

# THE PHYSIOLOGY OF COMPRESSED GAS DIVING

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## INTRODUCTION

The breathing of compressed gas while immersed and exposed to increased ambient pressure imposes significant homeostatic challenges on the body. This chapter discusses the important mechanisms of these challenges, with particular reference to the respiratory system.

## RESPIRATORY SYSTEM

### Aspects of compressed gas breathing equipment and its function.

SCUBA diving equipment is the most commonly used compressed gas system in civilian diving and illustrates the important features and function relevant to diving physiology. Basic SCUBA equipment consists of a cylinder of air at high pressure, a demand valve regulator, and a device for holding this equipment on the diver's back. In the modern context the latter is usually an inflatable jacket called a buoyancy control device (BCD) whose dual function it is to allow buoyancy adjustment in-water and carriage of the tank and regulator. Together with the wetsuit necessary for temperate water diving and weightbelt, this apparatus may constitute a significantly restrictive force over the diver's chest and abdomen.

The regulator reduces the cylinder high pressure air to ambient pressure and supplies air on demand. Thus, at a depth of 30m where the absolute pressure is 4 bars, the regulator supplies air at 4 bars and the air is 4 times as dense as air at sea level (1 bar). The ambient pressure is "measured" by the regulator second stage (attached to the mouthpiece) which, in the upright diver, is approximately 20cm above the centre of the chest. The water pressure acting on the chest will therefore be approximately 20cm H<sub>2</sub>O higher than that of the inspired gas, creating a negative transmural pressure which is greatest at the lung bases.

Regulator breathing resistance relates inversely to quality of manufacture and standard of maintenance. Further, breathing resistance tends to increase with depth as denser air flows through the regulator mechanism.

Finally, it should be noted that the internal volume of a portion of the regulator second stage is effectively an extension of anatomical respiratory dead space.

### Mechanics of breathing.

**Changes in compliance.** Changes in compliance are seen in the lung and chest wall. The negative transmural pressure across the chest wall of the upright scuba diver causes some pulmonary capillary engorgement. This effect is enhanced by the relative centralisation of blood volume that occurs with immersion, especially in cold water (see Cardiovascular system). This

engorgement of the pulmonary capillary vasculature causes a reduction of compliance in the lung tissue. This reduces the vital capacity of the lung by 10-15%.

SCUBA equipment, wetsuits, and weightbelts exert a restrictive force on the chest wall and abdomen. This effect is potentially marked if equipment is excessively tight fitting. The compliance of the chest wall is reduced and diaphragmatic breathing is impeded.

**Changes in airways resistance.** Airways resistance is affected by changes in gas density. Resistance is defined as  $\Delta P/V$ , where  $\Delta P$  = the pressure decrease across a tube and  $V$  = flow. In laminar flow, flow is largely independent of the density of the gas. In turbulent flow however, flow is inversely related to the square root of gas density. Therefore, in turbulent flow, for a given  $\Delta P$ ,  $V$  will be decreased if gas density is increased, and by definition resistance to flow will be greater.

According to Reynold's number predictions, flow within the lungs and airways is largely laminar although this assumption is likely to be invalid because of the vortices which occur in inspired air at each division of the bronchial tree. Indeed, it is likely that turbulent flow occurs widely in the large airways, particularly during rapid breathing when flow rates are much higher.

**Changes in the work of breathing.** Work of breathing in diving is consequently increased. Work is performed by the respiratory muscles in stretching the elastic tissues of the lungs and chest wall, moving inelastic tissues, and moving air through the respiratory passages. The preceding discussion demonstrates that in the immersed scuba diver there is an increase in elastic work (viz; decreased compliance in lung and chest wall), the work of moving inelastic tissues (viz; constrictive equipment), and the work of moving air through airways (viz; increased air density). Further, the airways resistance component of this increase in work of breathing is depth dependent.

### **Ventilation/perfusion matching in diving.**

The single most important determinant of efficient gas exchange is the matching of alveolar ventilation ( $V_A$ ) to perfusion of alveolar capillaries ( $Q$ ). The optimum ratio of  $V_A$  to  $Q$  is unity. Under-ventilated and over-perfused lung units ( $V_A/Q < 1$ ) represent a "right to left" shunt. The admixture of hypoxic blood from low  $V_A/Q$  units into systemic arterial blood is an important cause of a significant alveolar-arterial oxygen gradient.

