14

Decompression Theory and Applications

RICHARD D. VANN

After almost 100 years of diving and research, the mechanisms of decompression are still not completely understood, and the development of decompression procedures remains too much a matter of trial and error. Nevertheless, experience has made diving a practical and reasonably safe occupation. This chapter reviews the physical and physiological principles believed to be important in decompression, surveys current decompression practice as defined by several diving manuals, and discusses problems in the development of decompression schedules. A method of schedule calculation is introduced, and a series of decompression trials is described.

BUBBLE FORMATION

Although other mechanisms have been proposed, undissolved gas has been suspected as the cause of decompression sickness from the earliest days of decompression research. Bubbles are still believed to be the most probable cause, but it is now known that they have physiological as well as mechanical effects which can be equally or more dangerous and can persist for long periods of time.

The tendency for bubbles or cavities to form in a liquid is measured by the supersaturation (ΔP), which is defined as the difference between the sum of the dissolved gas tensions (ΣPi) and the barometric pressure (P_B):

$$\Delta P = (\Sigma Pi) - P_B \qquad (1)$$

The minimum supersaturation needed for formation of at least one bubble is known as the threshold supersaturation.

There are two principal mechanisms by which cavitation is thought to occur. In 1837 Schoenbein proposed that bubbles originate from pre-existing gas cavities now known as gas nuclei (Bateman & Lang 1945). In 1937 Doring postulated that bubbles form *de novo* from microscopic voids generated by random molecular motion (Hemmingsen 1978). Both mechanisms appear to be valid, but *de novo* nucleation requires higher supersaturations than are generated in diving (Gerth & Hemmingsen 1976). Gas nuclei, on the other hand, grow into bubbles at supersaturations of as little as 0.21 ATS (Bateman & Lang 1945). The evidence for gas nuclei is reviewed below.

In vitro cavitation

The application of hydrostatic pressure to water and gelatin, particularly at rapid compression rates, reduces the number of bubbles forming upon subsequent decompression. This is thought to occur because compression dissolves some gas nuclei (Harvey *et al.* 1944; Yount & Strauss 1976). Hydrostatic pressure treatment is now used as a specific test for gas nuclei.

Filtering gelatin before compression also reduces subsequent cavitation (Yount *et al.* 1979). Figure 14.1 shows that a filter with 0.45 μm radius pores decreases bubble formation by half and increases the threshold supersaturation from 1.5 to 2.5 ATS. A 0.18 μm filter diminishes bubble formation by a factor of almost 10 and raises the threshold to 6 ATS.

To explain these observations, Yount *et al.* (1979) suggested that gas nuclei are associated with particles having a range of sizes. The size of each nucleus determines the critical supersaturation at which it becomes a bubble. Figure 14.1 shows that the large nuclei have low critical supersaturations.

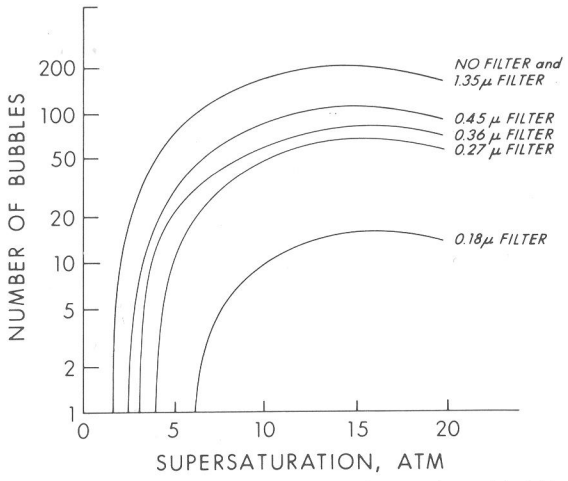

FIG. 14.1. The effect of filtration on the number of bubbles forming per gelatin sample after decompression from elevated pressure to 1 ATA (redrawn from Yount *et al.* 1979). Gas nuclei are associated with particles which can be removed by filtration. There are no nuclei with radii larger than 1.35 μm. Half the nuclei have radii between 1.35 and 0.45 μm. Larger nuclei grow into bubbles at lower supersaturations

As the supersaturation increases, smaller nuclei are recruited and more bubbles form.

A central question in the theory of gas nuclei is how they survive surface tension. If a gas nucleus were a spherical bubble, it would dissolve, because surface tension raises the internal pressure above the barometric pressure as described by Laplace's law (Fig. 14.2a).

Yount (1979) proposed that a gas nucleus is a spherical bubble stabilized against surface tension by a skin of surface active molecules (Fig. 14.2b). As a nucleus dissolves or is compressed, the molecules of the skin become packed together, forming a gas-tight barrier. If the mechanical strength of the skin is overcome by excessive compression, the nucleus collapses and is dissolved. During decompression the nucleus expands into a stable bubble when the internal pressure exceeds the pressure due to surface tension.

Quite a different explanation was proposed by Harvey *et al.* (1944), who postulated that a gas nucleus is a gas-filled crevice in a solid surface (Fig. 14.2c). The crevice is hydrophobic, so that it is not easily wetted by water. Upon compression the gas–liquid interface becomes concave, and the pressure in the crevice decreases, as required by Laplace's law. Thus, surface tension acts to stabilize rather than to dissolve the nucleus. The nucleus dissolves when excessive compression forces liquid into the crevice. It grows into a bubble during decompression when the gas–liquid interface becomes larger than a hemisphere (Fig. 14.2d). Expansion of the interface beyond a hemisphere is unstable, because the pressure due to surface tension falls as the radius increases.

Models of gas nuclei such as these are useful for interpreting experimental data. However, the data are not yet adequate to determine which model is most likely to be valid.

In vivo cavitation

The hydrostatic pressure test has been used to look for evidence of gas nuclei in animals. Evans and Walder (1969) studied bubble formation in transparent shrimp. They decompressed three groups of 50 shrimp from sea level to 0.079 ATA. One group was pressure treated at 389 ATA before

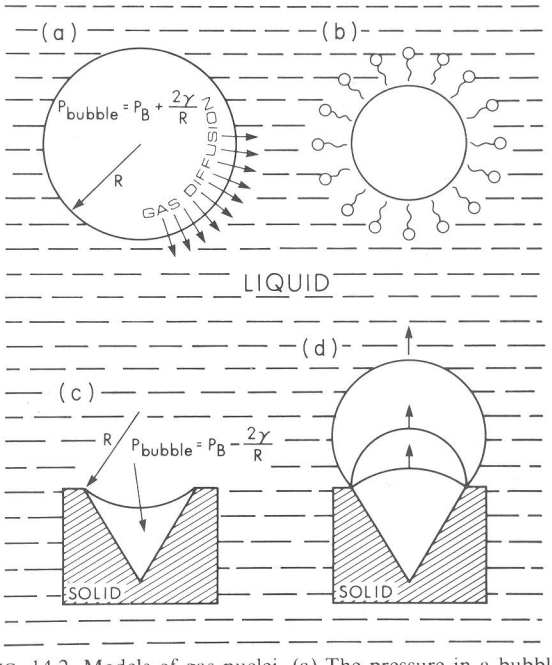

FIG. 14.2. Models of gas nuclei. (a) The pressure in a bubble exceeds the ambient pressure (P_B), and gas diffuses out of the bubble, causing it to dissolve. (b) A *skin-stabilized nucleus* in which a skin of surface active molecules prevents the outward diffusion of gas (Yount 1979). (c) A *crevice nucleus* in which a pocket of gas is stabilized in a crack in a hydrophobic solid (Harvey *et al.* 1944). The liquid does not advance into the crevice, because the crevice wall is not easily wetted, and a concave gas–liquid interface develops. By Laplace's law, the pressure in the bubble is less than the ambient pressure, and the gas is stabilized rather than dissolved by surface tension. (d) Bubble formation from a crevice nucleus. During decompression, the gas–liquid interface expands from the crevice (shown here as a cone), and its radius decreases, while the pressure due to surface tension increases. This pressure attains its maximum value when the interface is hemispherical. Further expansion causes the radius to increase and the pressure due to surface tension to fall, and a bubble forms

decompression. A second group was not pressure treated. Bubbles were observed in 4 pressure-treated shrimp and in 48 nontreated shrimp. A third group was pressure treated and then electrically stimulated to induce physical activity. Sixteen of these shrimp developed bubbles after decompression.

By the pressure test criterion, the bubbles observed in shrimp originated from gas nuclei. Similar experiments were performed to test the involvement of gas nuclei in decompression sickness (Vann *et al.* 1980). Three groups of rats made 2 h test dives to 240 ft (73 m), as shown in Fig. 14.3. One group, with an initial excursion to 1000 ft (303 m), had a 64% incidence of decompression sickness after the test dive. A second group made a 600 ft (183 m) excursion and had a 74% incidence. A third group, with no excursion, had an 83% incidence.

Again, the pressure test indicated the presence of gas nuclei. The observation that higher-pressure treatment gave greater protection suggests that, as in gelatin, gas nuclei are not alike in their responses to pressure change.

Beyer *et al.* (1976) conducted another experiment which tested the involvement of gas nuclei in decompression sickness. Fish were internally supersaturated by compression to and decompression from elevated pressure, or they were externally supersaturated at sea level by placing them in supersaturated water. Internal supersaturation includes a pressure treatment which is not present in external supersaturation. Fish that were internally supersaturated by exposure to air at 133 ft (41 m) had the same incidence of decompression sickness as fish externally supersaturated at a depth of 50 ft (15 m). The greater exposure depth for internal supersaturation was presumably a result of the destruction of gas nuclei during compression.

Adaptation

Regular exposure to pressure causes a reduced susceptibility to decompression sickness which is known as adaptation, acclimatization or habituation. Haldane was aware of the adaptation phenomenon as early as 1936 and recommended that compressed air workers be used on half-shifts during initial exposures (Rubenstein 1968). Walder (1968, 1975) presents evidence for compressed air adaptation indicating that the incidence of decompression sickness decreases with a half-time of 7 ± 4 days during daily exposures. If regular exposure stops, adaptation disappears in about 10 days. Adaptation is specific for each pressure and recurs with each increase in working pressure.

Walder (1969) suggested that adaptation is due to a gradual depletion of gas nuclei. Different populations of nuclei are eliminated at specific working pressures. Adaptation is lost when nuclei reaccumulate. Walder's hypothesis is supported by the observations of Yount *et al.* (1979) and Vann *et al.* (1980) that nuclei have dissimilar responses to decompression and to pressure treatment.

Adaptation has also been reported in diving. Hempleman (1967) and Elliott (1969) stress the importance of regular diving or 'work-up dives' to reduce the risk of decompression sickness. In addition, adaptation must be considered during the testing of decompression schedules. If the same divers are used too frequently, the resulting schedules will be unsafe for unadapted divers.

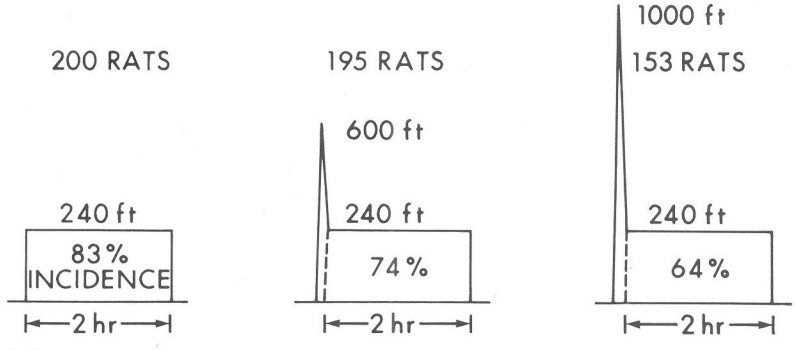

FIG. 14.3. Pressure treatment profiles for rat experiments (Vann *et al.* 1980). As the depth of the pressure treatment increased, the incidence of decompression sickness decreased, suggesting that pressure treatment destroys the gas nuclei which are responsible for bubbles causing decompression sickness

Negative pressure

In the sixteenth century Torricelli discovered that the height to which a suction pump can lift water is limited to about 30 ft (9 m) by cavitation (Derry & Williams 1960). Hayward (1970) demonstrated evidence for gas nuclei when he found that the lift could be increased to 56 ft (17 m) by pressure treating the water at 300 ATA.

The weight of the water column hanging in a suction pump exerts a tension or negative hydrostatic pressure at the top of the column. This negative pressure (P_N) increases the tendency for bubble formation. Supersaturation (equation 1) can be redefined to include P_N as

$$\Delta P = (\Sigma Pi) + P_N - P_B$$

Gent and Lindley (1958) demonstrated that mechanical tension in an elastic solid causes cavitation and that the effects of mechanical and gas tensions are additive. Similar results may occur in muscle and tendon.

Negative pressures are also generated by the relative motion of solids and liquids. Reynold's cavitation occurs when a liquid flowing in a tube encounters a constriction (Harvey 1950). Reynold's cavitation is a consequence of the Bernoulli principle, in which the pressure in a constriction decreases as the liquid velocity increases. The Bernoulli principle is fundamental to the negative pressures which cause cavitation on propellers and in pumps (Pearsall 1972) and may cause negative pressures in the circulatory system.

Tribonucleation, another source of negative pressure and cavitation, is the result of viscous adhesion between solids separating in a liquid (Hayward 1967; Campbell 1968). Tribonucleation has been observed on bearings (Floberg 1964), on a rolling ball (Ikels 1970) and during manipulation of tissue (Harvey *et al.* 1946). Tribonucleation occurs *in vivo* in cracking joints (Unsworth *et al.* 1971) and may be the cause of gas which is detected in joint spaces after diving but which is not associated with decompression sickness (Webb *et al.* 1944; Hanson *et al.* 1974; Bondi *et al.* 1977).

Many physiological processes generate negative pressures, and these would be expected to augment bubble formation. However, negative pressure also may be important in the creation of gas nuclei. This was suggested by the increased number of bubbles seen after decompression of shrimp which were electrically stimulated to produce movement and muscular activity (Evans & Walder 1969). A better understanding of how nuclei are generated might suggest methods to decrease their number and so increase decompression safety.

INERT GAS EXCHANGE

Diving involves the exchange of both dissolved and undissolved gases, and the two processes are quite different. Dissolved gas exchange has been studied principally by circulatory physiologists interested in blood flow measurement, while undissolved gas exchange has been studied by respiratory physiologists interested in the behaviour of gas-filled cavities.

Dissolved gas exchange

In 1897 Zuntz estimated the half-time for whole body nitrogen uptake during diving to be 10 min (Kety 1951). This estimate was based on the assumptions that the inert gas tensions in blood and tissue are in complete diffusion equilibrium and that the cardiac output is evenly distributed to all tissues. Boycott *et al.* (1908) retained the assumption of diffusion equilibrium, but postulated that regional gas exchange rates are unequal because cardiac output is unevenly distributed. These assumptions have been generally supported in subsequent investigations (Jones 1950; Johnson *et al.* 1952; Thompson *et al.* 1958, 1959; Johansson *et al.* 1964; Häggendal *et al.* 1965; Linder 1966; Bassingthwaighte *et al.* 1968; Paradise *et al.* 1971; Sparks & Mohrman 1977; Ohta *et al.* 1978), but there are circumstances in which diffusion can be a significant limiting factor, and these deserve further discussion.

Inert gas dissolved in blood is distributed by the circulation to the capillaries, from where it diffuses into tissue. If tissue offers little resistance to diffusion, gas is absorbed as rapidly as supplied by the circulation, and the tissue and venous tensions are equal. This is known as perfusion or blood-flow limited gas exchange, and the rate of gas absorption is determined entirely by blood flow. Conversely, exchange is diffusion limited when tissue offers substantial diffusion resistance, and the circulation supplies gas faster than it can be absorbed. In this case there are concentration gradients in tissue, and the tissue is not equal to the venous tension.

The magnitude of the tissue gradients depends

upon gas diffusivity, blood flow and diffusion distance.

Rapidly diffusing gases have smaller concentration gradients than have slowly diffusing gases. Diffusivity in tissue appears to be 20–50% of diffusivity in water (Krogh 1919; Suenson *et al.* 1974; Kawashiro *et al.* 1975).

Concentration gradients are more likely to exist at high rather than low blood flow. At low flow the capillary residence time of dissolved gas molecules is long, and diffusion equilibrium between blood and tissue is easily established. At high flow the residence time is short and may be insufficient to permit blood–tissue equilibrium to occur. These effects have been demonstrated for molecules which are diffusion limited at the capillary wall, such as urea and potassium (Landis & Pappenheimer 1963). Since they are not lipid-soluble like the inert gases, these molecules cannot dissolve in the endothelium and must cross the capillary walls at cell junctions.

Gradients within the capillary domain can have both radial and longitudinal orientation. Radial diffusion distances of 7–200 μm and longitudinal distances of 180–1000 μm have been reported (Altman & Dittmer 1971; Middleman 1972; Bassingthwaighte *et al.* 1974; Renkin *et al.* 1981). Calculations have indicated that radial gradients are small (Hill 1928; Kety 1951; Roughton 1952; Blum 1960; Johnson & Wilson 1966; Perl & Chinard 1968; Johnson 1970; Levitt 1970, 1971, 1972; Hennessy 1971, 1974; Bassingthwaighte 1974), but longitudinal gradients may be substantial in tissues with parallel capillaries and concurrent blood flow (Thews 1953; Blum 1960; Bassingthwaighte *et al.* 1970; Levitt 1972; Hennessy 1974). Large gradients are most likely to exist in tissues with diffusion distances of the order of millimetres, such as bone, articular cartilage (Anson 1966), or the eye (Kronheim *et al.* 1976).

Kety and Schmidt (1945) introduced a method for calculating cerebral blood flow based upon measurements of nitrous oxide uptake by the brain. This method, which has been adapted for use with other gases in various tissues, assumes that there are no concentration gradients between blood and tissue and that gas exchange is entirely perfusion limited.

Since blood flow determinations by the Kety–Schmidt method will be inaccurate if concentration gradients are present, many investigations have been conducted to compare calculated with directly measured flows. These were found to be in good agreement for hydrogen, xenon, krypton and argon in kidney and heart at blood flows as high as 500 ml/min per 100 g, where diffusion limitation should be most evident (Aukland *et al.* 1964; Ross *et al.* 1964; Tauchert *et al.* 1972).

The study which has best defined the influence of diffusion, however, made simultaneous measurements of the uptake of helium, nitrous oxide and krypton in canine myocardium (Klocke *et al.* 1972). From these measurements three independent values of blood flow were calculated. Since the gases are dissimilar in diffusivity by a factor of 3 or 4, it would be expected that the calculated flows would differ not only from the measured flow, but also from each other if their exchanges were limited by diffusion. No significant differences were found for any gas in normal heart, but when infarction was produced by ligating one coronary artery, calculated flow exceeded measured flow by 22% for nitrous oxide ($P < 0.05$) and by 28% for krypton ($P < 0.01$). No difference was noted for helium.

