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DIVING AND THE LUNG

Richard Moon and Bryant Stolp

Key Words
Physiology, pulmonary barotrauma.

Introduction

The respiratory system is affected by diving via a number of mechanisms. The increased flow resistance

engendered by breathing dense gas (Fig 1) and the additional mechanical load of the breathing apparatus added to the changes in pulmonary compliance caused by water immersion may significantly reduce ventilatory capacity. Dense gas breathing also engenders a greater likelihood of impairment of gas exchange due to diffusion problems in the alveolus. In addition, the lung is potentially subjected to damage during decompression by both pulmonary overexpansion and the effects of venous gas embolism. Finally, there is an uncommon syndrome in which young healthy individuals develop pulmonary oedema shortly after immersion at the beginning of a dive.

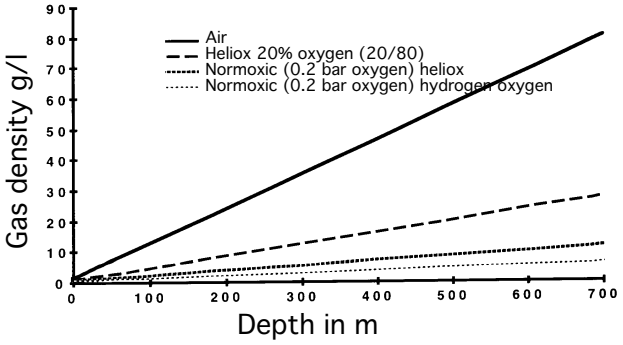

Figure 1. Gas density as a function of depth.

The densities of normoxic helium-oxygen ($He-O_2$) and hydrogen-oxygen (H_2-O_2) are displayed assuming a constant partial pressure of O_2 of 0.2 ATA. Gas temperature is 37°C.

external pressure gradient of the water column. This results in a redistribution of blood from the extremities into the thorax ranging from 500 to 800 ml. Some of this blood is retained within the great vessels and the heart, while a proportion of it engorges the pulmonary vessels, causing an increase in central venous and pulmonary artery pressures.[7] This results in a reduction in lung volume, particularly functional residual capacity (FRC)[8] and expiratory reserve volume (ERV), and a reduction in MVV of 5-10%.[1] When experiments are carried out in a hyperbaric chamber, immersion while the chamber is at pressure results in less decrement than at the surface.[9] The effects of immersion

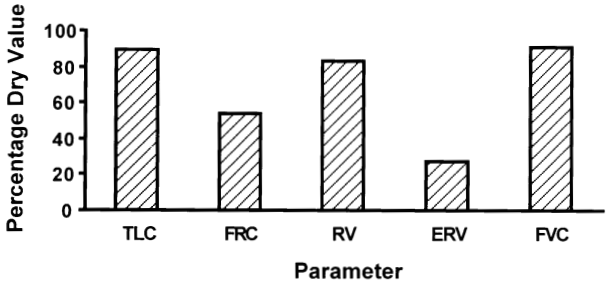

Figure 2. Effect of immersion on lung volumes.

Total lung capacity (TLC), functional residual capacity (FRC), residual volume (RV), expiratory reserve volume (ERV), forced vital capacity (FVC). Immersion reduces lung volumes predominantly by causing redistribution of blood from the legs into the thorax (see text).

Immersion

PHYSIOLOGICAL EFFECTS

During head-up immersion, the normal tendency for blood to pool in the legs due to gravity is immediately reversed as the hydrostatic pressure gradient within the venous system is almost exactly counterbalanced by the are fully exemplified by immersion to the neck and there is no additional load engendered by further descent into the water column. Changes in lung volume which occur on immersion are depicted in Fig. 2.

The engorged pulmonary vessels impinge upon the airways and increase airway resistance. Morrison and Taylor reported that subjects at rest experienced a 3 fold increase

TABLE 1
DENSITY OF BREATHING FLUIDS

Fluid	Depth (msw)	Density (g/l)
Air	0	1.1
$He-O_2$ (20% O_2)	0	0.4
Air	40	5.6
Air	50	6.8
Air	90	11.4
$He-O_2$ (20% O_2)	50	2.3
Trimix-10 (10% N_2 0.5 ATA O_2, balance He): (the highest gas density at which arterial blood gases have been measured during exercise)	650	17.1[1]
Ne-He-O_2 (0.21 ATA O_2, balance 76.8% Ne, 23.2% He): (the highest gas density breathed by man)	377	25.2[2]
Water		1000.0 [3-6]

in flow resistive work of breathing when they were immersed in water.[10] Upon immersion to the neck in water, pulmonary dynamic compliance (Cdyn) is reduced approximately 50% but static compliance (Cst) is unchanged.[11] The effect appears to be due to the immersion-induced reduction in lung volume that occurs because pressure at the mouth is lower than at the lung centroid. When mouthpiece pressure is increased to a value equal to the hydrostatic pressure at the lung centroid, thus restoring lung volume to control value, both dynamic and static compliance are returned to normal. Static compliance is a measure of the change in static lung volume for a given change in transpulmonary pressure ($\Delta V/\Delta P$) whereas dynamic compliance, measured during breathing or panting includes both respiratory compliance and airway resistance. The reduction in Cdyn with immersion is therefore probably due to the change in airway resistance and not due to altered lung tissue compliance secondary to engorged pulmonary vasculature.

