Chapter 2

The Decompression Syndrome

The body can withstand a very wide range of hydrostatic pressure provided it is applied isometrically to avoid any tissue deformation. This is achieved by ensuring that the 'hydraulic fluids' comprising the external environment and the breathing mix can reach all boundaries of the incompressible part of the body; these include the surfaces of the lung airways, the middle ear and the sinus cavities—in addition to the skin.

Dissolved gases

However, in order to increase the absolute pressure of the breathing mix to maintain this balance across the chest wall during compression, it is necessary to increase the partial pressure of at least one of the component gases. The genesis of real problems in abnormobaric environments is therefore the resulting increase in the arterial blood tension of at least one gas unless the subject breathes a liquid unsaturated with gases (see p. 209). Thus there are increased blood concentrations of those gases with their subsequent availability to the tissues in abnormally large quantities.

The uptake of nitrogen or inert gases of higher molecular weight can lead to narcosis (Behnke and Yarbrough, 1939), while extensive compression on a helium–oxygen mix can lead to various neurologic disorders characterized by a tremor which Brauer *et al.* (1966) have termed the high-pressure nervous syndrome (HPNS). At elevated partial pressures, oxygen can prove toxic to give either pulmonary distress or convulsions (p. 217); while air compression can lead to joint pains known generally as hyperbaric arthralgia (p. 206).

These manifestations of gas uptake are described later (Chapter 8) with particular reference to the limitations which each can impose upon the available means for preventing or treating 'the bends'. Each can also be avoided: narcosis by switching the inert gas to helium; HPNS and hyperbaric arthralgia by slowing the compression; and oxygen toxicity by reducing the fraction of oxygen in the inspired breathing mix. However, there is no such simple means of avoiding decompression. If sufficient, decompression can lead to gases separating from solution in the various body fluids and tissues in which they have accumulated at pressure. If the combination of exposure pressure and time exceeds certain critical limits (fig. 12), then rapid decompression can have numerous undesirable effects known collectively as decompression sickness; while excessive decompression can result in permanent disablement or death. The limits are remarkably well defined considering the usual range of biological variation, any contingencies being described by Behnke (1951) as 'perhaps five feet in depth or several thousand feet in altitude ascent separating injury from a state of well-being'.

When the exposure has exceeded these safe limits, it is necessary to decompress more gradually in attaining the lower pressure. The all-important question is—how gradually? What are the optimal changes to be made to ensure safety and yet minimize the wearisome time spent by a diver or caisson worker in returning to normal pressure—let alone the economic advantages of reducing the time involved?

However, before using the rest of this book

in attempting to answer that question, it is first desirable to be aware of the possible consequences if the prescribed decompression procedure proves inadequate.

Symptomatology of Inadequate Decompression

Decompression sickness is a very general term which has been loosely applied to encompass almost any undesirable effect which could be caused by decompression. This is less specific than most definitions of the syndrome since it is not yet certain that all of the symptoms usually listed are attributable to decompression *per se*. In returning to normal pressure, there must be as much compression as decompression, so that it is not always certain which of these phases of the exposure was primarily responsible for those effects first noticed after the whole excursion has been concluded.

The symptoms which have been listed are many (Table 2). The predominant feature is

Table 2 Symptoms of decompression sickness and their relative incidence

	Symptom		Survey (as percentage)						
			A	B	C	D	E	F	G
I	Localized pain (around joints)	'Limb bends'	70–92	98·6	44	53	71	91·8	90
I	Lymphatic occlusion								
I	Urticaria (wheal)			1·0	7			7·5	
I	Pruritus (itching)	'Skin bends'						14·9	
I	Paraesthesia (burning)							21·2	
I	Subcutaneous emphysema								
II	Hemiplegia	'Spinal bends'	0·6	↑	↑	↑	↑		↑
II	Paralysis		21	0·1				6·1	1·3
II	Numbness								
II	Aphasia		0·3–1						
II	Visual disturbances		5		2			6·8	
II	Convulsions								
II	Muscular twitching								
	Headache					22	15·9	32·8	
	Collapse								
	Syncope								
	Unconsciousness			↓	↓	↓	↓		↓
III	Vertigo								
III	Dizziness							8·5	
III	'The staggers'						9·2	5·4	3·6
III	Nausea	'Vestibular bends'						7·9	
III	Vomiting								
III	Nystagmus								
III	Incoordination								
III	Tinnitus (ringing in ear)	'Cochlear involvement'							
III	Partial deafness								
IV(?)	Dysbaric osteonecrosis (bone rot)								
	'The chokes'		1·6–6	0·3	5	14	0	1·0	1·0
	Aviators (a), Divers (d) or Caisson workers (c)		a	a	a	a	d	d	c

Other symptoms: Muscular weakness (fatigue), urinary disturbances, personality changes, irritability, restlessness or agitation, haemoptysis (bloody sputum), abdominal and intestinal pain.

Surveys: A: Haymaker (1957); B: Motley *et al.* (1945); C: Russell (1943); D: Cotes (1952); E: Slark (1962); F: Rivera (1964); G: Kleinfeld and Wilson (1956).

their tremendous diversity, not only in the nature of the reaction of the body to the primary insult(s) but in the severity and combinations in which they occur. This point is well illustrated by Behnke (1951) who, in reviewing 55 cases investigated by Masland (1943), concluded that 'on no single point do all cases agree'.

Classification

Symptoms which have been attributed to inadequate decompression can be conveniently grouped into categories of similar physiological mediation of the likely primary insult(s)—although these have not been identified unequivocally. The first two can be conveniently taken as the two major types of decompression sickness according to the classification adopted by the Decompression Panel of the Medical Research Council, London (Golding *et al.*, 1960).

(I) Limb pain: this is a local pain of widely varying intensity generally known as the 'bends' and described in more detail later since it is by far the most common symptom of decompression sickness.

Griffiths (1969) points out that, apart from this pain in one or more limbs, the man neither feels nor looks ill. While he restricts the classification of Type I decompression sickness to cases where limb pain is the only symptom, Kidd and Elliott (1969) include cutaneous and lymphatic manifestations within this category as essentially local reactions of the body. Hence this category can include the rash or patchy mottled appearance of the skin indicative of irregular cutaneous vasodilatation often preceded by intense itching (pruritus). These manifestations are often termed 'skin bends'.

In about five per cent of Type I cases there is tenderness of the limb associated with lymphatic occlusion as evidenced by the 'orange peel' appearance of the skin distal to the obstruction.

(II) Symptoms related to the central nervous system (CNS): these are less common than Type I but are much more serious, the subject feeling and appearing to be ill. Spinal lesions are among the most dangerous and can start with paraesthesia or numbness and lead to paresis (often hemiparesis), paralysis (often hemiplegia), unconsciousness and, ultimately, to death. Various other neurologic manifestations can also occur, including aphasia, severe headache, various visual disorders and impaired cerebration in general. There is some feeling that this category should be divided into two: one for obvious spinal involvement and the other for cerebral symptoms (p. 64). Comprehensive clinical descriptions of CNS manifestations of inadequate decompression have been presented by Fryer (1969), Griffiths (1969) and Elliott *et al.* (1974).

The occurrence of a Type II 'hit' is the most serious medical event which can occur in any of the pressure-related occupations, in view of the frequency with which such cases have led to death or permanent paralysis usually affecting the lower limbs and sphincter control of the bladder and rectum.

(III) Otologic disorders: these include symptoms involving dysfunction of the vestibular mechanism such as nausea, vomiting, vertigo, nystagmus and those associated more with the cochlear mechanism, viz. tinnitus and partial deafness. All are associated with the effects of primary insult to the inner ear, either to the end organ, or at any point along the spinovestibular tract to the cerebellum. These 'vestibular bends' are becoming such a major subdivision of Type II decompression sickness with the advent of very deep diving that they would now seem to warrant their own category. Other manifestations are collapse and may include a condition commonly termed 'the staggers' by caisson workers.