These studies indicate that diffusion limitation can be of consequence for the exchange of gases having diffusivities as low as those of nitrous oxide or krypton in tissues with limited vascularity, such as infarcted heart. In normal heart, which is highly vascular, exchange is perfusion limited. Most tissues are of intermediate vascularity, and the principal diving gases, helium and nitrogen, are more diffusible than are nitrous oxide or krypton. Thus, it would appear likely that, for diving, perfusion is the primary factor controlling dissolved gas exchange in the majority of body tissues and that unequal exchange rates are largely the result of regional differences in blood flow. Whether this is true also of the tissues susceptible to decompression sickness is yet to be determined.

While diffusion does not seem to have a substantial influence on gas exchange within the capillary domain, it may be more important between capillary domains or in unevenly perfused tissues. Diffusion shunts have been proposed between opposite ends of capillary domains (Aukland *et al.* 1967; Sejrsen & Tonnesen 1968) and have been observed between arterial and venous vessels (Duling & Berne 1970). Diffusion will also occur between adjacent tissues having unequal exchange rates (Behnke, in Momsen 1942; Perl *et al.* 1960; Hempleman 1963; Perl *et al.* 1965) or within a single tissue that is unevenly perfused (Crone & Garlick 1970). Such intertissue diffusion may be important in diving, where a slowly exchanging fat

deposit could act as an inert gas reservoir for an adjacent rapidly exchanging aqueous tissue which is sensitive to decompression sickness.

Oxygen window

Haldane (1922) pointed out that a bubble in the body is absorbed because its nitrogen partial pressure is greater than the nitrogen tension in the arterial blood. This difference is the driving force for the elimination of undissolved gas and has been called partial pressure vacancy (Momsen 1942), the inherent unsaturation (Hills 1966) or the oxygen window (Behnke 1967, 1975). The simplest term—oxygen window—will be used henceforth. The importance of the oxygen window to diving is illustrated in Fig. 14.4.

Figure 14.4(a) shows the partial pressures of oxygen, nitrogen, water vapour and carbon dioxide in the lungs and tissues of a diver equilibrated with air at 1 ATA. The alveolar nitrogen partial pressure ($P_{A_{N2}}$) is equal to the tissue nitrogen tension ($P_{t_{N2}}$), because the diver is saturated with inert gas. The tissue oxygen tension ($P_{t_{O2}}$) is less than the alveolar partial pressure ($P_{A_{O2}}$) due to metabolic oxygen consumption, and the tissue carbon dioxide tension ($P_{t_{CO2}}$) is greater than the alveolar partial pressure

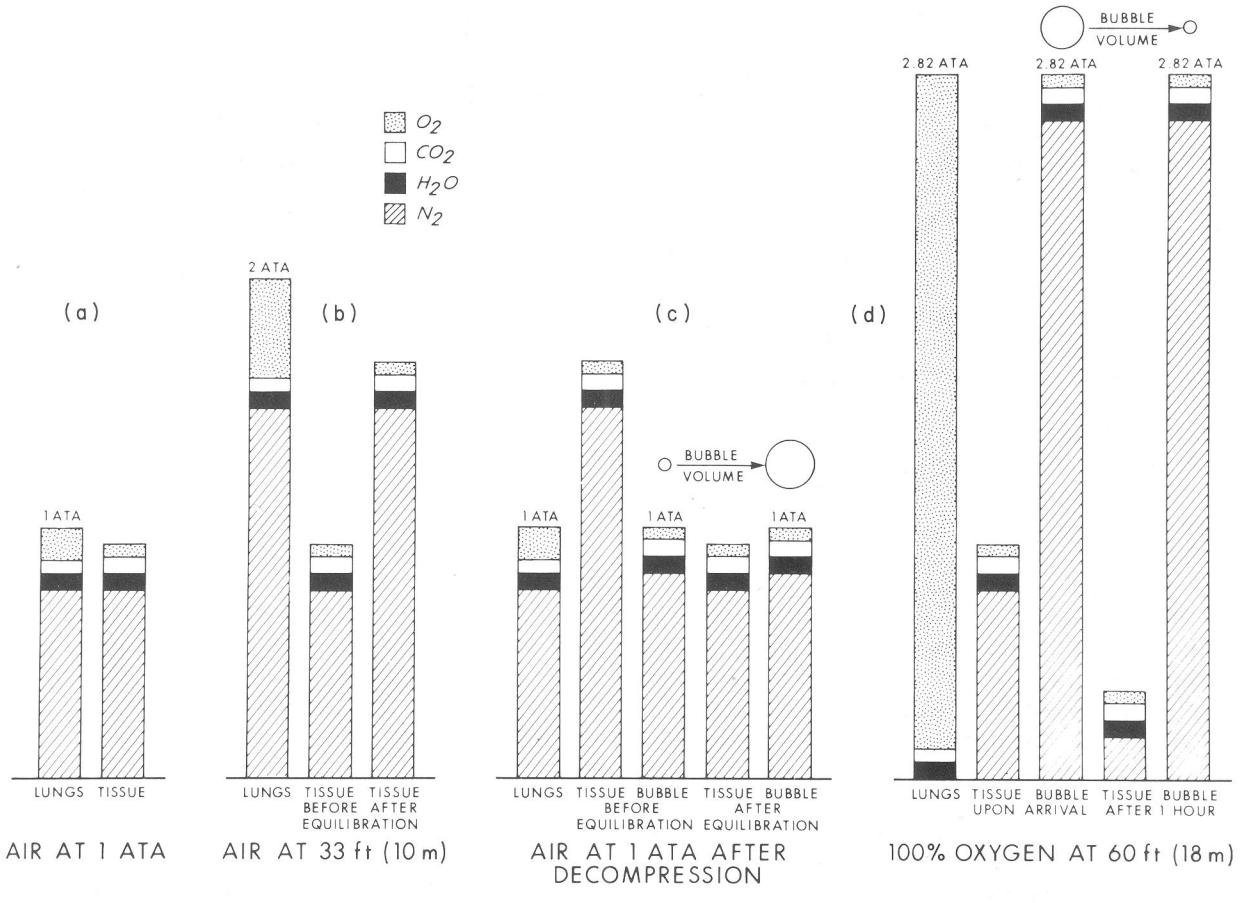

FIG. 14.4. The oxygen window principle as illustrated by lung, tissue and bubble gases. Complete equilibrium is assumed between alveolar gas and arterial blood. Mixed venous values are used for tissue gas tensions. The effects of surface tension and tissue elasticity on bubble pressure are omitted. (a) Air breathing at sea level. The sum of the gas tensions in tissue is less than 1 ATA because of tissue metabolism. (b) Air breathing at 33 ft (10 m). Before equilibration, the tissue nitrogen tension is the same as it was on the surface. After equilibration, it is equal to the nitrogen partial pressure in the lungs. (c) Air at sea level. A gas nucleus has grown into a bubble as a result of decompression. The excess inert gas in tissue diffuses into the bubble or is carried to the lungs by the circulation. The bubble expands when the tissue nitrogen tension exceeds the bubble partial pressure and shrinks when the difference is reversed. (d) Recompression to 60 ft (18 m) breathing 100% oxygen. Oxygen, carbon dioxide and water vapour partial pressures in the bubble are controlled to tissue levels. Nitrogen makes up the remaining pressure in the bubble, and the large gradient between bubble and tissue causes the bubble to dissolve rapidly

($P_{A_{CO_2}}$) due to carbon dioxide production. The decrease in oxygen is greater than the increase in carbon dioxide, because carbon dioxide has a higher solubility and because there are fewer carbon dioxide molecules produced than there are oxygen molecules consumed. As a result, the sum of the tissue gas tensions is less than 1 ATA.

In Figure 14.4(b) the diver breathes air at 33 ft (10 m). In his lungs $P_{A_{CO_2}}$ and the water vapour pressure (P_{H_2O}) are the same as at 1 ATA, but $P_{A_{O_2}}$ and $P_{A_{N_2}}$ have increased to make the total alveolar pressure equal to 2 ATA. In tissue, $P_{t_{O_2}}$ and $P_{t_{CO_2}}$ are unchanged as they are metabolically determined, but $P_{t_{N_2}}$ increases as a result of nitrogen carried from the lungs by the circulation. After about 12 h, the diver is saturated with inert gas, and $P_{A_{N_2}}$ and $P_{t_{N_2}}$ are equal.

In Figure 14.4(c) the diver is decompressed to 1 ATA and $P_{A_{O_2}}$ and $P_{A_{N_2}}$ return to their original values. The tissue is now supersaturated by 2/3 ATS as defined by equation (1), and a gas nucleus having a critical supersaturation of 2/3 ATS or less grows into a bubble.

By Dalton's law the sum of the partial pressures in the bubble equals the barometric pressure. (Surface tension and tissue elasticity, which would increase the bubble pressure, are not considered here.) P_{H_2O} is constant. The oxygen and carbon dioxide partial pressures in the bubble ($P_{b_{O_2}}$ and $P_{b_{CO_2}}$) are metabolically controlled to tissue values. Nitrogen is the only gas whose partial pressure is not fixed, and it makes up the difference between the other gases and the absolute pressure.

Excess nitrogen in tissue diffuses into the bubble or is carried by the circulation to the lungs. The bubble reaches its maximum volume shortly before nitrogen equilibrates between the lungs and tissue. After equilibration $P_{b_{N_2}}$ exceeds $P_{t_{N_2}}$ by the oxygen window, and the bubble is slowly absorbed.

If the diver develops decompression sickness, he is recompressed to 60 ft (18 m) breathing 100% oxygen, as shown in Fig. 14.4(d). The absolute pressure in his lungs and the bubble increases to 2.82 ATA, and the bubble volume is reduced by about 30%, as required by Boyle's law. The partial pressures of all gases in the bubble increase, but metabolism returns $P_{b_{O_2}}$ and $P_{b_{CO_2}}$ to tissue levels within a short time (Van Liew *et al.* 1965). The remainder of the pressure in the bubble must be made up by nitrogen to satisfy Dalton's law. The large nitrogen gradient from bubble to tissue causes the bubble to be rapidly absorbed. This gradient increases as tissue nitrogen is carried to the lungs by the circulation.

Using subcutaneous gas pockets in rats, Van Liew *et al.* (1965) demonstrated that

$$\text{oxygen window} = P_{A_{O_2}} - P_{b_{O_2}} + P_{A_{CO_2}} - P_{b_{CO_2}} \quad (2)$$

where the last three terms are essentially constant at low inspired oxygen partial pressures ($P_{I_{O_2}}$). At high $P_{I_{O_2}}$, however, tissue metabolic requirements are met entirely by dissolved oxygen (the venous haemoglobin is saturated), and $P_{b_{O_2}}$ rises at the same rate as $P_{A_{O_2}}$. When this occurs, the oxygen window reaches its maximum value and is constant despite further increase in $P_{I_{O_2}}$. The maximum value is determined by the arteriovenous oxygen extraction and is low in tissues with low extraction.

Figure 14.5 illustrates Van Liew's predictions of how the oxygen extraction affects the oxygen window as $P_{I_{O_2}}$ increases. Independent calculations of the oxygen window were made by Momsen (1942) and by Hills (1966) and Hills and

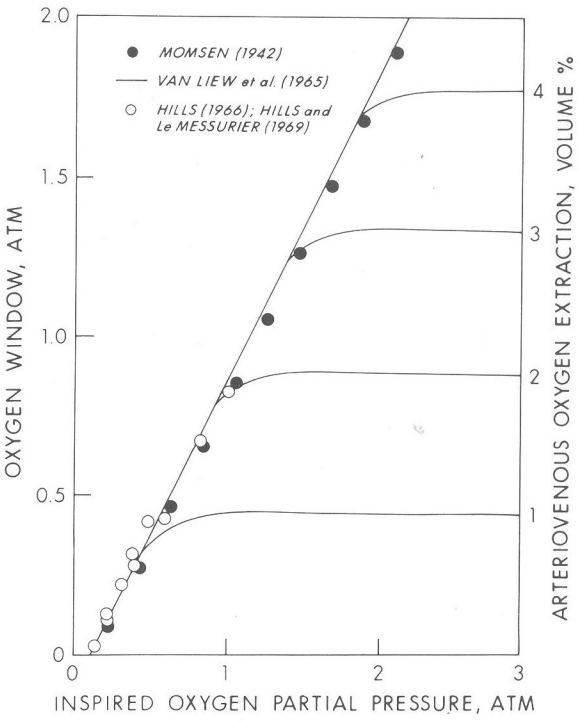

FIG. 14.5. The oxygen window as a function of inspired oxygen partial pressure. The values from Momsen (1942) are predictions, while the values from Hills (1966) and Hills and Le Messuier (1969) are measurements. The oxygen window in tissue does not increase indefinitely but reaches a maximum value, which is determined by the arteriovenous oxygen extraction. (Redrawn from Van Liew *et al.* 1965)

LeMessurier (1969), who also measured it in rabbits. These data are shown in Fig. 14.5. Until reaching its maximum value, the oxygen window is a linear function of P_{IO_2}, as defined by

$$\text{oxygen window} = P_{IO_2} - 92 \text{ mmHg}$$

after substituting

$$P_{AO_2} = P_{IO_2} - P_{H_2O}$$

and venous and arterial values for the last three factors in equation (2).

Undissolved gas exchange

The inert gas partial pressure in a bubble is greater than the inert gas tension in tissue, because of the oxygen window. As a result, a concentration gradient develops around a bubble as outwardly diffusing gas is dissolved in an increasingly large tissue volume and is carried away by the circulation. The gradient around a bubble can be estimated by assuming that the circulation absorbs inert gas at a rate proportional to the local difference between blood and tissue tensions (Van Liew 1968). Figure 14.6 shows gradients computed for oxygen, nitrogen and helium.

When a bubble forms in tissue, the driving force for the elimination of inert gas is reduced from the difference between arterial and tissue tensions to the magnitude of the oxygen window. This reduction causes the rate of gas elimination to fall (Hills 1970, 1978). The rate is further reduced by diffusion resistance which develops around the bubble (Van Liew & Hlastala 1969; Van Liew 1971a; Hlastala & Van Liew 1975). Thus, gas exchange during decompression is a slower process than exchange during time on the bottom. Evidence of reduced exchange efficiency during decompression has been found in humans (Willmon & Behnke 1941; Tobias *et al.* 1949; Kindwall *et al.* 1975), goats (Hempleman 1960), dogs (D'Aoust *et al.* 1976) and guinea-pigs (Hills 1978).

Dissimilar gases have been observed to be eliminated from subcutaneous pockets in rats at unequal rates, but the exit rate of each gas is proportional to its partial pressure and to the oxygen window (Van Liew 1968; Van Liew *et al.* 1968, 1973). Neither pure perfusion limited nor pure diffusion limited exchange adequately explained the elimination rates of helium, hydrogen and nitrogen (Piiper *et al.* 1962). The best description was found to be given by mixed exchange that

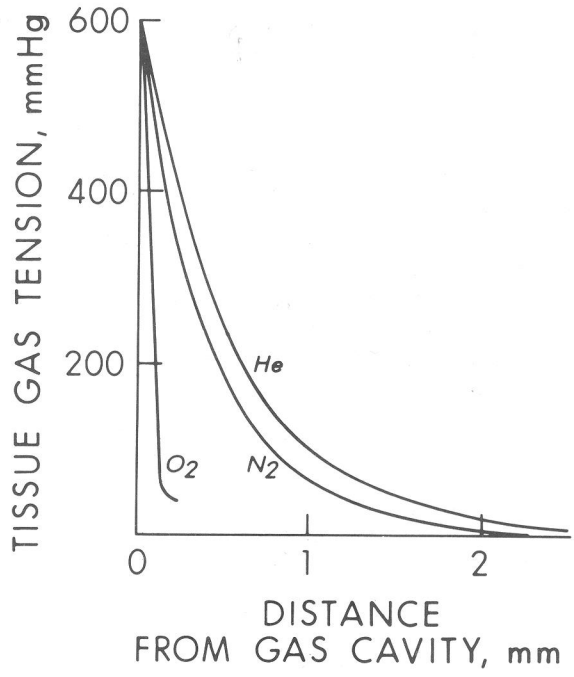

FIG. 14.6. Tissue tension gradients around a gas cavity (redrawn from Van Liew 1968). The oxygen gradient is steepest, because oxygen is removed both by the circulation and by tissue metabolism, whereas nitrogen and helium are removed only by the circulation. The helium gradient extends further than the nitrogen gradient into tissue, because helium diffuses faster than nitrogen

was 43, 51 and 67% diffusion limited for helium, hydrogen and nitrogen. Other experiments determined that sulphur hexafluoride, helium, hydrogen, nitrogen, argon and nitrous oxide were diffusion limited by 22, 61, 74, 91, 94 and 94%, respectively (Tucker & Tenney 1966). The degree of diffusion limitation can vary between experiments, as it depends on conditions in the gas pocket, such as blood flow and diffusion distance, as well as on gas solubility and diffusivity.

Subcutaneous gas cavities also are useful for studying counterdiffusion and multiple inert gas exchange. Sulphur hexafluoride cavities in rats were observed to increase in volume when the animals breathed air. This was ascribed to a faster uptake of nitrogen than elimination of low-solubility sulphur hexafluoride (Tenney *et al.* 1953; Tucker & Tenney 1966; Van Liew & Passke 1967). Van Liew and Passke (1967) postulated that the risk of decompression sickness would be greater if a diver was shifted from a slowly eliminated gas to a rapidly absorbed gas after decompression. Van Liew (1971b) found that the decompression sickness incidence of rats increased if the animals breathed

high-solubility nitrous oxide mixtures after decompression from exposure to elevated air pressure.

AIR DECOMPRESSION

It is a common belief that air decompression schedules are so precisely determined that decompression sickness will result if a schedule is not followed exactly. The error of this view is illustrated below with examples drawn from a number of diving manuals. Procedures for repetitive diving and surface decompression are also discussed.

No-stop dives

A dive which is short enough not to require decompression stops is called a no-stop, no-decompression or minimal decompression dive. Such dives are preferred by many diving manuals over decompression dives because of a lower risk of decompression sickness. However, as illustrated in Fig. 14.7, there is disagreement as to what should be the safe time limit at each depth. This disagreement may be, in part, the result of tests conducted under different exposure and exercise conditions.

Air decompression in the water

Considerable differences are also found in published decompression tables. Figure 14.8 shows seven schedules for a 20 min dive to 200 ft (60 m). These schedules can be divided into three groups which are roughly representative of the tables from which they were taken. (The Haldane schedules become shorter than the other schedules at long bottom times.)

The 1968 RN schedule is the longest, has an intermediate first stop depth and represents the most recent decompression calculation method (Hempleman 1975). The French, USN and RN Table 11 schedules are the shortest and shallowest, and reflect calculation procedures of the 1950s. The schedules from the Russian manual, RN Table 12 (deep air) and Boycott *et al.* (1908) are the deepest and longest as a group and probably represent earlier calculation methods.

