TALES FROM THE DEEP: THE PHYSIOLOGICAL CHALLENGES OF DEEP DIVING

Dr Simon Mitchell

Auckland City Hospital Auckland

Background

On 1 November 2008, Morgan Saxton was flying a Robinson R22 helicopter over Lake Wanaka when he crashed into the lake for reasons unknown. Over the following days, a small slick formed by oil leaking from the engine gave a clue to the location of the wreckage. It appeared to lie on a steeply sloping lake floor close to the shore, and in 80m (270 ft) of water. This was confirmed by a Royal New Zealand Navy remote operated vehicle (ROV); effectively an underwater robot equipped with a video camera and capable of diving to ~ 200m. The robot found the main airframe of the helicopter, with Morgan still in the cockpit. The operators managed to get a line onto the wreckage and both the wreck and pilot were retrieved to the surface.

Unfortunately, the engine of the helicopter had become detached from the main airframe during the crash. A further search with the ROV located it further up the slope in 74m depth, and an attempt to attach a line to the engine failed. This was a significant disappointment to the accident investigators since the engine and its drive linkages to the rotors might hold important clues to the cause of the crash. Not surprisingly, thoughts turned to sending divers down to find the engine and attach a lifting line to it. However, a dive to 70 - 80m imposes multiple physical and physiological challenges not present in normal recreational air diving, and neither the police nor the Navy dive teams were capable of mounting a diving operation to that depth in the Wanaka location. Most of these challenges (and their solutions) pertain to issues that are either directly or at least tangentially relevant to the science and practice of anaesthesia.

Pressure Changes With Depth

Fundamental to understanding the challenges of a deep dive is the concept that pressure increases rapidly during descent through the water column due to the 'weight of water' on top of the diver. Indeed, in approximate terms, pressure increases by 1 atmosphere for every 10m depth in sea water. Specifically:

1 atm abs = 101.325 kPa = 10.13 msw = 33.08 fsw = 760 mmHg

Where: atm abs = atmospheres absolute; kPa = kilopascals; msw = metres of seawater; fsw = feet of seawater; mmHg = millimetres of mercury.

Freshwater is less dense, and the increase in pressure is slightly slower, but the difference is sufficiently small as to be ignored in the present discussion. Thus, at a depth of 80m the ambient pressure would be approximately 9 atmospheres absolute (atm abs) (1 atm generated by the atmosphere above the water, and 8 atm generated by the water pressure). As depth and pressure increase, self contained underwater breathing apparatus (SCUBA) automatically adjusts the respired gas pressure supplied at the mouthpiece to match ambient pressure. If it did not, the gas pressure in the airways would be less than the ambient pressure and breathing would rapidly become impossible with even small increases in depth. This means that at 80m a diver breathing from standard scuba equipment would be breathing air at 9 atm abs. There are several problems associated with this.



Gas Density

The Problem

68 If air were respired at 80m (9 atm abs), it would be 9 times as dense as at the surface (1 atm abs). In absolute terms, the density at the surface would be 1.293 g L⁻¹ vs 11.637 g L⁻¹ at 80m. The effect of such changes in density on ventilatory capacity have been well researched. Maximum voluntary ventilation (MVV) declines significantly with depth (Figure 1); indeed MVV during air breathing at 30m (4 atm abs) is only 50% that at the surface.



Figure 1. Changes in MVV during air breathing as depth and gas density increases. From Camporesi and Bosco (2003)¹

This reduction in MVV can be explained by changes in resistance to flow and dynamic airway compression when gas density is greater. It is widely agreed that laminar flow is unlikely during respiration of gas at equivalent density to air at \geq 4 atm abs, even during quiet breathing in distal airway generations where it is usually predicted to occur. Under non-laminar flow conditions the resistance to flow (*R*) and pressure drop (ΔP) along the lower airway can be described by equations that include terms for density and viscosity as follows:

$$\Delta P = K(\mu\rho)^{\frac{1}{2}} \dot{V}^{\frac{3}{2}}$$
$$R = K(\mu\rho\dot{V})^{\frac{1}{2}}$$

Where: *K* is a constant dependent on lung anatomy and volume; μ = viscosity; ρ = density; and *V* = flow.