(IV) Dysbaric osteonecrosis ('bone rot'): this is an insidious manifestation of exposure to compressed air and possibly to other breathing mixes under hyperbaric conditions. Radiographically identifiable bone lesions are common in caisson workers and divers, occurring in both the shaft and the ends of the long bones—especially in the head of the humerus

and both ends of the femur. Those in juxta-articular locations are particularly serious since they can lead to collapse of the articular surface of the joint and to disablement in which surgery is usually of limited benefit. Particularly comprehensive descriptions of the lesions have been given by Golding *et al.* (1960) and McCallum *et al.* (1966). Since there is some measure of doubt in attributing dysbaric osteonecrosis to inadequate decompression, this category is treated apart from the others (p. 195).

Miscellaneous symptoms

There are a few less common symptoms which are difficult to fit into the above classification until their aetiology is better understood. These include 'the chokes', various psychosomatic disturbances, unusually severe fatigue with muscular weakness and acute lower abdominal pains which have been the principal feature in a few cases leading to death (Fryer, 1969). 'The chokes' is a substernal burning pain leading to coughing which may recede or progress to tachypneoa, hypoxia, shock and tussive syncope. For more details of the symptoms of inadequate decompression the reader should consult the comprehensive clinical descriptions compiled by Fryer (1969) and Kidd and Elliott (1969).

Incidence

The overall incidence of decompression sickness varies according to the adequacy of the decompression and hence upon the validity of the theoretical basis for the procedure adopted; any errors in using that procedure in practice; and any undue variation of the individual beyond the tolerances of the underlying model. The last of these is discussed later in connection with individual susceptibility (p. 41). However, much data is available upon the relative distribution of symptoms. Duffner *et al.* (1946) found that two symptoms occurred simultaneously in 26% of cases and more than two in 4·4% while pain of some kind failed to occur in only 5·3%. In analysing these complexes, Fryer (1969) points out that in 87% of the occurrences of limb pain, this is the only symptom, whereas 'chokes' and collapse rarely occur alone.

A recent review of 2500 U.S. Navy 'diving accidents' indicates that there was vestibular involvement diagnosed in 10–20% of these and in 30% of those which would be classified as Type II cases under the MRC two-category system (Kennedy and Diachenko, 1975). Another review of U.S. Navy diving records has indicated spinal cord involvement in about three times more diving accidents than attributed to other forms of neurologic disorder (Hallenbeck *et al.*, 1975).

The relative incidence of symptoms is influenced to some extent by the nature of the exposure and decompression but the results of several surveys are summarized in Table 2. These are difficult to compare because the symptom groupings are different but the overall trend seems to be much the same for divers, caisson workers and aviators. All show that by far the most common symptom is localized limb pain.

Limb 'bends'

One of the best descriptions of limb 'bends' is offered by Behnke (1951) as 'a dull throbbing type of pain, progressive and shifting in character, and frequently felt around the joints, or deeply in muscles and bones'. If untreated, the pain will increase in intensity and then regress, often reaching a 'peak' several hours after onset—sometimes of unbearable intensity (Aldrich, 1900). It has been known to persist for days. The pain does not seem to occur within the joint itself but around it and, as Ferris and Engel (1951) point out, appears to be strictly limited to those parts of the body primarily concerned with locomotion. This caused Fryer (1969) to comment upon how remarkably sparing is the affliction of certain joints such as the sterno-clavicular, the temporo-mandibular and those of the vertebral column.

On any occasion that Type I decompression sickness becomes manifest clinically, there is no predicting the limb or the side of the body

on which pain will occur. However, the distribution of limb 'bends' is not completely random, the preferred sites including the shoulders, elbows, wrists, thighs and knees.

Symptoms and occupation

Bends, essentially identical in character to those described by divers, are experienced by aviators. Behnke (1942) reported that men with mild symptoms were unable to distinguish between pain induced in a low-pressure chamber and that following diving. However, pain is more likely to occur in the shoulders of divers (Kidd and Elliott, 1969; Rivera, 1964; Slark, 1962) but in the lower extremities of aviators (Fryer, 1969) and caisson workers (Golding *et al.*, 1960; Rose, 1962). This difference is consistent with the nature of the work involved in each of the pressure-related occupations and the selective exercise incurred by each. The effects of exercise are discussed on p. 45.

When they occur, Type II symptoms of decompression sickness are often preceded by Type I manifestations. The relative rareness of spinal cord involvement with aerial decompression and more rapid amelioration of symptoms with recompression could indicate that any difference is essentially one of degree. This apparent limit to the severity of the manifestations of decompression sickness experienced by aviators has been attributed to the absolute value of normal atmospheric pressure (Piccard, 1941), i.e. aviators cannot experience a pressure change of more than one atmosphere.

The implication that Type II symptoms are an exacerbation of the same phenomenon underlying Type I is not certain and is discussed in much more detail on p. 60. In this connection K. H. Smith (in a personal communication) has made the interesting comment that a very close physical examination of a man with limb bends will usually reveal minor CNS involvement. The two may differ simply in onset time with Type I becoming apparent and requiring treatment before Type II can become manifest clinically.

Onset time

Symptoms can occur either during decompression or after the diver or caisson worker has returned to normal atmospheric pressure, those appearing before 'surfacing' being more common with helium–oxygen diving. This may not be attributable to the use of helium but to the need to use this gas for deeper dives which offer a much more exacting test of any calculation method and therefore emphasize any deviations of the calculated format from the 'ideal' decompression procedure. Over a greater depth range, these errors have more opportunity to accumulate to the extent that they become manifest as symptoms before the surface is reached.

Naval data from Canadian (Kidd and Elliott, 1969), British (Slark, 1962) and American (Rivera, 1964) sources indicates that 10–30% of cases generally occur in divers before surfacing, roughly half of these being limb bends alone. Of all cases, 60–90% occur within one hour of surfacing but there is some discrepancy in the number of Type II cases presenting within this period. This can be easily explained by the wide variation in exposures and decompressions involved in diving by comparison with aviators or even caisson workers.

Even in these occupations, the time of onset and rate of bends propagation are variable. This applies not only to the difference between individuals but to repetition of the same exposure on the same individual following a decompression profile of marginal adequacy.

Typical values for the time of onset of symptoms in caisson workers are given in Table 3. These data can be summarized by a much more recent statement (Griffiths, 1969) that, 'in the great majority of cases, the symp-

Table 3 Time of onset of symptoms in caisson workers

Time of onset	Number of cases	
First hour	64·2%	60%
Second hour	17·7%	35%
Third hour	6·9%	3%
Fourth hour	3·2%	
Fifth–eighteenth hour	7·6%	
After twelfth hour		2%
Total cases	280	300
Source of data	Levy (1922)	Thorne (1941)

toms commence within three quarters of an hour of the completion of decompression and only rarely are they delayed for a period of several hours'. Moreover, the potentially more serious cases usually develop early.

Exercise hastens onset appreciably (see p. 45). For this reason certain pearl divers in the Arafura Sea chose to work hard after returning to the deck. They argue that any impending symptoms will then come on quickly so that the treatment can be started by going back to depth before the ship moves to water too shallow for adequate recompression or too deep to anchor. Some Greek sponge divers also take this attitude while others claim that smoking a cigarette has the same effect of advancing onset. Few seem to take the attitude that by sitting quietly after return to the deck they might avoid a marginal bend. It is their experience that delayed symptoms seem to take longer to treat. This is compatible with the findings of Doll and Berghage (1967) that the longer the latent period, the longer and deeper the treatment needed.

