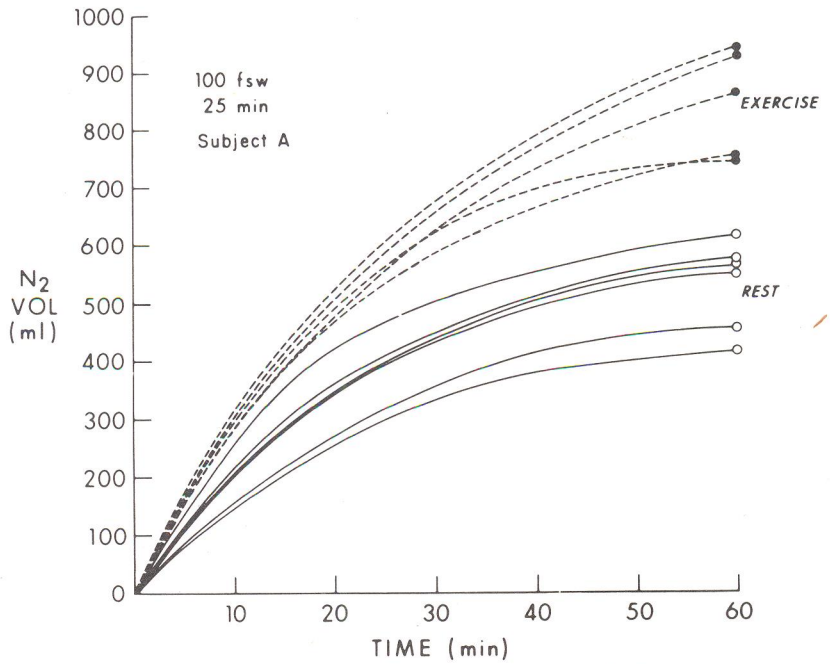

FIG. 14.8. Respiratory nitrogen elimination measured during rest at sea level after resting and exercising dives to 30 m (100 ft) for 25 min. (Reproduced with permission from Dick *et al.* 1984)

No difference in incidence was noted, however, between dry and wet exposures during decompression dives (Thalmann 1985; Weathersby *et al.* 1990). For decompression diving, increased gas elimination during decompression may cancel increased uptake at depth with no net effect on the risk of decompression illness.

The total volume of nitrogen eliminated through the lungs does not appear to be correlated with pain-only decompression illness, as indicated in Fig. 14.9 (Vann & Gerth, in press). Nitrogen elimination was measured during 180 min of oxygen breathing before altitude exposure. The *x*-axis represents nitrogen elimination over the entire 180 min period and during 30 min intervals. The *y* axis is the ratio of nitrogen eliminated by subjects who did not develop decompression illness to those who did. Over the entire period (labelled 0–180), the subjects without decompression illness eliminated only slightly more nitrogen than subjects with decompression illness. When the nitrogen elimination ratio was examined, in 30 min intervals, this was also true for the first 2 h, but subjects free from decompression illness were found to eliminate 70% more nitrogen in the third hour.

The absolute volume of nitrogen eliminated in the third hour was small. Thus, the tissues responsible for mild decompression symptoms at altitude would appear to exchange nitrogen slowly and to have a low nitrogen storage capacity. The large increase in nitrogen uptake produced by exercise at depth (Fig. 14.8) probably reflected rapid uptake by muscle with which mild decompression symptoms are not normally associated. Large capacity tissues may contribute their nitrogen to venous gas emboli.

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 bubbles and has been called the *partial pressure vacancy* (Momsen 1942), the *inherent unsaturation* (Hills 1966) and the *oxygen window* (Behnke 1967, 1975). The oxygen window is a direct consequence of the metabolic conversion of oxygen into carbon dioxide.