The lungs of the SCUBA diver are subjected to changes in both perfusion and ventilation. There is an increase in perfusion of lung units due to capillary engorgement (particularly at the bases) and the relative centralisation of blood volume. There is a decrease in ventilation due to reduced lung and chest wall compliance, abdominal constriction, and increased airways resistance. The net effect is toward an increase in low  $V_A/Q$  units and thus the shunting of blood from "right to left".

### **Changes in gas transport.**

**Oxygen.** Oxygen is transported in the blood either bound to haemoglobin (Hb) or dissolved in plasma. The solubility of  $O_2$  in plasma is low (0.003/mmHg/100mL) and in normobaric conditions by far the greatest proportion of  $O_2$  is transported bound to Hb.

The volume of O<sub>2</sub> bound to Hb can be calculated from:

$$\text{Hb bound O}_2 = \text{Hb concentration} \times \text{HbO}_2 \text{ capacity} \times \text{Hb saturation}$$

(mL %)                      (g%)                      (1.34 mL/g)                      (%)

Assuming a Hb concentration of 15% and saturations of 97% and 75% for arterial (PO<sub>2</sub> = 100mmHg) and venous (PO<sub>2</sub> = 40mmHg) blood respectively, values for arterial (CaO<sub>2</sub>) and venous (CvO<sub>2</sub>) oxygen contents are;

$$\text{CaO}_2 = 19.5\text{mL}\% \text{ (Hb bound)} + 0.3\text{mL} \text{ (dissolved)} = 19.8\text{mL}\%.$$

$$\text{CvO}_2 = 15.0\text{mL}\% \quad + 0.12\text{mL}\% \quad = 15.1\text{mL}\%.$$

The relative quantitative importance of O<sub>2</sub> transported by Hb is demonstrated. It is also apparent that under these typical circumstances, that 5mL% of O<sub>2</sub> is extracted from arterial blood to meet the body's needs.

Since Hb is normally 97% saturated breathing air at 1 bar, there is little potential for increasing O<sub>2</sub> transport on Hb by increasing the PO<sub>2</sub>. In contrast, dissolved O<sub>2</sub> increases linearly with PO<sub>2</sub> although it is only in hyperbaric conditions with a high FIO<sub>2</sub> that the dissolved fraction becomes significant. For example, breathing air at 3 bars gives a dissolved O<sub>2</sub> of 1.3mL%, and breathing 100% O<sub>2</sub> at 3 bars results in 6.7mL% of dissolved O<sub>2</sub> (0.003ml/dl/mmHg PO<sub>2</sub>). At 3 bars breathing air, there is still only a relatively small amount of dissolved O<sub>2</sub>. However, breathing 100% O<sub>2</sub> at 3 bars results in a dissolved fraction sufficient to meet the body's needs at rest in the absence of Hb. Hence the value of hyperbaric O<sub>2</sub> in conditions where O<sub>2</sub> delivery is compromised (**viz**; anaemia, carbon monoxide poisoning).

**Carbon dioxide.** Unlike O<sub>2</sub> which is supplied at increasing partial pressures at depth, the number of molecules of CO<sub>2</sub> that are produced remains constant for a given workload irrespective of depth. However, transport of the CO<sub>2</sub> load from tissues to lungs may be less efficient in the hyperbaric environment where an increased PO<sub>2</sub> causes a fall in reduced Hb in venous blood. Reduced Hb forms carbamino compounds with CO<sub>2</sub>, and buffers the H<sup>+</sup> resulting from hydration of CO<sub>2</sub> in red blood cells. However, these are two of the quantitatively less important mechanisms of CO<sub>2</sub> transport and the significance of this disturbance is questionable.

### **Changes in control of respiration.**

The precise anatomy and physiology of the control of breathing are still unknown but there is little doubt that CO<sub>2</sub> and O<sub>2</sub> levels in CSF and arterial blood, monitored by central and peripheral chemoreceptors are important determinants of the rate and depth (minute volume) of respiration. A rise in the PCO<sub>2</sub> or a fall in the PO<sub>2</sub> of arterial blood increases the level of brain stem respiratory centre activity, and changes in the opposite direction have an inhibitory effect.

In the hyperbaric environment, the increased PO<sub>2</sub> is thought to produce a slight depression of respiratory drive. Further, many divers, especially older career divers, show a reduced response to raised CO<sub>2</sub> levels. The mechanism of this reduced response is unknown but it has been suggested, without good supportive data, that it is a learned response in some divers.

At a cortical level, some divers deliberately override their insensible control mechanisms in an attempt to extend their underwater duration by conserving air supply. This unsafe practice in which ventilation is intentionally slowed or punctuated with short periods of apnoea is called "skip breathing".