From the viewpoint of establishing the inherent distribution of onset times, the most reproducible data should be that afforded by aerial decompression. Subjects who have been previously breathing air for an effectively infinite time at normal pressure can be regarded as at steady state before hypobaric exposure. Ferris and Engel (1951) present much data which they summarize as showing that pain generally reaches unbearable intensity after 15 min at 35,000 feet, while it rarely develops after 90 min.

Intensity

This raises the question of how to judge the severity of a case. One obvious yardstick is the form of manifestation, with Type II decompression sickness being regarded as more serious by virtue of the greater likelihood that it may lead to death or residual effects. The other aspect is to try to estimate the intensity of pain as the most common symptom.

Unfortunately the quantitative means available for estimating the severity of limb bends seem very arbitrary—usually dependent upon individual ability to withstand pain or upon personal descriptions of its intensity. One of these criteria is whether or not the subject seeks treatment. Since *treatments* are entered into the diving logs, or the records of the medical locks provided at the sites of tunnel construction, their preponderance tends to be taken as the incidence of decompression sickness.

In aviation terminology, a 'forced descent' is recorded when the pain reaches unbearable intensity at a particular altitude, or simulated altitude, to necessitate recompression to normal pressure. Much data relating the incidence of 'forced descents' to onset time has been given by Grenell *et al.* (1944), Ryder *et al.* (1945), Stewart *et al.* (1943), Robinson (1943) and Russell (1943). Attempts to analyse these data quantitatively have not ventured beyond use of the simple exponential function—i.e. a simple linear relationship between the rate of increase of cumulative incidence and onset time. Such simplistic approaches have indicated a latent period of 10–25 min preceding an increase in the number of cases which, if exponential, would have a half time of 10–30 min.

Having discussed the symptoms of inadequate decompression, their incidence, onset and intensity, as they have occurred 'in the field', it is now desirable to look at the factors which can influence them under controlled conditions which enable the major influences to be delineated.

A classification which has proven particularly convenient in previous surveys (Hills, 1966; Fryer, 1969; Kidd and Elliott, 1969) divides these factors into those inherent in the constitution of the individual as opposed to those describing the environmental history leading to decompression sickness.

Environmental Parameters

The imminence of decompression sickness is determined by the whole environmental history but the predominant parameters are pressure and time.

Any individual making a pressure excursion

must start and finish at atmospheric pressure. If decompression precedes compression, this represents aerial ascent. Moreover, if the subject was breathing air beforehand, then he can be assumed to be starting from a steady state, so that rapid ascent to a given altitude provides one of the few opportunities to look at the effects of decompression *per se*.

Decompression per se

There is unanimous agreement that greater decompression increases the imminence of symptoms. Each individual can be regarded as having his own threshold altitude which will vary slightly with body factors together with a random element inherent in almost any mechanism. The thresholds of a higher proportion of individuals of any group, therefore, are exceeded upon continuing decompression to greater altitudes, thus increasing the incidence. Moreover, the severity of symptoms increases in any one individual as the pressure is further reduced below that threshold or 'minimum bends altitude'. To give specific values, about 13% of a very large number of Air Force cadets were found to bend at 23,000 feet (0·404 ATA) but 50% at 32,000 feet (0·271 ATA) according to Gray (1951). However, there is the odd case reported at 18,500 feet (Fryer, 1964) or even 10,000–12,000 feet (Allan, 1945).

If compression precedes decompression in the environmental excursion, then it is only feasible to compare the effect of decompression *per se* if we standardize the previous pressure history. This presents an infinite number of possibilities but one of the more meaningful for investigating the underlying mechanism is the case where the subject remains for sufficient time on a fixed breathing mix at an absolute pressure P_1, say, to attain a steady state—whatever this might represent intrinsically. Steady state is used purposefully in perference to the more popular term 'saturation'. Although an inert gas probably equilibrates between body and atmosphere, saturation in the true physico-chemical sense cannot be obtained in a living tissue by virtue of the inequality introduced by the metabolic gases (see p. 239).

There is some doubt concerning the time required to attain steady state at elevated pressure but 8–12 hours is generally regarded as long enough for overall gas content to approach to within 95–99% of its final (asymptotic) value (Hempleman, 1969; Crocker *et al.*, 1951). However, if this pressure (P_1) is approached from higher rather than lower pressures, then there is evidence to suggest that this period could be considerably longer (Barnard, 1975). This is attributable to the residual, yet often asymptomatic, effects of previous decompressions.

On rapid decompression from a steady state at P_1, there is a remarkably well defined threshold pressure (P_2) at which the subject will develop bends if kept at that pressure for up to two hours (see fig. 11). This is well demonstrated by the work on goats by Hempleman (1957) who 'titrated' P_2 to obtain threshold values for various values of P_1. The relationship between P_1 and P_2 for marginal symptoms is discussed in detail later and shown in fig. 35.

If P_2 is normal atmospheric pressure, $P_1 - P_2$ now represents the least hyperbaric exposure, which can precipitate symptoms in that subject. This 'minimum bends' depth is, therefore, one of the best indices available to use in assessing the inherent susceptibility of an individual diver. Actual values are discussed in detail later but the original estimate of Boycott *et al.* (1908b) put the value for air-breathing naval

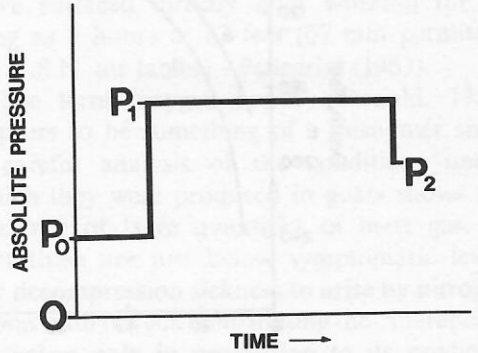

Fig. 11 Illustrating a simple exposure to an absolute pressure P_1 followed by rapid decompression to P_2. Normal atmospheric pressure is P_0

divers as around 38 feet. In 'weaker' subjects less likely to be pre-selected for their bends tolerance, the minimum bends depth is closer to 33 feet (Behnke, 1951).

The minimum bends depth is the hyperbaric counterpart of the minimum bends altitude in aerial work, both having the great advantage of presenting an index of individual tolerance essentially independent of time. However, if insufficient time is allowed to attain a steady state at P_1, a distinct threshold value of P_2 can still be obtained by titration (Hempleman, 1957) but the new value of P_2 is less than before —i.e. more decompression can be tolerated. Thus the imminence of bends for any given decompression is as much a function of the time as the depth—at least, for times insufficient for the attainment of a steady state.

Exposure pressure and time

Because divers would like to return to the surface rapidly, without invoking gradual decompression, there is naturally much interest in the case where P_2 is normal atmospheric pressure and P_1 is titrated to give a marginal condition upon surfacing. If this titration is repeated for different exposure times on the same breathing mix—air for instance—then the locus of these conditions represents a unique relationship between pressure and time of great fundamental significance in attempting to develop a quantifiable model for decompression sickness. This is termed the 'no-stop decompression' or the 'bounce dive' curve, because it represents the limits of an exposure whose depth–time profile is 'square'—that is one preceded by rapid descent to the 'bottom depth' and succeeded by rapid return to the surface. Curves for naval divers breathing air (Van der Aue *et al.*, 1951) and 80% helium + 20% oxygen (Duffner, 1958) are shown in fig 12. If the combination of 'bottom' depth and exposure time at that depth exceed the limits shown by this curve, then more gradual decompression is needed.