Metabolism converts a relatively insoluble gas, oxygen, into carbon dioxide, which is some 21 times more soluble. Figure 14.10 shows the effect on dissolved gas tension of exchanging oxygen for

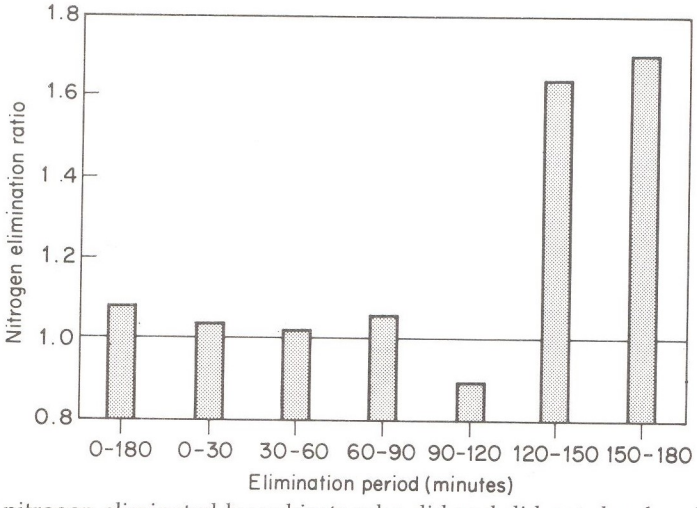

FIG. 14.9. The ratio of nitrogen eliminated by subjects who did and did not develop decompression illness. Nitrogen elimination was measured during oxygen breathing at sea level before altitude exposure at 9144 m (30 000 ft). Elimination ratios are shown for total nitrogen elimination over 180 min and during 30 min intervals. The subjects who did not develop decompression illness at altitude eliminated more nitrogen during the third hour (Vann & Gerth, in press)

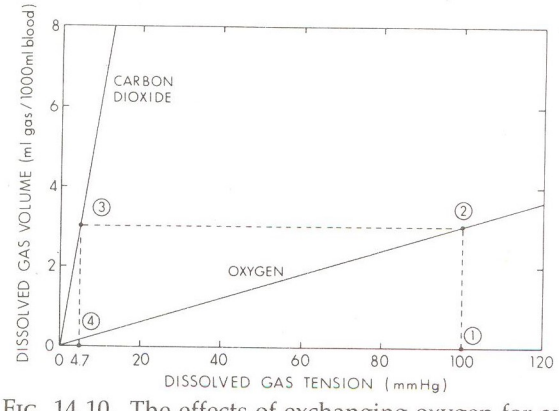

FIG. 14.10. The effects of exchanging oxygen for carbon dioxide on dissolved gas tension. When oxygen is converted into carbon dioxide, the gas tension falls from 100 to 4.7 torr (13.2 to 0.6 kPa), but the dissolved gas volume or content remains unchanged because carbon dioxide is more soluble than oxygen. (Reproduced with permission from Vann 1989c)

carbon dioxide. The steeper line is the relationship between carbon dioxide tension and carbon dioxide content. Its slope is the effective carbon dioxide solubility. The other line represents the same relationship for oxygen with a gradual slope reflecting a lower oxygen solubility. An oxygen tension of 100 torr (13 kPa; point 1) corresponds to a content of 3 ml O_2 per 1000 ml blood (point 2). If each oxygen molecule were exchanged for a carbon dioxide molecule (point 3), there would be no change in the dissolved gas volume, but the tension would fall to 4.7 torr (0.6 kPa; point 4). This is because carbon dioxide is more soluble than oxygen.

The metabolic exchange of oxygen for carbon dioxide in tissue is illustrated in Table 14.2 for a diver equilibrated with air at sea level. The sum of the alveolar partial pressures is 760 torr (101 kPa), while the total arterial gas tension is slightly less because of ventilation–perfusion inequalities (West 1971). The alveolar, arterial and venous nitrogen tensions are equal since the diver is equilibrated with air. The arterial oxygen tension falls from 95 torr (13 kPa) to a venous level of 40 torr

TABLE 14.2
Representative alveolar, arterial and venous gas tensions (mmHg)

	Alveolar	*Arterial*	*Venous*
Carbon dioxide	40	40	45
Oxygen	104	95	40
Water vapour	46	46	46
Nitrogen	570	570	570
	760	751	701

Oxygen window = 760 − 701 = 59 mmHg.

