, and providing 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 there is blood in equilibrium with the pressure of the gas breathed in the lungs that 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 examination of most tissues reveals a very large number of capillaries per unit volume; the figure varies from several hundreds in well-vascularized tissues to perhaps only one patent capillary per cubic millimeter 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 millimeter; 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, let us assume 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 Figure IV-3.

Suppose the volume of arterial blood flowing in to the tissues is ν ml/sec: then the volume of venous blood flowing out must also be ν ml/sec; otherwise the tissue would progressively swell or shrink. Let the solubility of the inert gas in blood be s_1 ml (at

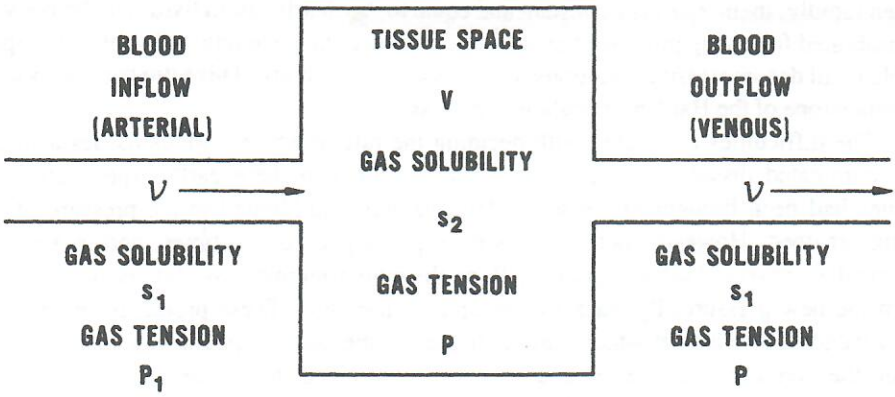

Figure IV-3. Concentration of dissolved gas in arterial, capillary, and venous blood. [(Adapted from Flynn and Lambertsen 1971).]

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 atm, then the total quantity of dissolved gas entering the tissue per sec is $P_1 \cdot s_1 \cdot \nu$ ml. If the tissue is considered as possessing a dissolved gas tension of P atm where P is uniform throughout the tissue (as noted above) and is therefore also the tension in the outgoing venous blood, then the total quantity of dissolved gas leaving the tissue per second is $P \cdot s_1 \cdot \nu$ ml.

The quantity accumulating in the tissue per second is the difference between that entering and that leaving, i.e., $(P_1 - P)s_1 \cdot \nu$. 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 $\Delta P \cdot s_2 V \cdot$ ml. In this small time the blood has delivered $(P_1 - P)s_1\nu \cdot \Delta t$ ml of dissolved gas. These two amounts must be equal, i.e.,

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

or

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

and s_1, ν, s_2, and V are all constants for any particular tissue; therefore $s_1\nu/s_2V$ is constant and will be called K:

$$\Delta P/\Delta t = K\,(P_1 - P)$$

as Δt becomes smaller,

$$dP/dt = K\,(P_1 - P) \qquad (1)$$

Students of elementary calculus will know that the variables are separated

$$dP/(P_1 - P) = K\,dt$$

and then integrated

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

or, expressing this differently,

$$P_1 - P = c\, e^{-Kt} \qquad (2)$$

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

$$\therefore P_1 = c\, e^0 = c$$

as $e^0 = 1$. Rearrange Equation 2 using this value of c,

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

This is the fundamental equation of the original Haldane analysis, and it 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 on the K-value chosen, and $K = s_1 v / s_2 V$ requires a substantial knowledge of the tissue physics and physiology 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 no one yet knows which tissue, or tissues, cause the bends, as becomes apparent later.

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 $t\infty$, then 2 t_1 will be three-quarters of the way to this value, 3 t_1 will be seven-eighths, and so on. Each additional half time takes the value halfway between the previous value and the infinite value. A table of the percentage saturations of a Haldane-like tissue in terms of the number of half times is shown in Table IV-2. 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 decompression 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.

D. *Using the Haldane Concepts*

The best way to reveal how these decompression procedures were obtained is to give, in its original wording, an account by G. C. C. Damant (n.d.) 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 seawater and the pressure stages, or *stops* as they are frequently called, were placed at 10-fsw (3.05-msw) increments. When reading this account, bear in mind that 0.7 msw or 2.25 fsw is equivalent to 1 psi of pressure and therefore the two calculations that follow refer to 6 hr of exposure at 57 fsw or 17.5 msw

and 1.5 hr at 68 fsw or 21 msw. 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 are 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 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 minutes respectively. Table *A* [IV-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 B [IV-3] gives, for various working pressures, the pressure to which the air lock may be reduced in the first rapid stage of decompression.

Table IV-2

TABLE A. Giving the Percentage Saturation at 1-min Intervals of Tissues (75T etc) That Become Half-Saturated in 75, 40, 20, 10, and 5 min[a]

Time, in min					Saturation	Time, in min					Saturation
5T	10T	20T	40T	75T	percentage	5T	10T	20T	40T	75T	percentage
	0.14			1	1		4.74		19	36	28
	0.29		1	2	2		4.94			37	29
	0.44	1	2	3	3		5.14	10	20	38	30
	0.59			5	4		5.35		21	40	31
	0.74		3	6	5		5.56	11	22	42	32
	0.89			7	6		5.78		23	43	33
0.5	1.04	2	4	8	7	3	5.99	12	24	45	34
	1.20			9	8		6.21		25	46	35
	1.36		5	10	9		6.43	13	26	48	36
	1.52	3	6	11	10		6.66		27	50	37
	1.68			13	11		6.90	14	28	52	38
	1.84		7	14	12		7.13			53	39
1.0	2.01	4	8	15	13		7.37		29	55	40
	2.18		9	16	14		7.61	15	30	57	41
	2.34			18	15		7.85		31	59	42
	2.51	5	10	19	16	4	8.11	16	32	61	43
	2.68		11	20	17		8.36		33	63	44
	2.86			21	18		8.62	17	34	65	45
	3.04	6	12	23	19		8.89	18	36	67	46
	3.22		13	24	20		9.16		37	69	47
	3.40		26	21			9.43	19	38	71	48
	3.58	7	14	27	22		9.71		39	73	49
	3.77		15	29	23	5	10.0	20	40	75	50
2	3.96	8	16	30	24		10.3		41	77	51
	4.15			31	25		10.6	21	42	79	52
	4.34	9	17	32	26		10.9	22	44	82	53
	4.54	9	18	34	27		11.2		45	84	54

Table A—*Cont.*

Time, in min					Saturation	Time, in min					Saturation
5T	10T	20T	40T	75T	percentage	5T	10T	20T	40T	75T	percentage
	11.5	23	46	86	55	11	21.8	44	87	163	78
	11.8		47	88	56		22.5	45	90	168	79
6	12.2	24	49	91	57		23.2	46	93	173	80
	12.5	25	50	94	58	12	23.9	48	96	179	81
	12.8	26	51	96	59		24.7	49	99	185	82
	13.2		53	99	60		25.5	51	102	191	83
	13.6	27	54	102	61	13	26.4	53	105	198	84
7	14.0	28	56	105	62		27.4	54	109	205	85
	14.3		57	107	63	14	28.4	56	113	213	86
	14.7	29	59	110	64		29.4	59	117	221	87
	15.1	30	60	113	65	15	30.6	61	122	230	88
	15.6	31	62	117	66	16	31.9	63	127	239	89
8	16.0	32	64	120	67		33.2	66	132	249	90
	16.4	33	66	123	68	17	34.7	69	138	260	91
	16.9	34	68	127	69	18	36.4	73	145	273	92
	17.4	35	70	130	70	19	38.3	77	153	287	93
9	17.9	36	72	134	71	20	40.6	81	162	304	94
	18.4	37	74	138	72	22	43.2	86	173	324	95
	18.9	38	76	142	73	23	46.4	93	185	348	96
	19.4	39	78	146	74	25	50.6	101	202	380	97
10	20.0	40	80	150	75	28	56.4	113	225	424	98
	20.6	41	82	154	76	33	66.4	133	265	498	99
	21.2	42	85	159	77						

[a]From Damant (n.d.).

ABBREVIATIONS

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

WP *Working Pressure* to which a man has been 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" Highest working pressure from which a man can be rapidly (say in two minutes) decompressed to atmospheric pressure [147 psi] without danger of resultant compressed air illness, however long the shift may have been. [The term "p" was used, differently defined, by Boulton (1942).] Decompressions are calculated so that by the time LP reaches zero, SP will have fallen to 18 lb. Haldane considered 18 lb [a 2.2:1 ratio] 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) [a 2.5:1 ratio] for "p", whilst others believe that a lower value is necessary to ensure complete safety [a strict 2.0:1 ratio, or 14.7 lb].

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

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 hours or more at a WP of 25 lb. For practical 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. 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 ("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, and we may disregard 40T, etc., knowing that by the time 75T has reached 18 lb they will have fallen to some still lower SP.

Table B [IV-3] shows that we can begin the men's decompression by allowing the LP to fall rapidly from 25 lb to 5 lb when the situation will be: 75T has SP 25 lb, with LP 5 lb, makes DP 20 lb. Therefore, 75T must desaturate from SP 25 lb to SP 18 lb, or by 7 lb, which is 35% of the DP and (by Table A [IV-2]) would require 46 minutes' time if the LP remained steady at 5 lb.

