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# Deep diving mammals: Dive behavior and circulatory adjustments contribute to bends avoidance

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**Abstract**

A mathematical model was created that predicted blood and tissue $N_2$ tension ($P_{N_2}$) during breath-hold diving. Measured muscle $P_{N_2}$ from the bottlenose dolphin after diving repeatedly to 100 m (*Tursiops truncatus* [Ridgway and Howard, 1979, Science, 4423, 1182–1183]) was compared with predictions from the model. Lung collapse was modelled as a 100% pulmonary shunt which yielded tissue $P_{N_2}$ similar to those reported for the dolphin. On the other hand, predicted muscle $P_{N_2}$ for an animal with a dive response, reducing cardiac output by 66% from surface values (20.5 to 6.8 l·min$^{-1}$), also agreed well with observed values in the absence of lung collapse. In fact, modelling indicated that both cardiovascular adjustments and dive behaviour are important in reducing $N_2$ uptake during diving and enhancing safe transfer of tissue and blood $N_2$ back to the lung immediately before coming to the surface. In particular, diving bradycardia during the descent and bottom phase together with a reduced ascent rate and increase in heart rate reduced mixed venous $P_{N_2}$ upon return to the surface by as much as 45%. This has important implications as small reductions in inert gas load (∼5%) can substantially reduce decompression sickness (DCS) risk by as much as 50% (Fahlman et al., 2001, J. Appl. Physiol. 91, 2720–2729).


*Keywords:* Decompression sickness; Modelling; Inert gas flux; Pressure; Decompression stop; Allometry

## 1. Introduction

During exposure to elevated environmental pressure increased amounts of inert gas dissolve in the tissues of air-breathing animals. The amount of inert gas dissolved is a function of the pressure and the duration of exposure. The tissue tension of dissolved inert gas continues to increase until equilibration occurs, at which time the organism is said to be saturated. If the decompression phase is too rapid, elevated levels of $N_2$ in blood or tissues exceed their solubility at the reduced pressure, a term called supersaturation. When $N_2$ becomes supersaturated in tissues, it comes out of solution possibly forming bubbles. In fact, detectable bubbles have been found in half the human subjects

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after decompression following a saturation dive to 3 m (1.3 ATA; Eckenhoff et al., 1990). At this time the tissue and blood $N_2$ tensions ($P_{N_2}$) should be ~1 ATA. Bubbles can block blood vessels and damage tissue, producing the medical condition called decompression sickness (DCS) or the bends. Some level of supersaturation and bubble formation may be tolerated, but current research suggests that any hyperbaric exposure has a finite probability of DCS (Weathersby et al., 1984, 1992).

The time it takes for a tissue to reach saturation, or equilibrium, after a change in pressure is dependent on the blood flow through that tissue (Hlastala and van Liew, 1975). This makes it possible to separate tissues into rapidly (e.g. heart) or slowly (e.g. fat) saturating tissues. Consequently, diving related vasoconstriction and bradycardia increases time to saturation and reduces the likelihood of reaching a tissue $P_{N_2}$ that will cause bubble formation during decompression. Still, current estimates indicate that blood and tissue $P_{N_2}$ in seals can be as high as 2–3 ATA during a single hyperbaric exposure without causing bends on decompression (Kooyman et al., 1972; Falke et al., 1985). Thus, in animals that dive repeatedly, elevated $P_{N_2}$ levels could predominate for most of their lives. These $P_{N_2}$ levels can cause severe DCS incidence in a significant proportion of non-diving mammals (Dromsky et al., 2000). Early research suggested that decompression was safe unless the rate of decrease in ambient pressure ($P_{amb}$) exceeded a certain critical value (Hills, 1977) but, it now seems that the occurrence of DCS is somewhat arbitrary, seldom either a certainty or zero for decompression from any given hyperbaric exposure (Weathersby et al., 1992). In addition, the probability of DCS increases with body mass ($M_b$, Berghage et al., 1979; Lillo et al., 1985, 1997, 2002; Lillo, 1988). Therefore, one "safe" tissue $P_{N_2}$ ($P_{N_{2_{tiss}}}$) cannot be assumed to apply to all breath-hold diving animals.

While repetitive breath-hold dives to 15–20 m have been reported to cause DCS in humans (Paulev, 1965, 1967), seals or dolphins are not reported to develop the bends during continuous diving. This suggests either adaptations that deal with an elevated bubble load or an ability to avoid supersaturation. An adapted immune system, which is less reactive to bubbles, could prove to be important since a connection between immune function and DCS has been shown (Ward et al., 1987; Kayar et al., 1997). Lung collapse has been suggested as a universal mechanism that reduces supersaturation and therefore, the risk of DCS in breath-hold diving mammals. Indirect measurements suggest that in seals and dolphins alveolar collapse occurs at approximately 40–80 m, reducing the amount of $N_2$ available to equilibrate with the tissues (Ridgway and Howard, 1979; Falke et al., 1985). Weddell and elephant seals are reported to dive upon exhalation further reducing the available $N_2$.

Thus, a better understanding of the inert gas flux of diving animals would enhance our understanding how they can perform repeated dives without developing $P_{N_{2_{tiss}}}$ levels that may be harmful. Therefore, we created a mathematical model to describe $P_{N_2}$ in blood and tissues during repeated breath-hold dives. The model was a simplified version of that used to trace metabolic and inert gases in human breath-hold divers (Olszowka and Rahn, 1987). However, our model accounts for changes in blood perfusion and also incorporates the possibility of a pulmonary shunt where blood bypasses the blood–gas exchange surface. We validated model predictions against data for the bottlenose dolphin (Ridgway and Howard, 1979), as this is the only animal in which both dive sequence and muscle $P_{N_2}$ have been reported for voluntary repeated breath-hold diving.

## 2. Materials and methods

### 2.1. Model

The model was adapted from the human breath-hold model developed by Olszowka and Rahn (1987) and is outlined in Fig. 1. The body was partitioned into five different compartments or stores; blood, brain, fat, muscle and a central circulatory compartment. The central circulatory compartment included heart, kidney, liver and alimentary tract while the fat compartment included fat, skin, bone and connective tissue.

Gas exchange occurred between lung and blood and between blood and each compartment (Fig. 1A–D). The $N_2$ store in the lung consisted only of a gas phase and was assumed to be perfectly mixed. We assumed that there was no diffusion resistance at the lung surface (Farhi, 1967). All pressures were corrected for water vapor pressure, assuming that the respiratory system was fully saturated at 37 °C. We