DECOMPRESSION THEORY

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Decompression theory describes the processes which determine the uptake and elimination of inert gases by tissues, and the theories of bubble formation and growth.

THE UPTAKE OF INERT GASES IN DIVING

Prior to a dive, the gas partial pressures in a diver's lungs, blood and tissues are in balance, a state of equilibrium. In diving parlance this is called **saturation,** and at sea-level pressure the partial pressure of nitrogen (PN_2) in tissues (PtN_2) can be calculated from:

$$PtN_2 = Pabs \times FtN_2 \text{ (**Dalton's Law**)} \qquad \textbf{Eq. 1}$$

$$= 1 \text{ bar} \times 0.8 = 0.8 \text{ bars},$$

where Pabs is the absolute pressure and FtN_2 is the fractional concentration of N_2.

If a diver, breathing 20% oxygen and 80% nitrogen, swims down to 30 MSW the inspired PN_2 (PiN_2) is calculated from equation 1 as:

$$PiN_2 = 4 \text{ bars} \times 0.8 = 3.2 \text{ bars}.$$

The lung alveolar PN_2 (P_AN_2) will be slightly lower than this, but for the sake of simplicity we will assume that it also is 3.2 bars. The venous blood being brought to the lungs will have a PN_2 (PvN_2) of 0.8 bars as it will be in equilibrium with the tissues and N_2 will diffuse from the alveoli into the blood. Equilibrium is quickly reached such that the arterial blood leaving the lung has a PN_2 (PaN_2) of 3.2 bars. Although some venous blood by-passes the lungs (a venous admixture or right to left shunt), again for simplicity it is assumed that the arterial blood entering the aorta from the left ventricle has a PN_2 (PaN_2) of 3.2 bars. It can be seen that ventilation is assumed to not be rate-limiting in the body's uptake of inert gases during diving. The blood that reaches the tissues has a PN_2 of 3.2 bars, and nitrogen will diffuse into the tissues, which have an initial PN_2 (PtN_2) of 0.8 bar, until such time as the PtN_2 also reaches 3.2 bars (equilibrium or saturation). The factors which determine how quickly the tissues reach equilibrium are:

1. the blood supply of the tissues (perfusion in mls of blood/100gm of tissue/min);

2. the solubility of the gas in the tissue relative to its solubility in blood;

3. the rate at which the gas diffuses through the tissue;

4. the temperature of the tissue (which influences both gas solubility and diffusion rates, as well as regional perfusion);

5. the tissue PCO_2 ($PtCO_2$) [which influences regional perfusion]; and,

6. the local energy consumption (which is related to workload and influences both $PtCO_2$ and regional perfusion).

With the exception of the Royal Navy and Royal Naval Physiological Laboratories (RNPL) and their derivative (e.g. BSAC) decompression schedules (which consider diffusion to be the rate-limiting process), all other decompression schedules are based on gas uptake being primarily influenced by tissue perfusion (the greater the blood flow the faster equilibrium is reached) and by the solubility of the gas in the tissue (the greater the solubility the slower equilibrium is reached). The attraction of these so-called "perfusion models" is that they make it easy to calculate the gas partial pressures in the tissues at any time of the dive using a single-function exponential for each tissue. For example, after a time "T", this can be calculated for N_2 for a specific tissue as (EQUATION TWO):

$$PtN^T_2 = PtN^o_2 + (PaN_2 - PtN^o_2)(1-e^{-q\alpha B/\alpha t}), \qquad \textbf{Eq. 2}$$

where PtN^T_2 is the PtN_2 at time = T, PtN^o_2 is the PtN_2 at time zero, PaN_2 is the arterial PN_2, q is the blood supply to the tissue, α_B is the solubility of N_2 in blood and α_t is the solubility of N_2 in the tissue.

A time-constant for each tissue (half-life, T½) can be derived and is based on its blood supply (q) and on the relative solubility of the gas in the tissue (α_t) compared with blood (α_B). The decompression schedules are then calculated by considering the body to be comprised of a number of tissue types with a range of half-lives.

Although these models are attractively simple, they do not accurately describe gas uptake as all of the 6 processes listed above influence the rate at which equilibrium is reached and their contributions are inter-related. Also, in some tissues such as tendons, blood vessels open and close periodically (i.e. perfusion is intermittent), such that a simple exponential description of gas uptake by these tissues is inappropriate.

THE ELIMINATION OF INERT GASES IN DIVING

At the completion of the dive described previously, the PtN_2 will vary between 0.8 and 3.2 bars dependent upon the duration of the dive, the time since the dive, the tissue, and the effects of the 6 processes listed above. Again for simplicity, we will assume that the tissues have reached equilibrium (viz; PtN_2 = 3.2 bars).

While it takes only about 20 minutes to saturate the highly perfused brain with N_2, it takes about 24 hours to achieve this for adipose tissue, as this has a poor blood supply and N_2 is fat-soluble. Helium is not fat-soluble and diffuses quickly; and, hence total body equilibrium is reached much more quickly with this gas.

