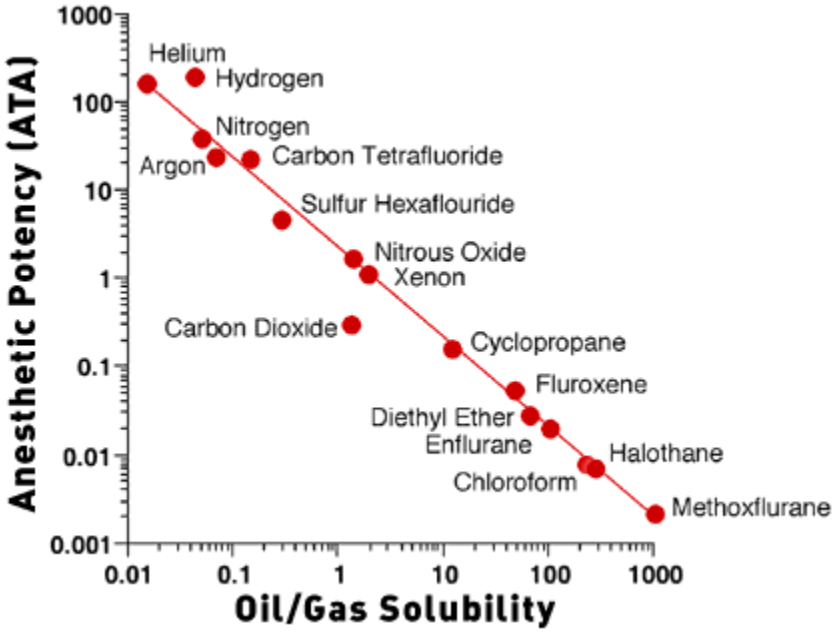
**CARBON DIOXIDE, NARCOSIS,
AND DIVING**

BY JOHNNY E. BRIAN JR., M.D.

Carbon dioxide (CO_2) is the gaseous end product of the aerobic metabolism of oxygen. CO_2 is highly soluble in body tissues, and readily diffuses from cells to blood, where circulation transports it to the lungs for elimination. Divers often ignore carbon dioxide, as CO_2 is a normal part of life. However, CO_2 may have definite and detrimental effects if a diver accumulates an excessive amount of CO_2. Understanding how CO_2 can become elevated, the symptoms, and the consequences of elevated CO_2 can only make us safer divers.

Air contains only 0.03% CO_2; therefore, under normobaric conditions, air inspired into the lungs is almost devoid of CO_2. This creates a large difference in the partial pressure of CO_2 (PCO_2) between blood and inspired air, promoting CO_2 to diffuse rapidly from blood into the gas phase of the lungs. At rest, ventilation is controlled by the PCO_2 in the ventilatory control center of the brain. The nervous system adjusts ventilation to maintain arterial blood PCO_2 ($PaCO_2$) constant, which at rest ranges from 35-45 mmHg (average 40 mmHg). Venous blood entering the lungs has a CO_2 partial pressure ($PvCO_2$) approximately 5 mmHg higher than arterial blood, or 45 mmHg. Because CO_2 is very soluble in blood, a large volume of CO_2 exists in a dissolved state in blood. This means that to lower blood PCO_2 any given amount, a large amount of CO_2 must be removed. As CO_2 diffuses into the gas space (alveoli) of the lungs, an equilibrium is established when the alveolar gas phase partial pressure of CO_2 ($PaCO_2$) and blood PCO_2 reach 40 mmHg. The volume of gas breathed per minute (minute ventilation) controls removal of CO_2 from the blood perfusing the lungs. When CO_2 production increases during exercise at 1 ATA, minute ventilation also increases to maintain $PaCO_2$ constant. With severe exercise at 1 ATA, $PaCO_2$ may decrease slightly. During exercise, if minute ventilation does not increase to match the increase in CO_2 production, then arterial PCO_2 will increase.