The nature of the exposure can also have some effect upon the type of symptom observed. For example, short deep dives are believed to predispose the diver towards CNS involvement;

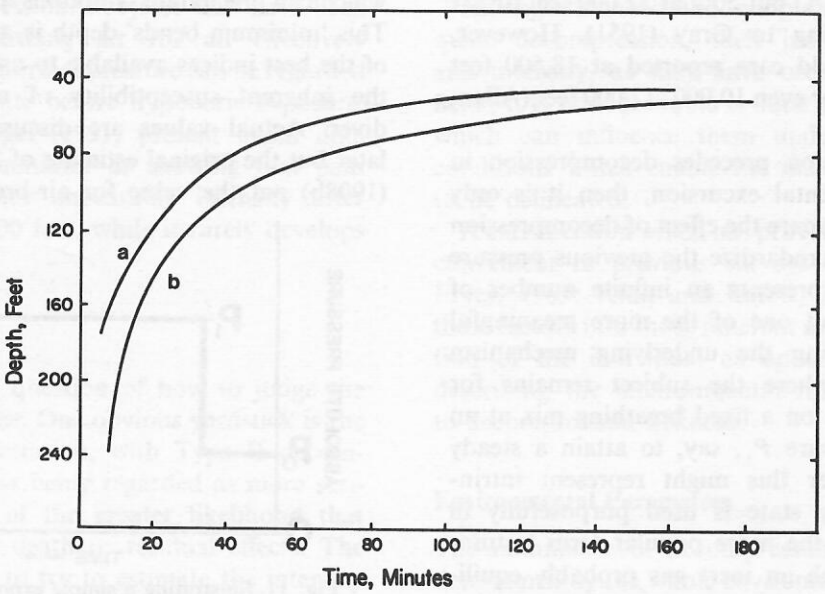

Fig. 12 The no-stop decompression limits, or 'bounce-dive' curves for men breathing (a) air—data from Van der Aue *et al.* (1951) and (b) 80:20 He:O_2—data from Duffner (1958) depicting his 'probable safe' minimal decompression curve

while the author has seen several vestibular 'hits' when heavy exercise has preceded minimal decompression. Unconfirmed reports also indicate a propensity for these category III symptoms to occur soon after switching from heliox to air at 120–180 feet during decompression—particularly following a 'saturation' exposure.

Vestibular manifestations tend to become the factor limiting decompression for depths in excess of 200–400 feet, Gehring and Bühlmann (1975) describing 12 cases of inner ear disorder following 24 exposures up to 1000 fsw. In 82 cases of decompression sickness following 211 of these very deep exposures, the only symptoms in 11 classified as 'neurologic' were vertigo, nausea, vomiting and tinnitus. The history of vestibular derangement induced by diving has been followed in detail by Kennedy (1974).

Oxygen partial pressure

There is no doubt that the substitution of oxygen for inert gas in the breathing mix reduces the likelihood of symptoms occurring upon subsequent decompression. As early as 1935, Hawkins *et al.* demonstrated that switching from air to oxygen two minutes before no-stop decompression enabled bottom time to be extended considerably (see Table 4). That is, the bounce dive curve for air (fig. 12) shifted to the right, so extending the safe range of no-stop exposures.

The switch from air to pure oxygen prior to aerial ascent offers undisputed protection against decompression sickness. One hour of this pre-oxygenation can reduce the number of cases in resting men taken to a simulated altitude of 38,000 feet by 95%; while two hours of oxygen-breathing reduces the incidence to one per cent of the previous value (Gray, 1942). The protection afforded by oxygen is not quite so spectacular in subjects who are exercising (see Table 5).

The protection also tends to be less if the oxygen is administered at lower absolute pressure, that is at altitude as opposed to pre-decompression. The incidence of cases of decompression sickness occurring in a large group of men at 35,000 feet was reduced from

Table 4 Comparison of exposure times with and without two minutes breathing 100% oxygen

Maximum exposure for direct ascent (min)			
		Air decompressions	
Depth (feet)	2 min on O_2* before ascent	U.S.N. tables (1954)	Miles (1962)
100	42	25	26
150	27½	15	11
167	16½	5	8
185	13½	5	7
200	13	—	6

*Data from Hawkins *et al.* (1935).

45% to 21% by interposing a stop of one hour at 20,000 feet during which they breathed pure oxygen. However, the incidence was further reduced to 12% when the hour of oxygen-breathing was switched to 10,000 feet, i.e. spent at a higher absolute pressure. In general, Gray (1944c) considers oxygenation at pre-bends altitudes to be '½–¾ as effective as at sea-level'; while Fryer (1962) selects 25,000 feet as the lowest pressure for gaining any significant protection from pre-oxygenation. The theoretical implications of the facts are discussed later (p. 138).

Instead of switching to pure oxygen, the addition of oxygen to breathing mixes can greatly extend the maximum depth of a diver for the same exposure time and decompression procedure. Thus, by increasing the partial pressure of oxygen to 1·6–1·8 kg. cm^{-2}, men have surfaced directly after working for as long as 7 hours at 82 feet (67 min permitted on U.S.N. air tables)—Pellegrini (1963).

The term 'oxygen bends' (Donald, 1955) appears to be something of a misnomer since a careful analysis of the conditions under which they were produced in goats shows the presence of large quantities of inert gas. In fact these are just below symptomatic levels for decompression sickness to arise by nitrogen alone, with oxygen contributing the difference—and then only in proportion to its predicted *venous* tension rather than its much higher arterial level (Hills, 1966).

There is no disputing the great benefit

Table 5 Reduction in aerial symptoms with pre-oxygenation

Original source of data—Author	Numbers involved		Conditions		Cases as percentage of those without pre-oxygenation: Mild symptoms		Severe bends	
	men	trials	Exercise level	Altitude (feet)	1 hour pre-ox.	2 hours pre-ox.	1 hour pre-ox.	2 hours pre-ox.
Ferris *et al.* (1943)	—	41	working	35,000	90–35%	45–50%	67–72%	25–28%
Clarke *et al.* (1945)	15	44	working	38,000	50–55%	17–19%	48–53%	13–15%
Henry *et al.* (1944)	—	52	working	38,000	20–22%	13–15%	20–25%	8–9%
Gray (1942)	80	—	resting	38,000	4·8–5·1%	< 1%	1·2–1·6%	< 0·1%

Data from Bateman (1951).

to be derived by using oxygen in the treatment of symptoms (see Chapter 8). These enormous advantages of oxygen might suggest eliminating the problems of decompression sickness entirely by diving upon pure oxygen alone. Unfortunately, it becomes toxic at elevated partial pressures with some particularly undesirable manifestations so that it becomes necessary to limit its usage and prudent to dispense it in an optimal manner (see p. 227).

While the substitution of oxygen for nitrogen in the breathing mixture clearly protects the individual, this does not necessarily hold for hyperoxia *per se*.

Hyperoxia per se

If, in place of substituting oxygen for nitrogen in the breathing mixture, the tissue nitrogen content is kept constant, then raising the inspired P_{O_2} above normal permits the effect of hyperoxia *per se* to be studied.

Analysis of the data for 'oxygen bends' in goats reported by Donald (1947) has indicated that, for the same partial pressure of nitrogen, elevating the oxygen partial pressure tends to increase the likelihood of decompression sickness (Hills, 1966). This marginal potentiation by oxygen and the propensity of Type II symptoms is consistent with this author's findings that goats can withstand less decompression if exposed for 8 hours to 200 feet on air than to 174 feet on 90 : 10 nitrogen : oxygen, i.e. at the same nitrogen partial pressure (186 feet). In both studies symptoms were predominantly Type II—a finding discussed on p. 68.

This work refers only to the oxygen level 'on the bottom' to try to isolate the effect of hyperoxia *per se* from the obvious kinetic advantages of oxygen in facilitating inert gas elimination *during* decompression discussed in the preceding section. This suggests that oxygen substitution is effective because it reduces the inert gas levels of tissue rather than combating any anoxic pain which could be produced by gas emboli lodging in arteries. This is consistent with the findings for hypoxia.