Immersion also causes a tendency for airways to close at a higher lung volume (increased closing volume).[12-15] Airway closure during immersion tends to occur at lung volumes greater than FRC in older individuals.[15,16] It has been suggested that if closing volume is greater than functional residual capacity, gas exchange units subtended by closed airways would increase venous admixture, causing a reduction in arterial PO_2. Cohen et al. reported that alveolar-arterial gradient (P_AO_2-P_aO_2) increased from 7 to 16 mm Hg when subjects (mean age 23 years) were immersed to the neck in water.[17] However, a study in which blood gases and V_A/Q of the lung were measured, immersion caused neither an increase in shunt nor blood flow to low V_A/Q units nor a reduction in PaO_2.[14]

IMMERSION PULMONARY OEDEMA.

Immersion pulmonary oedema is a syndrome in which divers develop dyspnoea and cough productive of pink, frothy sputum shortly after beginning a dive.[18-20] Initially it was believed to occur only in cold water, which supported the observation that the normal increase in forearm vascular resistance upon cold exposure was exaggerated in affected individuals, several of whom subsequently developed hypertension.[18] However, the syndrome can also develop in warm water, and cold exposure does not always cause an exaggerated increase in forearm vascular resistance.

Although the cause is not fully understood there are several possible factors which could promote pulmonary oedema. The increase in pulmonary vascular pressures secondary to blood redistribution from the periphery to the central compartment is enhanced by exercise,[7] and probably also by cold induced peripheral vasoconstriction. It has been suggested that this increases airway resistance, which then augments the effects of dense gas breathing (see below) and the effect of external breathing resistance. During inspiration, when the intrathoracic pressure is more negative than usual, the left ventricular transmural pressure required to eject blood (afterload) is increased. A higher afterload on the left ventricle, when the pulmonary vasculature is already engorged due to immersion, could perhaps precipitate a critical increase in pulmonary venous, and hence capillary, pressure. This mechanism has been implicated in negative pressure pulmonary oedema during emergence from general anaesthesia[21] Finally, high vascular pressure in conjunction with elevated pulmonary blood flow has been hypothesised to cause direct endothelial damage and capillary leak due to high shear stress.[22,23]

The effects of increased gas density.

Density and viscosity are primary determinants of the resistance to gas flow through a pipe. While gas viscosity is not significantly altered by pressures within the range of human diving, there is a linear increase in gas density with ambient pressure. The theory of constant flow in an infinitely long tube predicts that resistance increases in direct proportion to density. Measurements in divers indicate that airway resistance is greater during expiration than inspiration, and increases approximately in proportion to the square root of the density.[24,25]

Under normobaric conditions exercise is typically limited by the functional capacity of the cardiovascular system. However, at higher barometric pressure, and hence gas density, exercise may be limited by the ability to move gas into and out of the lungs. One way of quantifying the effect of increased gas density on pulmonary capacity is to measure the maximum voluntary ventilation (MVV). This represents the total amount of gas per minute that can be voluntarily moved in and out of the lungs with maximal effort. MVV has been measured systematically over a range of depths and gas densities and its relationship to ambient pressure (in atmospheres absolute) can be described as follows (Fig. 3):

$$MVV_{ATA} = MVV_0\rho^{-k}$$

where: MVV_{ATA} = maximum voluntary ventilation at depth (measured as pressure in atmospheres abolute)

MVV_0 = MVV at the surface

ρ = gas density (g/l)

k = constant (0.3-0.5)

The mechanical effects of dense gas have been vividly illustrated by Drs Larry Wood and Charles Bryan, who performed isovolume pressure-flow measurements on themselves breathing air at the surface and at equivalent depths of 30 and 90 meters in a chamber (Fig. 4). At depth expiratory flow limitation occurs at lower transmural pressures and higher volumes when compared with surface controls. Since maximum expiratory flow is determined