Except for extreme exposures, the decompression stops of the early schedules were deep and long by modern standards (Hawkins *et al.* 1935; Van Der Aue *et al.* 1951; des Granges 1957; Crocker 1958; Davis 1962). As experience was gained, however, it was found that they were often deeper and longer than necessary. Accordingly, USN schedules were revised twice (Hawkins *et al.* 1935; Des Granges 1957) and RN schedules at least once to reduce the first stop depth and shorten the total stop time of many dives (Crocker 1958).

Data on the decompression sickness incidence of the USN schedules were compiled by Berghage and

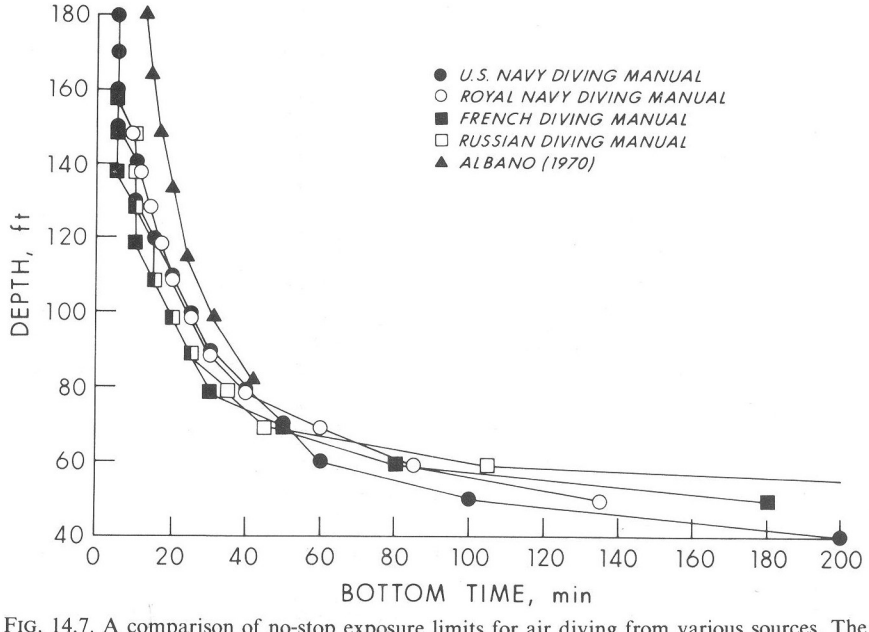

FIG. 14.7. A comparison of no-stop exposure limits for air diving from various sources. The differences may arise from dissimilar test conditions and exercise levels

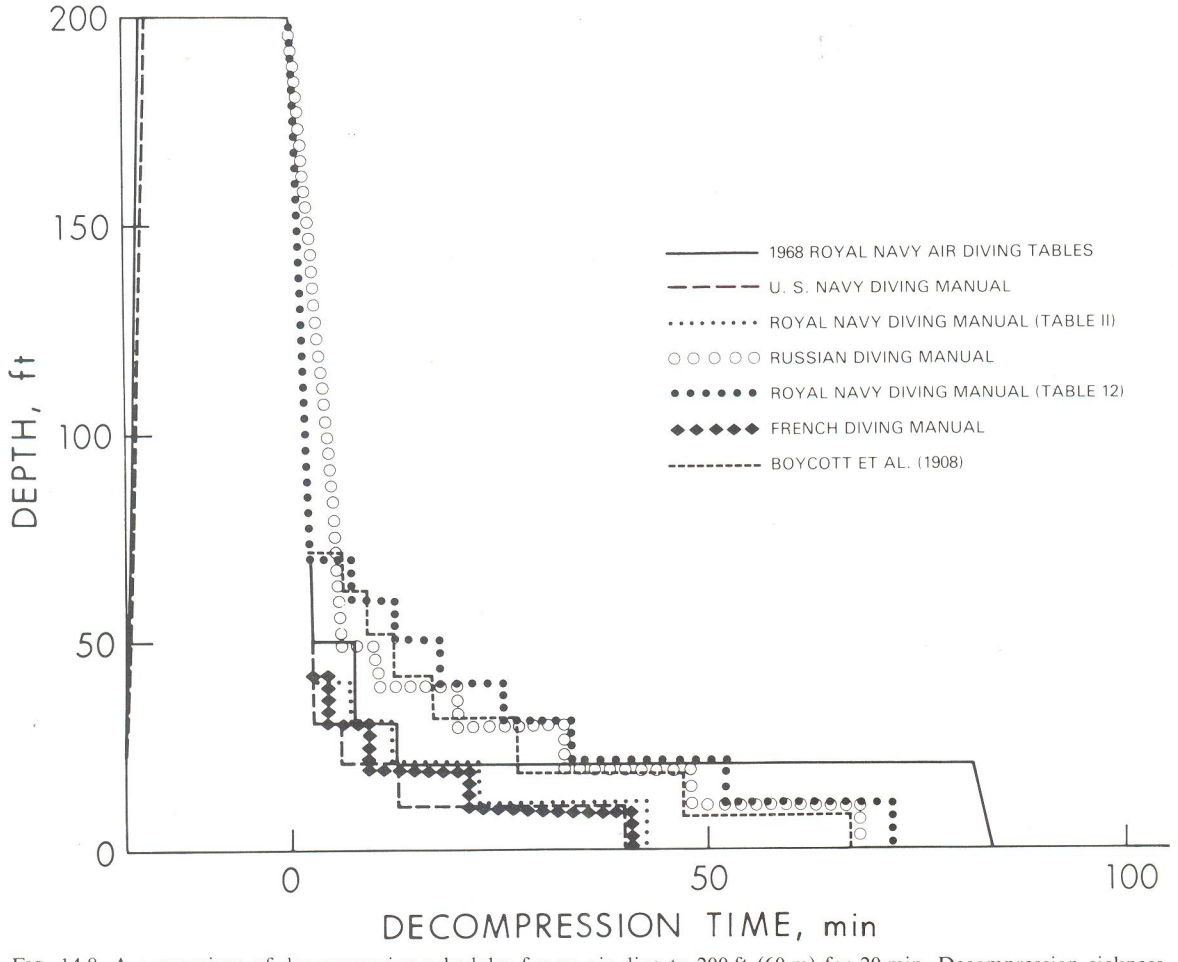

FIG. 14.8. A comparison of decompression schedules for an air dive to 200 ft (60 m) for 20 min. Decompression sickness occurred on the longest of these schedules after a resting dive to 190 ft (57 m). The diver had apparently restricted the circulation to his arm by lying on it. Even the most conservative schedules can be compromised by unfavourable conditions

Durman (1980) from 16 170 dives. In this survey 43 out of the 295 schedules were used more than 100 times but half were not used at all. The overall incidence was 1.25%. For schedules with at least 50 dives the incidence ranged from 0 to 4.8%. Another report reveals that decompression sickness with USN schedules is usually associated with unfavourable conditions such as cold water, rapid ascent, heavy work and repetitive diving (Arntzen & Eidsvik 1980).

Air schedules in the RN manual are more conservative than are USN schedules, yet tests of some of these have produced incidences of over 20% (1968 RN Air Tables). The 1968 RN Air Tables were developed to reduce this incidence of decompression sickness and apparently have been quite successful (Hempleman 1975). However, even these long schedules will fail under adverse conditions. One such failure occurred during decompression from a 20 min resting, chamber dive to 190 ft (57 m) using the 200 ft (60 m) schedule shown in Fig. 14.8. The diver developed decompression sickness in an arm which 'fell asleep' as a result of his lying on it during the long 20 ft (6 m) stop.

It appears likely that many published schedules will work under ideal conditions but all will fail in severe circumstances. It would seem difficult, if not impossible, for a decompression schedule to be both short and safe in all situations.

Repetitive dives

When dives are made in close succession, the decompression requirements of a second dive are increased as a result of residual nitrogen remaining from the first. The residual nitrogen is gradually

eliminated over a surface interval between dives of about 12 h. At the end of this time, the decompression requirements are generally thought to be the same for both dives.

The Russian diving manual does not mention repetitive diving. The RN manual computes the decompression for a second dive from the sum of both bottom times taken at the greater depth. The French manual gives a different table for each surface interval.

The USN manual has the most flexible, but complex, repetitive dive procedure. Each schedule and no-stop exposure is assigned a repetitive dive group which is a measure of residual nitrogen remaining at the end of a dive. Nitrogen elimination during the surface interval moves the diver to a repetitive group having less nitrogen. The new group is converted into a residual nitrogen time at the depth of the next dive and is added to the planned bottom time. This total time is used to select the next decompression schedule.

The USN repetitive dive procedure has been adopted by military, commercial and sport divers world-wide. Arntzen and Eidsvik (1980) have added a similar procedure to the more conservative RN Table 11.

Graver (1977, 1978) believes that the USN repetitive dive methods could be beneficially employed in multilevel diving where time is spent at several depths during a single dive. By current USN rules, the decompression requirements for a multilevel dive are determined from the total bottom time taken at the greatest depth, even if only a few minutes were spent there. By applying the repetitive dive procedure to a multilevel dive, Graver argues that considerable savings in decompression time could be safely achieved.

Oxygen decompression in the water

A reduction in decompression time of about 40% is possible by switching from air to oxygen during shallow decompression stops. Table 13 in the RN manual begins oxygen breathing at 60 ft (18 m). The Russian, 1968 RN and French tables shift to oxygen at 50, 40 and 20 ft, respectively (15, 12 and 6 m). The USN manual has no air decompression table that uses oxygen breathing in the water.

Surface decompression

Diving jobs frequently occur under conditions in which decompression in the water is impractical. Rough seas or strong currents make depth control impossible, and long periods in cold water cause hypothermia and increase decompression requirements.

The expedient solution to these problems has been to remove the diver from the water and to decompress him in a surface chamber where depth and temperature can be properly maintained. Yet this procedure is not without hazard, as the 'double-dive' manoeuvre has been shown to select Type II over Type I decompression sickness in animals (Hills 1971; Griffiths *et al.* 1971; Gait *et al.* 1975). Nevertheless, the benefits appear to outweigh the risks, and the practicality of surface decompression has been firmly established in many years of diving.

The RN, USN and Russian surface decompression tables using air are derived by applying a set of rules to their corresponding air tables. The RN manual has an additional table which uses oxygen in the chamber at 60 ft (18 m) and at shallower stops. Neither RN table uses decompression stops in the water.

Without question, however, the most successful procedure is the USN surface decompression table using oxygen. This table removes the diver from the water after he completes his 30 ft (9 m) stop and recompresses him in a chamber to 40 ft (12 m) breathing 100% oxygen, where he stays for up to an hour before direct return to the surface. The single 40 ft (12 m) oxygen stop is more effective than successive shallower oxygen stops, because the driving force for inert gas elimination (the oxygen window) remains high, as with an oxygen treatment table (Fig. 14.4). Schedules in the surface oxygen table are an average of 20% shorter than schedules in the USN Standard Air Table.

The best testimonial to the success of the USN surface oxygen table is its wide acceptance in commercial diving, where an unsatisfactory procedure is quickly dropped. With few exceptions, it is the only air diving table used in Norway or the North Sea (Arntzen & Eidsvik 1980). It is even used with the USN repetitive dive tables and has been extended from the normal limit of 170 ft (51 m) to 210 ft (63 m) by some diving companies. While these modifications are not endorsed by the US Navy, an analysis by Freitag and Hamilton (1974) suggests that using the repetitive tables with the surface oxygen table is not unreasonable.

The USN surface oxygen table is not entirely free from decompression sickness, however, and one North Sea company reported a 5% incidence in

582 dives. Again, these cases were associated with extreme conditions of cold water, hard work, rapid ascent, daily diving, deeper and longer dives, and surface intervals of over 3 min. To reduce this incidence of decompression sickness, the table was modified, with deeper and longer water stops, longer oxygen breathing periods and a slower ascent to the surface in the chamber. These modifications have resulted in a 0.7% decompression sickness incidence in 700 dives (Arntzen & Eidsvik 1980).

MIXED GAS DECOMPRESSION

While air is the most convenient and commonly used diving gas, artificial mixtures are valuable for reducing the decompression requirements of shallow dives and are essential for deep dives to prevent nitrogen narcosis. Helium–oxygen and multiple inert gas decompression are particularly challenging areas in which there is a great need for new developments.

Equivalent air depth

By increasing the oxygen percentage in air to above 21%, it is possible to achieve a considerable decompression advantage while remaining within the safe oxygen exposure limits. The Equivalent Air Depth theory provides a method by which existing decompression tables can be used to determine the decompression requirements for oxygen-enriched air.

The Equivalent Air Depth (EAD) is the depth of an imaginary air dive that would have the same nitrogen partial pressure as an actual dive on oxygen-enriched air. The EAD is given by

$$\mathrm{EAD} = \frac{(1 - F\mathrm{I}_{O2})(D + 33\ \mathrm{ft})}{0.79} - 33\ \mathrm{ft}$$

where D is the actual depth on a mixture with an inspired oxygen fraction $F\mathrm{I}_{O2}$. If the actual depth is 130 ft (39 m) and $F\mathrm{I}_{O2}$ is 0.32, the EAD will be 107 ft (32 m) and decompression schedules for 110 ft (33 m) should be used.

All dissolved gases, including oxygen, carbon dioxide and water vapour, contribute to the formation and growth of bubbles. The Equivalent Air Depth theory assumes that the partial pressures of these gases do not change with depth or gas mixture. Tissue metabolism normally holds $P\mathrm{t}_{O2}$ and $P\mathrm{t}_{CO2}$ at nearly constant levels. However, as indicated earlier, $P\mathrm{t}_{O2}$ will increase if $P\mathrm{I}_{O2}$ becomes too high. A corrected EAD (EAD′), which is applicable to long dives, may be found from

$$\mathrm{EAD}' = \mathrm{EAD} + \frac{\Delta P\mathrm{t}_{O2}}{0.79}$$

where $\Delta P\mathrm{t}_{O2}$ is the change in $P\mathrm{t}_{O2}$ due to elevated $P\mathrm{I}_{O2}$. $\Delta P\mathrm{t}_{O2}$ can be estimated from Fig. 14.5 as the difference between the predicted and actual oxygen window. For example, if the oxygen extraction is 4 vol.% and $P\mathrm{I}_{O2}$ is 2 ATS, $\Delta P\mathrm{t}_{O2}$ will be 0.17 ATS and EAD′ will be 7.1 ft (2.1 m) deeper than the uncorrected EAD. If the extraction is 3 vol.%, EAD′ will be 24 ft (7.2 m) deeper than EAD. The oxygen extraction is evidently an important determinant of a tissue's response to decompression.

A number of experiments have indicated that a $P\mathrm{I}_{O2}$ of 3–3.5 ATA contributes significantly to the incidence of decompression sickness (Donald 1955; Rashbass & Eaton 1957; Eaton & Hempleman 1973; Berghage & McCracken 1979a,b). In their study with rats Rashbass and Eaton concluded that 25–30% of the oxygen at 3.3 ATA behaves as inert gas. Applying these values to Fig. 14.5, it is estimated that oxygen extraction in tissues critical for decompression sickness in the rat is between 4 and 5 vol.%.

In testing the EAD theory in humans, Logan (1961) was unable to find a statistically significant difference in the incidence of decompression sickness for EAD dives with a $P\mathrm{I}_{O2}$ of up to 2 ATA, as compared with standard air dives. Logan concluded that while oxygen-enriched air might increase the risk of decompression sickness slightly, the risk was not great enough to abandon the EAD procedure.

Nitrogen–oxygen mixtures

The RN and USN diving manuals offer three nitrogen–oxygen mixtures, 40%–60%, 60%–40% and 67.5%–32.5%, for use with semi-closed circuit SCUBA. However, the operating characteristics of this equipment require that the inspired oxygen fraction be independently determined before EAD methods can be applied.

The NOAA manual defines EAD procedures for 68%–32% nitrogen–oxygen in open circuit SCUBA. The no-stop limits for this mixture have been tested safely in a small series of trials (Dunford *et al.* 1979).

The Russian manual gives a decompression table

for a 60%–40% nitrogen–oxygen mixture and a table which uses this mix on the bottom but air during decompression. Comparison of these tables indicates a 20% saving in decompression time when the mix is used throughout the dive. Some commercial diving companies are reported to use oxygen-enriched air with considerable savings in decompression time, but few details are available.

The recent manufacture of closed circuit mixed gas SCUBA has introduced another form of mixture diving—constant oxygen partial pressure. The new equipment has an electronic control system which maintains a constant oxygen partial pressure close to a predetermined set point that is independent of depth. Decompression schedules for this equipment have been tested at oxygen set points of 0.7 and 1.4 ATS. These are discussed by Thalmann *et al.* (1980) and Thalmann (1981), and also in this chapter.

Helium–oxygen mixtures

Helium was first used in the 1920s during diving experiments by the US Navy and the Royal Navy (Momsen 1942). These studies resulted in many cases of decompression sickness, because decompression schedule calculations assumed that helium and nitrogen acted independently in causing bubble formation. The experiments were discontinued, as it appeared that helium might be unsuitable for diving.

Helium experiments were resumed in the 1930s, when End (1937, 1938) demonstrated the practicality of helium–oxygen diving to 400 ft depths, and the US Navy developed their first helium partial pressure table (Momsen 1942). This table began oxygen breathing at 60 ft (18 m), with a final decompression stop at 50 ft (15 m) before surfacing. Decompression could be conducted in the water, in a submersible chamber, or using surface decompression techniques. The revised helium partial pressure table in the present USN diving manual uses oxygen at 40 ft (12 m). This table is reported to have had a decompression sickness incidence of about 2.4% in 366 dives (Hamilton & Kenyon 1976), but it does not have a good reputation in commercial diving.

The USN manual also has helium–oxygen tables for semi-closed circuit SCUBA. These tables include oxygen and repetitive dive procedures, and are substantially shorter than the partial pressure table. During their development, Workman and Reynolds (1965) noted that deeper first stops were required for helium–oxygen than for air decompression schedules.

The early RN helium–oxygen schedules employed a change to air at the first decompression stop, which took place as deep as 200 ft (60 m) in a submersible decompression chamber (Crocker & Hempleman 1957). Oxygen was used at 60 ft (18 m). Later schedules did not use air and reduced the depth of oxygen breathing (Hempleman & Trotter 1963, 1967). Some of these schedules were quite satisfactory for chamber dives with short bottom times, but Hempleman and Trotter were dissatisfied with the results of longer dives and open sea trials.

Helium–oxygen diving is by far most commonplace in the commercial diving industry. However, the decompression schedules used and their effectiveness are proprietary information and are rarely released for study. In general, it appears that during early development in the 1960s, helium and multiple inert gas schedules were short and used a great deal of oxygen (see, e.g., Keller & Bühlmann 1965) but in recent years have become longer and use less oxygen as the emphasis in the industry has shifted from speed to safety.