But, in practice, instead of maintaining the LP at 5 lb, 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 the present case, while the LP is falling by 5 lb, the SP must fall by 7 lb. 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 stages, remaining steady at each just long enough for the SP to fall by 1.4 lb. Thus:

1st Stage			
SP 25 lb	LP 5 lb	DP 20 lb	SP to fall 1.4 lb or 7% of DP takes 8 minutes
2nd Stage			
SP 23.6 lb	LP 4 lb	DP 19.6 lb	SP to fall 1.4 lb or 7% of DP takes 8 minutes
3rd Stage			
SP 22.2 lb	LP 3 lb	DP 19.2 lb	SP to fall 1.4 lb or 7% of DP takes 8 minutes
4th Stage			
SP 20.8 lb	LP 2 lb	DP 18.8 lb	SP to fall 1.4 lb or 7% of DP takes 8 minutes
5th Stage			
SP 19.4	LP 1 lb	DP 18.4 lb	SP to fall 1.4 lb or 8% of DP takes 9 minutes
Total time required			41 minutes

Taking the case of men who have become fully saturated under a WP of 40 lb. Table B [IV-3] shows that the LP may be rapidly reduced to 12 lb. Then, while it is slowly falling from 12 lb to zero, the SP must fall from 40 to 18 lb, or by an amount of 22 lb, which we can conveniently divide up into eleven decrements of 1.8 lb, and a twelfth of 2.2 lb, and the working, briefly expressed, becomes:

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

[It is of interest to note in this example that the decompression ratio (DR) from SP 40 to LP 12 is 2.05:1, or (40 + 14.7) ÷ 12 + 14.7), whereas that of SP 20.2 to LP 1, the last stop, is 2.22:1, or (20.2 + 14.7) ÷ 1 + 14.7).]

SHORT EXPOSURES

So far, we have only considered long exposures after which the desaturation of the slowest tissues, 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 hours at WP 30 lb, Table A [IV-2] shows that the saturations of different parts of the body would be:

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

Since 75T has not reached the danger limit "p" of 18 lb, it need not be considered at all, but 40T and 20T will both need gradual decompression. Inspection or a rough calculation shows that, though 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 a LP of 7 lb, as shown by Table B [IV-3], we see that 40T has to fall from 24 to 18 lb, while LP is falling from 7 lb to zero.

Dividing the decompression up into seven 1-lb stages as before, we get:

40T SP 24 lb	LP 7 lb	DP 17 lb with SP to fall by 0.5 lb, or 3%	takes 2 minutes
40T SP 23.5 lb	LP 6 lb	DP 17.5 lb with SP to fall by 0.5 lb, or 3%	takes 2 minutes
40T SP 23 lb	LP 5 lb	DP 18 lb with SP to fall by 1 lb, or 6%	takes 3 minutes
40T SP 22 lb	LP 4 lb	DP 18 lb with SP to fall by 1 lb, or 6%	takes 3 minutes
40T SP 21 lb	LP 3 lb	DP 18 lb with SP to fall by 1 lb, or 6%	takes 3 minutes
40T SP 20 lb	LP 2 lb	DP 18 lb with SP to fall by 1 lb, or 6%	takes 3 minutes
40T SP 19 lb	LP 1 lb	DP 18 lb with SP to fall by 1 lb, or 6%	takes 3 minutes
Total time required			19 minutes

[In this example the 40T or from SP 24 to LP 7 is only 1.78:1, whereas that from SP 19 to LP 1, the last stop, is 2.14:1.]

Table IV-3

TABLE B. 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.[a]

Working pressure, lb	May be reduced in 2 min to lb	Working pressure, lb	May be reduced in 2 min to lb
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

[a]From Davis (n.d.).

Haldane issued three separate air diving tables. The first table was for all those dives requiring a decompression time of less than 30 min. The second table was for all air dives requiring a decompression time of more than 30 min, and the third table was for deep air diving 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 feet per minute 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, 100 ft or 30 m (4 ATA), it is easy to see that it will double its size if the diver ascends through the water a distance of 66 ft or 20 m, to a depth of 33 ft or 10 m, i.e., 2 ATA pressure. The pressure on the bubble has been halved and therefore the volume has been doubled (Boyle's law). Similarly, if we imagine a small bubble at 33 ft (2 ATA), then rapid ascent to the surface (1 ATA) will also double the bubble's size. In the first case, however, it had been necessary to ascend 66 ft or 20 m to achieve a double of size, whereas in the second case only an ascent of 33 ft or 10 m 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 into account such possible bubble expansions and is therefore potentially hazardous. The acid test of any procedure, however, is, "Does it work?" The answer became quite clear that for the range of depths and bottom times commonly used 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 U.S. Navy, devised by French and Stilson in 1915 (see French 1916; Stilson 1915), were based on the Haldanian concepts of a decompression ratio and also used oxygen decompression to achieve depths between 200 fsw and 300 fsw. These tables were known as the Bureau of Construction and Repair tables (C & R tables). The C & R tables were used successfully in the 1915 salvage of the sunken submarine *F4* at a depth of 306 fsw.

E. *Post-Haldane Difficulties*

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 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 of better-designed and more reliable diving suits, it became possible to descend deeper and stay longer on the bottom than had 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 in Figure IV-4. If a dive of duration and depth such as 25 min at 100 ft is planned, then it is seen that decompression stages are required at 30, 20, and 10 ft, involving a total decompression time of 19 min. Reference to the current U.S. Navy Standard Air Diving Tables reveals that 25 min at 100 ft can be safely followed by direct ascent to the surface at a generally applied rate of 25 ft/min. 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 with a 2-hr bottom time, it is necessary to use Haldane's "Table II" (a page of which is reproduced in Figure IV-5), and it is seen that the total decompression time is 92 min for this particular dive. Again using the 1956 U.S. Navy Standard Air Diving tables for comparison with modern practice, it is seen that such a dive requires just over 132 min of decompression, although most diving supervisors faced with a dive of this nature will 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" (Figure IV-5) 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 his "Table I" was mostly oversafe and his "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 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 etiology 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. In most countries the authorities promulgating regulations relating to work in compressed air by tunnelers and caisson workers are 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 psi gauge) and the time at pressure not very great (not in excess of 4 hr), 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 is seen later in this chapter that the problems encountered by compressed air workers began to influence the diving scene in more recent times.

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.

Figure IV-4. Replica of a page taken from Table I of a British diving manual BR 155/43, after data by Haldane. [From Davis (1935).]

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	1½ to 1½ hrs.	—	—	—	—	—	—	10	25	37
			1½ to 1⅝ hrs.	—	—	—	—	—	—	10	30	42
			1⅝ to 2 hrs.	—	—	—	—	—	—	15	30	47
			2 to 2½ hrs.	—	—	—	—	—	—	20	30	52
			2½ to 2⅝ hrs.	—	—	—	—	—	2	23	30	57
			2⅝ to 2⅞ hrs.	—	—	—	—	—	3	27	30	62
			2⅞ 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	42⅜	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	53¼	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

Figure IV-5. Replica of a page taken from Table II of a British diving manual BR 155/43, after data by Haldane. [From Davis (1935).]

From 1932 onward for the next 25 years diving research effort was almost entirely confined to the U.S. Navy. The names of A. R. Behnke, C. W. Shilling, J. A. Hawkins, O. D. Yarbrough, O. E. VanDerAue, T. L. Willmon, M. Des Grange, J. V. Dwyer and, more recently, R. D. Workman, are principally associated with a quite remarkable series of experiments, generally involving human volunteers, that helped to define some of the boundary conditions of the decompression problem in a quantitative manner for the first time.

To pursue a calculation using the Haldane method it is necessary to know the time constants (K-value of Eq. 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 such knowledge was certainly not available in Haldane's day, and indeed it is still very difficult to obtain reliable, accepted, 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 behavior of his animals and, as he himself was well aware, it is always unsatisfactory to extrapolate from animal results and apply them to human beings.

Before proceeding with discussion of the human experimentation it would therefore 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 hr and then decompressed them to some lower pressure in order to establish his rule of a ratio of 2:1. 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 hr or thereabouts was quite sufficient, but investigators were soon led to enquire whether this was true. Many years later (Eaton and Hempleman 1962) 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 decompressed 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 than the first. If bends did occur after the original exposure, the experiment was repeated at a pressure 5 ft (1.5 m) lower in an attempt to obtain a troublefree 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 Figure IV-6. 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 hr of exposure at pressure, and certainly after 6 hr, there seems to be no change in the levels of threshold bend pressure.
3. There is a wide variability in individual response at any particular combination of pressure and time.

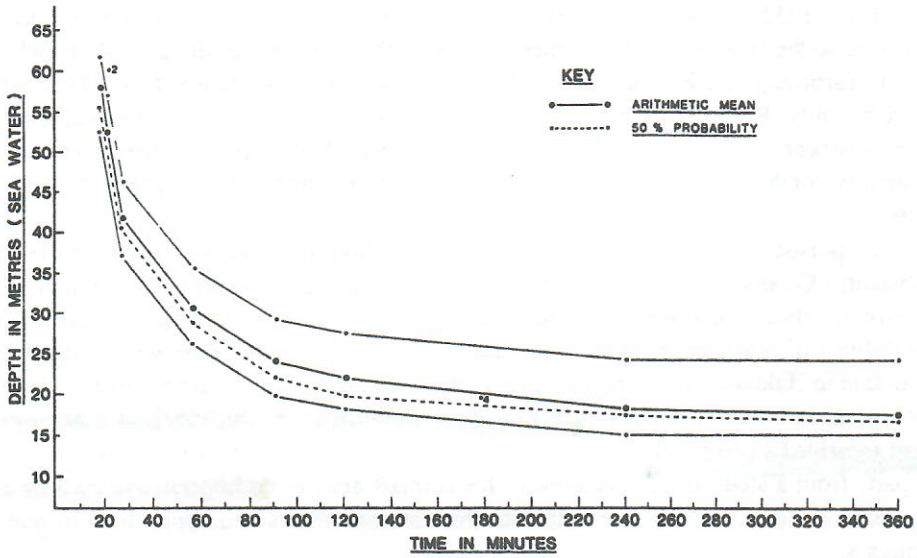

Figure IV-6. Population curve for goats, showing arithmetic mean and the 50% bend probablity. [From Eaton and Hempleman (1962).]

Such experimental results were not available to Haldane, and consequently his idea that 2 hr, 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. There is clearly a difference between the threshold bend level at a 3-hr duration and the threshold level at 6 hr. 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 on the use of animals with some of their tissues not equilibrated with gas, which 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.