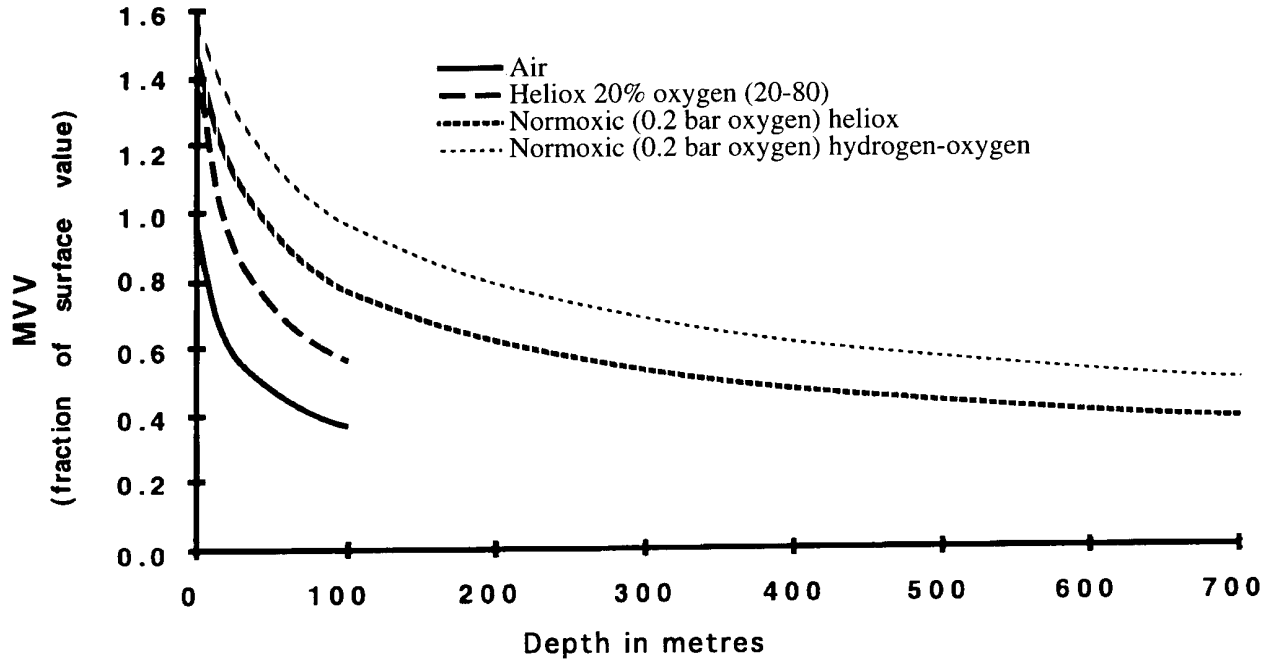

Figure 3. Effect of dive depth and breathing gas on maximum voluntary ventilation.

Air and heliox 80-20 are not shown deeper than 100 m as use of these breathing gases at deeper depths is limited by oxygen toxicity.

primarily by the elastic recoil pressure of the lung, this experiment illustrates the breathing strategy necessary to maximise ventilation at depth: increase lung volume and shorten inspiratory time to allow maximum time for exhalation. Expiratory flow-volume curves were recorded by the same investigators (Fig. 5).[26]

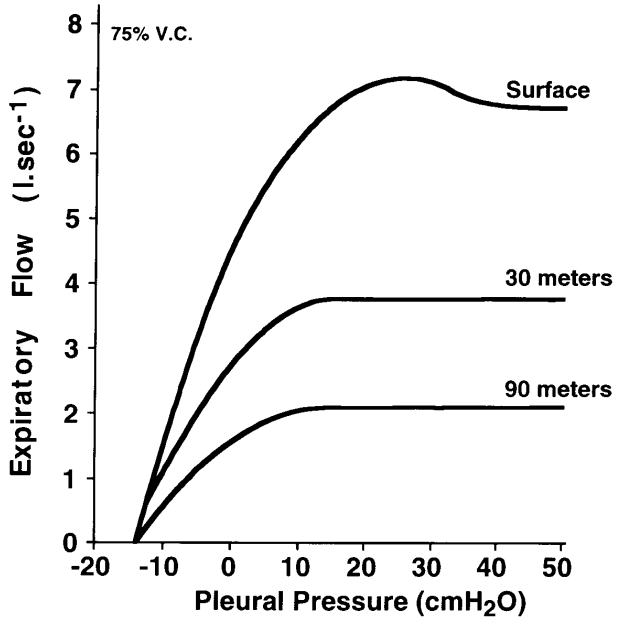

Figure 4. Isovolume pressure-flow relationship in one subject breathing air in a dry hyperbaric chamber as a function of depth (at 75% vital capacity).