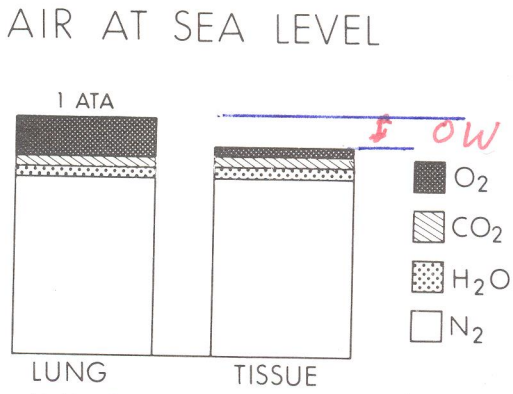

FIG. 14.11. Gases in the lungs and tissues of an air breathing diver at sea level. The metabolic exchange of oxygen for carbon dioxide results in a total tissue gas tension which is less than the ambient pressure. This difference is the oxygen window. (Reproduced with permission from Vann 1989c)

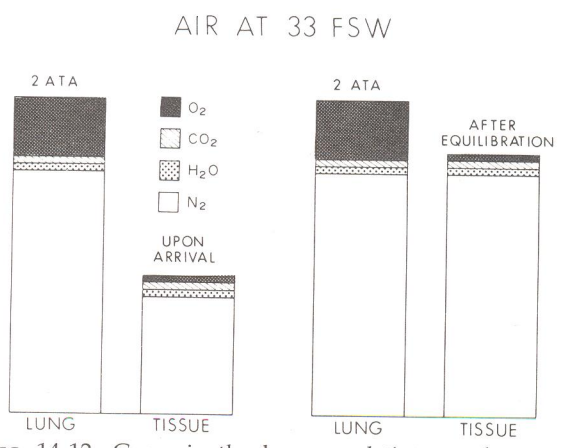

FIG. 14.12. Gases in the lungs and tissues of an air breathing diver at 10 m (33 ft). Initially, the tissue nitrogen tension is the same as in Fig. 14.11, but after sufficient time at depth, tissue nitrogen equilibrates with the 2 ata (202 kPa) of air in the lung. (Reproduced with permission from Vann 1989c)

(5 kPa), while the arterial carbon dioxide tension rises from 40 torr (5 kPa) to a venous level of 45 torr (6 kPa). The total gas tension in venous blood is 701 torr (93 kPa), 59 torr (8 kPa) less than the absolute pressure. This difference in gas tension is the 'oxygen window'. The magnitude of the oxygen window increases linearly with the inspired oxygen partial pressure (Momsen 1942; Van Liew *et al.* 1965; Hills 1966; Hills & LeMessurier 1969).

The gas tensions in Table 14.2 are shown as bar graphs in Fig. 14.11. The bar on the left represents gases in a diver's lungs at sea level. Dalton's Law of partial pressures requires that the sum of these gases be 1 ata (101 kPa). The bar on the right is the gases in the diver's tissues whose sum is less than 1 ata (101 kPa) because of the O_2 for CO_2 exchange.

In Fig. 14.12, the diver breathes air at 10 m (33 ft). The bars on the left show gases in his lungs and tissues upon arrival at depth. The oxygen and nitrogen partial pressures in his lungs have increased to make the sum of all gases equal to the absolute pressure of 2 ata (202 kPa), but his tissues have absorbed no additional nitrogen. The bars on the right show the lungs and tissues after nitrogen equilibration. The tissue nitrogen tension now equals the alveolar nitrogen partial pressure.