F. *U.S. Navy Initiative*

By a different chain of reasoning these weaknesses in the Haldane approach became apparent to C. W. Shilling and his colleagues (Hawkins et al. 1935). In essence these U.S. Navy experimenters undertook in the mid-1930s a set of exposures using human volunteers; these were very similar to the much-later experiments with goats just described. Volunteers were exposed to raised pressures of air and then decompressed back to surface pressure without decompression stages. The results of these experiments are set out in Table IV-4. As with the goats, so with the men; it can be seen that short exposures allow a quite deep dive, whereas longer bottom times can only safely take place at shallower

Table IV-4
Summary of Results from Exposure of Human Volunteers to Air Dives of Varying Depths and Times[a]

No. of tests	Depth, ft	Individual exposures	Initial exposure time, min	Final time, min	Decompression sickness			
					Time to 1st case, min	No. of cases	No. of exposures	
							Before 1st case	After 1st case
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	178	361

[a]From data of Hawkins et al. (1935).

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 analyzed. Consequently after Hawkins, Shilling, and Hansen analyzed their findings in 1935, they concluded that each tissue half time was associated with a particular unique decompression ratio, and their theoretical analysis is given in Table IV-5.

A little later, in 1937, Yarbrough 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, so he pressed ahead with his theoretical framework given in Table IV-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 (55 m) or greater, were calculated by Yarbrough and issued for use by the U.S. Navy in 1937. The ratios Yarbrough used for the 20-, 40-, and 75-min half times were somewhat lower than those of Hawkins et al. because Yarbrough based them on dives involving exercise at depth; he concluded that this gave a greater loading of dissolved nitrogen to the tissues and 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 U.S. Navy tables gained worldwide acceptance alongside the original old Haldane tables, which were still in use despite their obvious deficiencies.

Table IV-5
Theoretical Analysis of Tissue Half Times[a] Determined From Exposures of Human Volunteers

Tissue half-time	Hawkins et al.[b]	Yarbrough[c]
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

[a] Time required for a tissue to absorb or eliminate one-half the equilibrium amount of inert gas.

[b] Data from Hawkins et al. (1935).

[c] Data from Yarbrough (1937).
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 ratio of 2.0 becomes 1.58.

Let us now ask the same question of these tables that we did for the Haldane tables: 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, including more useful work on the bottom and less air usage. Nevertheless, when they were required for long bottom times at deeper depths, these tables suffered the same fate as Haldane's "Table II" (Figure IV-5) because they just did not offer sufficient protection from decompression sickness. This defectiveness was first properly examined by VanDerAue and associates (1945) in a prolonged and extensive series of experiments designed to give the U.S. Navy the capability of performing safe surface decompression diving.

It will be helpful to understand the problem facing VanDerAue at that time. If a diver is sent down and works for a sufficient length of time so that he can no longer ascend to the surface without needing decompression stages in the water, then if an emergency occurs, like a storm blowing up or enemy action in the vicinity, he is in an extremely dangerous situation. It was decided that in such a situation the diver should be rapidly decompressed through the water and hauled up onto the deck of the support ship for surface decompression. Clearly, if he stayed on deck at atmospheric pressure in these circumstances, he was due to 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. This procedure presents three immediate problems: (1) How much time can be spent between leaving the bottom to starting recompression on deck? (2) To what pressure must the diver be recompressed in the deck chamber? Back to full pressure of his dive? Back to the pressure of what should have been first stop if he had not been hauled up? Or to some intermediate value between these two extremes? (3) What decompression is necessary after settling questions (1) and (2)?

In considering the last question first, two opposing views present themselves. It could be said that because the diver had been grossly underdecompressed for part of this

procedure, he was near to being a bends patient and therefore required a decompression that ought to be very prolonged and close to a therapeutic recompression procedure. On the other hand it could be argued that the rapid pull to the surface had created a huge, although 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 that VanDerAue solved in his own way. The nature of his theoretical solution does not now concern us, but the practical procedure he used to justify his approach became the starting point for the next onslaught in the decompression battlefield. He decided to compare the effectiveness of his proposed surface decompression procedures with the existing standard air diving procedures (Yarbrough 1937) 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. Clearly this was an extremely bad result and, as pointed out by VanDerAue, it meant that the basis for the calculation of the Yarbrough tables needed urgent reappraisal. This important experimental work was completed in the period from the close of World War II to 1951.

Again, why did a set of procedures that are so patently inadequate manage to survive satisfactorily for so long? The answer is not far away 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 mintues of decompression. Such dives do not represent a test for the adequacy of the total theoretical framework, as they only involve short-tissue half times. The great contribution of the group of Hawkins et al. (1935) was to delineate the limits of short bottom-time diving; hence, not surprisingly, the Yarbrough tables were troublefree with this type of diving. Unfortunately no similar experimental data had been assembled for long bottom-time dives, and therefore the calculations were necessarily informed guesswork. The guesses had clearly not been sufficiently near to the truth, as VanDerAue discovered. However, these long bottom-time and deep air dives were quite rare, although when tried they invariably gave an unacceptable incidence of bends, these results were submerged in any overall survey of the effectiveness of the Yarbrough tables because of the vast numbers of troublefree no-stop diving results. A mere glance at the statistics thus gives a false sense of security. Incidentally, this same weakness still applies to a great deal of data currently issued on the incidence of decompression sickness.

The next step was therefore fairly obvious. Some adjustment of the calculating procedures had to be made in order to keep the gains made by Yarbrough for the short bottom-time dives but render the decompression safer for long bottom times. A mathematician, Jolly V. Dwyer (1955), was brought into the small team of Des Granges, Dwyer, and Workman (Des Granges 1956); he analyzed 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 and time dependent. (Yarbrough had introduced the idea that the ratio was only time dependent.) Second, they decided that Hawkins et al. (1935) had been correct in considering tissues with half-times as short as 5 min, and the spectrum of tissue half times was extended to 5, 10, 20, 40, 75, and

120. Significantly a tissue half time (120 min) considerably greater than the longest considered by Haldane and Yarbrough was now reintroduced after VanDerAue's earlier consideration of its theoretical existence. (O. E. VanDerAue, unpublished data on calculations made in 1946–1948 and used in dives at the Experimental Diving Unit, Washington, D.C., in 1951).

In outline the procedure they evolved for calculating the new tables was relatively simple, but the computations involved were exceedingly tedious, and because this really was not a task on which human beings should waste their lives, it was appropriately left to the computer. The tables can be calculated as follows: The nitrogen tension in a tissue is obtained by the method given earlier in the quoted Damant (n.d.) examples. Armed with this nitrogen tension and knowing the duration of the dive, it is possible to obtain the depth of the decompression stop from a relationship that Dwyer (1955) proposed between the tension of nitrogen in a particular tissue and the permitted supersaturation ratio. In essence, then, various tissue half times exist, each with a characteristic supersaturation ratio relationship that varies with the amount of dissolved gas in it. A notable 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. (This was also the conclusion of VanDerAue.) The calculation is therefore totally dependent on knowledge of the tissue half times and a set of ratio relationships and has now become 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 easily stated, but if the calculations are 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 U.S. Navy in 1956 and are still in use today. In fact these tables have become the most widely used procedures to date.

Once again the question must be asked whether the air decompression problem has been solved by these 1956 procedures. Regrettably the answer must be no. They represent another step forward from the Yarbrough tables, but if long dives (e.g., 1 hr bottom time) are required at depths in excess of about 120 ft (36 m), then too great a number of bends occur. Beckmann (1976) has described a large air diving contract at depths of this order using the U.S. Navy air diving tables, and it is quite clear from his experience that a reexamination of the theoretical basis to these tables must be undertaken if air diving by the U.S. Navy is to be usefully extended in the future. To illustrate the point made previously, it should be noted, however, that when reporting on the 1956 tables the Naval Safety Center gave an overall incidence of bends of only 0.65% in 1976. Before leaving consideration of the present U.S. Navy 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 bottom-time dive of 360 min to a depth of 140 ft (43 m) there were two serious cases of bends among the U.S. Navy volunteer divers. Despite this somewhat discouraging result the tables were issued, but only for emergency use.

All the previous systems of calculation have 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 (1955), 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 than to vary the ratio. R. D. 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 is 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, which we call Pti. Now, according to the Workman system there is for each tissue a unique value of Pti that will allow the diver to ascend safely to a particular stop value. Suppose we wish to ascend to a stop at a gauge depth D; extensive analysis of available data reveals that if these critical values of Pti referred to above are called M-values, then $M = M_0 + a \cdot D$, where M_0 is the maximum permitted excess tissue inert gas tension allowed when D (depth) = 0 (i.e., when the diver surfaces) and a is a constant that like M_0 depends on the tissue being considered.

The M-values for various tissue half times for any particular stop can easily be calculated from Table IV-6.

When calculating a decompression schedule for a particular dive Workman would make an allowance for the ascent time to the first stop. This refinement is omitted in this explanation of his method. The steps in the calculation follow:

1. Calculate the depth of the first stop, working to the nearest 10-ft (3-m) increment that is safe for the diver (see examples in Table IV-6).
2. Use the absolute depth of this stop to calculate its pressure (D + 33) in feet or (D + 10) in meters, and calculate the inert gas partial pressure present at the

Table IV-6
Equations for Calculating M-Values[a] of Various Tissues[b]

Half-time,[c] min	Equation
5	$M = 104 + (1.8 \cdot D)$[d]
10	$M = 88 + (1.6 \cdot D)$
20	$M = 72 + (1.5 \cdot D)$
40	$M = 56 + (1.4 \cdot D)$
80	$M = 54 + (1.3 \cdot D)$
120	$M = 52 + (1.2 \cdot D)$

[a] M-value, critical value of inert gas tension in tissue.

[b] Examples of use of equations: What is the M-value for the 80-min tissue at a 20-ft stop? Answer: $M = 54 + (1.3 \cdot 20) = 80$ ft. Or, if 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? Answer: $(80 - 54)/1.3 = 20$ ft. [From Workman (1965).]

[c] Half time, time required for a tissue to absorb or eliminate one-half the equilibrium amount of inert gas.

[d] D, gauge depth in ft.

stop, i.e., $(D + 33) \cdot F$, where F is the fraction of inert gas in the breathing gas; e.g., $F = 0.79$ for air.