There is a progressive fall in maximum expiratory flow, which remains constant at pleural pressures greater than 10-20 cm H_2O. Flow in this region is limited by dynamic airway compression, and can be explained by wave speed limitation.[69] Data shown are from Wood and Bryan.[70]

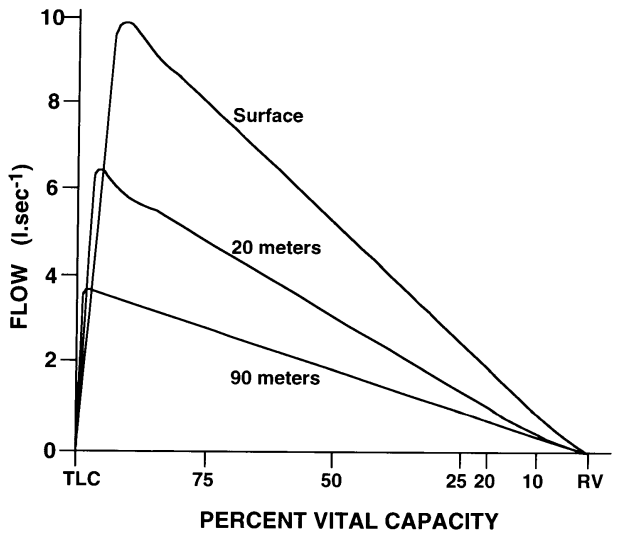

Figure 5. Expiratory flow-volume curves at various depths in a dry chamber breathing air.

Peak flow and the slope of the linear portion of the curve are highly dependent upon gas density. The data indicate that at least some density dependent (turbulent) flow exists in the lung under almost all conditions. Data from Wood and Bryan.[26]

Maximum expiratory flow rate and lung conductance (G, the reciprocal of resistance), have been measured over a range of gas densities (Figs. 6 and 7).

$G \propto \rho^{-c}$

where:

c is a constant (0.39 during tidal breathing and 0.47 during hyperventilation).

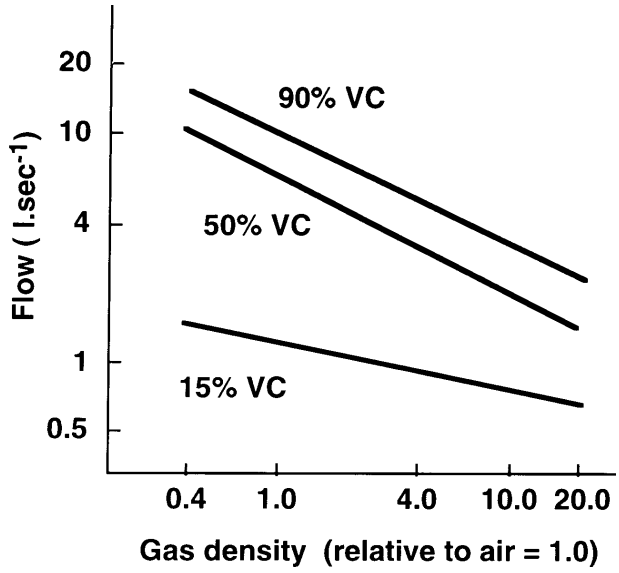

Figure 6. Maximum expiratory flow as a function of gas density and lung volume.

Data from Anthonisen.[24]

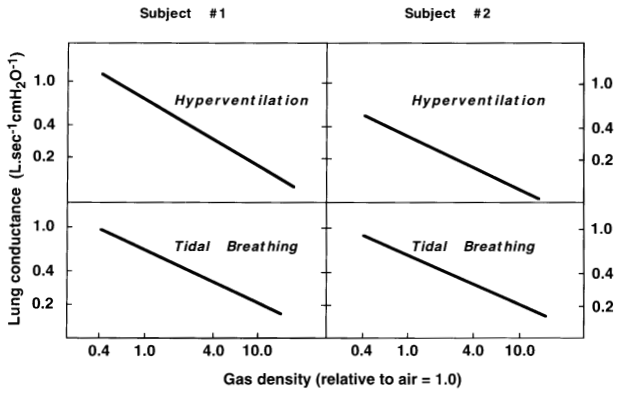

Figure 7. Lung conductance as a function of density in two individuals.

The lower panels represent inspiratory conductance during hyperventilation. During tidal breathing conductance (G) was proportional to $r^{-0.39}$; during hyperventilation G was proportional to $\rho^{-0.47}$, where ρ = gas density. The authors hypothesized that during hyperventilation flow was more turbulent and hence dependent on gas density to a greater degree. Data from Anthonisen.[24]

Resistance during inspiration is typically lower than it is during expiration (Fig. 8), suggesting that in order to achieve maximum ventilation (or minimise resistive work of breathing) a diver should use a short inspiratory time and breathe at a high lung volume.

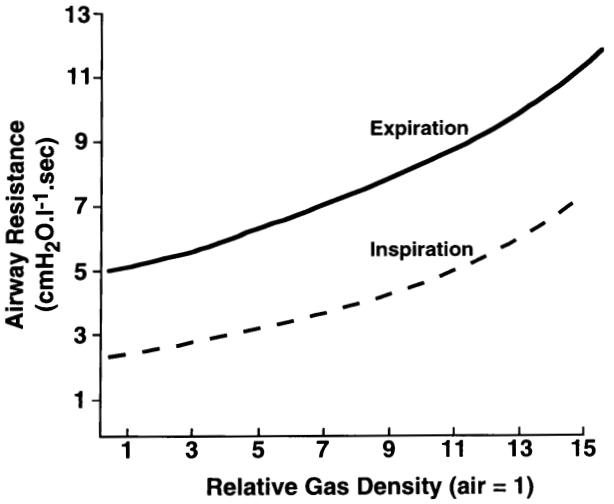

Figure 8. Inspiratory and expiratory flow resistance as a function of gas density during voluntary hyperventilation.