Suppose bubbles form in the diver's tissues after he returns to sea level (Fig 14.13). By Dalton's Law, the sum of the partial pressures in the bubble is 1 ata (101 kPa). (Surface tension and tissue elasticity would increase this pressure but are omitted here.) The water vapour pressure is constant and

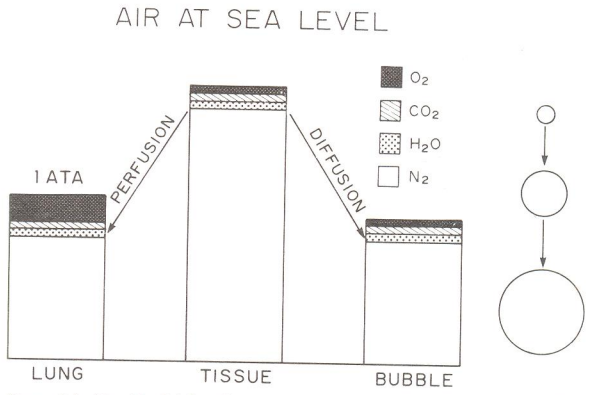

FIG. 14.13. Bubble formation and growth after decompression from 10 m (33 ft) to sea level. The bubble grows by inward diffusion of supersaturated nitrogen from tissue. Dissolved nitrogen is also carried to the lungs by the circulation. (Reproduced with permission from Vann 1989c)

the oxygen and carbon dioxide partial pressures are metabolically controlled to their tissue levels (Van Liew *et al.* 1965). Since the nitrogen tension in tissue is elevated, nitrogen diffuses both into the bubble and into the blood. Nitrogen diffusing into the blood and remaining dissolved is carried to the lungs and eliminated harmlessly, but nitrogen diffusing into the bubble causes it to expand.

Should expanding bubbles cause clinical decompression illness, divers are usually placed on 100% oxygen and recompressed to 18 m (60 ft) where the

absolute pressure is 2.82 ata (285 kPa) in the bubble and lung (Fig. 14.14). This raises the nitrogen partial pressure in the bubble, and nitrogen diffuses out of the bubble rapidly because of the large concentration gradient (oxygen window). This is the central concept underlying recompression therapy with 100% oxygen.

Bubble–Tissue Diffusion

The inert gas partial pressure in a bubble can be greater than the inert gas tension in tissue because of the oxygen window (Fig. 14.14). As a result, a concentration gradient develops around a bubble as outwardly diffusing gas is dissolved in an increasingly larger tissue volume and is carried away by the circulation. Figure 14.15 shows simulated nitrogen and helium gradients around a bubble. These gradients were computed by assuming the circulation absorbs inert gas at a rate proportional to the local difference between blood and tissue tensions (Van Liew 1968). The gradients dissipate at a distance of several millimetres.

When a bubble forms in tissue, the driving force for the elimination of inert gas is reduced from the difference between the arterial and tissue tensions to the magnitude of the oxygen window. This reduction causes the rate of gas elimination to fall (Hills 1978). The rate is further reduced by diffusion resistance around the bubble (Van Liew & Hlastala 1969; Van Liew 1971a; Hlastala & Van Liew 1975). The gas in a bubble must diffuse back into tissue before removal by blood flow. Thus gas exchange during decompression might be expected to be slower than exchange during time at depth. Evidence of reduced exchange efficiency during decompression has been found in humans (Willmon & Behnke 1941; Tobias *et al.* 1949; Kindwall *et al.* 1975; see Fig. 14.3), goats (Hempleman 1960, 1975), dogs (D'Aoust *et al.* 1976) and guinea-pigs (Hills 1978).

Dissimilar gases were observed to be eliminated from subcutaneous pockets in rats at unequal rates that were proportional to the oxygen window and the gas partial pressure (Van Liew 1968; Van Liew *et al.* 1968, 1973). A bubble with completely diffusion-limited inert gas exchange appears to have an exchange rate governed by the product of diffusivity and tissue solubility (permeability), whereas a bubble which is completely perfusion-limited has an exchange rate determined by the product of perfusion and blood solubility (Piiper *et al.* 1962).