3. Write down each tissue separately and note the value Pti (each tissue's inert gas tension) and each M value for the next lower stop. Find the inert gas "extraction" pressure gradient, i.e., $Pti - (D + 33) F$, 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 determine when this allows the diver to ascend to the next shallower stop. To accomplish this aim it is first necessary to assess what fraction (f) of the inert gas extraction pressure gradient needs to be lost by each tissue, i.e.,

$$f = \frac{Pti - M}{Pti - (D + 33) F}$$

When f is determined, 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. Once 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 equal to 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 constitutent of the diver's breathing gas are given in Tables IV-7 and IV-8.

Air diving or helium diving tables can now be calculated with relative ease provided sets of M-values are available. If those 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 can, 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, particularly those who serve commercial diving groups as advisers on decompression procedures.

G. *Diffusion vs. Perfusion*

While the U.S. Navy were engaged in revising the Yarbrough tables and preparing to introduce the 1956 tables an interesting development was being pioneered elsewhere. A simple single-tissue approach was suggested (Hempleman 1952) that could provide a

Table IV-7
Maximum Permissible Tissue Tensions in Use of Nitrogen Gas before Ascent to Next Stop[a]

Tissue half time,[b] min	Depth of decompression stop, fsw									
	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

[a] Data from Workman (1965).
[b] Time required for a tissue to absorb or eliminate one-half the equilibrium amount of inert gas.

Table IV-8
Maximum Permissible Tissue Tensions in Use of Helium Gas before Ascent to Next Stop[a]

Tissue half time,[b] min	Depth of decompression stop, fsw									
	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

[a] Data from Workman (1965).
[b] Time required for a tissue to absorb or eliminate one-half the equilibrium amount of inert gas.

satisfactory solution to the decompression problem. This idea, and some of the subsequent developments from it, are outlined to illustrate several further points of importance.

It seemed 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 deep dive with short bottom time or a shallow dive of long duration. This was also true for animals, as was revealed in the goat experiments described earlier. This evidence strongly suggests that only one tissue type is principally involved and that there is a critical quantity of gas that this tissue can tolerate without pain.

If only one tissue is involved, however, 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 cross section of a joint it is striking to observe that perfusion of some parts is very sparse indeed, and 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 the idea 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. Suppose we simplify the physical factors involved and see where this leads. Let us 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, and the elementary physical laws of diffusion can be applied.

Suppose 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

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

(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 this can be found in any standard text book on differential equations. For the purposes of this model it was supposed that at time $t = 0$ the gas tension in the cartilage had 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

$$\alpha = 1 - \frac{8}{\pi^2}\left(e^{Kt} + \frac{1}{9}e^{-9Kt} + \frac{1}{25}e^{-25Kt} + \ldots\right) \qquad (4)$$

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 modeling 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 afterward, 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 and critical excess quantity of gas that 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 to 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 been proved to give a remarkably good fit of the U.S. Navy no-stop dive data for all dives with bottom times of less than 100 min. Table IV-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 *U.S. Navy Diving Manual*. Consequently it was decided to use this model as the basis for calculating diving tables. It is much less versatile, however, than the Workman M-value treatment just described, but it has a certain appealing simplicity. Once the K-value is fixed in Eq. 1, then the fractional saturation is also fixed and is solely a function of time, whereas on the M-value scheme there are seven tissues available to fit the data!

Table IV-9
Bottom Time at Various Depths According to U.S. Navy Tables Compared with Hempleman Calculations[a]

	Bottom time, min	
Depth, ft	U.S. Navy table[b]	Q = 500 prediction[c]
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

[a] Table derived from equation in Hempleman (1952).
[b] Based on M-value scheme of Workman (1965).
[c] Expansion of equation developed by Hempleman (1952).

One other important feature of the Hempleman system of calculating is also worth attention. Anyone who has taken part in experimental diving observes that some dives can cause a persistent, low-level pain in a joint, which is called a *niggle* or *inkle*, for example. Such niggles can sometimes remain with the diver for 2 or 3 days. An extreme example was provided by an Ocean Systems diver reported by Hamilton et al. (1966) 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 in 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, doubts arise whether such complexes are not also, but 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 one *K*-value was used, 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 was also used to support the idea of elimination being 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 because of some similar external factor. Likewise, during decompression there will be those who eliminate dissolved gas rather more slowly than others. Because 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.

The idea of a single-diffusion–limited tissue, as well as the view that the rate of uptake of dissolved gas at pressure and its release on decompression are not the same, was a radical departure from the current thinking. It might reasonably be expected that such a 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 U.S. Navy tables. The only noticeable differences arise when deeper, longer dives, e.g., 1 hr 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 of the United Kingdom have become very popular with contractors doing such arduous dives, as they undoubtedly lead to less decompression sickness than the U.S. Navy tables, especially if the version with oxygen stops is used.

The important point for the impartial observer to notice is that two philosophies are possible in approaching the decompression problem. The first philosophy can be summed up in the question, "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 leads to an attempt to identify a tissue or tissues responsible, e.g., the cartilage diffusion 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 it to be a relatively thick avascular piece of tissue, e.g., cartilage, tendon, bone. However, B. A. Hills (1966) challenged the accepted values of the diffusion coefficient for dissolved inert gas in tissues and used

values a thousand times smaller than the generally accepted Krogh (1918) values. This drastically alters the K-values in the solution to the differential equation given above (Eq. 4). 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 as well as poorly vascularized tissue. Hills accordingly 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 outward 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 short 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 onto the original idea of a 2- or 3-mm-thick slice of avascular tissue in order to satisfy the time scales involved. In the course of his analysis, however, 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 IV-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 internal bubble pressure, being less than the outside pressure on the body, would cause such a bubble to start to shrink and continue shrinking until it disappeared completely under the influence of this excess hydrostatic pressure.

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 Loeschcke (1956), was termed *the inherent unsaturation* by Hills. Quite independently, A. R. Behnke (1967) also drew attention to the fact that oxygen usage created a dis-

Table IV-10
Gas Pressures and Tensions in Blood and Tissue at Atmospheric Pressure

	Oxygen		Nitrogen		Carbon dioxide		Water vapor	
	Pressure, mmHg	Tension, mmHg	Pressure, mmHg	Tension, mmHg	Pressure, mmHg	Tension, mmHg	Pressure mmHg	Tension, mmHg
Air (dry)	152		608		—			
Alveolar gas	103		569		41		47	
Arterial blood		88		569		41		47
Venous blood		37		569		47		47
Tissue cells		10		569		49		47

equilibrium in gas tensions; 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, it should lead to bubble shrinkage and is therefore potentially 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 to 3 atm and the oxygen pressure is very nearly 0.6 atm, or 460 mmHg. Because nearly all oxygen is carried in blood as oxyhemoglobin, 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 atm in the oxygen pressure of the breathing gas, very little extra oxygen is carried to the tissues.

The situation from the viewpoint of tissue oxygen tension 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 atm. 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-limited ideas and diffusion-limited ideas is now occurring. As was pointed out in 1963 (Hempleman 1963) and independently but earlier by Perl (1962), it can be misleading to think of a tissue as some isolated unit with a particular half time dependent upon its vascularity, as was supposed in the analysis given at the beginning of this chapter. 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 obvious 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. The only 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-limited 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 lack of understanding on this point that led to the rejection of the single-tissue concept. If a single tissue has only 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.

H. *Data from Tunnel Workers*

So far the discussion on theoretical concepts underlying decompression procedures has been largely restricted to those used by divers. As mentioned earlier, however, 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 individual

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 W. M. D. Paton and D. N. 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 hr were undertaken at pressures exceeding 22 psi gauge (50 ft or 15 m). Accordingly, steps were taken to reduce the incidence of decompression sickness—for example, by lowering the threshold of 22 psi gauge (50 ft or 15 m) stated by Haldane as requiring only 2 min of decompression, i.e., virtually a no-step exposure, to 18 psi gauge (40 ft or 12 m). This is typical of the history of compressed air working in tunnels. A decompression procedure is adopted, 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 have therefore 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 (e.g., pressure, temperature) were quite stable. A number of 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 thus the bubbles were expanding beyond their size at normal atmospheric pressure. This was quite a reasonable supposition, but barometric pressure showed no correlation with the fluctuations in the numbers of bends. After investigation of several such hypotheses it was found that the only one that could be strongly supported by analysis was the relationship between the influx of new laborers and the incidence of bends. It became apparent that "new starters" in compressed air were at a greater risk than those who had been regularly working in compressed air. Paton and Walder decided this increased resistance to decompression sickness was due to "acclimatization." Since then, other and 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 work force can lead to a noticeable decrease in incidence of decompression sickness. 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 investigators 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 was confirmed as an influence on the incidence of the bends, and it was discovered that whenever there were long absences from regular work the men had lost their acclimatization. Opportunity arose to analyze more closely the time scale for this process of deacclimatization, and it was found that a 7- to 11-day half time would adequately describe it. That is, if a group of workers was absent from regular work for about a week, then the incidence of bends on return to work was about halfway between that of the new starters and the fully acclimatized workers. This was a very interesting finding, and although it added complications to understanding the etiology 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. Nevertheless, 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 they were not acclimatized. 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 investigators 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 of large-scale radiographic surveys (Medical Research Council 1966) that established the fact that this disorder is widespread in these workers. This establishment of a link between hyperbaric exposure and subsequent osteonecrosis was one of the turning points in the history of hyperbaric medicine, and particularly of decompression procedures. Osteonecrosis became far more worrisome both to 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 onto nonproductive activities, they did not lead to the large claims for compensation that followed 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 worrisome, picture emerged here. It was apparent that vast numbers of ordinary divers never suffered either acute attacks of decompression sickness or osteonecrosis. The main source of osteonecrosis among divers was from either professional divers who indulged in repetitive air diving over many years (e.g., Japanese pearl fishers) or deep heliox divers using inadequately established procedures. Several surveys of such professional groups are given in Table IV-11.

At first glance these results in Table IV-11 are alarming, but two factors have since emerged that now render the problem less severe than might be thought from examining these gross data. First, there are various manifestations of osteonecrosis; only juxta-articular involvement is the real hazard to the worker, and even juxta-articular lesions will not always lead to breakdown of the articular surface and thus to 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 1970–1980 decade (personal communications). The osteonecrosis problem drew attention to the way that decompression schedules had been constructed—with the objective of only the avoidance of bends.