If the diver now returns to the surface, the PiN_2 falls to 0.8 bars, and allowing for all the previous assumptions, so does the PAN_2. The venous blood returning to the lungs will have a PN_2 of 3.2 bars (equilibrium with the tissues), and nitrogen will diffuse from blood to lungs to be exhaled. Again, the blood leaving the lungs and from the left heart to the aorta will have a PN_2 that approaches 0.8 bars, and when this arterial blood reaches the tissues, nitrogen will diffuse out of the tissues and into the blood along its concentration gradient (PtN_2- PaN_2 = 3.2- 0.8 = 2.4 bars).

The rate at which gas is eliminated from tissues and an equilibrium at PN_2 = 0.8 bars is re-established is influenced by exactly the same 6 processes as those that affect uptake; except that for some unknown reason it is very much slower. Unfortunately, the majority of decompression schedules are based on the assumption that gas uptake and elimination by tissues are mirror-images of each other and hence occur at the same rate.

Also, if bubbles form in the tissues, the gradient for inert gas to diffuse out of the tissues is significantly reduced. This is because the pressure inside the bubbles (P_B) will only be slightly higher than the ambient pressure (Pabs) to which the diver is subjected. In this case, P_B will be only slightly greater than 1 bar. As the bubble will initially contain oxygen, water vapour and the other constituents of the gas mixture being breathed and in similar fraction, the PN_2 in the bubble (P_BN_2) can be calculated from **Dalton's Law:**

$$P_BN_2 = P_B \times F_BN_2 \qquad \textbf{Eq. 3}$$

$$= \sim 1 \text{ bar} \times 0.8$$

$$\sim 0.8 \text{ bar.}$$

This bubble exists in a tissue which has PN_2 of 3.2 bars and it is clear that nitrogen will diffuse into the bubble, expanding it and lowering P_B (see Equation 5 below), and that less nitrogen will diffuse into the blood to be eliminated by the lungs.

It follows that the ideal decompression from a dive is the one that creates the biggest possible gradient (PtN_2- PaN_2) without causing bubbles to form. The effect of bubble formation on inert gas elimination demonstrates the intrinsic weakness of repetitive diving practices, and is the reason why multiple ascents (decompressions and hence bubble formation) are probably the single biggest risk-factor for decompression illness.

It is also clear from the preceding discussions of gas uptake and elimination that available decompression schedules are based on conceptual models that do not reflect the biological reality and that the choice of a particular schedule should be based on its demonstrated success in the field at preventing decompression illness and not on the theory that underlies it.

BUBBLE FORMATION AND GROWTH

If a bubble is to form in a tissue, there must be sufficient energy to overcome both the effects of the surface tension (γ) of the tissue liquid, which will act to collapse any bubble, and for gas to come out of solution. For a spherical bubble to form in a liquid of surface tension, γ, the total energy required (E) can be calculated from the Gibbs Free Energy Equation (EQUATION FOUR).

$$E = 4\pi r^2\gamma + 4/3\pi r^3 L_N \frac{PtGas}{Pamb}, \qquad \textbf{Eq. 4}$$

where L_N is the natural logarithm of the ratio of tissue gas tensions (PtGas) to ambient pressure (Pamb) and r is the radius of the bubble.

The pressure due to the tissue surface tension (P_γ) increases as the size of the bubble decreases in accordance with the **Laplace equation.** For a spherical bubble of radius, r, in a tissue of surface tension, γ, and where the bubble is not in contact with a solid surface (contact angle is 0°), the magnitude of P_γ is (EQUATION FIVE):

$$P_\gamma = \frac{2.\gamma}{r} \qquad \textbf{Eq. 5}$$

It follows that very high values of P_γ act on small bubbles and it is assumed that for bubbles to form in a diver's tissues that the P_γ is reduced by initial bubble formation occurring in defects or crevices of solid surfaces (viz; blood vessel walls), or on the basis of pre-existing small bubbles (called **nuclei**) which are formed continually in the body (viz; in areas of turbulent blood flow). There is experimental evidence to support the concept of such nuclei.

The linear relationship between the amount of gas in solution in a tissue and the Pgas to which that the tissue is exposed, is described by **Henry's Law**. This explains the bubble formation in carbonated drinks when the cap is removed and the Pabs (and hence PCO_2 in accordance with **Dalton's Law**) is lowered.