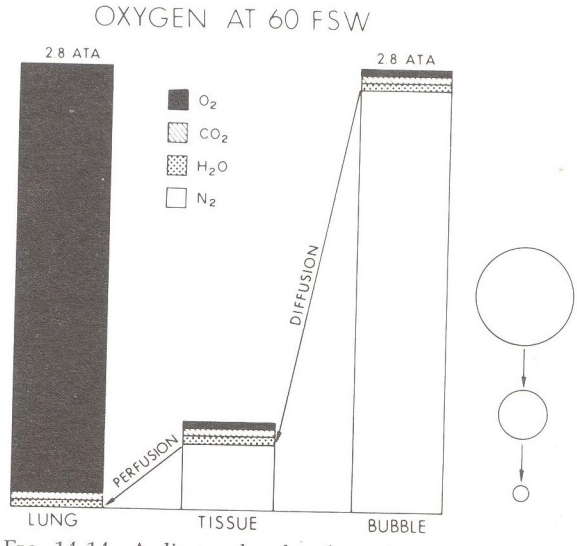

FIG. 14.14. A diver who develops decompression illness is recompressed to 18 m (60 ft) while breathing 100% oxygen. The sum of the partial pressures of all gases in the bubble increases to 2.82 ata (285 kPa) but outward diffusion of excess oxygen and carbon dioxide reduces their partial pressures to tissue levels. Since oxygen, carbon dioxide and water vapour are controlled to their tissue values, the nitrogen partial pressure must rise until the sum of all partial pressures equals 2.82 ata (285 kPa). The oxygen window is the concentration gradient between nitrogen in the bubble and in the tissue down which nitrogen diffuses. The size of the oxygen window increases as the tissue nitrogen tension is reduced by perfusion. (Reproduced with permission from Vann 1989c)

The exchange of nitrogen and helium in subcutaneous gas pockets was influenced by both diffusion and perfusion, but diffusion was more important for nitrogen (Piiper *et al.* 1962).

Van Liew and Passke (1967) postulated that shifting from a slowly permeating gas to a rapidly permeating gas after decompression might increase the risk of decompression illness. Bubbles would expand because the faster gas would enter before the slower gas could leave. This hypothesis was supported by an increase in the incidence of decompression illness when rats were shifted after decompression from air to highly permeable nitrous oxide (Van Liew 1971b).

A similar observation was made for humans who were saturated on air and shifted isobarically to helium–oxygen. Knee pain occurred 5–7 h after the gas shift (see 'Helium–Oxygen Diving'). Reference to Table 14.1 indicates that the permeability of helium in water is 14 times greater than that of

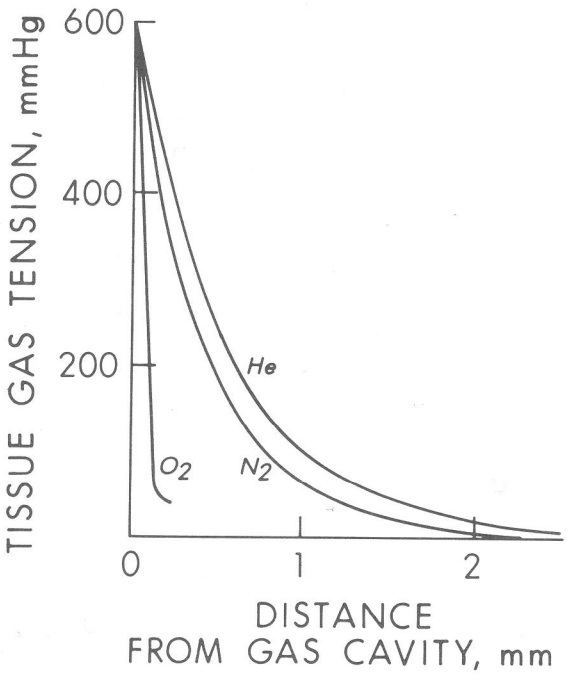

FIG. 14.15. Dissolved gas concentration gradients around a bubble filled with oxygen, nitrogen or helium. The oxygen concentration gradient is steepest because oxygen is metabolized in tissue as it diffuses out of the bubble. Nitrogen and helium are metabolically inert and are removed from tissue only by perfusion. Helium extends further into tissue than nitrogen because it diffuses more quickly. (Redrawn from Van Liew 1968)

nitrogen. This might implicate a diffusion-limited aqueous tissue as the site of the knee pain if it were caused by an expanding bubble.