A widely used commercial procedure is to switch from helium–oxygen to air at depths of 100–200 ft (30–60 m). Such a switch is advantageous because it saves helium, improves communications, keeps the diver warmer and accelerates decompression (Keller 1967). However, the air switch can cause inner ear decompression sickness if made too deep or too rapidly. Momsen (1942) noted that a direct change from helium–oxygen to air should not be made deeper than 165 ft (49.5 m), because of 'adverse' effects, but that a gradual change was permissible. A number of cases of inner ear decompression sickness are reported by Hamilton (1976). The shallowest of these occurred after a rapid air switch at 110 ft (33 m).

The reasons for this decompression sickness are not clear, as investigations of counterdiffusion phenomena (Chapter 15) have indicated that a helium to nitrogen gas switch should be beneficial rather than harmful. However, after a rapid shift to air, nitrogen may enter the blood and tissues of the inner ear faster than helium can be eliminated from the gas space of the middle ear. Because helium diffuses more rapidly than nitrogen, it would enter the inner ear faster than nitrogen could diffuse out, and the inner ear would become supersaturated.

Commercial schedules use both bell and surface

decompression procedures, and seem to be as long as or longer than the USN partial pressure schedules. Dives which are deeper than 400–600 ft (120–180 m) or longer than 1 or 2 h generally employ saturation diving techniques, because schedules for shorter dives are rarely satisfactory.

Perhaps the best document on the state of the art of helium–oxygen decompression is the report of the Undersea Medical Society workshop on the *Development of Decompression Procedures for Depths in Excess of 400 Feet* (Hamilton 1976). This publication discusses the variety of calculation methods that have been used and illustrates the diversity of schedules which they produce for a 30 min dive to 500 ft (150 m). Figure 14.9 reproduces this illustration.

These schedules are difficult to compare, because so many different gas mixtures are used. However, the schedule presented by Mueller and Oser (also discussed by Cabarrou *et al.* 1978) stands out from the others for its fast pressure reduction and short duration. The success of this schedule is surprising in light of the many cases of decompression sickness on other schedules with longer decompression times, such as the series of 173 man-dives reported by Bennett and Vann (also discussed by Bennett *et al.* 1978). Mueller and Oser's DFVLR schedule has several notable features:

1. The initial fast pressure reduction is contrary to the current belief that medium pressure reductions are best.
2. The first decompression stop, a 'recreation phase', is reported to be essential to the success of the schedule but is arbitrary and is not predicted by the modified Haldane decompression model that is used. This recalls the procedure of Momsen (1942), who used an empirical 7 min first stop in the USN helium partial pressure tables to accommodate the 'initial outrush' of helium.
3. The average oxygen partial pressure is about 2 ATA, which is higher than in most other schedules.
4. Oxygen is employed during the last decompression stop at 40 ft (12 m), which is much deeper than in all other schedules. This promotes

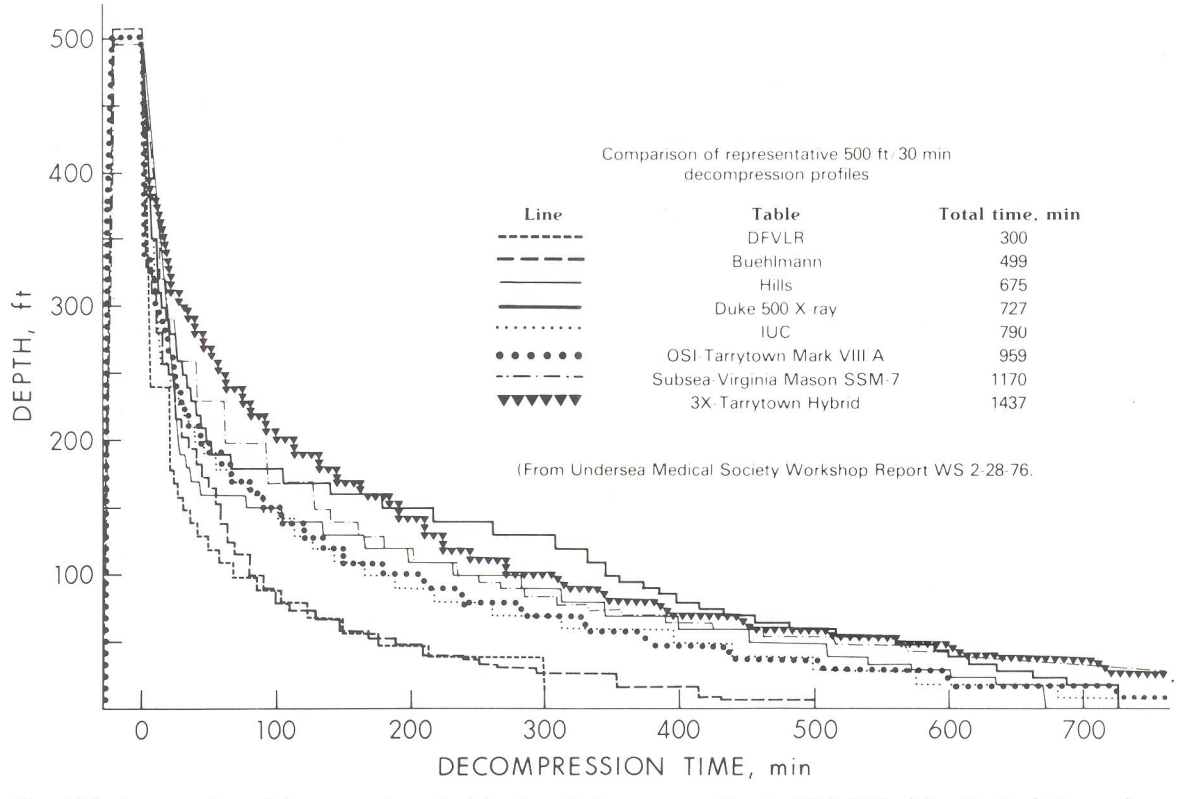

FIG. 14.9. A comparison of decompression schedules for a helium–oxygen dive to 500 ft (150 m) for 30 min (redrawn from Hamilton 1976). The shortest of these schedules appears to be one of the more successful

more efficient gas washout by maintaining a large oxygen window.

DECOMPRESSION MODELLING

While the study of decompression physics and physiology can be properly called a science, the development of decompression schedules is more of an art. All calculation models have their share of questionable assumptions, and even simple models are cumbersome to apply. Physiologically accurate models have many unknown parameters and require numerical solutions which can be painfully slow on even the largest computers. Furthermore, human trials under controlled conditions are mandatory—but potentially hazardous—before experimental schedules can be released for general use. The results of these trials, however, have always been inconclusive, because high cost has prohibited adequate tests to achieve statistical significance. Nevertheless, divers continue to need new decompression schedules or to find old schedules unsatisfactory, and new schedules and calculation methods continue to be devised.

The principles discussed earlier are employed below to formulate a decompression calculation model. Simplifying assumptions are used to keep the mathematics relatively approachable. The calculated decompression schedules were tested under various conditions, and the results of these tests are described.

Critical bubble volume hypothesis

As in the work of Nims (1951), Hills (1966) and Hennessy and Hempleman (1977), the principal assumption is that undissolved gas is the primary cause of decompression sickness. If the undissolved gas exceeds a critical volume in a susceptible tissue, minor symptoms such as joint pain will occur. This is the critical bubble volume hypothesis. No assumption needs be made as to whether the gas is intravascular or extravascular. Furthermore, as proposed by Hempleman (1975), it is postulated that if minor symptoms can be avoided consistently, major symptoms will be rare.

A dive is considered to be constructed of constant-pressure exposures separated by instantaneous pressure changes. A diver absorbs dissolved gas during his time on the bottom. Bubbles form upon ascent to the first decompression stop. These bubbles are absorbed during the stop but re-form on ascent to the next stop. This sequence of events is continued through subsequent stops to the surface. At no time are the bubbles allowed to exceed the critical volume which would cause decompression sickness.

Bubble formation

Figure 14.10 illustrates the process of bubble formation in tissue during ascent. A tissue containing gas nuclei (indicated by dots) but no bubbles is decompressed from an absolute pressure P_{B_1}. Each nucleus has a specific supersaturation at which it grows into a bubble. As the pressure is reduced, the larger nuclei are the first to become bubbles. Upon decompression to P_{B_2}, smaller nuclei are recruited and more bubbles form. If the bubbles are distributed uniformly, the tissue can be described as a collection of identical units, each one of which represents tissue as a whole. Such a unit is designated by the dotted line at pressure P_{B_2} in Fig. 14.10.

Now consider a tissue cell which is decompressed from P_{B_1} to P_{B_2}, as illustrated in Fig. 14.11. What is the lowest pressure that may be achieved without causing decompression sickness? At this safe ascent pressure the bubble reaches its critical volume (V_c). By the critical bubble volume hypothesis, further decompression causes additional expansion, and decompression sickness occurs. Hills (1966) offered the first solution to this problem. Hennessy and Hempleman (1977) refined Hills's solution. The derivations below extend its applicability.

Upon decompression from P_{B_1} to P_{B_2}, gas dissolved in the tissue unit diffuses into the bubble, and it grows until the tissue nitrogen tension is equal to the nitrogen partial pressure in the bubble. Diffusion is assumed to be instantaneous. The number of moles of nitrogen in the bubble (n_b) is defined by the ideal gas law as

$$n_b = \frac{Pt_{N2}\, V_c}{RT} \qquad (3)$$

where V_c is the critical bubble volume, R is the general gas constant, T is temperature, and the nitrogen partial pressure in the bubble (Pt_{N2}) is equal to the tissue nitrogen tension.

The molar quantity of nitrogen dissolved in tissue (n_t) can be found from Henry's law. In terms of the bunsen solubility coefficient of nitrogen in tissue (α_t), which has units of ml gas per ml tissue per

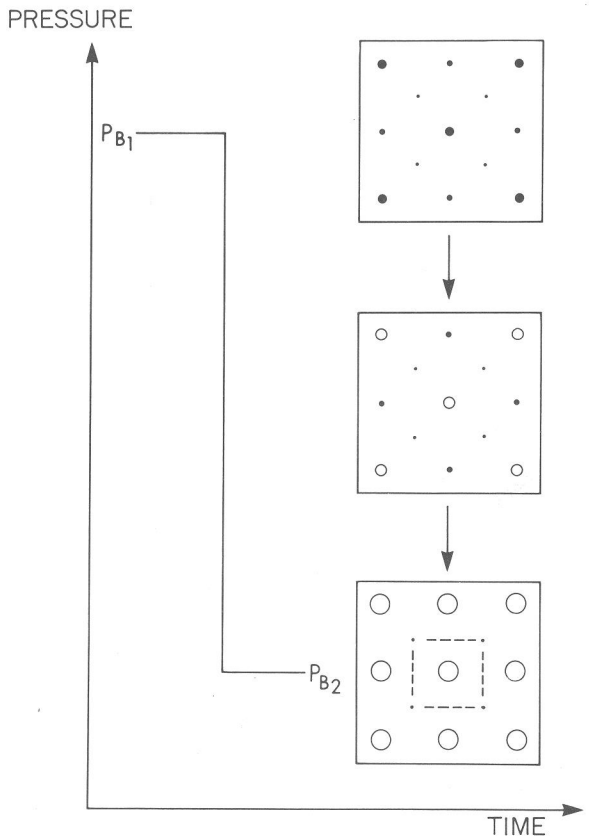

FIG. 14.10. Bubble formation during decompression. Before decompression, the tissue contains gas nuclei (dots) but no bubbles. When decompression begins, the larger more sensitive nuclei grow into bubbles. As decompression progresses, smaller less sensitive nuclei are recruited

ATS, Henry's law gives the volume of dissolved nitrogen in ml at a pressure of 1 ATA as

$$V_{N2} = \alpha_t Pt_{N2} V_t$$

where Pt_{N2} is measured in ATS and V_t is the tissue volume in ml. This gas volume can be converted into moles of dissolved nitrogen (n_t) by using the ideal gas law or

$$n_t = \left(\frac{1\text{ ATA}}{RT}\right)\alpha_t Pt_{N2} V_t \qquad (4)$$

The factor 1 ATA in equation (4) is omitted below, but its implied presence requires that all quantities measuring pressure must have units of ATA.

Since tissue to bubble diffusion is instantaneous, a reduction in pressure redistributes the gas molecules between bubble and tissue but does not alter their total number. A mass balance for inert gas before and after the pressure change is

$$n_{t1} = n_{t2} + n_{b2}$$

where the subscripts 1 and 2 refer to pressures P_{B_1} and P_{B_2}. Substitution of equations (3) and (4) into this leads to

$$(Pt_{N2})_2 = \left(\frac{\alpha_t}{\alpha_t + V_c/V_t}\right)(Pt_{N2})_1 \qquad (5)$$

where $(Pt_{N2})_1$ and $(Pt_{N2})_2$ are the nitrogen tissue tensions at P_{B_1} and P_{B_2}.

The bubble is acted on by an excess pressure (P_e) due to surface tension and tissue elasticity. Although this pressure varies with the bubble volume (Vann & Clark 1975), it is assumed here to be constant. The pressure in the bubble is the sum of the absolute pressure P_{B_2} and the excess pressure P_e but also equals the sum of the partial pressures of gases in the bubble, as required by Dalton's law, or

$$P_{B_2} + P_e = (Pt_{N2})_2 + Pt_{O2} + Pt_{CO2} + P_{H_2O}$$

where Pt_{O2}, Pt_{CO2} and P_{H_2O} are the tissue tensions of oxygen, carbon dioxide and water vapour. Thus,

$$(Pt_{N2})_2 = P_{B_2} + (P_e - P_{tg}) \qquad (6)$$

where

$$P_{tg} = Pt_{O2} + Pt_{CO2} + P_{H_2O}$$

Substituting equation (6) into equation (5), the safe ascent pressure is found to be

$$P_{B_2} = \left(\frac{\alpha_t}{\alpha_t + V_c/V_t}\right)(Pt_{N2})_1 - (P_e - P_{tg}) \qquad (7)$$

Hennessy and Hempleman (1977) applied the critical bubble volume hypothesis to saturation-excursion diving where a diver saturated at P_{B_1} is decompressed to P_{B_2}, as in Fig. 14.11. For a saturated diver, $(Pt_{N2})_1$ is equal to the alveolar nitrogen partial pressure $(P_{A_{N2}})_1$, which is defined with little error (Rahn & Fenn 1955) as

$$(P_{A_{N2}})_1 = P_{B_1} - P_{I_{O2}} - P_{H_2O} \qquad (8)$$

where $P_{I_{O2}}$ is the inspired oxygen partial pressure.

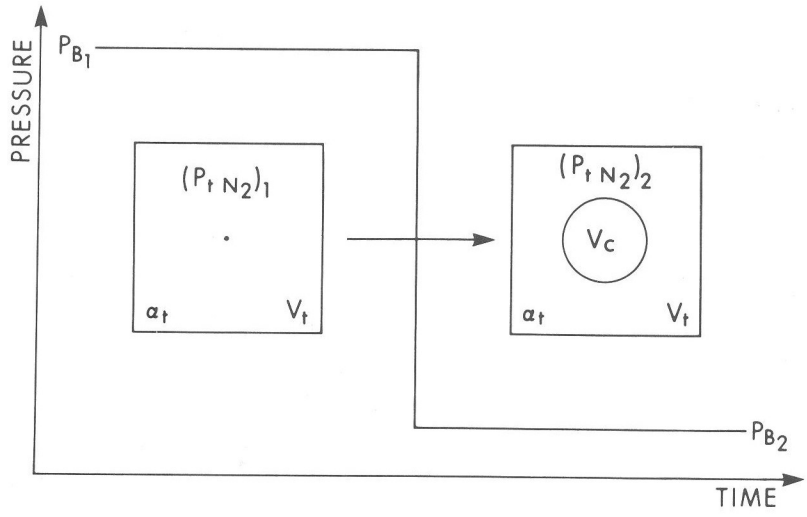

FIG. 14.11. Determination of the safe ascent pressure. A tissue unit containing a gas nucleus is decompressed, and a bubble forms. Gas diffuses into the bubble, and it grows until the nitrogen tissue tension and the bubble partial pressure are equal. The safe ascent pressure is reached when the bubble expands to the critical volume (V_c)

Substituting this for $(Pt_{N2})_1$ and equation (6) for $(Pt_{N2})_2$ in equation (5) gives

$$P_{B_2} = \left(\frac{\alpha_t}{\alpha_t + V_c/V_t}\right)(P_{B_1} - P_{I_{O2}} - P_{H_2O}) - (P_e - P_{tg}) \quad (9)$$

which is a linear relationship between the saturation pressure (P_{B_1}) and the safe ascent pressure (P_{B_2}). Hennessy and Hempleman (1977) found that this relationship was consistent with the helium–oxygen experiments of Barnard (1976) and was in reasonable agreement with nitrogen–oxygen decompression data. Equation (9) will be used to find some of the unknown parameters of the decompression model.

Gas exchange modelling

For the critical bubble volume hypothesis to be useful in decompression schedule calculation, it must include some form of inert gas exchange. The simplest representations of undissolved gas exchange are perfusion limitation (Fig. 14.12a) and diffusion limitation through a barrier (Fig. 14.12b; Van Liew 1967; Hennessy 1978).

These elementary models can account for the reduced driving force which accompanies bubble formation, but neither simulates the diffusion resistance which develops around the bubble. By including a diffusion barrier (Fig. 14.12c), the effects of both perfusion and diffusion are represented, although the number of unknown parameters is increased. Intertissue diffusion through a barrier can be added at the cost of still greater mathematical complexity (Fig. 14.12d). Diffusion, however, does not occur through barriers, as in Fig. 14.12, but is distributed throughout tissue (Van Liew 1967). Meisel *et al.* (1981) used a model incorporating distributed diffusion, but found that numerical integration was necessary even after tissue gradients were removed by assuming steady state conditions.

Selecting a gas exchange model is clearly a compromise between mathematical flexibility and physiological reality. Computational expediency argues for the simplest method unless greater complexity is unavoidable. The approach adopted here is to determine the capabilities and limitations of the simple models before proceeding to the more complex.

Although the elementary perfusion limited and diffusion limited models (Fig. 14.12a,b) are conceptually different, they have the same mathematical solution. This solution will be interpreted to represent perfusion limited exchange, since blood flow is the predominant factor controlling inert gas exchange in most body tissues.