Table IV-11
Incidence of Osteonecrosis Reported in Several Surveys

Survey	No. of divers	No. of divers with lesions	Incidence, %
Herget (1948)	90	29	32
Alnor (1963)	131	72	55
Ohta (1974)	301	152	50
Beckman (1976)	30	8	27
Totals	509	245	48

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 hematological investigation of J. Martin (Martin and Nichols 1971). Prior to his experiments it had been established by the Canadian workers Philp, Schacham, and Gowdey (1971) that there can be hematological changes during the course of exposure to diving pressures and sometimes shortly afterwards.

Martin, however, took these observations a step further. He used a standard dive that had always been troublefree from the viewpoint of the bends and that nevertheless was a substantial exposure to pressure, namely 1 hr at 100 ft (30 m). He used the Royal Navy Physiological Laboratory 1968 Air Diving Tables for his decompression procedure. No volunteers gave any indication of acute decompression sickness in the course of his 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 Figure IV-7. These observations have since been confirmed by other workers, and it must now be realized that asymptomatic changes are taking place in the body despite apparently adequate decompression. More recently G. Nichols (1979) has been showing gross changes in the erythrocyte sedimentation rate (ESR) during the course of prolonged decompressions from both heliox and nitrox saturation diving. Some of these changes take as long as 3 weeks after completion of decompression before they return to predive 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, changes have been noted by I. Rozsahegyi (1959) attributable to central nervous system damage. It should be mentioned, however, that the old Hungarian decompression procedures that 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 A. Palmer (1978) that animals (goats) given bend-producing schedules do exhibit spinal cord damage that can be seen histologically in animals killed several days, or even weeks, postdive. Thus there is further support for the view that hidden damage is taking place in a variety

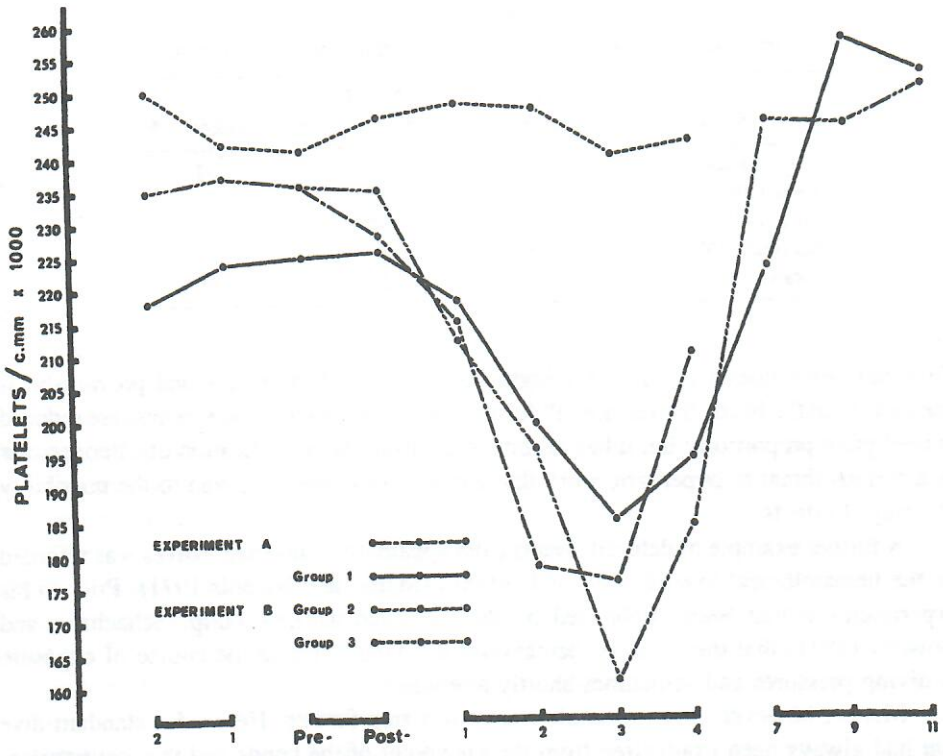

Figure IV-7. Platelet levels of several groups of divers (see text). The top curve is for a control group. [From Martin and Nichols (1971).]

of body tissues and that the adaptability of the body may be masking these problems except in those tissues where a pain results.

I. *Bubble Generation and Growth*

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 by A. R. Behnke (1951) 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 by using Doppler ultrasonic probes, asymptomatic bubbles could be detected in the bloodstream. The Seattle-based team headed by M. P. Spencer (Spencer and Campbell 1968) were foremost in establishing this technique as a major new contribution to the study of decompression procedures. Indeed the efficacies of various diving tables have 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 investigators. It does seem, however, that although these ultrasonic techniques for bubble analysis 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 D. J. Kenyon (1974) at the May 1974 Undersea Medical Society effectively states the conclusion of many 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 techniques other than Doppler, which requires moving bubbles for detection, are available. In particular there is ultrasonic imaging, pioneered by R. S. Mackay (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.

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

In 1971 G. D. Blenkarn and co-workers (1971) at Duke University 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 that switching breathing mixtures at a constant raised pressure gave enhanced possibilities for generating 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 effects on the skin.

The matter might well have rested on this explanation had it not been for some rather more dramatic experiments by C. J. Lambertsen and his co-workers (Graves et al. 1973), who in 1972 also subjected their volunteers to gas switching, but this time the pressure was much greater and equivalent to 1200 fsw (366 msw), and the gas being breathed within the chamber was heliox with switching to oxygen-neon or nitrox accomplished 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 period of delay, 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 outline of their hypothesis is illustrated in Figure IV-8.

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 that could exceed the hydrostatic pressure by as much as 30% according to these calculations. Following this initial explanation several other ingenious attempts have been made 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, ultrasonic Doppler probes were used to detect circulating bubbles in volunteers exposed for several hours to raised (132 ft or 40

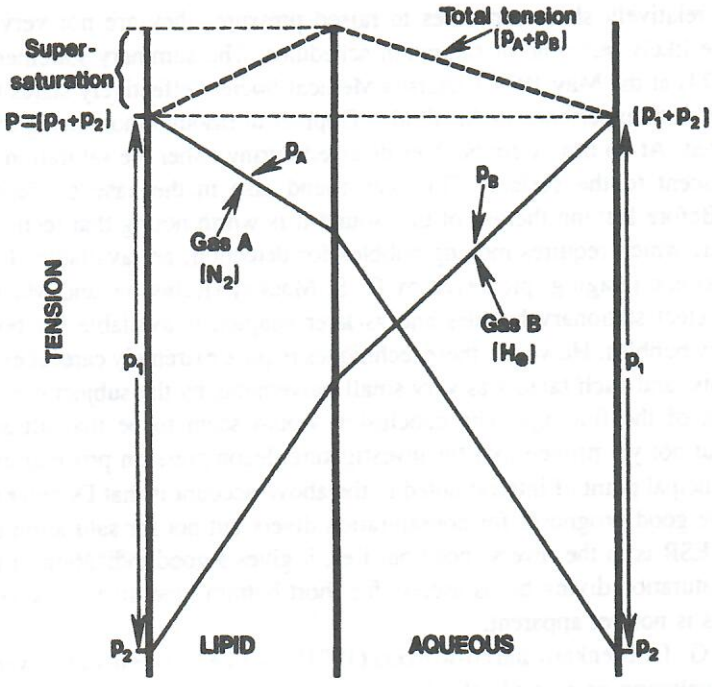

Figure IV-8. Initial explanation offered for isobaric decompression sickness signs caused by gas switching at pressure. For this idealized model the skin is bilayer with subcutaneous fat as the inner layer. The total ambient pressure is P, and the partial pressure of each inert gas is P_1. Gas A surrounds the body, Gas B is being breathed. When a steady state is reached Gas A diffuses linearly from the skin surface through each layer, and Gas B diffuses in the opposite direction. Both gases have different diffusion rates in the two layers, and their respective dissolved gas concentrations P_A and P_B are different in each layer. This could create supersaturation near to the lipid-aqueous boundary, as shown in the diagrammatic representation. [From Graves et al. (1973).]

m) pressures of air and then transferred with no change of pressure into a second chamber with an entirely new environment of heliox. 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 from the physician's viewpoint 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 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 the physician's awareness of the nature of the problems facing him.

If a beaker of pure water at constant temperature is exposed to a raised pressure of a gas such as nitrogen or helium, and if sufficient time is allowed to elapse so that equilibrium is reached, then rapid 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 if the liquid is stirred, then violent gas release takes place. All of this type of experimentation leads to the conclusion that a nucleus is necessary for promoting the formation of free gas from dissolved gas. In 1945 E. Newton-Harvey (Harvey 1945) demonstrated the same phenomenon in blood, namely that it is impossible to provoke bubble formation in supersaturated blood, but it can be made to effervesce profusely when stirred. He then went on to note that although he had demonstrated the virtual impossibility of producing bubbles in blood, it was, nevertheless, a matter of common observation that animals decompressed after exposure to only 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 that promotes bubble formation, and he introduced the idea of crevices of gas within the vessel walls that remain permanently stable unless subjected to enormous pressures; these "gas nuclei" act as sources for the formation of bubbles when dissolved gas diffuses into them. Furthermore, from experimentation with excised lengths of blood vessels he decided that arterial vessels are most likely to be responsible.

At first thought it might seem dubious to implicate the arterial system as the source of bubbles in blood. 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; as we saw earlier, this assumption is the basis of all the current decompression calculating systems. It must be remembered, however, that some arterial blood on its passage to those tissues which it supplied 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.

The evidence is quite convincing that if bubbles are generated in water supersaturated with dissolved gas, then when the bubble redissolves (by reapplication of pressure or because the water becomes undersaturated) a small deposit of impurity is left behind (Liebemann 1957). Should there be a further decompression, then a new bubble will form, using this small deposit as its nucleus. D. M. J. P. Manley (1960) deduced that the deposit had a volume between 10^{-4} and 10^{-5} mm^3 and when the bubble was very small this deposit provided a "skin" around the free gas. The skin slows the diffusion rate from small (<0.1-mm-diameter) bubbles, and this slowing down can be readily demonstrated. Since this early work by Manley and the theoretical analysis of bubble growth and decay by P. S. Epstein and M. S. Plesset (1950) numerous refinements have been made in the observations, but basically there have been no major changes in the general conclusions. More recently, the experiments of D. E. Yount (1978), using specially prepared sections of gelatin, have supported the organic skin concept of Manley.