Bubbles in adipose tissue and spinal white matter were observed to shrink, on the other hand, when rats were switched from air to helium–oxygen (Hyldegaard & Madsen 1989; Hyldegaard *et al.* 1991). James (1981) and Hills (1981) suggested that a nitrogen bubble in diffusion-limited adipose tissue would shrink during helium breathing because of greater nitrogen permeability. This would follow if the nitrogen permeability in adipose tissue were twice that of helium (assuming equal oil and adipose solubilities as in Table 14.1).

Hyldegaard *et al.* (1991) pointed out that spinal white matter is predominantly aqueous. A nitrogen bubble in aqueous tissue might shrink during helium breathing if the bubble were perfusion-limited. This follows when perfusion is the controlling factor because the solubility of helium in blood is less than that of nitrogen (Table 14.1). Thus, nitrogen would leave the bubble faster than helium could enter. The effects of gas-switching are important operationally (see 'Helium–Oxygen Diving') and possibly therapeutically (James 1981; Hills 1981).

'Oxygen Bends'

Previous sections have assumed that the tissue and venous oxygen tensions were equal to 40 torr (5.3 kPa) and remained constant at all inspired oxygen partial pressures. How valid is this assumption?

Most of the oxygen carried by blood is chemically bound to haemoglobin and, under normal conditions, only a small fraction is dissolved. Figure 14.16 shows the total oxygen content of blood in ml O_2 per 100 ml blood (a unit known as volume % or vol%) as a function of the oxygen tension. Point A1 is the arterial blood of a diver breathing air at sea level. Haemoglobin is nearly saturated with oxygen under these conditions. As the arterial blood passes through tissue, 5 vol% O_2 are removed and converted to CO_2. This causes the venous oxygen tension (indicated by point V1) to fall to 46 torr (6 kPa).

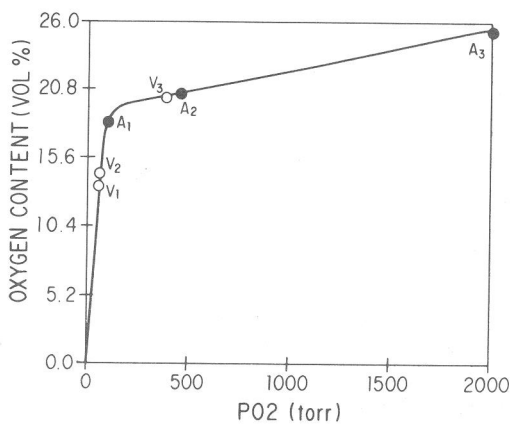

FIG. 14.16. The total blood oxygen content in vol% (ml O_2/100 ml blood) as a function of blood oxygen tension. Total content is the physically dissolved oxygen plus the oxygen chemically bound to haemoglobin. The points marked A1, A2, A3 and V1, V2, V3 are approximate arterial and venous oxygen tensions during air breathing at sea level, during air breathing at 3.5 ata (354 kPa) and during oxygen breathing at 3.5 ata (354 kPa). The oxygen extraction from blood is taken as 5 vol%. (Vann 1989d)

Now consider a diver breathing air at 3.5 ata (354 kPa). His alveolar oxygen partial pressure is 504 torr (67 kPa), but his arterial tension is only about 450 torr (60 kPa) as a result of ventilation–perfusion inequalities (Lambertsen *et al.* 1953). This is shown as point A2 in Fig. 14.16. When 5 vol% O_2 are extracted by tissue, the venous tension (point V2) falls to 53 torr (7 kPa). Now the diver switches to 100% O_2 at 3.5 ata (354 kPa) and his alveolar partial pressure rises to 2570 torr (342 kPa). Ventilation–perfusion inequalities reduce the oxygen tension in his arterial blood to around 2000 torr (266 kPa; Lambertsen *et al.* 1953). This is shown as point A3. Here, however, the venous tension (point V3) rises to 380 torr (51 kPa), far above the previous venous values. This unusually high venous tension occurs because the metabolic requirements of tissue are met entirely by dissolved oxygen. Venous haemoglobin remains saturated and on the flat rather than on the steep part of the oxygen content curve.