The greater drop in pressure along the airway when the respired gas is denser results in dynamic airway compression and effort independence during exhalation at much lower flow rates than normal.² The increase in airways resistance and the early onset of dynamic airway compression result in a tendency to breathe at higher lung volumes (and therefore larger airway diameters), but this puts the lung on an unfavorable part of the compliance curve, increasing the work of breathing. These factors, and particularly the early onset of dynamic airway compression, are thought to contribute significantly to the limitation of MVV.

If exercise ventilation intercepts a falling MVV at depth then the limits of normocapnic ventilation are reached. Wood and Bryan $(1969)^2$ raised the intriguing and frightening possibility of a related 'vicious circle' in which attempts by an exercising diver to increase ventilation beyond the point of effort independence merely produce more CO_2 with no increase in ventilation. The rising CO_2 would drive even greater effort to increase ventilation, thus establishing the vicious circle. This could ultimately result in death due to hypercapnia and there is strong circumstantial evidence that a scenario of this nature recently caused the death of a diver at extreme depth.³



Even during sub-maximal ventilation there are potential problems. There is considerable evidence that denser gas and the associated increase in work of breathing reduces the sensitivity of the respiratory controller to rising CO_2 , with a consequent tendency to hypercapnia; even when an increase in ventilation could be achieved.⁴ It is as though the controller prefers to allow CO_2 to rise, rather than drive the extra work required to maintain normocapnia; a phenomenon also seen in some COPD patients. This is problematic because even mild hypercapnia can predispose to narcosis and oxygen toxicity (see below).

In summary, increasing gas density during diving potentially reduces ventilatory capacity and predisposes to hypercapnia. Hypercapnia may predispose to oxygen toxicity and narcosis, and if progressive may ultimately result in incapacitation and unconsciousness. These respiratory issues are discussed in detail by Doolette and Mitchell (2009).⁴

The Solution

Deep divers substitute helium (a much lighter gas) for some or all of the nitrogen in the breathing mix in order to reduce gas density. This substitution is further discussed below.

Nitrogen Narcosis

The Problem

Nitrogen breathed under pressure has a narcotic effect and will ultimately produce hypnosis at a sufficiently high P_1N_2 . The mechanism of this effect is unknown, but it seems likely to have parallels with that of other anaesthetic gases. During air diving, narcosis is frequently described as first becoming apparent at 30m (4 atm abs) and the effects would almost certainly be incapacitating at 80m (9 atm abs). The narcotic effect of nitrogen is enhanced by hypercapnia.

The Solution

Conveniently, the solution to the gas density problem is also the solution to the narcosis problem; helium is not only light, it is also non-narcotic. Whilst it may seem sensible to substitute helium for all nitrogen in the respired mix, thus achieving the lightest and least narcotic gas possible for deep diving, this is usually not done for several reasons. First, helium is very expensive. This is less of a consideration during the use of rebreather devices (see below), but is certainly a concern during open circuit diving. Second, on short deep dives the use of higher fractions of helium usually results in longer decompressions. Finally, in very deep diving (beyond 150m) a phenomenon referred to as the 'high pressure neurological syndrome' (HPNS) may occur. This is manifest as various neurological symptoms but particularly tremors in the early stages. It is thought to be caused by a pressure effect on excitable membranes, and there are intriguing links to anaesthesia. Whereas some theories of volatile anaesthesia hold that it occurs, at least in part, because of membrane <u>expansion</u> by vapors, the HPNS may be explained by <u>compression</u> of membranes. Ironically, amelioration of HPNS can be achieved by giving an 'anaesthetic agent'; in this case nitrogen under pressure which may act by re-expanding the membrane. As in basic discussions of volatile anaesthesia these explanations are conceptual and simplistic, and the causation of HPNS is complex, controversial, and the subject of much ongoing research.