Perhaps some of the most significant experiments concerning bubble formation were those by Evans and Walder (1969), who took shrimps (*Crangon crangon*) at atmospheric

pressure and decompressed them to 60-mmHg pressure, at which pressure all the shrimps could be seen through their translucent shells to have bubbles. They were then sealed in a polythene bag full of seawater and compressed hydrostatically to about 100 bars. On decompression from this pressure back to atmospheric pressure very few of the shrimps could be made to exhibit bubbles on a 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 hr 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 that it can replace or reconstitute every few hours. If these observations are confirmed, then interesting possibilities for the etiology of decompression sickness are opened.

Also considered by Evans and Walder was the possibility that fissile material such as 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 it is not tenable as representing the principal etiologic 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 (Whitaker et al. 1945), which can involve very large shearing forces, or tribonucleation, i.e., movement of surfaces over one another (Ikels 1970), 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 (Laurens 1964). 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 for bubble formation 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, but a few simple physical points can be made. If the pressure on a bubble of diameter greater than 10 μm is doubled, 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 (sausagelike) 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 considered change within the tissue (see Figure IV-9). One other point that must be mentioned is that if bubbles decrease to a diameter less than that of a capillary (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. Furthermore, if the bubble is trapped in a

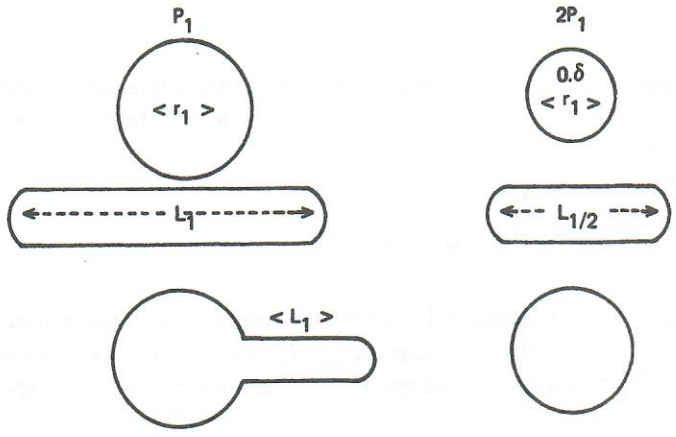

Figure IV-9. Three simplified tissue gas release possibilities showing that halving the volume can have quite different effects depending on the shape. Surface tension effects are being regarded as negligible.

blood vessel, then reducing its diameter to less than 10 μm will cause it to move through the capillary bed. The complexities of the problem can be seen, 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 1 atm) it was far from safe to use this same ratio value at several atmospheres of pressure. No controlled experimentation to discover the exact relationship between the ratio change and pressure was attempted until comparatively recently. In 1957 goats were exposed to raised pressures of air (P_1) for 6 hr, then decompressed rapidly to a new lower value (P_2), and held at P_2 to see whether a bend occurred (Hempleman 1957). In essence this repeated Haldane's old experiments but used 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 the conclusion was that Haldane was (near enough for practical purposes) correct and that P_1/P_2 was constant over a wide range of pressure values. Hills (1966), however, examined the data critically and showed that a relationship of the form

$$P_1 = a P_2 \div b$$

much more accurately described the results. Since his original analysis there have been other experiments using human volunteers and there is little doubt that a similar relationship describes these results also. For dives on heliox to pressures as great as 10 bars (300 ft or 90 m) the equation

$$P_1 = 1.397 P_2 + 5.7$$

satisfactorily describes the pressure P_2 (in meters) to which one can rapidly and safely decompress after exposure to pressure P_1 (in meters) for at least 24 hr 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 as a result of sudden drops in pressure changes from limb bends to mainly vestibular involvement.

For air the relationship is

$$P_1 = 1.361\ P_2 + 3.4$$

In all these equations the values of P_1 and P_2 are expressed in absolute terms, i.e., when $P_1 = 10$ the pressure is 10 bars, or near enough 10 atm pressure. The various predictions for P_1 and P_2 using this formula, and previous estimations by others, are shown in Figure IV-10.

J. *Diving Tables Today*

There are, as can be seen, several successful ways of constructing decompression tables despite the unavailability of basic underlying physiological facts. 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 still important ones, such as whether an attack of the bends can lead to, or be an indicator of, impending bone necrosis or CNS damage. From the point of view 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. Most of the air diving tables from the major navies of the world (U.S. Navy, French Navy, Royal Navy) are very satisfactory for the vast majority of air diving work (or sport). Indeed, if the bottom times, depths, and decompression times are compared, the similarity in international decompression requirements is obvious. However, if the decompression requirement exceeds about 1 hr, then all of these tables will begin to approach, or even greatly exceed, 1% incidence of bends. At this point it has been found by E. L. Beckmann (1976) that resort to extra decompression is mandatory, and he recommends following the 1968 air diving tables, which were issued in the United Kingdom by the Underwater Engineering Group of the Construction Industry's Research and Information Association, and using their Table II for oxygen breathing. Even these tables fail for really prolonged exposures to air pressures, and it is then necessary to consult the *NOAA Diving Manual* (Miller 1979).

Heliox 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 described earlier in this chapter. Experience has dictated which M-values give least trouble in a given set of circumstances. The U.S. Navy have had a set of bounce (i.e., short bottom time) helium diving tables for many years, but experience shows that use of these helium partial pressure tables is not successful at

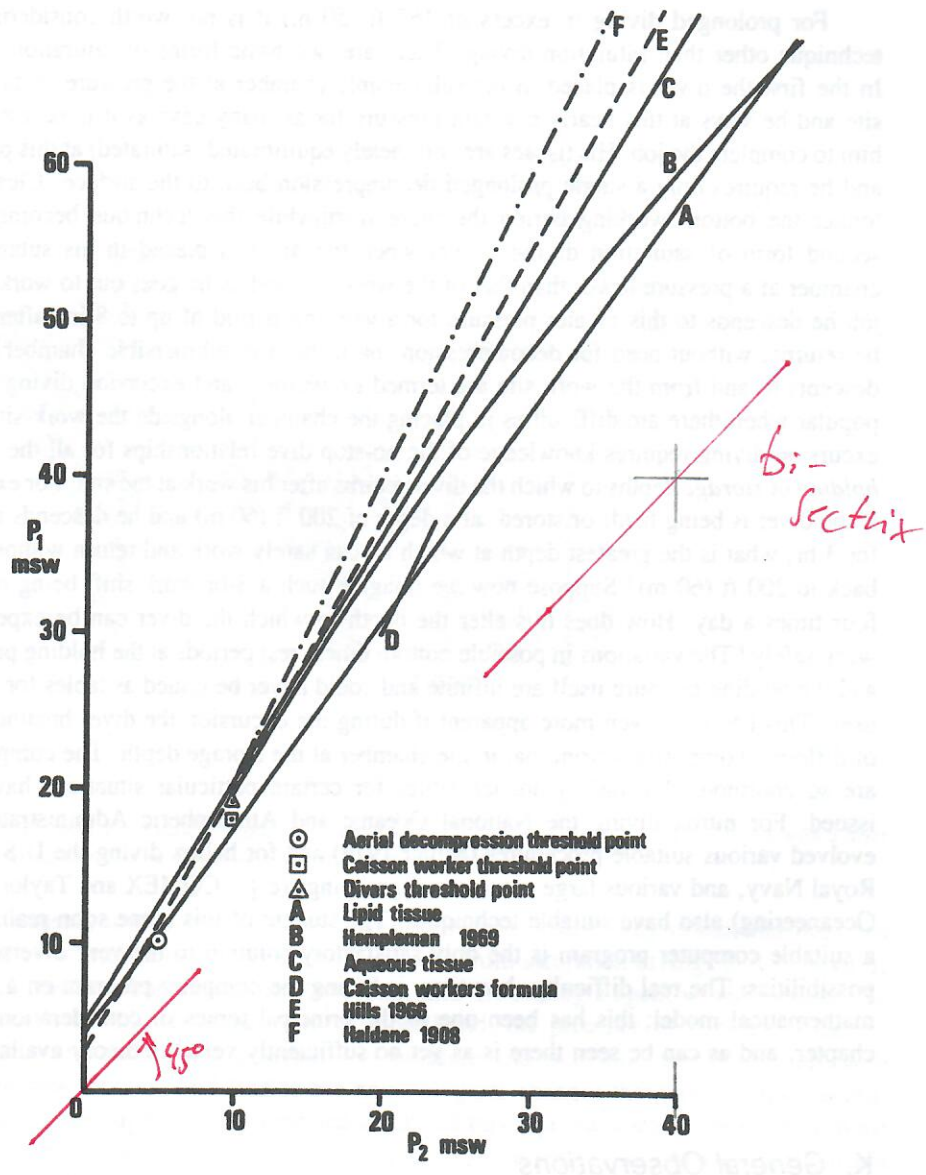

Figure IV-10. Absolute pressures are displayed. P_2 is the minimum pressure to which it is safe to ascend following a prolonged exposure to pressure P_1. Various estimates are tenable because insufficient direct evidence is available.

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 they are normally labeled "commercial-in-confidence," and it is difficult to obtain any objective appraisal of their success. It does seem, however, that no groups perform regular bounce diving to depths in excess of 500 ft (150 m) and that bottom times exceeding 1 hr are not yet considered sufficiently troublefree at depths of 250 ft (80 m) or greater.

For prolonged diving in excess of 165 ft (50 m) it is not worth considering any technique other than saturation diving. There are two basic forms of saturation diving. In the first 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 requires only 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 hr, 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 200 ft (60 m) and he descends to work for 3 hr, what is the greatest depth at which he can safely work and return without stops back to 200 ft (60 m)? Suppose now we imagine such a 3-hr 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 uses. 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 nitrox diving the National Oceanic and Atmospheric Administration has evolved various suitable procedures (Miller 1979) and for heliox diving the U.S. Navy, Royal Navy, and various large commercial groupings (e.g., COMEX and Taylor Diving Oceaneering) also have 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: The real difficulty, however, is basing the computer program on a reliable mathematical model; this 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.