Because of increased airway calibre during inspiration, inspiratory resistance is less than expiratory resistance. These data indicate that maximum ventilation is highly dependent upon the ventilatory strategy chosen by the diver. A short inspiration and prolonged expiration, carried out at high lung volumes will minimise the mechanical load imposed by high breathing resistance. Data from Vorosmarti.[25]

At the surface the maximum exercise ventilation is typically about half of the MVV. At 20 m depth (3 bar or ATA) breathing air, MVV is reduced by about 35 percent; at 40 m the MVV is reduced to about 50 percent of its surface value. Thus maximum exertion at depths in excess of 40 m is likely to be associated with relative hypoventilation as the ventilation required to eliminate metabolically produced CO_2 exceeds the maximum possible ventilation. This analysis tends to underestimate the predicted maximum exercise rate because maximum ventilation during exercise is approximately 10% higher than at rest,[27] and the respiratory control mechanism in exercising divers allows their arterial PCO_2 to rise.[1,28-30] On the other hand these factors may be offset by the increase in physiological dead space (see below).

However, the MVV may be an inaccurate predictor of maximum exercise capacity. The short term MVV does not require sustained respiratory muscle effort, as does the increase in ventilation required for exercise, and the

maximum sustainable ventilation is only about 50% of the 15 second MVV.[31,32] Stolp[33] and Shephard[34] attempted to predict maximum exercise ventilation as a function of sustained ventilatory capacity (SVC: sustained isocapnoeic MVV >3 minutes in duration) at high gas densities and found that when exercise ventilation exceeded 45-60% of SVC there appeared to be a respiratory limitation to exercise.

During diving exercise ventilation tends to be lower than at the surface, which can contribute to hypercapnoea.[29,35] While it would appear self evident that this is due to high airway resistance, some evidence suggests that it is ambient pressure rather than density that predicts hypercapnoea. Salzano, during simulated chamber dives at depths up to 650 m, actually observed higher ventilation during moderate exercise (see Fig. 9).[1]

While airway resistance may play a major role in determining ventilatory performance during diving, one must not forget the additional resistance that may exist because of the breathing apparatus. Warkander et al reported that adding external breathing resistance to divers exercising at 58 m resulted in elevation of end-tidal PCO_2 ($P_{ET}CO_2$) to 72 mm Hg. At the end of the exercise $P_{ET}CO_2$ was >90 mm Hg and loss of consciousness ensued.[36] Under resting conditions in healthy individuals $P_{ET}CO_2$ is an accurate reflection of arterial PCO_2, however during exercise $P_{ET}CO_2$ tends toward mixed venous PCO_2 levels, and it thus may exceed arterial PCO_2.[37] The relationship between end-tidal and arterial PCO_2 in diving, where there may be additional factors such as V_A/Q mismatch and impaired gas diffusion, is unknown. To date there are no published data directly comparing the two values during diving exercise.

Gas Phase Diffusion Impairment

At 1 ATA intra-alveolar diffusion of CO_2 and O_2 is believed to occur sufficiently rapidly that diffusion equilibrium occurs within each breath.[38] However, diffusion within the gas phase is slowed as gas density increases and it has been speculated that during diving this might result in impairment of CO_2 and O_2 exchange, resulting in hypercapnia and hypoxaemia. The Bohr dead space is calculated using the standard formula below (Bohr equation):

$$V_D = V_T \left[1 - \frac{P_E CO_2}{P_A CO_2} \right]$$

where: V_D = dead space
V_T = tidal volume
$P_E CO_2$ = mixed expired CO_2
$P_A CO_2$ = alveolar PCO_2

The Enghoff modification of the Bohr equation is to assume that $P_A CO_2$ = arterial PCO_2.

Direct measurement of arterial blood gases during experimental dives has revealed hypercapnia, which may be due to hypoventilation[35] or reduced efficiency of pulmonary CO_2 transport as measured by an increase in dead space/tidal volume ratio (shown in Fig. 9).[1,39]