The usual approach to decisions around the degree of nitrogen substitution with helium is to take account of the depth of the dive, the equipment utilised (rebreather vs open circuit – see below) and the amount of narcosis the diver is prepared to tolerate. Further discussion of this subject is beyond the scope of this paper, and may be found elsewhere.⁵





Oxygen Toxicity

The Problem

If a diver were to breathe air at 80m (9 atm abs) the P_1O_2 would be 9 x 0.21 = 1.9 atm abs. In fact, oxygen respired at a P_1O_2 > than \approx 1.3 atm abs may cause an unheralded generalized seizure which would likely be fatal during immersion, especially at extreme depth a long way from the surface. The risk increases with the P_1O_2 and the duration of exposure, and is also increased if there is hypercapnia; probably because this increases cerebral perfusion and consequently, the jugular venous oxygen tension.

The Solution

In addition to substituting helium for some or all of the nitrogen as described earlier, the diver must also reduce the oxygen content of the respired gas mix for deep diving. In general, divers try to maximize the amount of oxygen in the mix in order to reduce both inert gas uptake and decompression time, while at the same time managing the risk of oxygen toxicity. For example, if the diver accepts that the maximum safe P_1O_2 is 1.3 atm abs, then the ideal mix for use at 80m (9 atm abs) would contain a F_1O_2 of 1.3 / 9 = 0.14 (14%).

Gas Supply

The Problem

When using standard scuba equipment, gas is exhaled directly into the water and is lost. This is wasteful of gas, and the issue becomes significant when dive is deep and the gas contains helium (and is therefore expensive). As previously observed, the scuba regulator supplies gas at ambient pressure. Thus, at 80m (9atm abs) it is 9 times as dense as at 1 atm abs and an exhaled tidal breath will expand to 9 times its original volume by the time it reaches the surface. It follows that the duration of a cylinder of gas breathed at 80m will be only 1 ninth of that if breathed at the surface. Deep dives may be hours long (see below) and if performed on open circuit scuba equipment, there can be difficulty carrying sufficient gas.

The Solution

One approach is to carry large numbers of gas cylinders but there are obvious disadvantages, and the gas cost is high. Divers are increasingly adopting the same solution as anaesthetists trying to conserve anaesthetic gas: the use of a circle circuit. In diving parlance, circle circuits are referred to as 'rebreathers', and the layout of a popular type of rebreather is shown in Figure 2.



Figure 2. Typical electronic mixed gas rebreather. From Lippman and Mitchell (2005).⁵ See explanation below.

The rebreather 'loop' consists of a mouthpiece with one-way valves, a CO₂ absorbent canister (usually referred to as a 'scrubber') and a collapsible counterlung which the diver exhales into then inhales from, 'Fresh gas flow' comes from two sources, and unlike an anesthetic machine, is not continuous in this type of rebreather. There are 3 galvanic fuel cells (Figure 2) that measure the PO_2 and feed that data back to a microprocessor. The readings are averaged, and whenever the averaged reading falls below a 'set-point' chosen by the user, the microprocessor opens an electronic solenoid valve and oxygen is allowed to bleed into the loop. When the PO_2 is restored to the set point, the solenoid valve closes. This 'constant PO₂' approach means that the oxygen content of the mix changes with depth (and ambient pressure). For example, if the user selects a PO₂ set point of 1.3 atm abs, this means that there will be 14% oxygen in the loop at 80m (as discussed above), whereas at the final decompression stop at 3m (ambient pressure 1.3 atm abs), the loop will contain 100% oxygen. This has the advantage or ensuring the diver is breathing the maximum safe PO2 and the minimum Pinert gas at all times in the dive. During descent the counterlung is compressed as ambient pressure increases, and its volume is automatically maintained from a diluent gas cylinder containing an appropriate helium and nitrogen-based mix. During the period at depth and during ascent, the only gas consumed (and added to the loop) is the oxygen consumed by the diver; a situation somewhat akin to ultra-low flow anaesthesia. Clearly, this system is very economical in respect of gas consumption. A prudent rebreather diver will always carry one or more cylinders of bailout gas with scuba regulators in case of a rebreather failure.