When the diver described previously returns to the surface however (Pabs = 1 bar), the PtN_2 is still 3.2 bars. In this context bubbles will form, and will always form when the partial pressure of an inert gas in a tissue exceeds ambient pressure (Pabs). Since 1908, the relationship between Ptgas and Pabs (the second component of equation 4) that determines whether bubbles form or not has been actively debated (as it is vital to decompression schedule calculations), and yet still has not been resolved. Boycott, Damant and Haldane (1908) proposed that bubbles would not form unless PtN_2 was at least 1.6 times greater than Pabs (the famous 2:1 ratio for Pabs and air diving). Since then the critical relationship between Ptgas and Pabs has been claimed to be everything from a fixed ratio, a variable ratio, a fixed difference, to a variable difference. Indeed, it is likely that bubbles form whenever Ptgas exceeds Pabs. The difficulty at arriving at a sensible relationship is firstly that it is hard to know when bubbles form, as they form in tissues before they do in venous blood and return to the heart (where they can be detected ultrasonically); and secondly, ultrasonically detectable bubbles in the heart occur before, and often without, symptoms and signs of decompression illness. Modern sound technology may help to resolve this dilemma.

The second reason why it is difficult to determine when bubbles form is that the total of gas tensions in tissues and venous blood is less than atmospheric pressure. Consequently, this provides a "safety buffer", and is known as **"inherent unsaturation."** Inert gas tensions must therefore increase by at least this amount (the degree of unsaturation) before atmospheric pressure is even equalled, let alone exceeded. This phenomena is due to the consumption of oxygen in the tissues and the production of carbon dioxide. Even if one molecule of carbon dioxide is produced for every molecule of oxygen consumed, carbon dioxide is far more soluble than oxygen and hence the fall in oxygen partial pressure will be greater than the rise in carbon dioxide partial pressure (TABLE 1).

When 100% oxygen is breathed, and especially at hyperbaric pressures, this inherent unsaturation is significantly increased. This is called the "oxygen window" (TABLE 2).

Once formed, the total pressure in a bubble (P_B) must be at least equal to the sum of ambient pressure (Pabs), the pressure due to the surface tension of the tissue fluid (P_γ), and the pressure due to the elastic distortion of the tissue (Pe); all of which will act to collapse the bubble (EQUATION SIX):

$$P_B \geq Pabs + P_\gamma + Pe \qquad \textbf{Eq. 6}$$

As the P_γ increases with decreasing bubble size, the smaller the bubble the greater its total pressure. This explains why bubbles coalesce, that is why small bubbles (high pressure) bleed into larger bubbles (lower pressure) to expand the bigger bubble and lower its pressure further. In addition to increasing stability because of growth, and hence lowered P_γ, bubbles also become stabilised by surface active molecules (surfactants) being absorbed onto their surface. These surfactants are complex fats and exist in the lungs (to keep the alveoli dry and expanded), and line blood vessels and the gastro-intestinal tract. The hydrophobic (water-repellant) portions of these molecules are attracted to gas bubbles. Once stabilised in this fashion bubbles may possibly exist in tissues for weeks.

Compression of a diver in a recompression chamber with tissue bubbles will reduce the volume of these bubbles by an amount predictable from Boyle's Law (TABLE 3).

This demonstrates why most divers with decompression illness are treated at 18 MSW (2.8 bars); also, at this depth 100% oxygen can be administered. The physical reduction in bubble volume has the following effects:

1. increased P_γ acting on the bubble to collapse it (although this does not increase as much as suggested by the degree of volume reduction, as P_γ varies with bubble radius not volume, and the radius of a spherical bubble varies with the cube-root of its volume);

2. reduced length of a cylindrical bubble in an arteriole (as the length of such an embolus varies directly with its volume) such that it is more likely to redistribute through the capillary bed to the veins;

3. relatively increased bubble surface area (to volume) for gas exchange;

4. reduced compression and back-pressure in non-complaint tissues (viz; tendon, spinal cord, bone medulla); and

5. reduction of bubble volume below the volume-threshold for symptoms.

SUGGESTED READING

1. Vann RD, Thalmann ED. Decompression physiology and practice. In: Bennett PB, Elliott DH Eds. The physiology and medicine of diving, fourth edition. Saunders, London, 1992, pp 376-432.

TABLE 1: ALVEOLAR, ARTERIAL AND VENOUS GAS TENSIONS DURING AIR BREATHING

	Alveolar (mmHg)	**Arterial (mmHg)**	**Venous (mmHg)**
CO_2	40	40	45
O_2	104	95	40
H_2O	46	46	46
N_2	570	570	570
Total	**760**	**751**	**701**

TABLE 2: TYPICAL ALVEOLAR, ARTERIAL AND VENOUS GAS TENSIONS AFTER BREATHING 100% OXYGEN AT ATMOSPHERIC PRESSURE FOR 4 HOURS

	Alveolar (mmHg)	**Arterial (mmHg)**	**Venous (mmHg)**
CO_2	40	40	45
O_2	674	650	100
H_2O	46	46	46
N_2	0	0	20
Total	**760**	**736**	**211**

TABLE 3: THE RELATIONSHIP BETWEEN DEPTH, PRESSURE AND THE RELATIVE VOLUME OF A SPHERICAL BUBBLE

Depth (msw)	Pressure (bars)	Relative volume
0	1.0	1.0
10	2.0	0.5
20	3.0	0.33
30	4.0	0.25
40	5.0	0.2
50	6.0	0.17