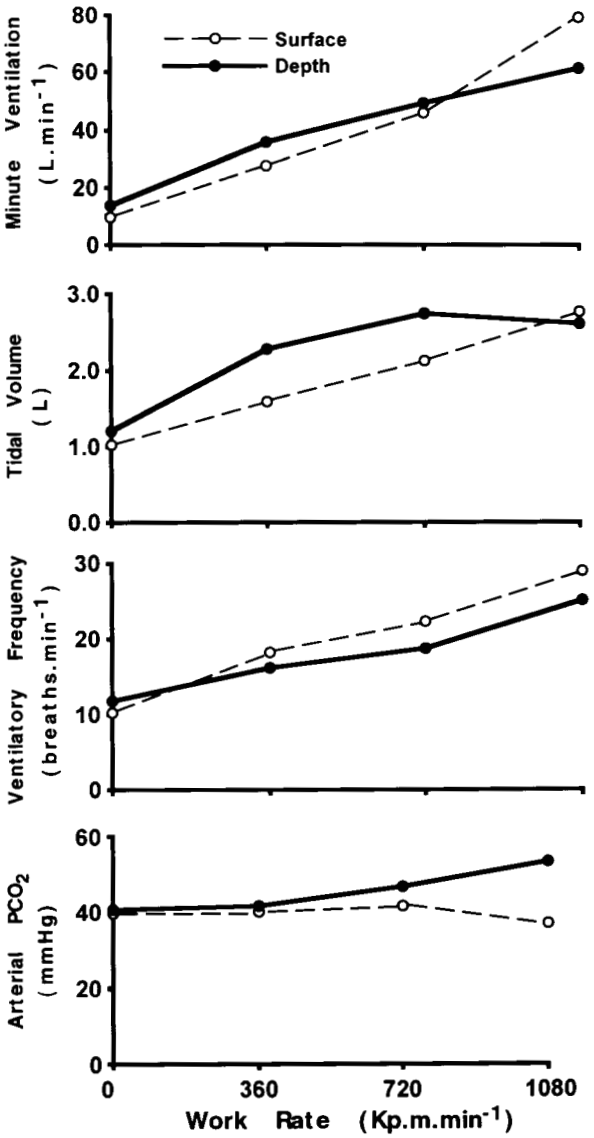

Figure 9. Exercise ventilatory response and arterial PCO_2 during bicycle exercise in a series of deep diving experiments.

Plotted data are mean values from 5 volunteers participating in experimental saturation dives to 460 and 650 m. Gas density at depth was 7.9-17.1 g/l. At rest and during moderate work loads, arterial PCO_2 at depth was maintained close to surface values, but because of greater physiological dead space a higher minute ventilation is required. At the highest work load minute ventilation approaches MVV and cannot increase further resulting in hypercapnoea. At depth the divers in this study demonstrate the typical breathing pattern of individuals with high airway resistance, higher tidal volume and lower breathing frequency. Data are from Salzano.[1]

Observations suggesting O_2 exchange impairment were made by Chouteau,[40] who noticed that goats in a chamber breathing normoxic heliox (atmospheric PO_2 = 0.22 bar or ATA) at 71-91 bar (700-900 m equivalent depth, 11-16 g/l) became ataxic and lost their footing. Increasing the chamber PO_2 reversed the situation, until, at 101 ATA (1000 m, 16.8 g/l), one animal died despite increasing the PO_2 to 0.9 ATA. Chouteau believed that this was due to impaired O_2 diffusion, and this was later referred to as the "Chouteau effect". Initially, in deep diving exposures using heliox in which humans experienced psychomotor impairment, nausea, vomiting and tremor it was suspected that hypoxia might be responsible. However, it became apparent that these symptoms, which were related to both ambient pressure and rate of compression, and became known as the high pressure nervous syndrome (HPNS), were more likely due to neuronal membrane effects and alterations in neurotransmitters unrelated to the PO_2.

Paradoxically, Gledhill observed a reduction in alveolar-arterial PO_2 gradient in subjects breathing SF_6.[41] Except in one study of three individuals, in which alveolar-arterial PO_2 difference at rest increased 2-3 fold at 300 m (heliox, PO_2 = 0.28 bar, inspired gas density 5 g/l),[42] direct measurement of arterial PO_2 in experimental dives has revealed either a reduced A-a gradient at a gas density of 3.2 g/l[39] or no significant change (up to 17 g/l).[1,35,43] Lambertsen reported a deep dive in which the ambient PO_2 in the breathing gas (up to 25.2 g/l) was maintained at 0.21 bar. Although arterial PO_2 was not measured, subjects did not report any difficulties which might have been attributable to hypoxia.[2] Actual measurement of arterial PO_2 at an inspired gas density slightly higher than in the Chouteau experiments, with an inspired PO_2 of 0.5 bar, revealed values of 200-300 mm Hg, even during exercise.[1] The Chouteau effect was therefore probably due to some phenomenon other than hypoxia, possibly HPNS. Interestingly, despite greater than adequate arterial PO_2 values, subjects in Salzano's study had significantly higher arterial lactate levels, an observation consistent with reduced O_2 delivery to exercising muscle.[1]

The issue of gas phase diffusion impairment during diving therefore remains an open one. Certainly if there is diffusion limitation to pulmonary O_2 transport it is of minor importance, at least in divers with normal lungs. Since most diving is performed using breathing mixtures with a fixed proportion of O_2, a built in safety feature during descent that will tend to offset possible problems with pulmonary O_2 exchange, is the rise in inspired PO_2 in parallel with the increase in gas density.