Hypoxia

Contrary to popular belief based largely upon infarction theories of decompression sickness (Fryer, 1969), the practical evidence indicates that hypoxia offers some protection in man—at least in aerial exposure (Motley *et al.*, 1945). Moreover, bends susceptibility in mice is lower in those more resistant to hypoxia (Yunkin, 1970).

Selection of inert gas

It was pointed out earlier that it is desirable to substitute helium for at least part of the nitrogen

of the breathing mix when diving deeper than 150–250 feet. This is due primarily to the improved pulmonary mechanics and reduced carbon dioxide retention by breathing a less dense gas—apart from its lower narcotic potency (see Chapter 8); but what effect does such a switch have upon the imminence of decompression sickness? There is really little information suitable for fundamental judgements. One piece of evidence in favour of helium is the greater minimum bends depth of 38–40 feet (Duffner, 1958) when mixed with 20% oxygen compared with 33 feet on air. However, the reverse seems to be true on aerial decompression, when previous ventilation with 80 : 20 helium : oxygen gave a lower minimum bends altitude than with air according to one report (Beard *et al.*, 1967).

Any inherent advantage in the use of helium, as suggested by the comparison of minimum bends depths, seems to be less if steady state is not attained before the start of decompression. In fact, as exposure time is reduced, there is the indication that the bounce dive curves for 80 : 20 helium : oxygen and air might cross (Hempleman, 1967), suggesting that a diver might go deeper on air than heliox for no-stop decompression if his bottom time were less than 15–20 min (see p. 188).

Bounce dive data for men breathing 80:20 helium : oxygen (Workman, 1965) tend to confirm the implication of Hempleman; but it is unfortunate from a mechanistic standpoint that these exposures were all safe and were not titrated to bends points to be sure that exposures could not match or exceed those for air. This implied greater susceptibility when using helium as the inert gas for short deep exposures is also reflected in the current U.S.N. (1974) diving tables. For 30 min at 200 feet, the air table requires only 73 min of total decompression time (in water) compared with 92–97 min on 80 : 20 helium : oxygen despite invoking oxygen-breathing at the 50- and 40-foot stops.

The fundamental ramifications of such observations are discussed in great detail in Chapter 6 but one is essentially balancing kinetic differences against solubility differences, with the latter —and hence helium—winning out as exposure time is increased, i.e. as a steady state is approached prior to decompression.

Limited data available for neon (Hamilton and Langley, 1971; Schreiner *et al.*, 1972) and hydrogen (Edel *et al.*, 1972; Edel, 1974) suggest that each of these reaches a slightly different compromise between kinetics and solubility which could make them slightly better suited than nitrogen or helium for particular ranges of exposure. However, the cost of neon and the explosive hazards of hydrogen indicate that these gases must still be regarded as novelties in diving.

Repetitive decompression

From the standpoint of elucidating the mechanism for bends, it is interesting to consider the case of repetitive decompression while breathing pure oxygen—a situation where no inert gas could be taken up. Houston (1947) found that the minimum bends altitude (normally at least 22,000–23,000 feet, p. 35) was reduced to 17,000–18,000 feet on successive rapid ascents, i.e. subjects became *more* susceptible with repetitive decompression despite there being no opportunity to take up more inert gas.

Repetitive hyperbaric exposures also appear to predispose the subject towards bends; although, in this case, it is more difficult to isolate the effects of repetitive decompression *per se* from those of repetitive exposure upon the cumulative uptake of gases. However, the best documented evidence refers to small animals (see p. 160). It has also been shown in goats that, beyond certain depth thresholds for *upward* excursions interposed between exposure and decompression, any symptoms are likely to be Type II rather than Type I (Hills, 1971a). On the other hand, *downward* excursion diving appears to follow much the same rationale as regular single exposures (Bornmann, 1970).

Acclimatization

So far, in this text, only situations where the repetition of decompression or exposure occurs

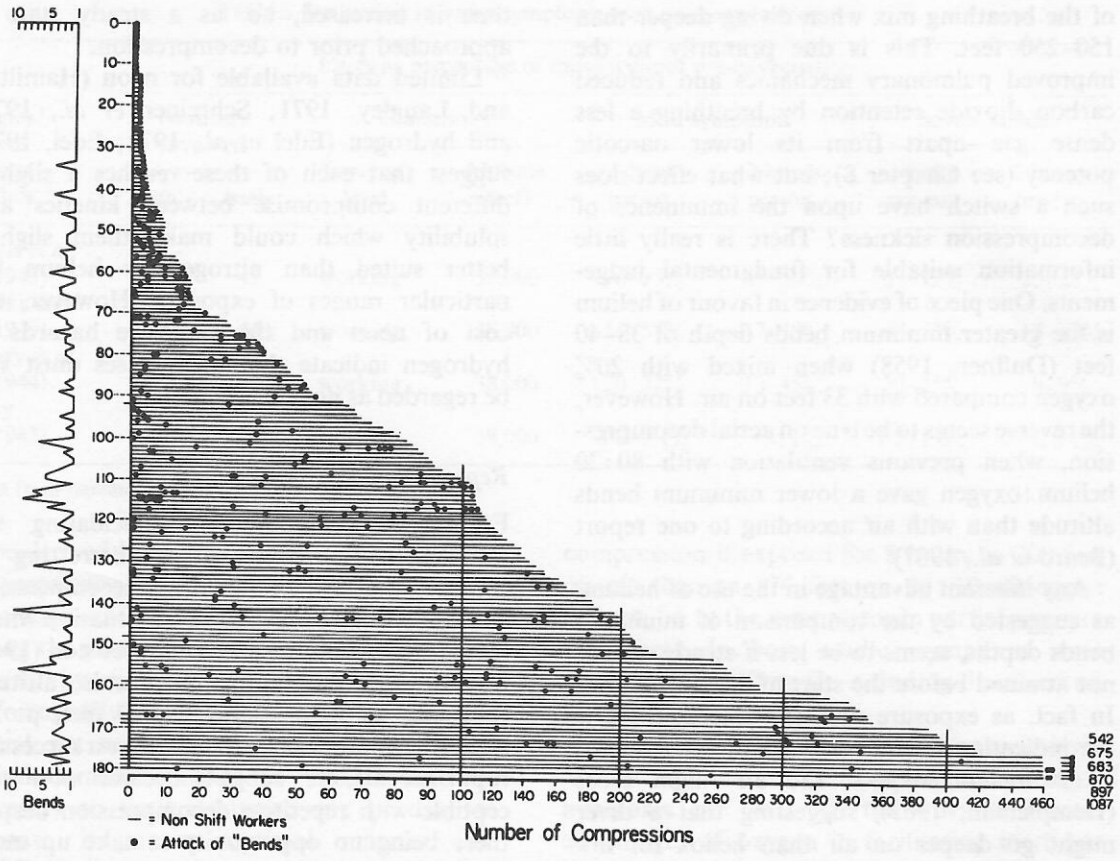

Fig. 13 'Bends' history of 181 compressed-air workers in the Tyne Tunnel. From Walder (1968) by permission of D. N. Walder. Black dots indicate attacks of 'bends'

within a few hours, if not minutes, have been considered. However, there is a long-term effect tending to induce the reverse effect, i.e. reducing the susceptibility with consecutive exposures.

The most specific evidence of adaptation is provided by data on the incidence of symptoms recorded in large numbers of caisson workers (Paton and Walder, 1954; Walder, 1968). For instance, if the construction of a tunnel has reached the stage at which the electricians are needed, they will incur the highest incidence of bends on their first decompression. In subsequent shifts, the number of cases requiring recompression is found to fall off daily in an approximately exponential manner. Careful analysis of these curves (fig. 13) shows that they are bimodal with the major component having a half-time of about five to seven days (Walder, 1968). There is also an indication that acclimatization to one pressure does not afford the same protection against exposure to a higher pressure. The other and much smaller component is suggestive of a long-term form of adaptation with a half-time of the order of several months.