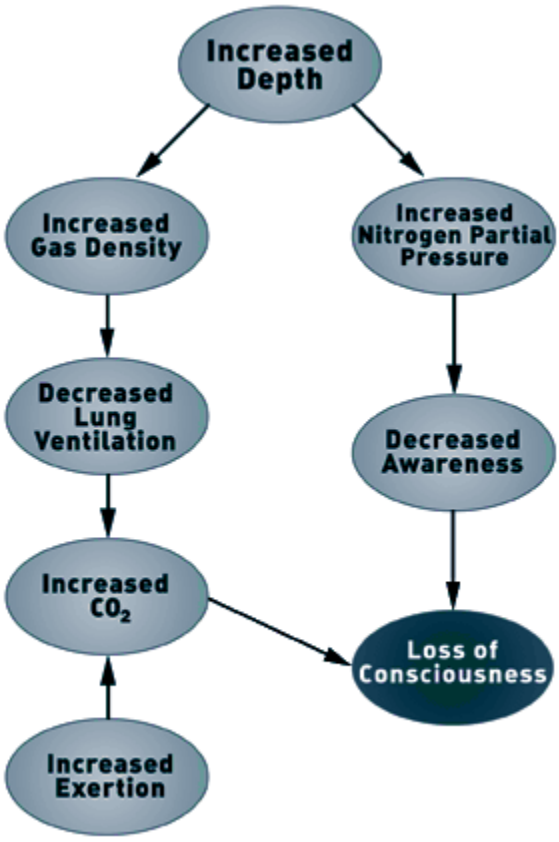
Carbon dioxide is a narcotic gas capable of depressing awareness to the degree of total loss of consciousness. In humans, acute elevation of arterial PCO_2 above 70-75 mmHg reduces the level of awareness (20), and $PaCO_2$ above 100-120 mmHg produces unresponsiveness (26). Severe elevation of $PaCO_2$, by inhalation of 30%-40% CO_2 (220-300 mmHg), produces surgical anesthesia in both animals and humans (14,25). In dogs, an arterial PCO_2 above 250 mmHg results in a state of general anesthesia (2). Carbon dioxide has not been useful as a general anesthetic, as severe elevation of PCO_2 produces marked derangement in acid-base balance. In addition, anesthetic levels of CO_2 produce seizures in both animals and humans (1, 14, 25).



Carbon dioxide is 25 times more lipid-soluble than nitrogen, and lipid solubility has been correlated with the narcotic potency of gases. Figure 1 is a plot of oil/gas solubility versus anesthetic potency of gases and inhaled anesthetics. These gases fall along the line that indicates a high degree of correlation between lipid solubility and anesthetic potency. Xenon and nitrous oxide have approximately the same lipid solubility as CO2. If the anesthetic effect of CO2 was produced only by lipid solubility, then the CO2 point should lie along the line with the nitrous oxide and xenon points. CO2, however, falls below the line, which means that anesthesia is produced by a lower partial pressure of CO2 than would be predicted from the lipid solubility. The anesthetic potency of CO2 is about 130 times that of nitrogen, much greater than the ratio of lipid solubilities of CO2 and nitrogen. This suggests that CO2 produces an anesthetic effect independent of lipid solubility.

Elevation of CO2 has been associated with a decreased level of consciousness during both hyperbaric chamber and wet dives. Case and Haldane used inspired CO2 to elevate arterial PCO2 at 1 ATA, and during hyperbaric exposures to 300 FSW, in human volunteers (1). Most data reported in this study are subjective impressions; therefore, the objective measurements are limited. However, the paper is a fascinating report of early diving research, including a description of spinal bends in Haldane after a He/O2 dive (1). At 1 ATA, 6%-8% (45-60 mmHg) inspired CO2 produced a marked increase in respiration, but little change in mental or physical skills (1). Although Case and Haldane did not measure arterial PCO2, it was most likely less than 80 mmHg under the conditions at 1 ATA. The exposures were then repeated after compression to 300 FSW. The subjects noted that there was much less increase in ventilation during inspired CO2 at 300 FSW. This suggests that the subjects were unable to increase minute ventilation to an equal level as during the 1 ATA exposure. One can only deduce that the increase in arterial PCO2 was more severe at 300 FSW than at 1 ATA. At 300 FSW during CO2 inspiration, there was severe impairment of both mental and physical skills. Subjects