Although the observed elevation in Bohr dead space is consistent with gas phase diffusion limitation, there are also other explanations. An elevation in anatomic dead space due to a breathing strategy in which breathing occurs at higher lung volumes could contribute. Impaired gas distribution, causing V_A/Q mismatching, could also contribute to a higher dead space. Finally, pressure-induced dysfunction of macromolecules facilitating gas transport, such as the enzyme carbonic anhydrase, may cause arterial PCO_2 to exceed pulmonary end-capillary PCO_2 ('blood phase diffusion impairment'), thus simulating gas phase diffusion impairment and similarly elevating measured dead space.

Perhaps the ultimate experiment to assess diffusion of gases in the medium of highest conceivable gas density (1,000 g/l) was performed by Dr Joannes Kylstra. Studies in humans during therapeutic lung lavage, and one volunteer, in whom one lung was filled with saline while the other was ventilated with 100% O_2 revealed only small differences between PCO_2 values in end-tidal expired saline and arterial blood.[4,5] Given the experimental conditions of low CO_2 elimination rate and extremely slow exhalation (<3 breaths per minute) these data were consistent with complete diffusive equilibrium between alveolar liquid and end-capillary blood.

Effects of Decompression on the Lung

BAROTRAUMA

Pulmonary overpressurisation during decompression results from breath holding or bronchial obstruction and distal air trapping. The most common manifestation is mediastinal emphysema; less common are pneumothorax and gas embolism. An intrapulmonary pressure exceeding 60-80 mm Hg is sufficient to cause pulmonary damage.[44,45] This pressure differential can occur if a diver takes a full breath of compressed gas and then ascends from a depth as shallow as 1-1.5 m. AGE has indeed been reported after a dive to one metre depth[46] and in scuba divers breathing compressed air near the surface while being washed over by large waves. It has also been observed in commercial divers exposed to underwater explosions.

The numerous instances of pulmonary barotrauma (PBT) not associated with breath holding have led to hypotheses regarding regional bronchial obstruction. Dahlback and Lundgren[12] have demonstrated that immersion induces intrapulmonary gas trapping, due in large part to the increase in central blood volume.[47] Forceful exhalation during ascent from a dive might therefore generate pulmonary barotrauma. It is possible that the physiological effects of immersion may be at least in part responsible for the relatively common occurrence of pulmonary barotrauma in divers in contrast to its extreme rarity in the dry chamber environment during decompression from hyperbaric oxygen therapy.

The effects of immersion to induce gas trapping may be compounded by lung pathology. Autopsy on a submariner who died during submarine escape training

revealed obstruction of the right middle lobe due to focal bronchial obstruction from a calcified lymph node.[48] Diffuse airways obstruction due to moderately severe asthma has been associated with decompression illness,[49,50] and has traditionally been a contraindication to diving.[51] However, an international panel reached the consensus that individuals with asthma in whom pulmonary mechanics can be rendered normal (including after a provocative test) by pharmacotherapy are probably not at substantially increased risk of DCI or PBT.[52] Individuals with focal air trapping due to cysts or bullae are probably at risk of pulmonary barotrauma and AGE.[53,54]

Colebatch et al. have demonstrated that divers with a history of AGE have less distensible lungs and increased recoil pressure than control divers.[55,56] In one diver, in whom spontaneous mediastinal emphysema had occurred when performing breath hold diving, after a maximum inspiration, transpulmonary pressure exceeded 70 cm H_2O, a pressure which is close to the level demonstrated to cause pulmonary rupture. The authors speculated that stiff airways may cause stress magnification at high lung volumes (i.e. during greatest stretch). It has been demonstrated that restricting lung expansion with an abdominal binder may protect against pulmonary barotrauma.[45,57] It is therefore not the increase in pressure that produces pulmonary barotrauma, but rather the stretch. It has therefore been suggested that during decompression from a dive, breathing at either high or low lung volumes should be avoided.[58,59]

EFFECTS OF VENOUS GAS EMBOLI (VGE).