The acclimatization seems to be largely reversible with no detectable protection remaining in the man who stops working in compressed air for about three weeks.

A similar phenomenon has been demonstrated in goats whose minimum bends depths are increased by 5–15 feet by regular exposure to compressed air at least three times per week (Hills, 1970a).

'Wet' versus 'dry' exposures

There are many sets of decompression tables which have proved satisfactory in chamber trials but have resulted in an unacceptable incidence of symptoms when tested in the ocean. Sometimes this can be attributed to failure to provide adequate simulation of exercise in 'dry' runs, but psychosomatic effects probably account for most of the difference.

There is also a difference depending upon whether the man is immersed in water or not. 'Skin bends' are much more common in 'dry' as opposed to 'wet' runs, i.e. in aviators or subjects in chambers simulating dives as opposed to divers in 'wet suits'. Rashbass (1956) found that he could induce pruritus in most subjects in a 'dry' chamber upon rapid decompression following an air exposure of 18 min at a simulated depth of 240 feet. However, it was prevented over all areas of the skin immersed in water at body temperature prior to decompression.

Skin contact

Symptoms identical to 'skin bends', presenting both urticaria and pruritus, have been produced *without decompression* over those parts of the body exposed to heliox while the subject breathed air at a simulated 200 feet (Blenkarn *et al.*, 1971).

Another aspect of skin contact is the diver who puts on weight and out-grows his 'wet suit' or who borrows one which is too small. On return to the surface, he is far more likely to get bends pain around a joint where the limb is 'pinched' by the suit. Similarly, an injury or a tourniquet applied to a limb during decompression predisposes that limb to bends—Ferris and Engel (1951), Haymaker (1957).

Temperature

Cold water has long known to predispose the diver towards bends.

Both Nims (1951) and Cook (1951) summarize earlier work which shows that an increase in environmental temperature produces a slight drop in the incidence of decompression sickness on aerial exposure. This has been particularly well demonstrated by Tobias *et al.* (1943) who found a lower incidence of bends in locally heated regions than in non-heated regions of the body, including contralateral sites.

A similar correlation between cold exposure and the incidence of fatal decompression sickness has been demonstrated in rats exposed to positive air pressures (Hempleman, 1968).

While there seems little doubt that increased environmental temperature during decompression tends to offer a marginal reduction in the incidence of bends, there is some evidence to indicate that body temperature can rise after the onset of symptoms (Masland, 1943; Goggio, 1943; Adler, 1950). However, the limb in pain usually appears cyanotic and feels hypothermic (Ferris and Engel, 1951).

Recompression

Recompression of the diver with overt symptoms almost invariably gives immediate relief of limb bends and other Type I manifestations of decompression sickness. It also gives relief for the vast majority of Type II (CNS) cases but the failure rate is higher. Since recompression is by far the most successful treatment, it is discussed in more detail later (see Chapter 8).

Individual Factors and Susceptibility

There have been many attempts at somatotyping in order to predict the men most susceptible to decompression sickness before employing them as divers, aviators or caisson workers. While most have proved unsuccessful, a few general trends have emerged from the large random scatter; but the less obvious have only reached statistical significance where large numbers of subjects have been tested. The principal factors of body constitution which have shown a clear correlation with susceptibility are obesity and age.

Obesity

As early as 1868, de Méricourt recommended

that no corpulent individual be employed as a sponge fisherman, while Smith (1873) noted the number of stout, heavily-built men among those taken sick in the New York caissons. Since these early observations there have been many attempts to obtain quantitative correlations between obesity and bends incidence. Fraser (1942) indicated a linear variation between corporeal density (body weight/surface area) and susceptibility while Gray (1951), using data from many sources, claimed relationships with linear density (weight/height) and ponderal index (weight/height^3) (see fig. 14a). These general trends in aviators have been confirmed by Welham *et al.* (1944) and Swann and Rosenthal (1944). The dates of these reports are significant since it is only in times of national emergency that 'volunteer' subjects are available in sufficient numbers to establish the less obvious trends.

However, subsequent surveys of caisson workers (Paton and Walder, 1954) and divers (Wise, 1963) have largely failed to substantiate these claims to statistically significant levels; although Wise did reach a one per cent level using the Sheldon somatotype index.

Even before Vernon (1907) aroused scientific interest in obesity by showing that nitrogen was five times more soluble in oils and fats than in water, there were animal studies which showed fewer lesions in lean than in fat animals after hyperbaric exposure (Heller *et al.*, 1900). Later studies essentially confirm these findings (Boycott and Damant, 1908a; Gersh *et al.*, 1945; Philp and Gowdey, 1964; Gowdey and Philp, 1965).

The problem really amounts to one of finding a better index to describe obesity. Hence it is disappointing that attempts to correlate symptoms in divers and caisson workers with skin-fold thickness Walder (1966b) and total body fat (Wise, 1963) proved negative.

Age

There is little doubt that susceptibility tends to increase with age, qualitative reports indicating this trend dating back to Catsaras (1890), Snell (1896), Heller *et al.* (1900) and later corroborated by Boycott and Damant (1908a) and Hill (1912).

From the USAF records of massive numbers of potential aviators tested during the Second World War, Gray (1951) has shown a roughly linear advance of susceptibility with age (fig. 14b), expressing the relationship as

$$\text{Relative susceptibility} \propto \text{Age (in years)} - 13{\cdot}4 \qquad (4)$$

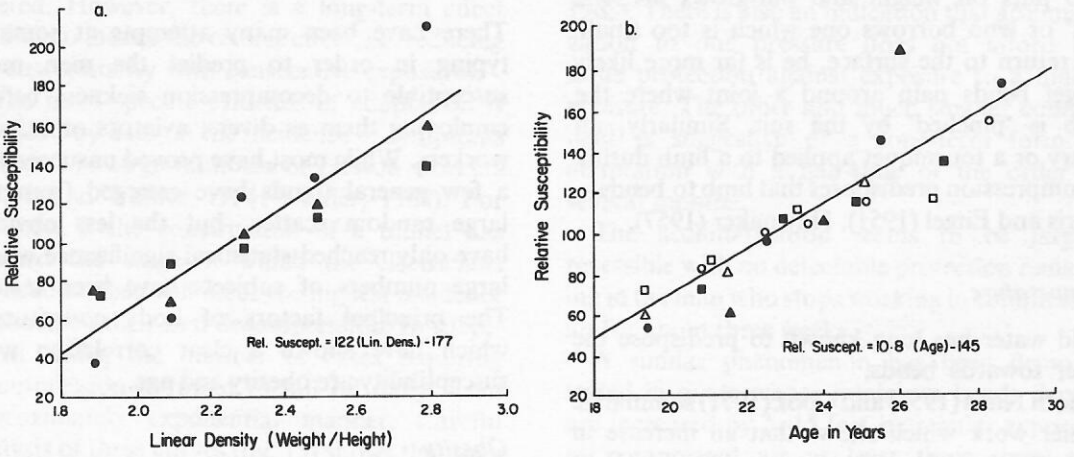

Fig. 14 Relative susceptibility to decompression sickness plotted against (a) linear density and (b) age. Data from Gray (1951)

On the other hand, Walder (1969) expresses the view that the bends rate is not obviously related to age for men below the age of 40 but rises thereafter with a particularly steep increase for those over 50. Taking caisson workers as a group with a relatively wide range of age, Paton and Walder (1954) have shown that, on the whole, the older the man the sooner he will experience decompression sickness after starting work in compressed air.

Physical fitness

The popular notion that physical fitness reduces susceptibility to decompression sickness does not seem to be substantiated by 'hard' scientific data.