noted that the "narcosis" was much more severe than with exposure to air alone at 300 FSW. When inspired CO2 was increased to a level of 0.8%-0.9% at 300 FSW (which equals 8-9% at 1 ATA; 60-70 mmHg), subjects quickly lost consciousness and some seized. Subjects were described as lapsing into unconsciousness "quietly and easily" Although PCO2 was not measured, arterial PCO2 was likely greater than 80 to 100 mmHg at 300 FSW. Case and Haldane theorized that the respiratory response to CO2 was suppressed by nitrogen narcosis.



Warkander et al. studied CO2 accumulation during exercise at 6.8 ATA, and reported 2 subjects that required rescue from a wet pot due to severe CO2-induced incapacitation (24). Both subjects had elevation of arterial PCO2 above 80-90 mmHg, and both were unaware of their incapacitation. In the same study, other subjects continued to function with similar elevation of arterial PCO2. This suggests that CO2-induced depression of awareness may vary greatly between individuals.

Carbon dioxide reduces mental and physical capacity at sub-anesthetic concentrations. Hesser et al. studied the effect of increased CO2 in volunteers under normobaric and hyperbaric conditions (6,7). They found that a modest increase of PCO2 to 50-60 mmHg significantly reduced the ability to perform mental skills such as arithmetic and color naming, as well as physical skills, such as manual dexterity and eye-hand coordination. They concluded that the effect of CO2 was additive to, but not synergistic with, nitrogen narcosis. Fothergill et al. also studied the effect of PCO2 elevation to 50-60 mmHg on a

battery of mental tests in volunteers, reporting that the modest increase in PCO2 reduced the number of correct responses principally by reducing the number of attempts at the tests (4). This suggests that increased PCO2 slows comprehension of presented information. The data also suggests that modest elevation of PCO2, which may occur during diving, may contribute to "narcosis" independent of elevation of PN2.

Although Case and Haldane theorized that narcosis limited the respiratory response to CO2, it is the increase in gas density, and not the narcotic properties of the gas, that limits the ventilatory response under hyperbaric conditions (5,10). As the rate of lung ventilation increases, exhaling becomes an active process, with increased intrathoracic pressure (the pressure inside the chest) increasing the rate of gas flow out of the lungs. Progressive increase in the force of exhalation will increase the gas flow rate, but only up to a point. The airways that conduct gas in and out of the lungs can be compressed and collapsed by pressure on the outside of the airways. The intrathoracic pressure is the pressure applied to the outside of the airways. During forced exhalation, intrathoracic pressure rapidly rises above the pressure at which airways collapse. When the airways begin to collapse, the flow of gas out of the lungs is obstructed. Thus, gas flow is slowed. The maximal possible expiratory gas flow rate occurs when the airways just begin to collapse. This means that the expiratory gas flow rate cannot be increased beyond the point when airways begin to collapse, regardless of how much effort is exerted. Exhalation is frequently termed "effort independent", as forced expiratory effort cannot overcome the expiratory obstruction due to airway collapse.

Under normobaric and hyperbaric conditions, the single factor that limits the ability to increase ventilation is the rate at which gas can be exhaled from the lungs. The ability to exhale gas is reduced during hyperbaric and diving conditions. As gas density increases, increased effort is required to exhale gas (i.e.,it takes more work to move a heavier gas). However, the amount of work that can be generated (the pressure differential) is limited by the collapse of the airways. Airways collapse at the same intrathoracic pressure under normobaric and hyperbaric conditions. This means that to exhale gas, the amount of work is fixed and equal under normobaric and hyperbaric conditions. Moving denser gas with the same amount of work means that airways begin to collapse at a lower expiratory gas flow rate. The result is that the maximal possible lung ventilation per minute is progressively reduced as gas density increases.