### **Net effects of respiratory alterations.**

Sustained work output by tissues is largely limited by their O<sub>2</sub> supply. In normal exercise, tissue O<sub>2</sub> supply is limited by cardiac output rather than ventilation or gas exchange. In the healthy person ventilation can be dramatically increased to 200L/min or more, and a rapid transit of blood through alveolar capillaries (normally about 0.35sec) in high output situations does not prevent equilibration of gases across the respiratory barrier. In addition, the VA/Q profile of the lung usually improves during exercise.

Underwater, even at the relatively modest depth of 30M (4 bars) commonly attained by sport divers, air density/airway resistance factors mediate a reduction in maximum voluntary ventilation to approximately half the surface value. This reduction in ventilatory capacity, the concomitant increase in the work and O<sub>2</sub> cost of breathing, the increase in low VA/Q units, and dead space effects, determine that underwater work may be ventilation rather than perfusion limited. It can be readily appreciated that a diver at modest depth, swimming into a 1 knot current (VO<sub>2</sub> for fin swimming at 1 knot = 2L/min), wearing ill fitting equipment, using a poorly maintained regulator, and being subject to the above physiologic compromise, might fail to sustain the required work to make progress.

Another important consequence of these respiratory alterations is the diver's predisposition to retain CO<sub>2</sub>. Factors contributing to this include: increase of work of breathing which increases CO<sub>2</sub> production and limits ventilation; decreased respiratory drive; decreased CO<sub>2</sub> sensitivity in divers; skip breathing; and dead space effects. The consequences of hypercapnia in divers include: unpleasant and dangerous symptoms such as dyspnoea, headache, nausea, and unconsciousness; and potentiation of nitrogen narcosis, oxygen toxicity, and decompression illness.

## **CARDIOVASCULAR SYSTEM**

### **Changes in blood volume distribution.**

When the diver is immersed, the haemodynamic effect of gravity is abolished and there is a consequent redistribution of peripheral blood into the central circulation. This effect is enhanced in cold water when peripheral vasoconstriction further enhances the central redistribution.

The relative central hypervolaemia increases the activity of stretch receptors in the walls of the great veins and right atrium, with receptors in the carotid sinus and aortic arch also involved if the blood shift is sufficient to increase mean arterial pressure. The increased stretch receptor activity mediates a decrease in ADH production from the hypothalamus/posterior pituitary. This results in an increased permeability to water in renal distal tubule cells and therefore increased urinary loss of water. The net result is an undesirable tendency toward dehydration which may be exacerbated by lack of adequate drinking water and/or seasickness.

### **Cardiac effects.**

Immersion has been demonstrated to increase cardiac output by up to 32% in thermoneutral water. The mechanism is an increase in venous return due to the centralisation of blood volume. The increased preload, manifest as increased stretching of the heart muscle fibres during diastole, invokes the Starling mechanism in which the force of contraction is raised to cope with the extra volume. Stroke volume is therefore increased. In colder water the increase in cardiac output is less, due to a concomitant bradycardia.

There is a bradycardia associated with immersion. The so-called "mammalian dive reflex" invoked by cold water contacting the face includes a bradycardia. In predisposed individuals the vagal outflow can be intense enough to produce asystole or arrhythmias (**viz**; unexplained drowning after leaping into cold water). However, a bradycardia is produced even in a dry chamber at elevated ambient pressure and other explanations for the immersion bradycardia in divers has been sought.

It has been demonstrated that an increase in PaO<sub>2</sub> and the narcotic effect of inert gases can produce a fall in heart rate averaging 10 beats/min in divers, independent of the "dive reflex". The degree of bradycardia is increased in colder water and concomitantly, so is the risk of arrhythmias in predisposed individuals. However, the role of these factors in diving fatalities is largely unknown.

Another mechanism of bradycardia of questionable significance in divers, but that is often mentioned in the diver training literature is the carotid sinus reflex. There is the theoretical possibility that if a wetsuit hood fits too tightly around the neck, it may stimulate a carotid sinus reflex (**viz**; carotid sinus massage) and therefore a bradycardia.

Views differ on the net effect of these mechanisms on mean arterial blood pressure. It is likely that blood pressure remains largely unchanged in health in the hyperbaric environment.

### **SUGGESTED READING:**

1. Edmonds CW, Lowry C, Pennefather JW. Diving and subaquatic medicine, 4th edition. Ch 3