K. *General Observations*

It is not good enough just to reach a satisfactory calculating system that has no physiological basis. This may be illustrated by considering the cross section of the shoulder joint (Figure IV-11), which was used earlier to show that the synovial membrane or slab of cartilage was a reasonable model for consideration. It requires very little extra speculation, however, 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. It is inside a bony capsule, and a large pressure could be generated inside the bone that would indubitably cause pain. Indeed, bubbles inside

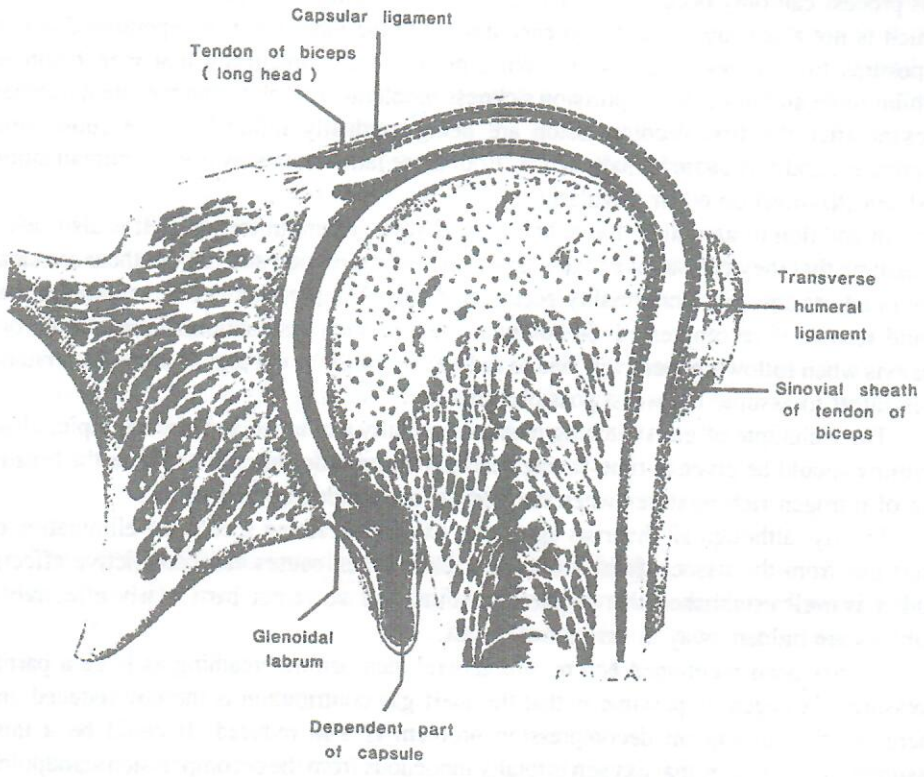

Figure IV-11. Section of shoulder joint to illustrate idea of viewing cartilage as a slab of tissue with a layer of blood along face of slab. See also reference in text to "whole joint" concept.

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 etiologic 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 and ischemia, would interfere with bone marrow function and lead to the hematological changes that have been observed. It could also 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, 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!

Given this situation the physician must maintain a stance that does not adhere to any particular set of beliefs and yet offers a rational approach to decompression or any subsequent therapies involved. The best concepts to adopt are therefore the most conservative. It is safest to assume that every decompression, without exception, is accompanied by a bubble. This bubble may, or may not, manifest itself overtly, but silent bubbles are always present. If men work for several days on diving or tunnel procedures, and if it can be noticed that they are not having as many attacks of bends due to acclimatization or adaptation to work in compressed air, then it is safest to assume that

this process can only occur by desensitizing and damaging some physiological response, which is not a desirable practice to encourage. On the other hand, if repetitive dives or exposures to compressed gases are taking place at such frequency that men begin to exhibit more and more decompression sickness problems, it is clear that the silent bubbles present after the first decompression are being gradually inflated by the subsequent exposures, and this again is malpractice. Both these latter situations (i.e., acclimatization and sensitization) do occur, regularly.

In addition to assuming that bubbles are always present in the body, it is also safest to assume that they can survive many days post-decompression and, unless there are clear contraindications, any abnormality occurring post-decompression is due to bubbles that could resolve if recompressed. In common with all such assumptions there will be occasions when following them will lead to failure, but until the etiology is better understood it is safest to assume the worst possible situation.

The technique of gas switching must be carefully examined and, for example, close scrutiny should be given to time-saving decompression procedures that rely on the breathing of nitrogen-rich mixtures when the body is surrounded by helium.

Finally, although all theorists agree that oxygen breathing speeds the elimination of inert gas from the tissues, it is also known that oxygen causes vasoconstrictive effects, and it is well established that oxygen breathing per se is not particularly effective if bubbles are hidden away in very slow tissues.

As has been mentioned before, the general idea behind breathing as large a partial pressure of oxygen as possible is that the inert gas contribution is thereby reduced and therefore the subsequent decompression problem is also reduced. It could be a false assumption, however, that oxygen is totally innocuous from the decompression standpoint. Reference to any standard textbook on physiology will reveal that the cardiovascular system responds markedly to large changes in arterial oxygen content. Many tissues suffer severe vasoconstrictive effects under high partial pressures of oxygen. For example, the exposed pial membrane of a cat can be seen to blanch when the animal is given raised oxygen pressures to breathe. The quantitative aspects of how the various tissue circulations respond to large changes in oxygen concentration is far from understood. Nevertheless, it can be said unequivocally that breathing pure oxygen or high partial pressures of oxygen does normally speed up the decompression. The only exception occurs when the schedule has given, or is close to giving, a bend. Oxygen on its own does not help here on most occasions. Only recompression, to remove or reduce the size of the offending bubbles, will be effective, and when accompanied by breathing of oxygen-rich mixtures is of course even more effective, but the possibility of oxygen toxicity must always be borne in mind. Limits for safe breathing of raised partial pressures of oxygen are given elsewhere in this book (see Chapter II).

Reference has been made earlier to the technique of surface decompression in attempts to shorten the "in water" decompression time required by the conventional diver. Although this is rarely used except by military divers, it is worth mentioning as a possible technique for emergencies, and clearly a physician will be involved at some point. A similar procedure is used by tunnel workers and is generally termed *decanting*. In this latter method the compressed air worker at the end of his shift period is rapidly decompressed to atmospheric pressure and then walks from the work chamber to a separate pressure chamber in which he is rapidly recompressed back to his former shift pressure.

He is normally then required to wait a few minutes at this maximum pressure, after which he is decompressed at the normal slow decompression rate demanded by his time at the full shift pressure. This procedure is used when for some technical reason it is not desirable to have the decompression lock directly attached to the main working chamber. Both surface decompression and decanting involve the highly undesirable practice of rapidly decompressing the man from his working pressure to atmospheric pressure, at which pressure he would suffer a serious attack of decompression sickness unless rapidly recompressed within a few minutes. Needless to say, for regular use these techniques require experienced teams to ensure success. Some groups attempt to render surface decompression less hazardous by completing one or two short deep stops in the water before surfacing directly, but although this is indubitably a move in the right direction, it still remains a technique for experienced teams of divers, and in any case is not to be encouraged except under compelling circumstances. As was pointed out when considering VanDerAue's testing of this technique, the areas of doubt about how the body is responding to the various violent pressure changes involved are too numerous for one to feel confident that lack of overt decompression sickness is necessarily accompanied by lack of hidden damage.

The day is not far distant when the theoretician will be able to offer divers or tunnel workers a set of versatile concepts that 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 the 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 or 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 that can detect the very first indications of impending trouble. This would make it possible to monitor the pressure-time course on an individual basis and would make less essential the understanding of detailed physiological mechanisms.

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References

ALNOR, P. C. 1963. Chronic changes in the bone structure of divers. *Bruns. Beitr. Klin. Chir.* 207: 475–485.

BATEMAN, J. B. 1951. Review of data on value of pre-oxygenation in prevention of decompression sickness. In: *Decompression Sickness*, edited by J. F. Fulton. Philadelphia: W. B. Saunders. Chap. 9, Pt. 1.

BECKMANN, E. L. 1976. Recommendations for improved air decompression schedules for commercial diving. Sea Grant Tech. Rep. UNIHI-SEA Grant-TR-76-02.

BEHNKE, A. R. 1951. Decompression sickness following exposure to high pressures. In: *Decompression Sickness*, edited by J. F. Fulton. Philadelphia: W. B. Saunders, p. 53–89.

BEHNKE, A. R. 1967. The isobaric (oxygen window) principle of decompression. In: *Trans. Third Annual Conference of the Marine Technology Society*. Washington, DC: Marine Technol. Soc.

BERT, P. 1878. *Barometric Pressure*, transl. by M. A. and F. A. Hitchcock. Columbus, OH: College Book Co., 1943, p. 5. (Republ. Bethesda, MD: Undersea Medical Society, 1978.)

BLENKARN, G. D., C. AQUADRO, B. A. HILLS, AND H. A. SALTZMAN 1971. Urticaria following sequential breathing of various inert gases at 7 ATA: a possible manifestation of gas-induced osmosis. *Aerosp. Med.* 42: 141–146.

BOULTON, G. O. 1942. The use of air locks. *J. Inst. Eng. Aust.* 14(1).

BOYCOTT, A. E., G. C. C. DAMANT, AND J. S. HALDANE 1980. Prevention of compressed air illness. *J. Hyg.* 8: 342–443.

CHRYSSANTHOU, C. P. 1973. Studies on the mechanism and prevention of decompression sickness. Progress Report of the Physiology Program. Arlington, VA: Office of Naval Research, p. 7–8.

DAMANT, G. C. C. (n.d.). *Calculating Decompressions on the Late Professor J. S. Haldane's System.* London: Siebe, Gorman and Co., Ltd.

DAVIS, R. H. 1935. In: *Siebe, Gorman & Co. Decompression Tables.* London: St. Catharine Press.

DAVIS, R. H. 1981. *Deep Diving and Submarine Operations* (8th Ed.). Gwent, Wales, U.K.: Siebe, Gorman and Co., Ltd.