Gas	Density gram/liter of gas
Nitrogen	1.1009
Helium	0.1573
Oxygen	1.2572
Neon	0.7930
Argon	1.5696

A common method to measure the respiratory response to CO2 is to allow a subject to breathe CO2 and measure the increase in lung ventilation. Nitrox at 4 ATA attenuates the increase in lung ventilation with inspired CO2; reducing gas density with He/O2 restores the CO2 response to the 1 ATA baseline (10). Breathing air at 4 ATA (99 FSW) reduces maximal expiratory gas flow rate and maximal lung ventilation per minute to one-half that present at 1 ATA (27). The effect on lung ventilation is more marked at greater ambient pressure or with gases of greater density. The ability to increase ventilation and eliminate CO2 during exertion may be significantly limited by increased gas density. Thus, maintenance of a normal PaCO2 may not be possible when breathing dense gas.

Elevated CO2 is normally a potent respiratory stimulus and, under normobaric conditions, causes increased respiratory rate (hyperventilation) and the sensation of shortness of breath. Further elevation of PCO2 leads to headache, dizziness, nausea, and eventually a reduced level of consciousness. Similar symptoms occur during diving and hyperbaric exposure, although some have reported that the sensations of hyperventilation and shortness of breath may not be noted (24). It is possible that, during diving, CO2-induced dizziness could be mistaken for nitrogen narcosis. Although increased CO2 is normally a potent respiratory stimulus, elevation of PCO2 to levels associated with a decreased level of consciousness (100- 200 mmHg or greater) progressively depresses respiration (18, 19). Thus, severe elevation of PaCO2 will cause further CO2 retention by reducing lung ventilation.

Gas density is a critical element in the respiratory response to exertion at depth. Table 1 lists densities of diving gases, while table 2 lists the composite densities of diving mixes. By summing the fractional gas densities of a mix, and then multiplying it by depth in ATA, the density of the mix can be calculated. Air at 99 FSW, 32% nitrox at 99 FSW, 16/55 at 200 FSW, and 10/70 at 300 FSW all have approximately the same density. The effect of these mixes on the ability to breathe and eliminate CO2 should be very similar. Oxygen is slightly denser than nitrogen, so substitution of oxygen for nitrogen slightly increases mix density relative to air.

At rest, while breathing nitrox at 4 ATA, PCO2 is normal, indicating adequate ventilation to eliminate CO2 (10). During exertion, however, the increase in lung ventilation is less than occurs at 1 ATA, and PCO2 rises to a significantly higher level than during exercise at 1 ATA (5,10). When lung ventilation approached the maximum possible at a given gas density, PCO2 must increase. The response of ventilation and PCO2 to exercising while breathing dense gas has been tested a number of times (3,10,11,22,27). These studies were directed more at commercial diving conditions, with short periods of exercise (minutes) and high levels of exertion. In addition, these studies were conducted under optimal respiratory conditions, with subjects breathing from very low resistance gas circuits. Because of the conditions of these studies, they are less helpful in determining allowable exercise levels in technical and cave diving, where exertion is less but over a longer period of time. In addition, in-water divers breathe from demand valve regulators, which may impose additional work when breathing.

When breathing air under optimal respiratory conditions at 4 ATA (or gas of equivalent density), the maximal possible ventilation for a very short time period (maximum voluntary ventilation) is 3 to 3.5 ft3/min (10,27). During exertion, lung ventilation can usually be sustained at 75% of the maximal voluntary ventilation (15), which would translate into 2.7 ft3/min with a low resistance breathing circuit. Real-life in-water diving is usually conducted under less than optimal conditions. It should therefore be expected that the maximal possible minute ventilation would be less. Divers swimming at 50-60 ft/min require a ventilation rate of about 0.6 ft3/min or less (13). Experience indicates that breathing less than 1 ft3/min of gas during technical and cave diving is tolerated without symptoms of CO2 accumulation. However, as gas consumption increases above 1 ft3/min, especially as gas consumption approaches 2 ft3/min, there is increased likelihood of CO2 accumulation and resultant deleterious effects.