By a mass balance for inert gas in Fig. 14.12(a),

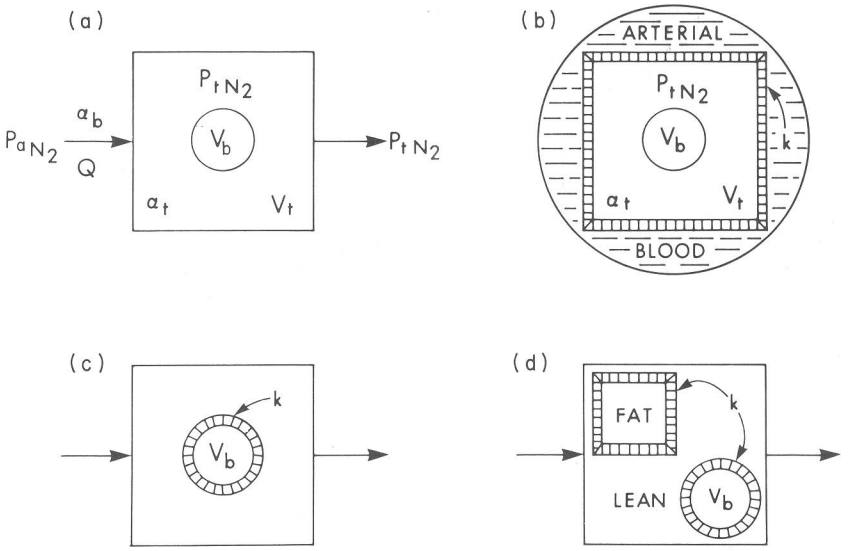

FIG. 14.12. Models of undissolved gas exchange. (a) Gas exchange is entirely perfusion limited. (b) Blood–tissue exchange is diffusion limited, and tissue–bubble exchange is perfusion limited. (c) Blood–tissue exchange is perfusion limited, and tissue–bubble exchange is diffusion limited. (d) the same as (c) but intertissue diffusion occurs between fat and lean components

the rate of change of gas stored in the bubble and tissue is equal to the difference between gas entering with the arterial blood and leaving with the venous blood or

$$\frac{d}{dt}\left(\frac{\alpha_t}{RT}V_t Pt_{N2}+\frac{Pt_{N2}V_b}{RT}\right) = \frac{\alpha_b}{RT}\dot{Q}(Pa_{N2}-Pt_{N2})$$

where t is time, $\dot{Q}$ is blood flow in ml/min, V_b is the bubble volume and Pa_{N2} is the arterial nitrogen tension. The tissue tension is equal to the partial pressure in the bubble, defined, as in equation (6), by

$$Pt_{N2} = P_B+(P_e-P_{tg})$$

Since P_B, P_e and P_{tg} are constant, the time derivative of Pt_{N2} is zero, and the mass balance reduces to

$$\frac{dV_b}{dt} = \alpha_b\dot{Q}\left(\frac{Pa_{N2}}{Pt_{N2}}-1\right)$$

Integrating with respect to time and dividing by V_t gives

$$\frac{V_b}{V_t} = \frac{V_{b0}}{V_t}+\alpha_b\frac{\dot{Q}}{V_t}\left(\frac{Pa_{N2}}{Pt_{N2}}-1\right)t \qquad (10)$$

where V_{b0} is the initial bubble volume. Thalmann (1981) has used a similar method to describe gas exchange during decompression. If $\alpha_b\dot{Q}$ is replaced by $\alpha_t k$, equation (10) defines the bubble volume for the diffusion limited model (Fig. 14.12b), where k is the diffusion barrier parameter.

When no bubble is present, such as during bottom time or during decompression after the bubble has been absorbed, the tissue cell becomes a single compartment of the Haldane decompression model (Boycott *et al.* 1908), and the tissue nitrogen tension varies exponentially with time, as defined by

$$Pt_{N2} = Pa_{N2}+((Pt_{N2})_0-Pa_{N2})e^{-(\alpha_b/\alpha_t)(\dot{Q}/V_t)t} \qquad (11)$$

where $(Pt_{N2})_0$ is the initial tissue tension.

Unknown parameters

The more unknowns a model has, the more difficult it is to use. To reduce the number of unknowns to a manageable level, appropriate values from the literature have been assumed where reasonable. Venous values are used for the tissue tensions:

$$Pt_{O2} = 40 \text{ mmHg}$$
$$Pt_{CO2} = 45 \text{ mmHg}$$
$$P_{H_2O} = 47 \text{ mmHg}$$

The tissue is assumed to have both fat and lean components, but because diffusion is instantaneous, tissue inert gas solubility is defined simply by

$$\alpha_t = \alpha_f f_f + \alpha_b(1 - f_f) \quad (12)$$

where f_f is the fat fraction, α_f is the fat solubility and α_b is the blood solubility. The blood and lean solubilities are taken to be equal. The nitrogen and helium solubilities in blood are as given by Dejours (1966):

$$\alpha_{bN2} = 0.0137 \text{ ml ml}^{-1} \text{ ATS}^{-1}$$
$$\alpha_{bHe} = 0.0087 \text{ ml ml}^{-1} \text{ ATS}^{-1}$$

For fat, the solubilities given by Altman and Dittmer (1971) for olive oil are used,

$$\alpha_{fN2} = 0.067 \text{ ml ml}^{-1} \text{ ATS}^{-1}$$
$$\alpha_{fHe} = 0.0159 \text{ ml ml}^{-1} \text{ ATS}^{-1}$$

Unfortunately, as Weathersby and Homer (1980) point out, accurate knowledge of solubility is poor for many tissues and represents a major weakness in decompression modelling. Thus, the parameter 'fat fraction' may serve as a fitting constant to account for solubility errors.

For a decompression model to be both conceptually and operationally satisfactory, its predictions must be consistent with documented diving experience. A particularly useful class of such data is derived empirically from experiments which are independent of any decompression model. These data are scarce, however, and reflect a wide variation in the susceptibility of divers to decompression sickness. For example, determinations of the no-stop air saturation depth range from 25 ft (7.5 m; 6 h exposure, Behnke & Jones 1974; 12 h exposure, Spencer 1976) to 36 ft (11.3 m; 12 h exposure; Duffner & Snider 1959). Similar determinations of the threshold exposure for 80% helium–20% oxygen vary from 36 ft (11.3 m) to 50 ft (15.7 m) (12 h exposure; Duffner & Snider 1959). Other model-independent data give the safe ascent depths from saturation exposures. Barnard (1976) reports ascents from 220 ft (69 m) to 150 ft (45 m) with helium–oxygen at an inspired oxygen partial pressure of 0.22 ATS, while Spaur *et al.* (1978) report ascents from 1000 ft (300 m) to 820 ft (249 m) at a P_{IO2} of 0.35 ATS.

The critical volume hypothesis, as defined by equation (9), is easily applied to these data to find values for the remaining unknown parameters fat fraction (f_f), excess bubble pressure due to surface tension and tissue elasticity (P_e), and critical bubble volume per unit tissue volume (V_c/V_t). This selection is not unique, however, and is refined further using the model-independent no-stop exposure limits shown in Table 14.1 for air (Van Der Aue *et al.* 1951) and those determined by Duffner for 80% helium–20% oxygen (Workman & Reynolds 1965). In applying the decompression model to these limits, P_e must have different values for nitrogen and helium. Since helium solubility is less than nitrogen, larger supersaturations are possible, more bubbles form and V_t is less for helium (Fig. 14.10).

TABLE 14.1
Air and 80% helium–20% oxygen no-stop exposure limits

Depth, ft (m)	*Exposure time (min)*	
	*Air**	*80% He–20% O_2†*
50 (15)	105	168
60 (18)	70	105
70 (21)	52	69
80 (24)	40	50
90 (27)	32	41
100 (30)	26	35
110 (33)	22	30
120 (36)	18	24
130 (39)	15	19
140 (42)	13	16
150 (45)	11	13
160 (48)	9	11
170 (51)	7	9

* Van Der Aue *et al.* (1951).
† Workman and Reynolds (1965).

By appropriate selection of parameters, the model can be tailored to a diver of any desired susceptibility as defined by model-independent data. A set of parameters which has proved useful in the development of nitrogen–oxygen decompression schedules is

$$f_f = 0.08$$
$$V_c/V_t = 0.0013$$
$$P_e = 0.45 \text{ ATS}$$

These values define a diver who has a no-stop air saturation depth of 27 ft (8.5 m), a no-stop 80% helium–20% oxygen saturation depth of 38 ft (11.9 m) and a safe ascent depth from saturation at 1000 ft (313 m) of 850 ft (266.1 m). The only remaining unknown parameter is the blood flow per unit tissue volume ($\dot{Q}/V_t$), which is discussed in the next section.

Decompression schedule calculation

A major difference between the Haldane, Van Liew, Hennessy or Thalmann models and the perfusion limited, critical bubble volume model is that in the latter case the exchange parameter (blood flow) is not constant but varies with the diver's exercise and thermal states. This hypothesis has practical implications. The wide range of published no-stop exposure limits for air shown in Fig. 14.7 may be partly due to differences in work rate. Higher work rates would produce shorter no-stop limits as a result of increased nitrogen uptake. Similarly, exercise on the bottom during a decompression dive would raise blood flow and increase the depth of the first decompression stop. Hypothermia during decompression, however, would reduce the blood flow and increase the length of the stops.

Blood flow has been estimated empirically for several exercise levels in a series of decompression trials which are described later. These estimates are shown in Table 14.2 and, with the parameters of the previous section, are applied below in a sample decompression schedule calculation. The calculation algorithm is easily adapted for use with a programmable calculator.

Consider a nitrogen–oxygen dive to 150 ft (45 m) using a closed circuit mixed gas SCUBA which maintains an oxygen set point of 1.4 ATS. The diver does 60 min of light work on the bottom, which is equivalent to a blood flow of 1.4 ml/min per 100 g (Table 14.2). The dive profile is illustrated in Fig. 14.13 with the changes that occur in the bubble volume and nitrogen tissue tension.

Nitrogen is absorbed as defined by equation (11), where Pa_{N2} and α_t are given by equations (8) and (12). To allow for error in oxygen set point calibration, P_{IO2} is taken as 1.35 ATS. Figure 14.13 shows that Pt_{N2} increases during the time at 150 ft (45 m).

After 60 min at this depth, the diver ascends to his first decompression stop, and a bubble forms. From equation (7), the safe ascent depth is found to be 29.8 ft (8.9 m), which puts the first decompression stop at 30 ft (9 m) after rounding off. The volume of the bubble which forms is determined by substituting V_b for V_c in equation (7) and solving for V_b/V_t, or

$$\frac{V_b}{V_t} = \alpha_t\left(\frac{Pt_{N2}}{P_{B_2}+(P_e-P_{tg})}-1\right) \tag{13}$$

where Pt_{N2} is the tissue tension just before ascent and P_{B_2} is the absolute pressure at the decompression stop. Figure 14.13 shows that the bubble volume increases from zero prior to decompression to 96% of the critical bubble volume (V_b/V_c) upon arrival at 30 ft (9 m).

The diver rests during decompression, and his tissue perfusion is reduced to 0.9 ml/min per 100 g (Table 14.2). The bubble is completely absorbed after 13 min at the 30 ft (9 m) stop. The absorption time (t_a) can be found by solving equation (10) for time and setting V_b/V_t equal to zero, or

$$t_a = \frac{-V_{b0}/V_t}{\alpha_b(\dot{Q}/V_t)(Pa_{N2}/Pt_{N2}-1)} \tag{14}$$

where V_{b0}/V_t is given by equation (13), Pt_{N2} is given by equation (6), and Pa_{N2} is defined by equation (8), with P_{IO2} equal to 1.35 ATS.

Once the bubble has dissolved, gas exchange is again governed by equation (11). The diver must remain at 30 ft (9 m) until Pt_{N2} is low enough to allow safe ascent to his next stop at 20 ft (6 m). The value of Pt_{N2} at which it is safe to ascend can be found by solving equation (7) for $(Pt_{N2})_1$, or

$$Pt_{N2} = \frac{(P_{B_2}+P_e-P_{tg})(\alpha_t+V_c/V_t)}{\alpha_t} \tag{15}$$

where P_{B_2} is the absolute pressure at 20 ft (6 m). If the tissue tension required for safe ascent is known, equation (11) may be solved for (t_r), the remaining time at 30 ft (9 m), so that

$$t_r = -\frac{\alpha_t V_t}{\alpha_b \dot{Q}} \ln\left[\frac{Pa_{N2}-Pt_{N2}}{Pa_{N2}-(Pt_{N2})_0}\right] \tag{16}$$

where

$$(Pt_{N2})_0 = P_{B_1}+(P_e-P_{tg})$$

TABLE 14.2

Empirically determined values of blood flow ($\dot{Q}/V_t$) for various dive conditions

Conditions	*Oxygen consumption (litres/min)*	*Blood flow ($\dot{Q}/V_t$) (ml/min per 100 g)*
Wet rest Dry rest	0.3–0.6	0.5 (cool)– 1.0 (warm)
Light work	1.0	1.4
Moderate work	2.0	1.6 to 1.7

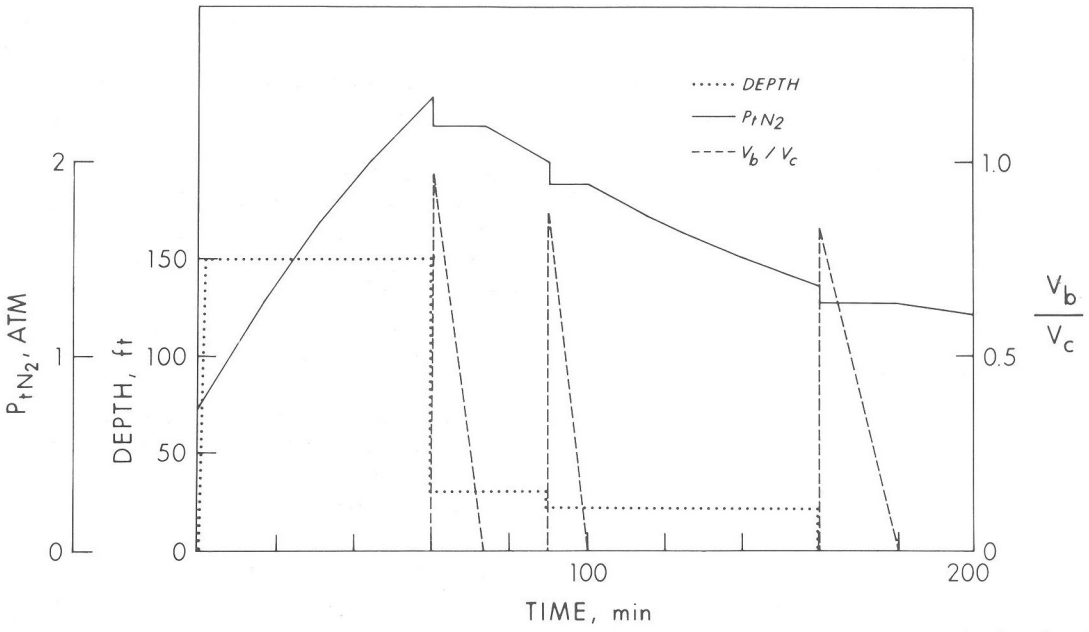

FIG. 14.13. A depth–time profile of a 60 min dive to 150 ft (45 m). The variations of the nitrogen tissue tension (Pt_{N_2}) and the ratio between the bubble volume and the critical bubble volume (V_b/V_c) are shown. During the time at 150 ft (45 m), Pt_{N_2} increases exponentially. Upon decompression to 30 ft (9 m), a bubble having 96% of the critical volume forms and is absorbed linearly during the first part of the decompression stop. While the bubble is present, Pt_{N_2} is constant. After the bubble is absorbed, Pt_{N_2} decreases exponentially. Each decompression stop is of sufficient length so that the bubble forming on ascent to the next stop will not exceed the critical volume. The rate at which the bubble is absorbed depends on blood flow and the inspired oxygen partial pressure

is the tissue tension at 30 ft (9 m) just after the bubble was absorbed. This time is found to be 15 min. Adding t_a and t_r and rounding off to an even 5 min gives a decompression stop time of 30 min at 30 ft (9 m).

Upon arrival at 20 ft (6 m), the oxygen set point is reduced to 1.3 ATS because of equipment limitations giving a PI_{O_2} of 1.25 ATS after allowing for calibration error. The staging time at 20 ft (6 m) before ascent to the surface is determined in the same manner as was the time at 30 ft (9 m). The bubble volume on arrival at 20 ft (6 m) is found to be 87% of the critical volume by using equation (13). Equation (14) gives the bubble absorption time as 11 min and equation (15) the nitrogen tissue tension with which it is safe to surface. From equation (16) the remaining time at 20 ft (6 m) is found to be 57 min, and the total time at this stop is 70 min after rounding off. Upon surfacing, the diver breathes air with lower PI_{O_2}, and the bubble is absorbed more slowly. This schedule has been tested safely 20 times.

DECOMPRESSION TRIALS

The experiments described below tested predicted decompression schedules for 60 min nitrogen–oxygen dives to 100 and 150 ft (30 and 45 m) under various conditions of exercise, oxygen partial pressure and, to a lesser extent, temperature. Dives were conducted in the wet and the dry chambers of the F.G. Hall Laboratory at Duke University with support provided by the US Navy.

The divers were US Navy combat swimmers, and the Mark XV UBA (Underwater Breathing Apparatus) was used. This equipment is a closed circuit mixed gas SCUBA designed to maintain a depth-independent oxygen set point of 0.7 ATS. Modifications allowed the use of other set points and permitted oxygen consumption measurements as an index of diver workload.

The divers exercised at oxygen consumptions of 1, 2 or 3 litres/min by swimming underwater against a trapeze in a wet chamber. These consumptions were subjectively defined as light, moderate and heavy work, and are approximately equal to swimming speeds of 0.6, 1.1 and 1.3 knots (Lanphier 1953). The water temperature was 23 °C and the divers wore wetsuits.

Statistical considerations

Susceptibility to decompression sickness is influenced by many factors and for some purposes may

be considered a statistical phenomenon. This is true when deciding how many trials to give an experimental schedule before approving it for general use. Berghage *et al.* (1974) found that the binomial distribution function adequately described animal experiments, and they suggested that it might also pertain to human diving. Thalmann *et al.* (1980) applied the binomial distribution to their results.

The number of trials a schedule should receive is found from the binomial distribution by specifying the desired decompression sickness incidence and the confidence level of achieving this incidence. For example, if a schedule is desired to have not more than a 5% incidence at the normally accepted level of statistical confidence (95%), the schedule must be tested 60 times without incident. If one incident is permitted, 90 trials are necessary.

These large numbers force the disappointing conclusion that decompression schedules cannot be tested to statistical confidence without substantial financial and manpower commitments. Indeed, sufficient trials have probably never been conducted. Boycott *et al.* (1908) tested each of their schedules twice, which served only as a check against catastrophic inadequacy. Four tests and six tests per schedule were conducted during development of the USN Standard Air Tables and the USN helium–oxygen SCUBA tables (des Granges 1957; Workman & Reynolds 1965). A number of laboratories report using 12 dives per schedule (Hamilton 1976). The greatest number of trials, 20–40 per schedule, were used in a USN Mark XV UBA decompression project (Thalmann *et al.* 1980).

For the Mark XV UBA dives reported here, a goal of at least 20 safe trials was set for each schedule tested, although as many as 30 trials were conducted. Using the binomial distribution, 20 tests gives a 64% confidence level that a schedule will not produce more than a 5% decompression sickness incidence, while 30 tests gives a 79% confidence level. If decompression sickness occurred on a schedule, that schedule was usually not tested again, because more trials are needed to achieve the same confidence level than with no incident.