During decompression, VGE are extremely common, occurring in a large proportion of scuba divers engaged in single[60] or repetitive dives.[61] A short lived decrease in carbon monoxide transfer factor (D_LCO) and arterial PO_2 occurring in parallel with the appearance of VGE have been described after a bounce dive to 55 m.[62] Hlastala demonstrated that intravenous infusion of gas in experimental animals caused an increase in high V_A/Q gas exchange units,[63] and Ohkuda et al. demonstrated in sheep that this can result in capillary leak and pulmonary oedema.[64] High levels of VGE during decompression from a dive can also produce pulmonary oedema in humans (cardiorespiratory decompression illness or "chokes").[65]

A group in which repetitive or continuous VGE have been observed is divers decompressing from saturation dives. Indeed, several reports have demonstrated that, after decompression, saturation divers have elevated respiratory dead space and reduced DLCO to a degree that correlates with a cumulative measure of VGE.[66,67]

Effects of Inspired Gases on the Lung

The pharmacological effect of a gas is a function of its partial pressure. Therefore, gas mixtures which may not be toxic at 1 ATA can induce lung injury during diving. Oxygen at a concentration of 21%, for example, can become toxic to the lung at ambient pressures greater than 3 bar (20 m) where the PO_2 = 0.6 bar. At that pressure many hours of exposure are ordinarily required, therefore this is not an issue except during saturation diving or during the treatment of decompression illness. Pulmonary O_2 toxicity manifests as substernal burning, a reduction in vital capacity, capillary leak (Adult respiratory distress syndrome or ARDS), and if exposure does not cease, death. Provided the inspired PO_2 is reduced, pulmonary O_2 toxicity is usually completely reversible. A detailed discussion of pulmonary O_2 toxicity by Clark is suggested for more detail.[68]

Conclusions

The lung is exposed to numerous stresses while diving. The lung is the origin of arterial gas embolism, and when large amounts of venous gas embolism are present it is a target organ for decompression sickness. The lung is at risk of injury due to toxic environmental gases. Finally, a testament to the remarkable flexibility of this complex anatomic structure, is the fact that the lung is subjected to gases with properties considerably different from those of ambient air and yet is able to maintain sufficient levels of bulk gas movement and exchange of both O_2 and CO_2.

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INTERPRETATION OF GAS IN DIVING AUTOPSIES

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Key Words

Accidents, bubbles, death, investigations.

Introduction

Recent autopsy protocols for diving fatalities have emphasised the importance of the detection of gas in the body to diagnose cerebral air gas embolism (CAGE), either by erect chest X-ray, CT Scan or by dissection underwater.[1-3]

Boyle's Law states that at a constant temperature the volume of a gas is inversely proportional to the pressure. Cerebral air gas embolism occurs during an uncontrolled ascent without exhalation because the volume of the gas in the lungs expands as the ambient pressure falls, forcing gas into the pulmonary circulation and thence into the cerebral circulation.

Unfortunately, very little critical analysis has been made of the significance of intravascular gas at autopsy. Intravascular gas was detected in 12 out of 13 diving fatalities autopsied at the NSW Institute of Forensic Medicine. In 5 of the 12, the history and autopsy findings did not suggest cerebral air gas embolism. What then is the significance of gas?

Could the gas be artefactual?

Forensic pathologists have long recognised that the process of decomposition causes gas formation. Bacteria proliferate in the dead body, particularly in the blood vessels, breaking down blood and tissues and generating gas in a process of putrefaction. If decomposition was responsible for the intravascular gas then this gas should also be seen in non-diving fatalities.

Resuscitation, using endotracheal intubation, positive pressure ventilation and intravenous cannulation, can cause subcutaneous emphysema and even air emboli. Eight out of 13 of the divers were subject to vigorous resuscitation. If resuscitation was responsible for the intravascular gas then it should also be present in non-diving fatalities.

Finally, at increased pressure the body absorbs nitrogen. Normally during ascent nitrogen diffuses out of the tissues and is breathed out, part of the process of decompression. However, if death occurs at depth and the body is brought rapidly to the surface, nitrogen bubbles will evolve in blood vessels and in soft tissues and are not removed because the circulation has stopped. Decompression would appear to be capable of generating intravascular gas in diving fatalities, either during or after death.

Methods

All diving fatalities in NSW are autopsied at the NSW Institute of Forensic Medicine. In the cases presented here erect chest and abdominal x-rays were taken before autopsy. Autopsies were commenced as soon as possible after death, however there were often delays in transporting the body. The body was positioned with a block under the upper back so that the chest was the highest point. The chest was opened first taking care not to cut the superficial veins of the neck. Gas was aspirated from the heart, using a Hamilton "gastight" syringe (Hamilton Company, Reno, Nevada 89502, USA).

The inferior vena cava and portal vein were opened once the block was removed from under the body. Air aspirated was analysed by the Department of Mineral Resources, Lidcombe. The diving equipment was examined and tested by NSW Police Divers, Sydney Water Police. Where dive computers were used they were down loaded and the dive profiles recorded. Air from the tanks was also tested by the Department of Mineral Resources.

Results

Twelve of 13 diving fatalities had intravascular gas.

The time between death and post mortem varied from 8 hours to 5 days. The average time was 41 hours. Two bodies were recovered from a wreck at 51 m after being missing for 3 days. If these two cases are excluded, the average delay to post mortem was 26 hours, still a significant delay.

In all 12 of the diving fatalities gas was present in the heart, neck veins, inferior vena cava and portal/hepatic veins, often with as much as 100 ml of gas in the right