A number of studies have reported that divers have an abnormal respiratory response to CO2 (8,9,12,17). Lanphier reported that US Navy divers swimming at about 75 ft/min exhibited abnormal elevation of PCO2 that averaged 55 mmHg (12,13). The study-subjects were hardhat divers, in whom inadequate helmet ventilation often causes CO2 rebreathing. These divers were later exercised at 1 ATA, where they also exhibited marked and abnormal elevation of PCO2 (12). Lanphier theorized that chronic CO2 rebreathing in these divers led to CO2 insensitivity. However, Kerem et al. studied open circuit scuba divers, and also reported a reduction of the respiratory response to CO2; (8)Sherman et al. (21) reported similar findings. These findings suggest that chronic CO2 rebreathing is not required for a diver to develop a depressed respiratory response to CO2. The depressed respiratory response to elevation of CO2 appears to vary greatly between individuals, with some divers being normal and other having a very depressed CO2 response (12, 16). Divers may consciously reduce their rate of ventilation to conserve gas, which would lead to CO2 accumulation. Because most diving mixes are relatively hyperoxic, hypoxia with reduced ventilation is unlikely. Lanphier et al. attempted to develop a normobaric screening test to identify individuals with reduced respiratory response to CO2. Unfortunately, only testing under hyperbaric conditions was successful (13). The existence and prevalence of impaired CO2 response in cave and technical divers is not known.

Scuba regulators can add additional resistance to breathing, limiting the ability to eliminate CO2. Almost all studies of respiratory dynamics at depth are conducted under optimal respiratory conditions. There has been very limited study of the effect of demand valve regulators on the ability to breathe at depth. Breathing air at 4 ATA significantly reduces the maximum possible minute ventilation; moreover, the addition of a demand valve regulator causes a very slight additional reduction in ventilation (23). Increasing the resistance of breathing at 4 ATA causes a slight increase in PCO2 during He/O2 breathing (12). However, Lanphier reported that during exertion and air breathing at 7.8 ATA, restriction of breathing rapidly resulted in unconsciousness, most likely due to CO2 retention (12). The overall impact of breathing through a modern, well-maintained scuba regulator on the response of PCO2 to exercise is unknown. Notwithstanding, breathing resistance should be kept to a minimum to reduce the possibility of CO2 retention.

The primary cause for CO2 elevation during diving, then, is exertion coupled with increased gas density. Stress increases the metabolic rate and can contribute to increased CO2 production. Rebreathing expired gas containing CO2 will also elevate PCO2. However, significant rebreathing seems unlikely with standard demand-valve scuba regulators, as they have minimal dead space. Devices with increased dead space, such as communication systems and full-face masks, may elevate CO2 by rebreathing. Rebreathers can also elevate CO2 due to malfunction of the one-way valves or exhaustion of the CO2 absorbent. The ability to perform exertion at 1 ATA should not be used as a guide for exertion at depth, as the ability to ventilate the lungs may be significantly limited by the increased gas density. Divers should monitor themselves and their buddies for signs and symptoms of elevated PCO2. Increased CO2 impairs mental and physical skills and may hamper self-rescue. Severe elevation of CO2 can depress the level of awareness and prevent a diver from recognizing and reversing the process. Divers have become incapacitated and lost consciousness due to CO2 retention without being aware of being in a life-threatening situation. Elevated CO2 also increases the likelihood of hyperoxic seizures.