Exercise during bottom time

Behnke and Willmon (1941) demonstrated that exercise on the surface increases the rate of whole body inert gas uptake. Van Der Aue *et al.* (1951) reported that air schedules which were safe for resting divers produced 20–30% decompression sickness incidences in working divers. Schibli and Bühlmann (1972; also see Bühlmann 1975) found that divers doing light work while breathing helium–oxygen required 20–40% more decompression time than did resting divers.

The first decompression schedule to be calculated for the Mark XV UBA trials was for a dive with 60 min of moderate work at 100 ft (30 m) followed by resting decompression. The effect of exercise was not initially appreciated, however, as illustrated in Fig. 14.14 by the seven cases of decompression sickness which occurred in six schedules. The sixth schedule had a total stop time of 115 min and was tested 20 times. On two occasions the divers did heavy work instead of the planned moderate work, and one case of decompression sickness developed.

The next dive was to 100 ft (30 m) for 60 min with the divers resting in a dry chamber. Analysis of the preceding schedules and of Van Der Aue's no-stop dives (Table 14.1) indicated that the decompression time would be short because gas uptake was low. A 40 min schedule was tested safely in 30 trials (Fig. 14.14).

Most 60 min working dives probably do not exceed light exercise, and moderate or heavy exercise is unusual. Two 100 ft (30 m), 60 min schedules were tested with light exercise (Fig. 14.14). At a decompression time of 80 min, one incident occurred in 5 dives. At 90 min, 29 dives were conducted without incident.

Exposures of 60 min at 150 ft (45 m) were also tested under dry and wet conditions. Of two resting schedules, the first produced one incident in 8 dives with 110 min of decompression. The second schedule, 130 min long, was tested safely 26 times. In the wet chamber, however, with light work on the bottom and resting decompression, 195 min of decompression time was insufficient to prevent decompression sickness. No further dives were tested under these conditions. A wetsuit provided insufficient thermal protection for exposures of this duration, even in relatively warm water.

The safety of a decompression schedule is determined not only by the total stop time, but also by the distribution of this time over depth. Stops too deep or too long are ineffective in eliminating excess inert gas, and may cause additional gas uptake. Stops too shallow or too short promote unsafe bubble growth. The dive profiles tested in the development of a schedule for moderate work

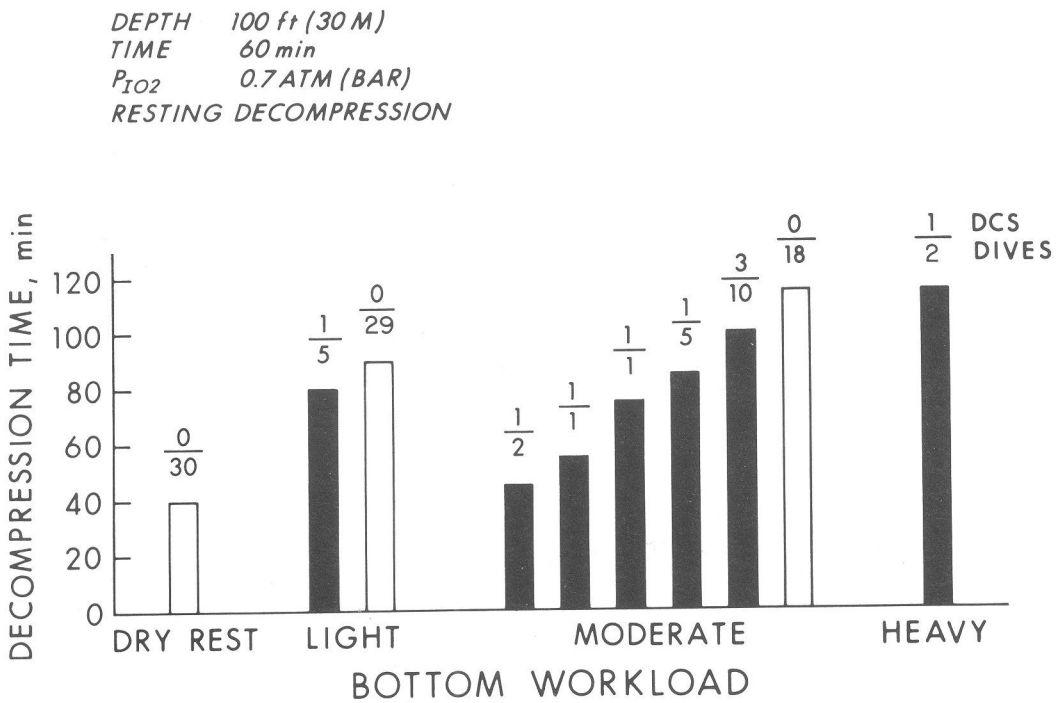

FIG. 14.14. The effect of exercise on the bottom on decompression time for a 60 min dive to 100 ft (30 m). The oxygen partial pressure was 0.7 ATS, and the divers rested during decompression. As the workload on the bottom increased, so did the decompression time

at 100 ft (30 m) are shown in Fig. 14.15. In the first two schedules there were no stops deeper than 30 ft (9 m). After the second schedule failed, it was postulated that deeper stops were needed. Accordingly, arbitrary stops were added at 40 ft (12 m) in the third schedule, and at 50 and 60 ft (15 and 18 m) in the fourth, fifth and sixth schedules. Decompression sickness occurred despite these measures and was not eliminated until the time at 10 and 20 ft (3 and 6 m) was extended. It was concluded that arbitrary decompression stops were unnecessary, and they were not used in subsequent schedules.

The effect of exercise during bottom time on the depth of the first stop and on the distribution of decompression time is shown in Fig. 14.16. The dry, resting schedules were shallowest and shortest. Evaluation of an operational schedule under these conditions is clearly inadequate. Exercise on the bottom increased the first stop depth and the total decompression time. This was probably the result of greater gas uptake due to the higher blood flows which occur with exercise. Estimated blood flows for moderate work, light work, dry rest, and wet resting decompression are given in Table 14.2.

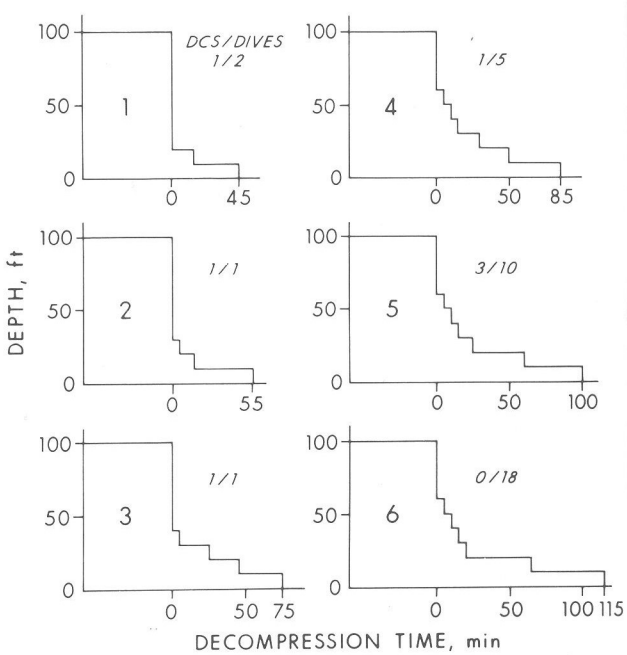

FIG. 14.15. Dive profiles tested for 60 min of moderate work at 100 ft (30 m). The oxygen partial pressure was 0.7 ATS, and the divers rested during decompression. Deep decompression stops were not as effective in preventing decompression sickness as were long, shallow stops

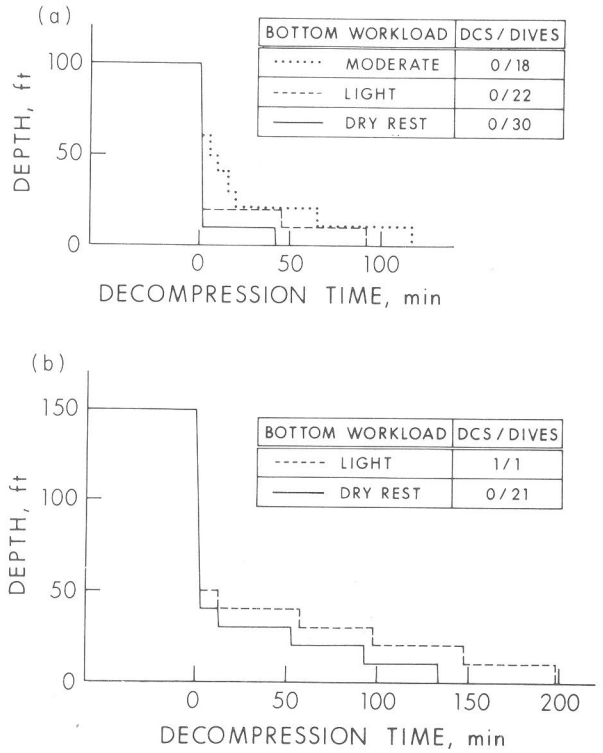

FIG. 14.16. The effect of exercise on the bottom. The oxygen partial pressure was 0.7 ATS, and the divers rested during decompression. (a) A dive to 100 ft (30 m) for 60 min. The depth of the first decompression stop and the total stop time increased as the workload on the bottom increased. (b) A dive to 150 ft (45 m) for 60 min. This dive was conducted safely at rest in a dry chamber, but failed with light exercise on the bottom and resting decompression. Hypothermia prevented further tests under wet conditions

Exercise during decompression

Work during bottom time unequivocally increases the risk of decompression sickness, but the effect of work during decompression is less certain. Theoretically and experimentally, exercise accelerates gas exchange. The possible beneficial effect of exercise was recognized at an early date, and RN divers not only exercised during decompression, but also had their air supply restricted to induce carbon dioxide retention and, consequently, increase circulation (Boycott *et al.* 1908). Many studies have since shown that carbon dioxide retention increases the risk of decompression sickness (e.g. Hodes 1946; Mano & D'Arrigo 1978).

Before World War II, USN divers routinely exercised during their decompression stops (Ellsberg 1927). Altitude decompression experiments during the war, however, showed that exercise increased the incidence and severity and reduced the onset time of decompression sickness (Gray & Masland 1946; Smedal *et al.* 1946). When diving experiments also indicated an increased incidence, Van Der Aue *et al.* (1949) recommended that exercise during or after decompression be abandoned.

These and similar studies are often quoted as evidence for avoiding exercise during decompression. However, closer inspection reveals that the subjects of the studies exercised not during but after decompression. Thus, the proper conclusion is that exercise following decompression is harmful, while the effect of exercise during decompression is unknown.

If exercise during bottom time accelerates the uptake of inert gas by increasing blood flow, it is reasonable to suppose that exercise during decompression will accelerate elimination if the gas stays in solution. In the event of substantial bubble formation, however, the rate of gas elimination will be reduced, because diffusion resistance increases and the driving force decreases. Mechanical factors relating to bubble formation or coalescence also may increase the risk of decompression sickness.

To assess the hypothesis that exercise during decompression can be beneficial, schedules were calculated and tested for 100 and 150 ft (30 and 45 m) dives with light work both during bottom time and during decompression. These schedules are shown in Fig. 14.17 with the corresponding schedules from Fig. 14.16 which used light work on the bottom but resting decompression.

The 100 ft (30 m) resting schedule was 90 min long (no incidents in 29 dives; one incident occurred after an 80 min schedule). The working schedule had a total time of 60 min (no incidents in 26 dives). The 150 ft (45 m) resting schedule was 195 min long and resulted in decompression sickness after its only trial. With work during decompression, however, the same diver safely completed a 155 min schedule. In 28 trials of this schedule, one case of decompression sickness developed in a diver of apparently unusually high susceptibility. This was discovered later when he complained of decompression sickness symptoms during several commercial air flights not associated with recent diving. These symptoms were reproduced during a laboratory exposure at an altitude of 8000 ft (0.75 ATA) and resolved with oxygen breathing and descent to sea level.

Since both resting and working schedules had the same workload on the bottom, the depths of their first decompression stop were also the same.

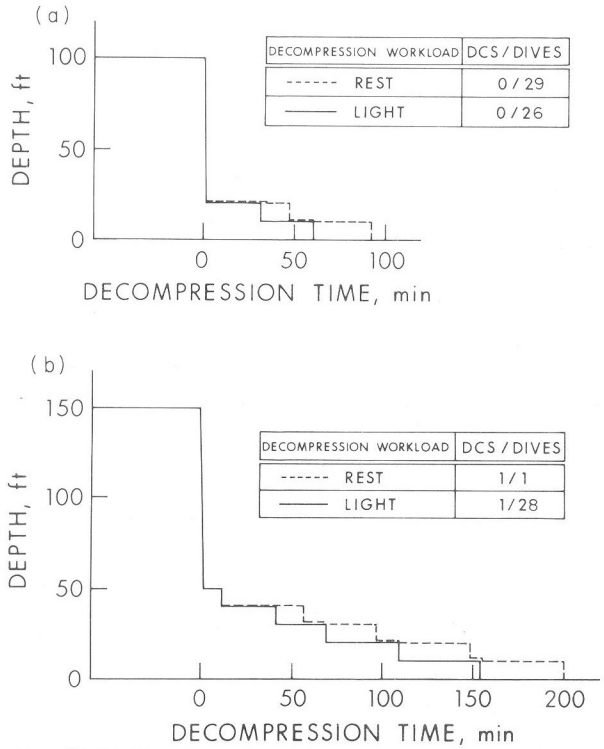

FIG. 14.17. The effect of exercise during decompression. The oxygen partial pressure was 0.7 ATS, and the divers did light work on the bottom. (a) A dive to 100 ft (30 m) for 60 min. Light work during decompression reduced the decompression time. (b) A dive to 150 ft (45 m) for 60 min. This dive was impractical with resting decompression but was possible with working decompression, even with a shorter decompression time

The principal difference between resting and working decompressions was that work shortened the length of the stops. This suggests that bubble growth during decompression was not excessive and that most inert gas remained dissolved, as indicated in Fig. 14.13. Thus, the increase in blood flow which accompanied exercise was effective in accelerating gas elimination and reducing decompression time.

Temperature

As with exercise, temperature appears to exert its influence on decompression through changes in blood flow, although variations in gas solubility may also be important. Cold and heat have opposite effects, depending on the phase of the dive in which they are encountered. These effects are generally interpreted to reflect decreased perfusion in cold environments and increased perfusion in warm environments.

Divers who are cold during bottom time seem to have a lower risk of decompression sickness than have warm divers. After no-stop exposures, cold divers had fewer intravascular bubbles than did warm divers (Dunford & Hayward 1981). In tests of surface decompression schedules where divers were in the water during their bottom time but in a warm chamber during decompression, there was less decompression sickness in cold than in warm water (Van Der Aue *et al.* 1951). Field experience has also shown that divers in wetsuits who are cold during time on the bottom are less likely to get decompression sickness than are divers in hot water suits who are warm (Long 1981).

During decompression, it is better to be warm than cold. Nitrogen elimination and xenon clearance are greater in warm water than in cooler water (Balldin 1973, 1978). In altitude exposures warm subjects eliminated krypton more rapidly and had a lower incidence of decompression sickness than did cool subjects (Griffin *et al.* 1946; Tobias *et al.* 1947, 1949). Oxygen breathing during warm water immersion before decompression to altitude was more effective in preventing decompression sickness than was pre-oxygenation in thermoneutral air (Balldin 1973). Indeed, immersion itself has been shown to increase the rate of nitrogen elimination (Balldin & Lundgren 1972).

Although the effects of temperature were not specifically studied in the Mark XV UBA dives, they could not be avoided. Even with full wetsuits, divers immersed in 21 °C water for 2.5 h had rectal temperature drops of as much as 1 °C and oral temperature drops of 2 °C or more. As a diver cools and his rate of gas elimination decreases, it would be expected that the length of his decompression stops would increase. The beneficial effect of exercise during decompression may be, in part, that it keeps the diver warmer than he would be at rest.

One series of Mark XV UBA experiments was conducted which suggested that warm decompression gives a lower susceptibility to decompression sickness. Schedule 5 in Fig. 14.15 had one DCS incident in five trials. The same schedule was tested safely 12 times, however, when the diver was removed from the water at the 30 ft (9 m) stop and decompressed in a dry chamber.

It is not known to what degree cooling reduces perfusion in tissues susceptible to decompression sickness. However, it was estimated from schedule calculations and trials that the blood flow varies from 1.0 ml/min per 100 g in a warm, resting diver to 0.5 ml/min per 100 g in a cold diver (Table 14.2). A reduction in flow of this magnitude would double the length of a decompression stop.

Oxygen partial pressure

While an oxygen partial pressure of 0.7 ATS is suitable for many purposes, it is not optimal for minimizing decompression time. Higher values reduce inert gas uptake on the bottom and accelerate elimination during decompression.

The employment of oxygen for short exposures is limited, however, by central nervous system toxicity. During the Mark XV UBA trials, an oxygen convulsion occurred at 100 ft (30 m) after 40 min of moderate to heavy work at a P_{IO_2} of 1.6 ATS. The diver later reported, and bench tests confirmed, that the breathing apparatus had excessive respiratory resistance. This can cause carbon dioxide retention and possibly early oxygen toxicity (Lambertsen 1974; Piantadosi *et al.* 1979). Accordingly, the apparatus was modified to reduce resistance, and the divers were instructed not to 'overbreathe' the equipment or work to the point of dyspnoea. The P_{IO_2} was also lowered to 1.4 ATS.

The results of the trials at 1.4 ATS and earlier trials of 0.7 ATS are shown in Fig. 14.18. The divers did light work on the bottom and rested during decompression. The 100 ft (30 m) dive had a decompression time of 90 min at 0.7 ATS (no incidents in 29 tests). One incident occurred in 11 trials of an 80 min schedule. At a P_{IO_2} of 1.4 ATS the time was 20 min (no incidents in 27 tests). The 150 ft (45 m) dive, attempted unsuccessfully at 0.7 ATS with a stop time of 195 min, had a decompression time of 100 min at 1.4 ATS (no incidents in 20 tests). One decompression incident occurred in 11 trials of a 90 min schedule.

The last decompression stop of the 1.4 ATS schedules was at 20 ft (6 m) instead of 10 ft (3 m). With the deeper stop, shorter decompression times were predicted, because of an increased driving force for gas elimination. Upon reaching 20 ft (6 m), the P_{IO_2} was reduced to 1.3 ATS (81% oxygen). The breathing apparatus could not achieve a higher oxygen percentage without exhausting gas into the water. The deeper stop also permits better depth control during open sea diving.