DES GRANGES, M. 1956. *Standard Air Decompression Table.* Res. Rep. 5-57. Washington, DC: U.S. Navy Experimental Diving Unit.

DWYER, J. V. 1955. *Calculation of Air Decompression Tables.* Res. Rep. 4-56. Washington, DC: U.S. Navy Experimental Diving Unit.

EATON, W. J., AND H. V. HEMPLEMAN 1962. The incidence of bends in goats, after direct surfacing from raised pressures of air. Rep. 209. London: Medical Research Council, R.N. Personnel Research Committee, Underwater Physiology Sub-Committee.

EPSTEIN, P. S., AND M. S. PLESSET 1950. On the stability of gas bubbles in liquid-gas solutions. *J. Chem. Phys.* 18: 1505–1509.

EVANS, A., AND D. N. WALDER 1969. Significance of gas macronuclei in the aetiology of decompression sickness. *Nature (London)* 222: 251–252.

FLYNN, E. T., AND C. J. LAMBERTSEN 1971. Calibration of inert gas exchange in the mouse. In: *Underwater Physiology IV. Proceedings of the Fourth Symposium on Underwater Physiology*, edited by C. J. Lambertsen. New York: Academic Press, p. 179–191.

FRENCH, G. R. W. 1916. Diving operations in connection with the salvage of the USS "F-4." *U.S. Nav. Med. Bull.* 10: 74–91.

GOLDING, F. C., P. D. GRIFFITHS, W. D. M. PATON, D. N. WALDER, AND H. V. HEMPLEMAN 1960. Decompression sickness during construction of the Dartford Tunnel. *Br. J. Ind. Med.* 17: 167–180.

GRAVES, D. J., J. IDICULA, C. J. LAMBERTSEN, AND J. A. QUINN 1973. Bubble formation resulting from counter-diffusion supersaturation: A possible explanation for isobaric inert gas 'urticaria' and vertigo. *Phys. Med. Biol.* 18: 256–264.

HALLEY, E. 1717. The art of living under water. *Phil. Trans. R. Soc. London.*

HAMILTON, R. W., J. B. MACINNIS, A. D. NOBLE, AND H. R. SCHREINER 1966. Saturation diving at 650 feet. Technical Memorandum B-411. Tonawanda, NY: Ocean Systems.

HARVEY, E. N. 1945. Decompression sickness and bubble formation in blood and tissues. *Bull. N.Y. Acad. Med.* 21: 505–536.

HARVEY, E. N. 1951. Physical factors in bubble formulation. In: *Decompression Sickness*, edited by J. F. Fulton. Philadelphia: W. B. Saunders, p. 90–114.

HAWKINS, J. A., C. W. SHILLING, AND R. A. HANSEN 1935. A suggested change in calculating decompression tables for diving. *U.S. Nav. Med. Bull.* 33: 327–338.

HEMPLEMAN, H. V. 1952. Investigation into the decompression tables. A new theoretical basis for the calculation of decompression tables. Rep. III, Pt. A. London: Medical Research Council, R.N. Personnel Research Committee, Underwater Physiology Sub-Committee.

HEMPLEMAN, H. V. 1957. Investigation into the decompression tables. Further basic facts on decompression sickness. Rep. 168. London: Medical Research Council, R.N. Personnel Research Committee. Underwater Physiology Sub-Committee.

HEMPLEMAN, H. V. 1963. Tissue inert gas exchange and decompression sickness. In: *Proceedings of the Second Symposium on Underwater Physiology*, edited by C. J. Lambertsen and L. J. Greenbaum. Washington, DC: Natl. Acad. Sci./Natl. Res. Council.

HERGET, R. 1948. Recent observations of barotraumatic chronic joint complaints in divers. *Arch. Klin. Chir.* 261: 330–360.

HILLS, B. A. 1966. A thermodynamic and kinetic approach to decompression sickness. Adelaide: Libraries Board of South Australia. Thesis.

IKELS, K. G. 1970. Production of gas bubbles in fluids by tribonucleation. *J. Appl. Physiol.* 28: 524–527.

JAMINET, A. 1871. *Physical effects of compressed air and of the causes of pathological symptoms produced on man, by increased atmospheric pressure employed for the sinking of piers in the construction of the Illinois and St. Louis bridge over the Mississippi River at St. Louis, Missouri.* St. Louis: Ennis.

KEAYS, F. L. 1909. Compressed air illness, with a report of 3,692 cases. *Publ. Cornell Univ. Med. Coll.* 2: 1–55.

KEAYS, F. L. 1912. Compressed-air Illness. *Am. Labor. Legisl. Rev.* 2: 192–205.

KENYON, D. J., M. FREITAG, AND M. R. POWELL 1974. Efficient decompression procedure for 1000 foot diving. *Undersea Biomed. Res.* 1: A7.

KROGH, A. 1918. The rate of diffusion of gases through animal tissues, with some remarks on the coefficient of invasion. *J. Physiol.* 52: 391.

LAURENS, P. 1964. Considerations sur l'origine des bruits du coeur. *Acta Cardiol. (Brux.)* 19: 327–344.

LIEBERMANN, L. 1957. Air bubbles in water. *J. Appl. Phys.* 28: 205–211.

LOESCHCKE, V. H. 1956. Über die Diffusion von Gas in mit Gas untersättigte Lösungen mit Durchrechnung biologischer Beispiele. *Z. Naturforsch.* 11B: 613–620.

MANLEY, D. M. J. P. 1960. Change of size of air bubbles in water containing a small dissolved air content. *Br. J. Appl. Phys.* 11: 38–42.

MARTIN, K. J., AND G. NICHOLS 1971. Changes in platelets in man after simulated diving. Rep. 5/71. Orpington, Kent, England: R. N. Physiological Laboratory.

MEDICAL RESEARCH COUNCIL DECOMPRESSION SICKNESS PANEL 1966. Bone lesions in compressed air workers with special reference to men who worked on the Clyde Tunnels 1958 to 1963. *J. Bone Joint Surg. (Lond.)* 48B: 207–235.

MILLER, J. W. (editor) 1979. *NOAA Diving Manual.* Washington DC: National Oceanic and Atmospheric Administration, U.S. Dept. of Commerce.

NICHOLS, G. 1979. Changes in erythrocyte sedimentation associated with saturation diving. *Annual Scientific Meeting, European Union of Biological Sciences Fifth Scientific Meeting, Bergen, Norway.*

OHTA, Y., AND O. SHIGETO 1974. *Symposium on Dysbaric Osteonecrosis*, edited by E. L. Beckmann and D. H. Elliott. Washington, DC: National Institute of Occupational Safety and Health.

PALMER, A. C., W. F. BLAKEMORE, J. E. PAYNE, AND A. SILLENCE 1978. Decompression sickness in the goat: nature of brain and spinal cord lesions at 48 hours. *Undersea Biomed. Res.* 5: 276–286.

PATON, W. D. M., AND D. N. WALDER 1954. Compressed air illness—An investigation during the construction of the Tyne Tunnel, 1948/1950. Spec. Rep., Ser. No. 281. London: Medical Research Council.

PERL, W. 1962. Heat and matter distribution in body tissues and the determination of tissue blood flow by local clearance methods. *J. Theor. Biol.* 2: 201–235.

PERL, W., H. RACKOW, E. SALANITRE, G. L. WOLF, AND R. M. EPSTEIN 1965. Inter-tissue diffusion effect for inert fat-soluble gases. *J. Appl. Physiol.* 20: 621–627.

PHILP, R. B., P. SCHACHAM, AND C. W. GOWDEY 1971. Involvement of platelets and microthrombi in experimental decompression sickness; similarities with disseminated intra-vascular coagulation. *Aerosp. Med.* 42: 494–502.

RAHN, H., AND W. O. FENN 1955. *A Graphical Analysis of the Respiratory Gas Exchange.* Washington, DC: Am. Physiol. Soc.

RASHBASS, C. R. 1955. Investigation into the decompression Tables. Rep. VI New Tables. Rep. 151. London: Medical Research Council, R. N. Personnel Research Committee, Underwater Physiology Sub-Committee.

ROZSAHEGYI, I. 1959. The late consequences of the neurological forms of decompression sickness. *Br. J. Ind. Med.* 16: 311–317.

RUBISSOW, G. J., AND R. S. MACKAY 1974. Decompression study and control using ultrasonics. *Aerosp. Med.* 45: 473–478.

SPENCER, M. P., AND S. D. CAMPBELL 1968. The development of bubbles in the venous and arterial blood during hyperbaric decompression. *Bull. Mason Clin.* 22: 26–32.

STILSON, G. D. 1915. *Report on deep diving tests-USN.* Washington, DC: U.S. Department of the Navy, Bur. of Construction and Repair.

U.S. NAVY 1978. *U.S. Navy Diving Manual (Change 2). Air Diving.* Washington, DC: U.S. Navy Dept., Vol. 1. (NAVSEA 0994-LP-001-9010.)

UNDERWATER ENGINEERING GROUP 1968. *Air Diving Tables*. London: Her Majesty's Stationery Office.

VANDERAUE, O. E., E. S. BRINTON, AND R. J. KELLER 1945. Surface decompression and testing of decompression tables with safety limits for certain depths and exposures. Res. Rep. Washington, DC: U.S. Navy Experimental Diving Unit.

WHITAKER, D. M., L. R. BLINKS, W. E. BERG, V. C. TWITTY, AND M. HARRIS 1945. Muscular activity and bubble formation in animals decompressed to simulated altitudes. *J. Gen. Physiol.* 28: 213–223.

WORKMAN, R. D. 1965. Calculation of decompression schedules for nitrogen-oxygen and helium-oxygen dives. Res. Rep. 6-65. Washington, DC: U.S. Navy Experimental Diving Unit.

YARBROUGH, O. D. 1937. Calculation of decompression tables. Res. Rep. Washington, DC: U.S. Navy Experimental Diving Unit.

YOUNT, D. E. 1978. Responses to the twelve assumptions presently used for calculating decompression schedules. In: *Decompression Theory*. Bethesda, MD: Undersea Medical Society. (Decompression Workshop.)