Tissue oxygen extraction is a function of metabolism and blood flow and ranges from 1.3 to 10.0 vol% depending on the tissue (Table 14.3; Folkow & Neil 1971). The effect of the oxygen extraction on venous oxygen tension is shown in Fig. 14.17 as a function of arterial tension. The lowest curve, which represents an extraction of 6 vol%, shows that the venous tension rises gradually at arterial tensions of up to 2000 torr (266 kPa). For extractions of 5 vol% and below, however, the venous tension increases precipitously. At the lowest extractions, the venous oxygen tension can contribute more than 760 torr (101 kPa) to the dissolved gas tension. Could this additional oxygen potentiate bubble growth when added to nitrogen already dissolve in tissue?

TABLE 14.3
Oxygen extraction in vol% (ml/100 ml blood) for various tissues and organs (Folkow & Neil 1971)

Heart	10.0
Brain	6.0
Gastrointestinal tract	6.0
Resting muscle	5.0
Kidney	1.3
Remainder	5.0

Donald (1955) addressed the possible contribution of oxygen to decompression illness by exposing seven goats to 60 min dives at 15 m (50 ft). None of the animals developed symptoms during air dives with an oxygen partial pressure of 0.53 ata (53.5 kPa). When the partial pressure was raised to 3.53 ata (356.5 kPa) with the same nitrogen partial pressure, however, six of seven animals developed serious but transient symptoms. Five recovered spontaneously at sea level and one needed recompression.

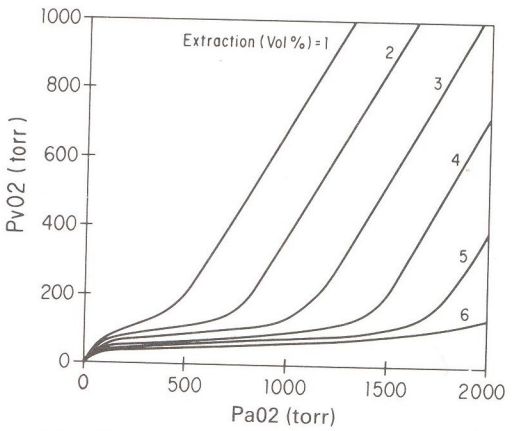

FIG. 14.17. The effect of oxygen extraction on venous oxygen tension (Pv_{O2}) as a function of arterial oxygen tension (Pa_{O2}). At higher oxygen extractions, Pv_{O2} remains relatively constant as Pa_{O2} rises. In tissues with lower extraction, Pv_{O2} rises steeply at high Pa_{O2}. This increase begins sooner at lower extractions. (Vann 1989d)

These effects, which Donald (1955) called 'oxygen bends', indicate that oxygen is not an innocuous gas for decompression at a partial pressure of 3.53 ata (356.5 kPa). Nevertheless, the spontaneous recovery from serious symptoms suggests that oxygen bends may be a less serious problem than nitrogen-induced symptoms because excess oxygen in the bubbles is rapidly absorbed upon decompression to sea level and return to normal oxygen partial pressure.

Equivalent Air Depth

Raising the oxygen fraction in a diver's breathing gas reduces nitrogen uptake at depth and increases allowable bottom time. As long as the venous oxygen tension remains nearly constant (Fig. 14.17), the decompression requirements should be determined only by nitrogen, and decompression schedules may be chosen according to the Equivalent Air Depth (EAD) theory. The

EAD theory allows existing air decompression tables to be used with nitrogen–oxygen mixtures having elevated oxygen fractions.