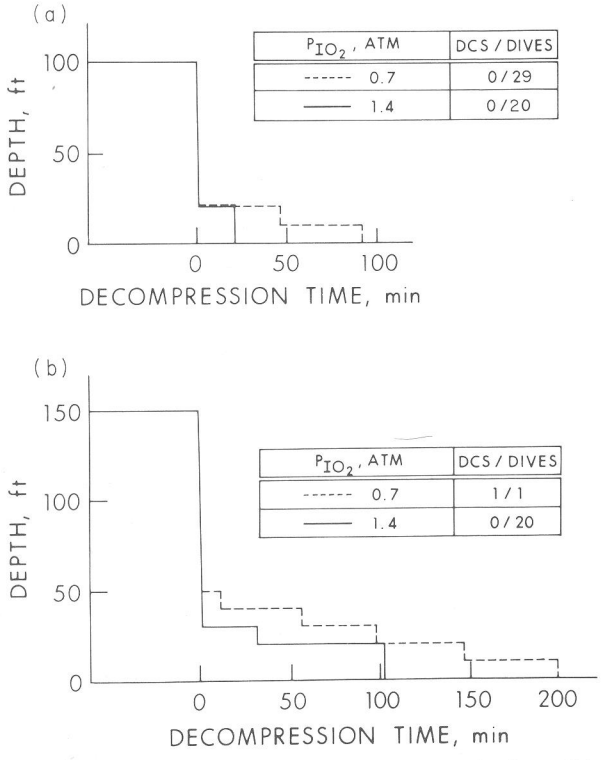

FIG. 14.18. The effects of oxygen partial pressure. The divers did light work on the bottom and rested during decompression. On the elevated oxygen dives, the P_{IO_2} was reduced from 1.4 to 1.3 ATS at 20 ft (6 m) to reduce gas leakage from the breathing apparatus. The last decompression stop was placed at 20 ft (6 m) rather than at 10 ft (3 m), because this increased the driving force for nitrogen elimination and reduced the decompression time. (a) A dive to 100 ft (30 m) for 60 min. (b) A dive to 150 ft (45 m) for 60 min

CONCLUSIONS

As in past human decompression trials, the results of the Mark XV UBA dives discussed above are statistically inadequate, and no firm conclusions can be drawn. However, whether from considerations of decompression physics and physiology, decompression practice, or decompression schedule development, there is a strong indication that the conditions under which a dive is conducted are as important in determining the decompression requirements as are depth, bottom time and gas mix.

The safest approach to the problem of designing decompression schedules is to assume extreme conditions, but such schedules would be unnecessarily deep and long for most dives and might be unacceptable to many divers. An alternative approach would be to design less conservative schedules with modifications for extreme conditions. The best solution is uncertain, and it is probably impossible to completely prevent decompression sickness, but the incidence will certainly be reduced with a better appreciation of factors affecting susceptibility.

REFERENCES

ALBANO, G. (1970). *Principles and Observations on the Physiology of the SCUBA Diver*. English translation. ONR Report Dr-150, Office of Naval Research. Department of the Navy, Arlington, Virginia.

ALTMAN, P. L. & DITTMER, D. S. (1971). *Respiration and Circulation*. Bethesda, Md: Fedn Am. Socs Exp. Biol.

ANSON, B. J. (1966). *Human Anatomy*. New York: McGraw-Hill.

ARNTZEN, A. J. & EIDSVIK, S. (1980). *Modified Air and Nitrox Diving and Treatment Tables*. Norwegian Underwater Institute Report No. 30-80.

AUKLAND, K., AKRE, S. & LERRAND, S. (1967). Arteriovenous counter-current exchange of hydrogen gas in skeletal muscle. *Scand. J. Clin. Lab. Invest.* **19** (Suppl. 99), 72.

AUKLAND, K., BOWER, B. F. & BERLINER, R. W. (1964). Measurement of local blood flow with hydrogen gas. *Circ. Res.* **14**, 164–187.

BALLDIN, U. I. (1973). The preventive effect of denitrogenation during warm water immersion on decompression sickness in man. In *Försvarsmedicin, 1st EUBS Meeting*, Karolinska Institute, Stockholm, June 13–15.

BALLDIN, U. I. (1978). Effect of immersion and ambient temperature on elimination of ^{133}Xenon from human adipose tissue. In *Underwater Physiology VI. Proc. VI Symp. Underwater Physiology*, pp. 329–334. Ed. C. W. Shilling & M. W. Beckett. Bethesda, Md: Fedn Am. Socs Exp. Biol.

BALLDIN, U. I. & LUNDGREN, C. E. G. (1972). Effects of immersion with the head above water on tissue nitrogen elimination in man. *Aerospace Med.* **43**, 1101–1108.

BARNARD, E. E. P. (1976). Fundamental studies in decompression from steady-state exposures. In *Underwater Physiology V*, pp. 263–272. Ed. C. J. Lambertsen. Bethesda, Md: Fedn Am. Socs Exp. Biol.

BASSINGTHWAIGHTE, J. B. (1974). A concurrent flow model for extraction during transcapillary passage. *Circ. Res.* **35**, 483–503.

BASSINGTHWAIGHTE, J. B., KNOPP, T. J. & HAZELRIG, J. B. (1970). Concurrent flow model for capillary-tissue exchanges. In *Capillary Permeability*, pp. 60–80. Ed. C. Crone & N. A. Lassen. Copenhagen: Munksgaard.

BASSINGTHWAIGHTE, J. B., STRANDELL, T. & DONALD, D. E. (1968). Estimation of coronary blood flow by washout of diffusible indication. *Circ. Res.* **23**, 259–278.

BASSINGTHWAIGHTE, J. B., YIPINTSOI, T. & HARVEY, R. B. (1974). Microvasculature of the dog left ventricular myocardium. *Microvasc. Res.* **7**, 229–249.

BATEMAN, J. B. & LANG, J. (1945). Formation and growth of bubbles in aqueous solutions. *Can. J. Res.* **23** (Sec. E), 22–31.

BEHNKE, A. R. (1967). The isobaric (oxygen window) principle of decompression. In *Trans. Third Annual Conference Marine Technology Society*. San Diego.

BEHNKE, A. R. (1975). Early quantitative studies of gas dynamics in decompression. In *The Physiology and Medicine of Diving and Compressed Air Work*, 2nd edn, pp. 392–416. Ed. P. B. Bennett & D. H. Elliott. London: Baillière Tindall.

BEHNKE, A. R. Jr. & JONES, J. P., JR. (1974). Preliminary BART tunnel results. In *Dysbarism-related Osteonecrosis*, pp. 25–40. Ed. E. L. Beckman & D. H. Elliott. NIOSH.

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

BENNETT, P. B., VANN, R. D., ROBY, J. & YOUNGBLOOD, D. (1978). Theory and development of subsaturation decompression procedures for depths in excess of 400 feet. In *Underwater Physiology VI. Proc. VI Symp. Underwater Physiology*, pp. 367–381. Ed. C. W. Shilling & M. W. Beckett. Bethesda, Md: Fedn Am. Socs Exp. Biol.

BERGHAGE, T. E. & DURMAN, D. (1980). *US Navy Air Decompression Schedule Risk Analysis*, p. 22. U.S. Nav. Med. Res. Inst., Rep. NMRI 80-1, Jan.

BERGHAGE, T. E. & MCCRACKEN, T. M. (1979a). Use of oxygen for optimizing decompression. *Undersea Biomed. Res.* **6**, 231–239.

BERGHAGE, T. E. & MCCRACKEN, T. M. (1979b). Equivalent air depth: fact or fiction. *Undersea Biomed. Res.* **6**, 379–384.

BERGHAGE, T. E., WOOLLEY, J. M. & KEATING, L. J. (1974). The probabilistic nature of decompression sickness. *Undersea Biomed. Res.* **1**, 189–196.

BEYER, D. L., D'AOUST, B. G. & SMITH, L. S. (1976). Decompression-induced bubble formation in salmonids: comparison to gas bubble disease. *Undersea Biomed. Res.* **3**, 321–338.

BLUM, J. J. (1960). Concentration profiles in and around capillaries. *Am. J. Physiol.* **198**, 991–998.

BONDI, K. R., MILLER, D. A., KNIGHT, D. R. & HARVEY, C. A. (1977). Joint crepitation after chamber air dives to 188 fsw. *Undersea Biomed. Res.* **4**, 89–94.

BOYCOTT, A. E., DAMANT, G. C. C. & HALDANE, J. S. (1908). The prevention of compressed-air illness. *J. Hygiene* **8**, 342–443.

BÜHLMANN, A. A. (1975). Decompression theory: Swiss practice. In *The Physiology and Medicine of Diving and Compressed Air Work*, 2nd edn, pp. 348–365. Ed. P. B. Bennett & D. H. Elliott. Baltimore: Williams and Wilkins.

CABARROU, P., MUELLER, K. G., FUST, H. D., OSER, H., KREKELER, H. & FINDELDEY, U. (1978). Development and testing of heliox dives in excess of 100 meters. In *Underwater Physiology VI. Proc. VI Symp. Underwater Physiology*. Ed. C. W. Shilling & M. W. Beckett. Bethesda, Md: Fedn Am. Socs Exp. Biol.

CAMPBELL, J. (1968). The tribonucleation of bubbles. *Br. J. Appl. Phys.* (J. Physics D), Ser. 2 **1**, 1085–1088.

CROCKER, W. E. (1958). *Proposals for Applying the New Standard Tables to Surface Decompression and Combined Dives*. RNPL Report 3/58.

CROCKER, W. E. & HEMPLEMAN, H. V. (1957). *The Decompression Problems of Diving to 600 Feet*. RNPL report 3/57.

CRONE, C. & GARLICK, D. (1970). The penetration of inulin, sucrose, mannitol and tritiated water from the interstitial space in muscle into the vascular system. *J. Physiol.* **210**, 387–404.

D'AOUST, B. G., SMITH, K. H. & SWANSON, H. T. (1976). Decompression-induced decrease in nitrogen elimination rate in awake dogs. *J. Appl. Physiol.* **41**, 348–355.

DAVIS, R. H. (1962). *Deep Diving and Submarine Operations*. London: Saint Catherine Press.

DEJOURS, P. (1966). *Respiration*. Oxford, New York: Oxford University Press.

DERRY, T. K. & WILLIAMS, T. I. (1960). *A Short History of Technology*. Oxford University Press.

DES GRANGES, M. (1957). *Standard Air Decompression Table*. NEDU Report 5-75.

DONALD, K. W. (1955). Oxygen bends. *J. Appl. Physiol.* **7**, 639–644.

DUFFNER, G. J. & SNIDER, H. H. (1959). *Effects of Exposing Men to Compressed Air and Helium-Oxygen Mixtures for 12 Hours at Pressures of 2–2.6 Atmospheres*. NEDU Report 1-59.

DULING, B. R. & BERNE, R. M. (1970). Longitudinal gradients in periarteriolar oxygen tension. *Circ. Res.* **27**, 669–678.

DUNFORD, R., COOPER, R., WELLS, M. & D'AOUST, B. G. (1979). Assessment of 32% O_2 in N_2 diving tables by means of ultrasonic Doppler monitoring. *Undersea Biomed. Res.* **6** (Suppl.), 17.

DUNFORD, R. & HAYWARD, J. (1981). Venous gas bubble production following cold stress during a no-decompression dive. *Undersea Biomed. Res.* **8**, 41–49.
EATON, W. J. & HEMPLEMAN, H. V. (1973). *The Role of Oxygen in the Aetiology of Acute Decompression Sickness.* RNPL Report 12-73.
ELLIOTT, D. H. (1969). Some factors in the evaluation of oxy-helium decompression schedules. *Aerospace Med.* **40**, 129–132.
ELLSBERG, E. (1929). *On the Bottom.* New York: Dodd, Mead.
END, E. (1937). Rapid decompression following inhalation of helium-oxygen mixtures under pressure. *Am. J. Physiol.* **120**, 712.
END, E. (1938). The use of new equipment and helium gas in a world record dive. *J. Ind. Hyg. Toxicol.* **20**, 511–520.
EVANS, A. & WALDER, D. N. (1969). Significance of gas micronuclei in the aetiology of decompression sickness. *Nature, Lond.* **222**, 251–252.
FLOBERG, L. (1964). Cavitation in lubricating oil films. In *Cavitation in Real Liquids*, pp. 138–146. Ed. R. Davies. New York: Elsevier.
FREITAG, M. & HAMILTON, R. W. JR. (1974). Comparison of the U.S. Navy air decompression tables by gas-loading analysis. *Undersea Biomed. Res.* **1**, 175–179.
GAIT, D., MILLER, K. W., PATON, W. D. M., SMITH, E. G. & WELCH, B. (1975). The redistribution of vascular bubbles in multiple dives. *Undersea Biomed. Res.* **2**, 42–50.
GENT, A. N. & LINDLEY, P. B. (1958). Internal rupture of bonded rubber cylinders in tension. *Proc. R. Soc., A* **249**, 195–205.
GERTH, W. A. & HEMMINGSEN, E. A. (1976). Gas supersaturation thresholds for spontaneous cavitation in water with gas equilibration pressures up to 570 atm. *Z. Naturforsch.* **31a**, 1711–1716.
GRAVER, D. (1977). A decompression table procedure for multi-level diving. In *Proc. 8th Int. Conf. Underwater Education (NAUI)*, pp. 126–131.
GRAVER, D. (1978). Multi-level decompression table procedure update. In *Proc. 9th Int. Conf. Underwater Education (NAUI)*, pp. 159–164.
GRAY, J. S. & MASLAND, R. L. (1946). Studies on altitude decompression sickness: II. The effects of altitude and of exercise. *J. Aviat. Med.* **17**, 483–493.
GRIFFIN, D. R., ROBINSON, S., BELDING, H. S., DARLING, R. C. & TURRELL, E. S. (1946). The effect of cold and rate of ascent on aero-embolism. *J. Aviat. Med.* **17**, 56–66.
GRIFFITHS, H. B., MILLER, K. W., PATON, W. D. M. & SMITH, E. B. (1971). On the role of separated gas in decompression procedures. *Proc. R. Soc., B* **178**, 389–406.
HÄGGENDAL, E., NILSSON, N. J. & NORBÄCK, B. (1965). On the components of ^{85}Kr clearance curves from the brain of the dog. *Acta Physiol. Scand.* **66** (Suppl. 258), 5–25.
HALDANE, J. S. (1922). *Respiration.* Yale University Press.
HAMILTON, R. W. JR. (Ed.) (1976). *Development of Decompression Procedures for Depths in Excess of 400 Feet*, p. 27. Bethesda, Md: Undersea Medical Society.
HAMILTON, R. W. & KENYON, D. J. (1976). Decompression work at Tarrytown. In *Development of Decompression Procedures for Depths in Excess of 400 Feet*, pp. 91–99. Ed. R. W. Hamilton. Bethesda, Md: Undersea Medical Society Report No. WS: 2-28-76.
HANSON, R. DE G., VOROSMARTI, J. & HARRISON, J. A. B. (1974). Gas-filled joints after oxygen-helium saturation dive. *Br. Med. J.* **3**, 154.
HARVEY, E. N. (1950). Bubble formation in liquids. In *Medical Physics*, Vol. 2, pp. 137–150. Ed. Glasser. Chicago: Year Book Publishers.
HARVEY, E. N., BARNES, D. K., MCELROY, W. D., WHITELEY, A. H., PEASE, D. C. & COOPER, K. W. (1944). Bubble formation in animals. I. Physical factors. *J. Cell. Comp. Physiol.* **24**, 1–22.
HARVEY, E. N., WHITELEY, A. H., COOPER, K. W., PEASE, D. C. & MCELROY, W. D. (1946). The effect of mechanical disturbance on bubble formation in single cells and tissues after saturation with extra high gas pressures. *J. Cell. Comp. Physiol.* **28**, 325–337.
HAWKINS, J. A., SHILLING, C. W. & HANSEN, R. A. (1935). A suggested change in calculating decompression tables for diving. *Navy Med. Bull.* **33**, 327–338.
HAYWARD, A. T. J. (1967). Tribonucleation of bubbles. *Br. J. Appl. Phys.* **18**, 641–644.
HAYWARD, A. T. J. (1970). Mechanical pump with a suction lift of 17 m. *Nature, Lond.* **225**, 376–377.
HEMMINGSEN, E. A. (1978). Effects of surfactants and electrolytes on the nucleation of bubbles in gas-supersaturated solutions. *Z. Naurforsch.* **33a**, 164–171.
HEMPLEMAN, H. V. (1960). *The Unequal Rates of Uptake and Elimination of Tissue Nitrogen Gas in Diving Procedures.* RNPL Report 5/60.
HEMPLEMAN, H. V. (1963). Tissue inert gas exchange and decompression sickness. In *Proc. 2nd Symp. Underwater Physiology*, pp. 6–13. Ed. C. J. Lambertsen & L. J. Greenbaum, Jr. NAS-NRC Publ. 1181, Washington, D.C.
HEMPLEMAN, H. V. (1967). Decompression procedures for deep, open sea operations. In *Proc. 3rd Symp. Underwater Physiology*. Ed. C. J. Lambertsen. Baltimore: Williams and Wilkins.
HEMPLEMAN, H. V. (1975). Decompression theory: British practice. In *The Physiology and Medicine of Diving and Compressed Air Work*, 2nd edn, pp. 331–347. Ed. P. B. Bennett & D. H. Elliott. Baltimore: Williams and Wilkins.
HEMPLEMAN, H. V. & TROTTER, C. (1963). *Deep Diving Trials.* RNPL Report 3/63.
HEMPLEMAN, H. V. & TROTTER, C. (1967). *An Account of Oxy-helium Diving Experiments Carried out at the Royal Naval Physiological Laboratory from January to April 1965 and Subsequent Sea Testing in the Toulon Area during April, May and June 1965.* RNPL Report 2/67.
HENNESSY, T. R. (1971). Inert gas diffusion in heterogeneous tissue. II: with perfusion. *Bull. Math. Biophys.* **33**, 249–257.
HENNESSY, T. R. (1974). The interaction of diffusion and perfusion in homogeneous tissue. *Bull. Math. Biol.* **36**, 505–526.
HENNESSY, T. R. (1978). A mathematical model for predicting decompression sickness from saturation exposures. In *Proc. Symp. Effects of Pressure.* Ed. S. Daniels & H. J. Little. Univ. of Oxford.
HENNESSY, T. R. & HEMPLEMAN, H. V. (1977). An examination of the critical released gas volume concept in decompression sickness. *Proc. R. Soc. B* **197**, 299–313.
HILL, A. V. (1928). The diffusion of oxygen and lactic acid through tissue. *Proc. R. Soc., B* **104**, 39–96.
HILLS, B. A. (1966). *A Thermodynamic and Kinetic Approach to Decompression Sickness.* Ph.D. thesis. Libraries Board of South Australia, University of Adelaide.
HILLS, B. A. (1970). *Vital Issues in Computing Decompression Schedules from Fundamentals. I—Critical Supersaturation Versus Phase Equilibrium.* RNPL Report 4/70.