Decompression

The Problem

Henry's law predicts that inert gas is taken up into blood and tissues from the respired gas when breathing at greater ambient pressures during a dive. The longer and deeper a dive, the more inert gas is absorbed. During ascent from a dive ('decompression') this gas can no longer be held in solution and is eliminated in the exhaled breath. However, some tissues wash gas out more slowly than others during decompression, and if the sum of tissue gas partial pressures exceeds the ambient pressure at any point, a state of 'supersaturation' is said to exist. Bubbles may form either in the tissues themselves or the venous system draining them. Depending on the nature



of the tissue, and the number and size of the bubbles, this may result in symptoms of decompression sickness.⁶ Most venous bubbles are filtered by the lungs, but they may arterialize across right to left shunts such as a patent foramen ovale, and then be distributed to sensitive tissues such as the brain, spinal cord and inner ear, giving rise to decompression sickness symptoms relevant to those organs.⁷

The Solution

Divers use planning software to incorporate 'decompression stops' into their ascents. This aims to allow adequate time for gas washout and to thereby avoid excessive tissue supersaturation. The use of progressively higher inspired fractions of oxygen during ascent whilst remaining within safe P_1O_2 limits (as occurs with a constant PO_2 rebreather) helps accelerate inert gas elimination. However, most of the algorithms upon which these planning tools are based are theoretical and are not properly validated by real world use. Moreover, susceptibility to decompression sickness is variable among individuals and this cannot be accounted for by these algorithms because the important differences between individuals are not understood. To make matters worse, there are clearly important but poorly understood factors that vary within individuals from day to day. A dive profile that is safe for an individual on one day may produce serious decompression sickness on another day. The reader can rightly conclude that there is no reliable 'solution' to the danger of decompression sickness.

Epilogue

A relatively small number of recreational divers at the lunatic fringe (of which the author is one) utilize the above techniques (and others) to visit deeper depths and / or remain at depth longer than would be afforded by standard scuba air techniques. Utilizing a rebreather and helium – nitrogen – oxygen mixtures the author visited the helicopter wreck site to search for the engine in May this year. The profile of one of two dives is shown in Figure 3.

Unfortunately, the lake had received a massive rainfall only 5 days prior and the visibility was markedly reduced from normal. On the bottom it was only possible to see about 1-2m and it was pitch dark. The engine was not located, but a second attempt will be made later this year when the lake's visibility improves.



Figure 3. Dive profile for a dive to search for Morgan Saxton's engine May 2009. Depth is measured on the left x axis and recorded by the solid black line; time is on the y axis; the loop PO₂ is measured on the right x axis and recorded on the irregular trace (reflecting small fluctuations in PO₂). Note the depth of 81m, the bottom time of just over 20 minutes, and the total dive time of 2 hours and 14 minutes. Note also that the PO₂ was controlled around 1.3 atm abs for most of the dive except at the end when surface supplied oxygen was breathed and the loop PO₂ was allowed to drift down.



References

- 1. Camporesi EM, Bosco G. Ventilation, gas exchange, and exercise under pressure. In: *Bennett and Elliott's Physiology and Medicine of Diving (5th ed)*, edited by Brubakk AO, Neuman TS. Edinburgh: Saunders, 2003.
- 2. Wood LDH, Bryan AC. Effect of increased ambient pressure on flow-volume curve of the lung. *J Appl Physiol* 27: 4-8, 1969.
- 3. Mitchell SJ, Cronje F, Meintjes WAJ, Britz HC. Fatal respiratory failure during a technical rebreather dive at extreme pressure. *Aviat Space Environ Med* 78: 81-86, 2007.
- Doolette DJ, Mitchell SJ. Gas exchange in hyperbaric environments. In: Wagner PD, Hlastala MP (eds), Handbook of Physiology, Section 3 (Respiration) Volume IV (Gas Exchange). Bethesda, MD. 2009, The American Physiological Society. In press.
- 5. Lippmann J, Mitchell SJ. *Deeper into Diving (2nd ed)*. Melbourne: Submariner Publications, 2005: 511pp.
- 6. Doolette DJ, Mitchell SJ. The physiological kinetics of nitrogen and the prevention of decompression illness. *Clin Pharmacokinet* 40: 1-14, 2001.
- 7. Mitchell SJ, Doolette DJ. Selective vulnerability of the inner ear to decompression sickness in divers with right to left shunt: the role of tissue gas supersaturation. *J Appl Physiol* 106: 298-301, 2009.