Mix	Density gram/liter of gas
Air at 1 ATA	1.138
Air at 99 FSW	4.552
32% Nitrox at 99 FSW	4.605
16/55 at 200 FSW	4.310
10/70 at 300 FSW	4.560

If a diver experiences symptoms of elevated CO2, they should stop their exertion and relax, if possible. This will reduce CO2 production, and should allow time for the

ventilation to eliminate the excess CO2. If this is not possible, then the dive should be terminated. Ascent to a shallower depth will be beneficial by reducing gas density and allowing more effective ventilation to eliminate CO2. Incapacitated but breathing divers should also be taken to a shallower depth for the same reason. Elimination of excess CO2 and recovery of consciousness may be possible once gas density is reduced.

The Impacts of Smoking on Diving

BY ART RANZ, DDS

Cigarette smoking is one of the largest preventable health and death risks in the United States. It receives enormous amounts of negative media attention and yet millions of people start smoking every year. Unfortunately, it is frequently difficult to have a prudent, scientific discussion about the risks of smoking with someone who is addicted to nicotine. The addiction leads smokers to rationalize or deny the risks of smoking. However, this "head in the sand" response allows them to ignore the obvious impact that smoking has upon their bodies and the more subtle ways it effects many aspects of their lives, such as scuba diving.

The effects of smoking are especially significant for persons who participate in scuba diving. A review of scientific literature about the body's reaction to smoking and nicotine addiction illustrates how smoking can effect diving performance. While the diving and health limitations imposed by tobacco use vary according to the degree of use, tobacco always has some impact on individual health.

The most extensive, long-term, prospective study on smoking and other health issues is the Framingham study. This ongoing study has followed 5,000 people for more than 34 years, providing a wide range of statistical information. For instance, the 30-year-old who smokes 15 cigarettes a day - or less than one pack - shortens his life by five years. Smokers experience a 20-fold increase in lung cancer and greatly increased cancer rates in other organs, including skin, bladder, pancreas, mouth and throat. Smokers have twice the risk of cardiovascular disease, 2.2 times the number of strokes and 3.5 times more intermittent claudication expressed as leg cramping due to a lack of circulation. At any given age, the risk of dying for any reason is twice that of a non-smoker. Smokers have seven times the normal incidence of airway damage and respiratory distress. Children who smoke beginning at age 14 only develop 92 percent of the lung function, on average, that a non-smoking child does. This loss of function is permanent. Obviously, efficient lung function is essential to managing stressful situations and promoting efficient inert gas removal from a diver's blood. Poor circulatory efficiency can have dangerous impacts on inert gas elimination and oxygen delivery to needy muscles, greatly effecting a diver's personal safety. Atherosclerotic plaques in blood vessels form twice as fast when smoking is added to a high fat diet.

There are great increases in the LDL ("bad cholesterol") that reduces circulatory efficiency and complicates inert gas removal. Inert gas (especially nitrogen) appears to lodge in fatty deposits, creating likely sites for bubble congregation and growth.

Furthermore, 90 percent of patients with infections after spinal surgery are smokers and bone marrow density in men is decreased almost 20 percent and in women 25-30 percent, while the incidence of back pain from a work related injury increases from one in five to one in two for smokers. Hyperbaric bone damage (osteonecrosis) has gained increasing concern among medical professionals as researchers strive to demonstrate the cause of occasional bone degradation. To be sure, reduced bone density due to smoking aggravates the problem and some researchers are suggesting a more careful analysis of the relationship between hyperbaric damage and tobacco smoking.

How does tobacco cause such dangerous repercussions?

There are four groups of dangerous substances present in cigarette smoke:

1. Carcinogens and co-carcinogens are mostly polycyclic aromatic alcohols that directly initiate cancer formation. These affect areas in direct contact with the smoke and also distant organs through absorption into the bloodstream.

2. Irritants cause immediate coughing and broncoconstriction, inhibit cilliary action in the lung and stimulate mucus secretion.

3. Chronic exposure to nicotine induces an increase in the number of nicotinic cholinergic receptors in the brain, causing structural and functional changes in the brain and nervous system. It induces tolerance and physical and psychological changes upon withdrawal. These are classic developments from an addictive drug.