Karpovich (1944) found no correlation between the incidence of symptoms and the scores of physical fitness tests taken by 197 AAF cadets. On the other hand, Ferris and Engel (1951) indicate a slight correlation between susceptibility and lung clearance from data recorded by Henry (1945) and Stevens *et al.* (1943). A more definite trend is claimed by Jones *et al.* (1942) who found a decrease in susceptibility with increased elimination rate of an inert gas from the body. While largely substantiated by Henry (1943), *et al.* (1943), the correlation is still described as 'poor' by Henry and Ivy (1951). These authors claim that the uptake of radioactive krypton by a hand shows 'more promise'.

One of the reasons for the lack of correlation may be the need to look at *relative* fitness rather than the ability to perform certain physical tasks. However, this raises the problem of how to estimate the ultimate level of achievement to be expected from a subject without his actually undertaking a course of physical training.

While the foregoing reasoning applies to men in essentially good health, there are numerous examples where prior disease or injury has increased susceptibility to decompression sickness (see Fryer (1969)). Sites of recent minor injury are particularly prone to limb bends (Thompson *et al.*, 1944).

Water balance

There is evidence to suggest that subjects with a naturally high rate of water turnover tend to be slightly less susceptible to decompression sickness—as shown in aviators (Warwick, 1943; Cook *et al.*, 1944; Walder, 1948). However, it remains a controversial issue whether a forced increase of imbibed fluid offers any protection.

Race and sex

Any inherent differences in susceptibility between groups of different race or national origin are certainly not obvious or, at least, no greater than otherwise predicted on the basis of obesity, acclimatization, etc. Thus Motley *et al.* (1945) estimated that severe symptoms in potential aviators occurred in 4·7% caucasians compared with 3% for negroes out of 4,600 American subjects similarly decompressed.

The author has seen a few Greek sponge divers and several Okinawan pearl divers who could tolerate less gradual decompression than most other men but they were particularly lean and well acclimatized. Moreover, they would start in shallow water and go deeper during the season, regularly diving at least twice per day and six days per week for 10 months per year in the case of pearl divers in Australian coastal waters. The season is 7–8 months in the Eastern Mediterranean. By comparison, the divers of most navies could be regarded as unacclimatized, so that no national comparisons suggest any intrinsic difference.

Relatively few women have been decompressed (Allan, 1945; Fryer, 1969) but their susceptibility showed no obvious difference from men.

However, there is some evidence to indicate that pregnancy offers marginal protection in goats (Gray, 1951).

Psychological aspects

Another factor little understood is the psychological stress of diving which can not only

affect the performance of a diver in completing a task underwater (Bachrach, 1975) but can also influence his susceptibility to decompression sickness. Hedgepeth (1976) describes certain individuals he considers temperamentally maladjusted to commercial diving who may fear that they will get 'bent' on a particular dive and surely do so. It has been this writer's experience that native pearl fishermen who tend to avoid certain parts of the ocean where they claim there is 'heavy water' will usually get 'bent' when diving in those areas and yet instrument surveys reveal no scientific reason. The same men show no undue sensitivity to decompression sickness when diving elsewhere (LeMessurier and Hills, 1965) and yet are most superstitious and will often refuse to dive if they have forgotten their rabbit's foot or other lucky charm. Thus the psychological aspects of diving are most complex and must remain a factor virtually impossible to feed into any computer for generating decompression schedules. Psychosomatic changes in blood enzyme levels have also been found with diving (Martin, 1972).

Time of day and year

Slight variations in bends susceptibility have been found with the season and time of day. Henry *et al.* (1943), after carefully standardizing their decompressions to simulated altitudes, found a slight yet distinct increase in symptoms occurring during morning trials compared with afternoon trials. This coincided with a decrease in the measured metabolic rate of the same subjects. Gray (1943) found that this diurnal bends-effect became more apparent at greater altitude, the number of forced recompressions at 38,000 feet reaching 70 in morning trials compared with 40 in the afternoon.

Distribution of susceptibility

The foregoing discussion has covered many factors which could influence tolerance to decompression to various degrees and there may be many more as yet undiscovered. Consequently, it would be an horrendous task in statistical analysis to try to isolate many of the less apparent trends when all act simultaneously to determine the susceptibility of the individual.

Whatever the contributing factors and their relative predominance, however, there is the practical need to characterize the net distribution in tolerances as far as possible. This would help in changing any decompression format from, say, a 5% to a 1% bends incidence whatever the validity or efficiency of the model underlying the calculation method. Two different approaches have been reported.

The incidence of decompression sickness in 288 mice exposed for 15 min to nitrogen : oxygen at 13·8 and 14·2 ATA has been shown to be consistent with a binomial distribution or, at least, departures from theoretical estimates can be explained on the basis of chance alone (Berghage *et al.*, 1974). However, these short exposures superimpose upon susceptibility another set of variables concerned with the *kinetics* of gas transfer in each individual.

An alternative approach has sought to avoid time-dependent factors by starting the critical decompression with the subject in a steady state, using the minimum bends depth of a diver (or the minimum bends altitude of an

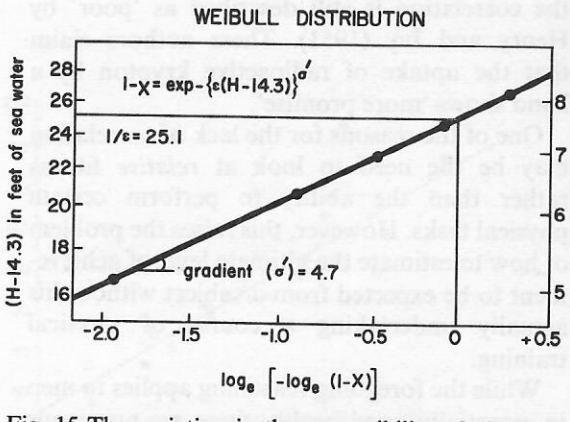

Fig. 15 The variation in the susceptibility of divers to decompression sickness plotted as a Weibull distribution (Hills, 1968a) expressing the cumulative fraction of cases (X) as a function of their minimum bends depth (H)—the maximum depth for safe return direct to the surface after eight hours exposure upon air. Data points are taken from Crocker *et al.* (1951)

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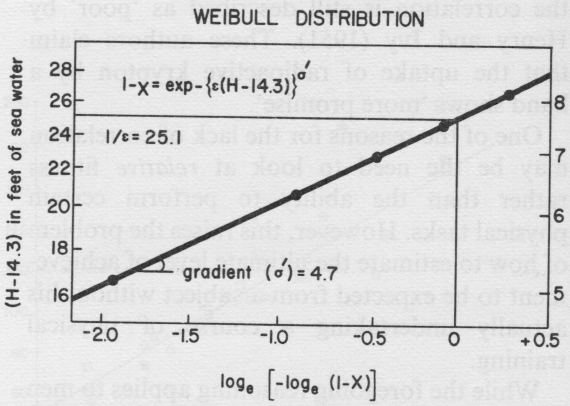

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aviator) as the best index of inherent susceptibility (see p. 35 for the full significance of these indices).

Accordingly an average individual has a minimum bends depth of 33 feet of sea water on air (Van der Aue *et al.*, 1945)—sometimes loosely termed a '33-foot man'—compared with a 38-foot average diver (Boycott *et al.*, 1908b; Behnke, 1951). Professional divers tend to be a somewhat preselected group of the population. Any individual particularly susceptibile to bends will tend either to drop out of the profession voluntarily or to lose his job if his employer is afraid that he may be prone to more serious symptoms in the future which might precipitate legal action for damages.