The EAD is the depth of an imaginary air dive that would have the same nitrogen partial pressure as an actual dive with a gas having an oxygen fraction greater than air. EAD is given by

$$\text{EAD} = \frac{(1 - F_{IO2})\,(D + 10\,\text{m})}{0.79} - 10\,\text{m}$$

where D is the actual depth, and F_{IO2} is the inspired oxygen fraction. If the actual depth is 39 m (130 ft) and the F_{IO2} is 32%, for example, the EAD is 32 m (107 ft) and air decompression schedules for 33 m (110 ft) should be used.

The EAD theory should be reasonable at low inspired oxygen partial pressures (P_{IO2}'s) where the venous oxygen tension is relatively unchanged but might fail at high partial pressures if the metabolic requirements of tissue are met by oxygen dissolved in the blood. Weathersby *et al.* (1986b) tested the EAD theory during no-stop dives at various depths for bottom times of 30, 60 and 240 min and P_{IO2}'s of 0.21–1.3 ata (21–131 kPa). The mean incidences of decompression illness are shown in Fig. 14.18 at high and low P_{IO2}'s. Each point represents the average of 76–82 dives. The incidence of decompression illness increased slightly at the higher P_{IO2} for one dive series and decreased in two series. This could indicate either higher or lower risk at elevated P_{IO2}, but neither conclusion was supported statistically. A lower risk might be explained by decreased tissue perfusion and nitrogen uptake due to oxygen-induced vasoconstriction.

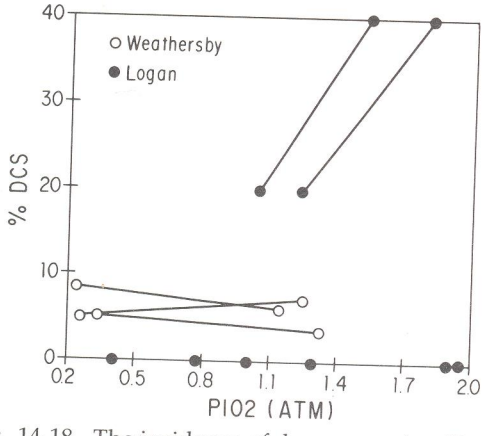

FIG. 14.18. The incidence of decompression illness as a function of P_{IO2} for humans from studies by Logan (1961) and Weathersby *et al.* (1986b)

Another study tested decompression dives with bottom times of 15, 30, 60 and 180 min at P_{IO2}'s of 1.0–1.8 ata (101–182 kPa; Logan 1961). The results of this study are also shown in Fig. 14.18. Three dive series were inconclusive as there was no decompression illness. During two series, one incident occurred in five trials at the lower P_{IO2} and two incidents in five trials at the higher P_{IO2}. These trials did not strictly test the EAD theory, however, as decompression took place with a higher P_{IO2} than in air. This would advantageously accelerate nitrogen elimination.

Logan (1961) concluded that oxygen partial pressures between 1.2 and 1.6 ata (121 and 162 kPa) make a small and statistically insignificant contribution to decompression risk and that this risk does not warrant abandoning the EAD theory. The results of Weathersby *et al.* (1986b) do not contest this conclusion, at least up to oxygen partial pressures of 1.3 ata (131 kPa). While the partial pressure at which the EAD theory begins to fail is unknown, oxygen partial pressures much higher than 1.3 ata (131 kPa) may not be altogether useful due to the increasing risk of central nervous system oxygen toxicity.

PROBABILISTIC MODELLING

A decompression model is a set of concepts which relates the occurrence or non-occurrence of decompression illness to changes in ambient pressure and breathing gas composition. The purpose of a model is to avoid an unacceptable incidence of decompression symptoms. A model may incorporate environmental or physiological factors such as exercise, temperature, body fat, etc., if sufficient data are available to justify their inclusion.

Models range from empirical mathematical functions with no physiological rationale to detailed descriptions of the biophysical processes believed to be relevant. The perfect model would predict the nature and onset time of every incident of decompression illness. Such a model does not yet exist and may never exist, but imperfect models have had reasonable success in computing useful decompression procedures (Chapter 13; Wienke 1991).