4. Toxic gases are inhaled, including carbon monoxide, hydrogen sulfide and hydrogen cyanide.

Smoking related cancer is tragic, costly and largely preventable, but the direct impact to diving is often less obvious. By way of illustration, the irritants present within smoke induce a chronic inflammation of the alveoli causing the body to produce proteolytic enzymes that eat away at the alveolar wall. Cilia are microscopic hairs that fan and carry harmful particles out of the lung. The irritants present in smoke impede these cilliary actions. With the addition of increased secretions, the lung has now lost a significant part of its defenses from outside agents. Chronic bronchitis develops, making smokers more susceptible to emphysema, viral and bacterial infections. As this process continues over the years and more alveolar damage occurs, there is a loss of capillaries in the walls which causes "ventilation-perfusion abnormalities."

This damaging chain of events leads to a reduction in the area of alveolar membrane available for gas exchange and also to perfusion of unventilated areas and ventilation of unperfused areas. In simple terms, gas exchange is compromised and air (or other gases) is not reaching the blood for exchange. General lung function is often severely compromised in the smoking population as is evidenced by several clinical measurements in the lung. The standard measure of lung function is the forced expiratory volume in one second or FEV1. This is the amount of air that can be exhaled in one second.

The Framingham study showed the FEV1 to be decreased to 80 percent of expected values in smokers. This decrement in lung function creates less efficient ventilation on

exertion and decreases the force of the cough (a vital protective mechanism for the lung) and may indicate a general degradation of lung health. The forced vital capacity (FVC) is another common measure of lung function and measures the amount of air one can expel from a full inhale to a full exhale. On average, smoking reduces FVC by 10 percent in moderate smokers. A 10 percent reduction in vital capacity is a significant indication of lung dysfunction and an obvious deterrent to pulmonary exchange in decompression.

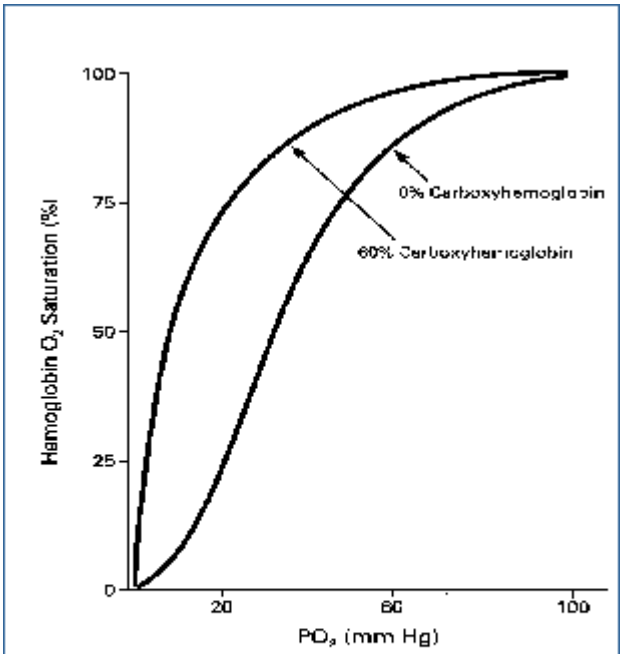


Figure 1. Oxygen–Hemoglobin Dissociation Curve.
The presence of carboxyhemoglobin shifts the curve to the left and changes it to a more hyperbolic shape. This results in a decrease in oxygen carrying capacity and impaired release of oxygen at the tissue level.