Bearing these considerations in mind, it is interesting to see how closely data for the distribution in the minimum bends depths of naval divers (Crocker *et al.*, 1951), goats (Davidson *et al.*, 1950) and the minimum bends altitude of pilots (Gray *et al.*, 1945) can be described (Hills, 1968a) by the Weibull function (see fig. 15). This is a statistical function which has proved particularly successful in renewal theory (Cox, 1962) to quantify such events as the breakdown of television sets or the failure of light bulbs.

The distribution of minimum bends depths has been obtained in practice by gradually increasing the air pressure (P_1) at which they have been permitted to reach steady state before rapid decompression to a pressure P_2, gradually eliminating men from further trials as they 'bent' (see fig. 11). If the cumulative fraction of cases up to P_1 is χ, then it is found (Hills, 1968a) that the 'survivor' function $(1-\chi)$ is related to minimum bends depth (H) by

$$1-\chi = \exp\{-[(H-14{\cdot}3)/25{\cdot}1]^{4{\cdot}73}\} \quad (5)$$

where $H = P_1 - P_2$ expressed in feet of sea water. Applied to the more general case where P_2 is not normal atmospheric pressure and the subject is breathing a gas mixture of which a volume fraction F_i is inert then

$$1-\chi = \exp\left\{\left[-\varepsilon\left[\frac{(P_1-46)}{(P_2+74)}-\frac{1}{F_i}\right]^{\sigma'}\right\}\right. \quad (6)$$

where ε and σ' are constants and P_1 and P_2 are now expressed in mm Hg.

Thus both statistical approaches have two degrees of freedom. The binomial has the mean and the degree of skewness, while the Weibull function has ε and σ' as the two constants for which one is free to select values to give the best fit. In view of the remarkable fit of decompression data offered by Equation 6, it is interesting to reflect that, in Reliability Physics, the Weibull function has generally proved of greatest benefit in describing what is essentially the failure of systems to which a multiplicity of factors can contribute.

Exercise

At first sight, the effects of exercise on the imminence of decompression sickness may appear confusing and apparently conflicting, until the incidence and onset time of symptoms are considered in the context of the phase of the environmental excursion at which the exercise is undertaken, i.e. whether, during any pre-oxygenation; at maximum or 'bottom' pressure; during decompression; or after decompression.

After decompression

There is little doubt that post-decompression exercise reduces the induction period for the onset of any impending symptoms. To support this statement there is a vast mass of data available—largely collected by the U.S. Air Force during the Second World War from which the following case has been selected as reasonably typical. Cook (1951) described an experiment on 474 men where a change from one to two sets of ten, nine-inch 'step-ups' every 30 seconds reduced the average onset time from 42·4 to 33·8 minutes at 30,000 feet and from 29·0 to 19·4 minutes at 38,000 feet.

From the same vast source of data, Gray (1943, 1944a) comes to the general conclusion that exercise taken aerially can reduce a man's minimum bends altitude by as much as 5,000 feet. This is in basic agreement with the feelings

of physicians attending divers and caisson workers that exercise *after* decompression tends to increase susceptibility, besides reducing onset time.

During pre-oxygenation

Exercise while breathing pure oxygen before decompression seems to offer marginal improvement in the unquestionable protection offered by pre-oxygenation (see p. 37).

While Robinson (1943) could find no difference, Gray (1942) claimed a reduction of incidence when men worked during pre-oxygenation; but inspection of the data shows that this was based upon a fall in the bends rate from 12·7% to 11·4%. On the other hand, Webb *et al.* (1944a) recorded 18 forced recompressions among 39 men exercising for one hour on oxygen before decompression compared with 26 for the same men resting during that period. Finally Evelyn (1941) claims that 30 min pre-oxygenation with muscular activity is equivalent to 60–90 min of the same preventive treatment while resting. Any improvement is easily explained by increased nitrogen washout with exercise.

At pressure

There is unanimous agreement that exercise *at depth* increases the likelihood of developing symptoms on subsequent decompression. As a guide to the magnitude of the effect, Behnke (1951) quotes 25 min as the safe working time at 100 feet to be followed by no-stop ascent compared with 35 min in the resting state. Once again, this can be easily interpreted on the basis of an increased rate of nitrogen transfer with exercise as opposed to any effect of exercise *per se*, since susceptibility was changed in the opposite direction to its effect during pre-oxygenation.

During decompression

This is the most interesting phase of an environmental excursion from a mechanistic viewpoint but, unfortunately, it is also the one in which the data is most conflicting. On the basis that exercise increases inert gas transfer, it would naturally be inferred (e.g. Haldane and Priestley, 1935) that it would do the same *during* decompression and so reduce the bends-rate. However, a distinct increase in the incidence of symptoms was observed when divers worked during their ascent on U.S.N. (1943) tables—Van der Aue *et al.* (1949).

On the other hand Japp (1909) found no adverse effect in letting caisson workers walk through the tunnel during decompression—but this was at a much deeper stop than advocated by the subsequent rationale published by Boycott *et al.* (1908) to become the basis of the U.S.N. tables. More recent findings seem to be exonerating exercise during decompression but these are also introducing deeper decompression stops. The theoretical compatibility of these observations is described on p. 251.

Carbon dioxide

Since this gas is produced in large amounts by the body during exercise, the effects described in this section have led to much theoretical speculation concerning its role in decompression. Carbon dioxide is then abundantly available in tissues although distributed between various forms of chemical association with body fluids so that quite a minority is actually present as simple molecular CO_2. Moreover, with such a high solubility, even this makes a minimal contribution to the total tension tending to induce bubble formation.

Gray (1944b), after recording every detail of all symptoms which developed at 38,000 feet in 204 subjects breathing 100% oxygen and another 204 simultaneously decompressed and breathing a mixture of 88% oxygen and 12% carbon dioxide, found that 'in no single respect was there any difference between the groups of even moderate statistical significance'. Thus Cook (1951) states that 'carbon dioxide has no effect whatsoever upon decompression sickness'.

This statement may well apply where the subject has already reached a *steady state* before decompression but there is some reason

to query it in cases where a man could be considered to be taking up inert gas at the time of increase of P_{CO_2}. There are data on caisson workers showing that men working in a poorly ventilated area of a tunnel are more susceptible to decompression sickness (Snell, 1896) while improved overall ventilation can reduce the incidence (Moir, 1896). The incidence is also higher where much carbon dioxide is released from the diggings (Kindwall, 1974). Momsen found that two divers who had bent in inadequately ventilated suits did not bend when they repeated the same dive the next day with much improved ventilation (Behnke and Yarbrough, 1938). These findings certainly implicate carbon dioxide but the effects do not seem to be large enough to warrant any explanation beyond the standard physiological response of the body to this gas as seen under normobaric conditions, viz. increased ventilation rate and cardiac output which could accelerate inert gas uptake. Unfortunately, there appear to have been no trials performed to test the effect of elevating the carbon dioxide only during decompression, when it should assist nitrogen elimination, or at the end of a so-called 'saturation' exposure.

In the absence of such decisive information, the aerial, tunnel and diving data can be said to be collectively compatible with the interpretation that carbon dioxide has no effect on decompression sickness beyond its ability to stimulate the cardio-respiratory system and so accelerate inert gas transfer.

However, it is possible that carbon dioxide may have a different role in the aetiology of dysbaric osteonecrosis (p. 202), bone representing a massive 'pool' for carbon dioxide storage as bicarbonate (Poyart *et al.*, 1975). It might also affect the lung as a bubble trap, facilitating the passage of 'silent' venous bubbles to the arterial system where they could induce Type II symptoms (p. 67).

General

This discussion, while by no means comprehensive, is intended as a summary selecting data considered most suitable to illustrate the effect of each factor isolated as far as possible from the host of other variables influencing the manifestation of inadequate decompression. Whereas their presentation has been purposefully divorced from mechanistic considerations, these basic facts can now be discussed as a whole in trying to determine the primary insult to the body which precipitates decompression sickness.