HILLS, B. A. (1971). Decompression sickness: a fundamental study of 'surface excursion' diving and the selection of limb bends versus CNS symptoms. *Aerospace Med.* **42**, 833–836.
HILLS, B. A. (1978). Effect of decompression per se on nitrogen elimination. *J. Appl. Physiol.* **45**, 916–921.
HILLS, B. A. & LEMESSURIER, D. H. (1969). Unsaturation in living tissue relative to the pressure and composition of inhaled gas and its significance in decompression theory. *Clin. Sci.* **36**, 185–195.
HLASTALA, M. P. & VAN LIEW, H. D. (1975). Absorption of in vitro inert gas bubbles. *Resp. Physiol.* **24**, 147–158.
HODES, R. (1946). The relation between alveolar carbon dioxide tension and susceptibility to decompression sickness. *Am. J. Physiol.* **147**, 603–615.
IKELS, K. G. (1970). Production of gas bubbles in fluids by tribonucleation. *J. Appl. Physiol.* **28**, 524–527.
JOHANSSON, B., LINDER, E. & SEEMAN, T. (1964). Collateral blood flow in the myocardium of dogs measured with krypton-85. *Acta Physiol. Scand.* **62**, 263–170.
JOHNSON, J. A. (1970). A model for capillary exchange with applications. In *Capillary Permeability*, pp. 206–217. Ed. C. Crone & N. Lassen. Copenhagen: Munksgaard.
JOHNSON, J. A., CAVERT, H. M. & LIFSON, N. (1952). Kinetics concerned with distribution of isotopic water in isolated perfused dog heart and skeletal muscle. *Am. J. Physiol.* **171**, 687–693.
JOHNSON, J. A. & WILSON, T. A. (1966). A model for capillary exchange. *Am. J. Physiol.* **210**, 1299–1303.
JONES, H. B. (1950). Respiratory system: nitrogen elimination. In *Medical Physics*, Vol. II, pp. 855–871. Ed. Glasser. Chicago: Year Book Publishers.
KAWASHIRO, T., CAMPOS CARLES, A. C., PERRY, S. F. & PIIPER, J. (1975). Diffusivity of various inert gases in rat skeletal muscle. *Pflügers Arch.* **359**, 219–230.
KELLER, H. (1967). Use of multiple inert gas mixtures in deep diving. In *Proc. 3rd Symp. Underwater Physiology*, pp. 267–274. Ed. C. J. Lambertsen. Baltimore: Williams and Wilkins.
KELLER, H. & BÜHLMANN, A. A. (1965). Deep diving and short decompression by breathing mixed gases. *J. Appl. Physiol.* **20**, 1267–1270.
KETY, S. S. (1951). The theory and applications of the exchange of inert gas at the lungs and tissues. *Pharm. Rev.* **3**, 1–41.
KETY, S. S. & SCHMIDT, C. J. (1945). The determination of cerebral blood flows in man by the use of nitrous oxide in low concentrations. *Am. J. Physiol.* **143**, 53–66.
KINDWALL, E. P., BAZ, A., LIGHTFOOT, E. N., LANPHIER, E. H. & SEIREG, A. (1975). Nitrogen elimination in man during decompression. *Undersea Biomed. Res.* **2**, 285–297.
KLOCKE, F. J., BUNNELL, I. L., WITTENBERG, S. M., GREENE, D. G. & FALSETTI, M. H. (1972). Validation of inert gas measurements of coronary blood flow and contrasting findings in patients with and without coronary artery disease. In *Int. Symp. Myocardial Blood Flow in Man, Methods and Significance in Coronary Disease*, pp. 321–335. Ed. A. Maseri. Turin: Minerva Medica.
KROGH, A. (1919). The rate of diffusion of gases through animal tissues, with some remarks on the coefficient of invasion. *J. Physiol. (Lond.)* **52**, 391–408.
KRONHEIM, S., LAMBERTSEN, C. J., NICHOLS, C. & HENDRICKS, P. L. (1976). Inert gas exchange and bubble formation in the eye. In *Underwater Physiology V*, pp. 327–334. Ed. C. J. Lambertsen. Bethesda, Md: Fedn Am Socs Exp. Biol.
LAMBERTSEN, C. J. (1974). Effects of excessive pressures of oxygen, nitrogen, carbon dioxide, and carbon monoxide: implications in aerospace, undersea, and industrial environments. In *Medical Physiology*, Ch. 66, pp. 1563–1597. Ed. V. B. Mountcastle. St. Louis: Mosby.
LANDIS, E. M. & PAPPENHEIMER, J. R. (1963). Exchange of substances through the capillary walls. In *Handbook of Physiology. Circulation*, Sect. 2, Vol. II, Ch. 29, pp. 961–1034. Washington, D.C.: Am. Physiol. Soc.
LANPHIER, E. H. (1953). *Oxygen Consumption in Underwater Swimming*. NEDU letter report 15-53.
LEVITT, D. G. (1970). Evaluation of the early extraction method of determining capillary permeability by theoretical capillary and organ models. *Circ. Res.* **27**, 81–95.
LEVITT, D. G. (1971). Theoretical model of capillary exchange incorporating interactions between capillaries. *Am. J. Physiol.* **220**, 250–255.
LEVITT, D. G. (1972). Capillary-tissue exchange kinetics: an analysis of the Krogh cylinder model. *J. Theoret. Biol.* **34**, 103–124.
LINDER, E. (1966). Measurements of normal and collateral coronary blood flow by close-arterial and intramyocardial injection of krypton-85 and xenon-133. *Acta Physiol. Scand.* **681** (Suppl. 272), 5–31.
LOGAN, J. A. (1961). *An Evaluation of the Equivalent Air Depth Theory*. NEDU Report 1-61.
LONG, R. W. (1981). Final workshop general discussion. In *Thermal Constraints in Diving*, p. 413. Ed. L. A. Kuehn. Undersea Medical Society Workshop, UMS Publ. 44 WS (TC) 4-1-81.
MANO, Y. & D'ARRIGO, J. (1978). Relationship between CO_2 levels and decompression sickness: implications for disease prevention. *Aviat. Space Environ. Med.* **49**, 349–355.
MEISEL, S., TALMON, Y. & KEREM, D. (1981). Evaluation of decompression tables by a model describing bubble dynamics in tissue. In *Proc. 7th Symp. Underwater Physiology*, pp. 775–784. Ed. A. J. Bachrach & M. M. Matzen. Bethesda, Md: Undersea Medical Society.
MIDDLEMAN, S. (1972). *Transport Phenomena in the Cardiovascular System*. New York: Wiley-Interscience.
MOMSEN, C. B. (1942). *Report on Use of Helium-Oxygen Mixtures for Diving*. NEDU Report 2-42, AD728758.
NIMS, L. F. (1951). Environmental factors affecting decompression sickness. in *Decompression Sickness*, pp. 192–222. Ed. J. F. Fulton. Philadelphia: Saunders.
OHTA, Y., SONG, S. H., GROOM, A. C. & FARHI, L. E. (1978). Is inert gas washout from the tissues limited by diffusion? *J. Appl. Physiol.: Resp. Environ. Exercise Physiol.* **45**, 903–907.
PARADISE, H. F., SWAYZE, C. R., SHIN, D. H. & FOX, I. J. (1971). Perfusion heterogeneity in skeletal muscle using tritiated water. *Am. J. Physiol.* **320**, 1107–1115.
PEARSALL, I. (1972). *Cavitation*. M & B monograph ME/10. London: Butler and Tanner.
PERL, W. & CHINARD, F. P. (1968). A convection-diffusion model of indicator transport through an organ. *Circ. Res.* **22**, 273–298.
PERL, W., LESSER, G. T. & STEELE, J. M. (1960). The kinetics of distribution of the fat-soluble inert gas cyclopropane in the body. *Biophys. J.* **1**, 111–135.
PERL, W., RACKOW, H., SALANITRE, E., WOLF, G. L. & EPSTEIN, R. M. (1965). Intertissue diffusion effect for inert fat-soluble gases. *J. Appl. Physiol.* **20**, 621–627.
PIANTADOSI, C. A., CLINTON, R. L. & THALMANN, E. D. (1979). Prolonged oxygen exposures in immersed exercising divers at 25 fsw (1.76 ATA). *Undersea Biomed. Res.* **6**, 347–356.
PIIPER, J., CANFIELD, R. E. & RAHN, H. (1962). Absorption of various inert gases from subcutaneous gas pockets in rats. *J. Appl. Physiol.* **17**, 268–274.

RAHN, H. & FENN, W. O. (1955). *A Graphical Analysis of the Respiratory Gas Exchange*. Washington, D.C.: Am. Physiol. Soc.
RASHBASS, C. & EATON, W. J. (1957). *The Effect of Oxygen Concentration on the Occurrence of Decompression Sickness*. RNPL Report 10/57.
RENKIN, E. M., GRAY, S. D., DODD, L. R. & LIA, B. D. (1981). Heterogeneity of capillary distribution and capillary circulation in mammalian skeletal muscles. In *Underwater Physiology VII*. Ed. A. J. Bachrach & M. M. Matzen. Bethesda, Md: Undersea Medical Society.
ROSS, R. S., UEDA, K., LICHTLEN, P. R. & REES, R. (1964). Measurements of myocardial blood flow in animals and man by selective injection of radioactive inert gas into the coronary arteries. *Circ. Res.* **15**, 28.
ROUGHTON, F. J. W. (1952). Diffusion and chemical reaction velocity in cylindrical and spherical systems of physiological interest. *Proc. R. Soc., B* **140**, 203–221.
RUBENSTEIN, C. J. (1968). *Role of Decompression Conditioning in the Incidence of Decompression Sickness in Deep Diving*. US Navy Experimental Diving Unit Report 12-68.
SCHIBLI, R. A. & BÜHLMANN, A. A. (1972). The influence of physical work upon decompression time after simulated oxy-helium dives. *Helv. Med. Acta* **36**, 327.
SEJRSEN, P. & TONNESEN, K. H. (1968). Inert gas diffusion method for measurement of blood flow using saturation techniques. *Circ. Res.* **22**, 679–693.
SMEDAL, H. A., BROWN, E. B., JR. & HOFFMAN, C. E. (1946). Incidence of bends pain in a short exposure to simulated altitudes of 26 000, 28 000 and 30 000 feet. *J. Aviat. Med.* **17**, 67–79.
SPARKS, H. V. & MOHRMAN, D. E. (1977). Heterogeneity of flow as an explanation of the multi-exponential washout of inert gas from skeletal muscle. *Microvasc. Res.* **13**, 181–184.
SPAUR, W. H., THALMANN, E. D., FLYNN, E. T., ZUMRICK, J. L., REEDY, T. W. & RINGELBERG, J. M. (1978). Development of unlimited duration excursion tables and procedures for helium-oxygen saturation diving. *Undersea Biomed. Res.* **5**, 159–177.
SPENCER, M. P. (1976). Decompression limits for compressed air determined by ultrasonically detected blood bubbles. *J. Appl. Physiol.* **40**, 227–235.
SUENSON, M., RICHMOND, D. R. & BASSINGTHWAIGHTE, J. B. (1974). Diffusion of sucrose, sodium, and water in ventricular myocardium. *Am. J. Physiol.* **227**, 1116–1123.
TAUCHERT, M., KOCHSIEK, K., HEISS, H. W., STAUER, B. E., KETTLER, D., REPLOH, H. D., RAU, G. & BRETSCHNEIDER, H. F. (1972). Measurement of coronary blood flow in man by the argon method. In *Int. Symp. Myocardial Blood Flow in Man, Methods and Significance in Coronary Disease*, pp. 139–143. Ed. A. Maseri. Turin: Minerva Medica.
TENNY, S. M., CARPENTER, F. G. & RAHN, H. (1953). Gas exchange in a sulfur hexafluoride pneumoperitoneum. *J. Appl. Physiol.* **6**, 201–208.
THALMANN, E. D. (1981). Refinement of 0.7 ATA in nitrogen decompression tables. *Undersea Biomed. Res.* **8** (Suppl.), abstract 2.
THALMANN, E. D., BUCKINGHAM, I. P. & SPAUR, W. H. (1980). *Testing of Decompression Algorithms for Use in the US Navy Underwater Decompression Computer*. Phase I. NEDU Report 11-80.
THEWS, G. (1953). Über die mathematische Behandlung physiologischer Diffusionsprozesse. *Acta Biotheoret.* **10**, 105–138.
THOMPSON, A. M., CAVERT, H. M. & LIFSON, N. (1958). Kinetics of distribution of D_2O and antipyrine in isolated perfused rat liver. *Am. J. Physiol.* **192**, 531–537.
THOMPSON, A. M., CAVERT, H. M., LIFSON, N. & EVANS, R. L. (1959). Regional tissue uptake of D_2O in perfused organs: rat liver, dog heart, and gastrocnemius. *Am. J. Physiol.* **197**, 897–902.
TOBIAS, C. A., JONES, H. B., LAWRENCE, J. H. & HAMILTON, J. G. (1949). The uptake and elimination of krypton and other inert gases by the human body. *J. Clin. Invest.* **28**, 1375–1385.
TOBIAS, C. A., LOOMIS, W. F. & LAWRENCE, J. H. (1947). Studies on skin temperature and circulation in decompression sickness. *Am. J. Physiol.* **149**, 626–633.
TUCKER, R. W. & TENNEY, S. M. (1966). Inert gas exchange in subcutaneous gas pockets of air-breathing animals: theory and measurement. *Respir. Physiol.* **1**, 151–171.
UNSWORTH, A., DOWSON, D. & WRIGHT, V. (1971). Cracking joints. A bioengineering study of cavitation in the metacarpophangeal joint. *Ann. Rheum. Dis.* **30**, 348–358.
VAN DER AUE, O. E., KELLAR, R. J. & BRINTON, E. S. (1949). *The Effect of Exercise During Decompression from Increased Barometric Pressures on the Incidence of Decompression Sickness in Man*. NEDU Report 8-49.
VAN DER AUE, O. E., KELLAR, R. J., BRINTON, E. S., BARRON, G., GILLIAM, H. D. & JONES, R. J. (1951). *Calculation and Testing of Decompression Tables for Air Dives Employing the Procedure of Surface Decompression and the Use of Oxygen*. US Navy Experimental Diving Unit Report 13/51.
VAN LIEW, H. D. (1967). Factors in the resolution of tissue gas bubbles. In *Underwater Physiology III*, pp. 191–204. Ed. C. J. Lambertsen. Baltimore: Williams and Wilkins.
VAN LIEW, H. D. (1968). Coupling of diffusion and perfusion in gas exit from subcutaneous pocket in rats. *Am. J. Physiol.* **214**, 1176–1185.
VAN LIEW, H. D. (1971a). Effect of a change of perfusion on exit of nitrogen and oxygen from gas pockets. *Resp. Physiol.* **12**, 163–168.
VAN LIEW, H. D. (1971b). Dissolved gas washout and bubble absorption in routine decompression. In *Proc. IV Symp. Underwater Physiology*, pp. 145–150. Ed. C. J. Lambertsen. New York: Academic Press.
VAN LIEW, H. D., BISHOP, B., WALDER, D. & RAHN, H. (1965). Effects of compression on composition and absorption of tissue gas pockets. *J. Appl. Physiol.* **20**, 927–933.
VAN LIEW, H. D. & HLASTALA, M. P. (1969). Influence of bubble size and blood perfusion on absorption of gas bubbles in tissue. *Resp. Physiol.* **7**, 111–121.
VAN LIEW, H. D. & PASSKE, M. (1967). Permeation of neon, nitrogen, and sulfur hexafluoride through walls of subcutaneous gas pockets in rats. *Aerospace Med.* **38**, 829–831.
VAN LIEW, H. D., RODNER, R. D. & OLSZOWKA, A. J. (1973). Steady-state composition of tissue gas pockets in rats in hyperbaric environments. *Aerospace Med.* **44**, 499–504.
VAN LIEW, H. D., SCHOENFISCH, W. H. & OLSZOWKA, A. J. (1968). Exchanges of N_2 between a gas pocket and tissue in a hyperbaric environment. *Resp. Physiol.* **6**, 23–28.
VANN, R. D. & CLARK, H. G. (1975). Bubble growth and mechanical properties of tissue in decompression. *Undersea Biomed. Res.* **2**, 185–194.
VANN, R. D., GRIMSTAD, J. & NIELSEN, C. H. (1980). Evidence for gas nuclei in decompressed rats. *Undersea Biomed. Res.* **7**, 107–112.

WALDER, D. N. (1968). Adaptation to decompression sickness in caisson work. In *Proc. 3rd Int. Biometerology Congress, Oxford*, pp. 350–359.
WALDER, D. N. (1969). The prevention of decompression sickness in compressed air workers. In *The Physiology and Medicine of Diving and Compressed Air Work*, 1st edn, pp. 437–450. Ed. P. B. Bennett & D. H. Elliott. London: Baillière, Tindall and Cassell.
WALDER, D. N. (1975). The prevention of decompression sickness. In *The Physiology and Medicine of Diving and Compressed Air Work*, 2nd edn, pp. 456–470. Ed. P. B. Bennett & D. H. Elliott. London: Baillière Tindall.
WEATHERSBY, P. K. & HOMER, L. D. (1980). Solubility of inert gases in biological fluids and tissues: a review. *Undersea Biomed. Res.* **7**, 277–296.
WEBB, J. P., FERRIS, E. B., JR., ENGEL, G. L., ROMANO, J., RYDER, H. W., STEVENS, C. D. & BLANKENHORN, M. A. (1944). *Radiographic Studies of the Knee During Bends*. NRC Comm. on Av. Med., Report No. 305.
WILLMON, T. L. & BEHNKE, A. R. (1941). Nitrogen elimination and oxygen absorption at high barometric pressures. *Am. J. Physiol.* **131**, 633–638.
WORKMAN, R. D. & REYNOLDS, J. L. (1965). *Adaptation of Helium-Oxygen to Mixed Gas Scuba*. NEDU Research Report 1-65.
YOUNT, D. E. (1979). Skins of varying permeability: a stabilization mechanism for gas cavitation nuclei. *J. Acoust. Soc. Am.* **65**, 1429–1439.
YOUNT, D. E. & STRAUSS, R. H. (1976). Bubble formation in gelatin: a model for decompression sickness. *J. Appl. Physics* **47**, 5081–5089.
YOUNT, D. E., YEUNG, C. M. & INGLE, F. W. (1979). Determination of the radii of gas cavitation nuclei by filtering gelatin. *J. Acoust. Soc. Am.* **65**, 1440–1450.

Diving Manuals

US Navy Diving Manual, NAVSEA 0994-LP-001-9010, January 1979.
Royal Navy Diving Manual, Ministry of Defence B.R. 2806, March 1972.
1968 Royal Navy Air Diving Tables, Royal Navy Physiological Laboratory, Alverstoke.
French Diving Manual—Bulletin Official du Ministère du Travail, Fascicule Special No. 74–48 bis, Mesures particulieres de protection applicables aux scaphandriers, 14 Juin 1977.
Russian Diving Manual—Handbook for Divers. Ed. Y. P. Shikanov. Joint Publications Research Service (JPRS) 60691, NTIS Springfield, Va., 4 December 1973.
NOAA Diving Manual, 2nd edn. Ed. J. W. Miller. U.S. Dept. of Commerce, December 1979.