Nicotine is not only a powerfully addictive drug, but a potent pharmacological agent. Nicotine promotes platelet aggregation and fibrinogen formation, which are precursors to the clots that obstruct small blood vessels. An obstruction initiates negative repercussions that increases the risk of diving and decompression. The heart rate increases, elevating oxygen consumption and the shrinking of small blood vessels increases total peripheral resistance. The resistance, in turn, causes more problems such as increased blood pressure and poor circulation in the periphery of the body. Peripheral circulation involves the miles of very small blood vessels all over the body. The vessels are problematic in efficient inert gas elimination. For example, the extremities contain numerous areas of reduced circulatory efficiency such as the joints (responsible for the majority of decompression sickness). When divers begin to get chilled, a natural reduction in blood circulation to the peripheral system occurs to maintain a reasonable core temperature. Smoking exacerbates this problem as studies show that the circulation in small blood vessels is reduced 19 percent after just two cigarettes. Poor gas exchange and increased risk of decompression sickness results.

The Problem with Carbon Monoxide

It is important to understand the Oxygen Dissociation Curve when reviewing the impact of smoking on oxygen transport mechanisms. This curve illustrates the assimilation of oxygen in large amounts even with low oxygen pressures in the lungs. Hemoglobin picks up the oxygen from the lungs and transports it to the tissues where it is released. Several factors control how easily the oxygen is released from its hemoglobin carrier. Higher concentrations of carbon dioxide in the blood cause the body to react as if there is poor ventilation and a greater need for oxygen. This environment initiates the release of more oxygen to the tissues. Under these conditions the hemoglobin affinity for oxygen is reduced, making it easier for oxygen to be released. In reference to the Dissociation Curve, this condition is sometimes referred to as a "shift to the right" and results in a greater supply of oxygen to the tissues. However, a "shift to the left" prevents oxygen

from being released to the tissues. This condition is prominent with the carbon monoxide accumulation that results from smoking.

The primary mechanism behind the risk of carbon monoxide impact is twofold. First it binds to hemoglobin 250 times better than oxygen, making a compound called carboxyhemoglobin. This compound replaces the oxygen in the hemoglobin molecule and prevents the leftward shift of the Oxyhemoglobin Dissociation Curve. The increased affinity of hemoglobin for oxygen results in a decrease in oxygen carrying capacity and impaired release of the oxygen once it reaches the tissues. Non-smokers have about one percent carboxyhemoglobin while smokers have close to 15 percent. To illustrate the severely harmful effects of CO in the blood, imagine that an individual has 50 percent of their hemoglobin bound to CO. Compare this individual with another person who has lost half of their hemoglobin (due to severely bleeding ulcers, chronic gastrointestinal bleeding or massive injuries, for instance).The individual who has 50 percent of their hemoglobin bound with CO will die. But, the person who has a 50 percent loss of hemoglobin will still not experience hypoxia while in a resting state.

Furthermore, chronic hypoxia (reduced oxygen) results from the smoking induced impairment of oxygen transport and causes the production of more red blood cells. The red blood cells are the containing mechanism for oxygen transport in the hemoglobin. The Framingham study has shown that smokers have a significant increase in the percentage of red blood cells in the blood (increased hematocrit). Normally the red blood cells are about 35-40 percent of the blood by volume. Smoking can cause this to increase by 20 percent, making the blood much more viscous, inducing obvious complications to efficient circulation. This problem is further aggravated by the pressures found below the surface and causes sludging of the red blood cells in the small capillaries, damaging the cells lining the blood vessels (endothelium).

The transport of hydrogen cyanide to the lungs during smoking creates additional decrements to health and diving safety. This noxious gas directly prevents use of oxygen by the cells by interfering with the cellular engine- the mitochondria. Even small amounts of hydrogen cyanide are deadly. The presence of this toxic substance causes direct injury to the lung by interfering with the alveolar enzymes normally responsible for maintaining the integrity of the alveolar membranes. Hydrogen sulfide is another dangerous substance in cigarette smoke and is a direct toxin to most all cell life, especially to tissues it directly contacts such as the lungs. The numerous impediments to a healthy circulatory and respiratory system establish an insidious cycle of unacceptable risk to safe diving practices.

For instance, when increasing environmental demands require the delivery of more oxygen, the smoker is at a serious disadvantage. An increased supply of oxygen in the inspired air does not help delivery of more oxygen to the tissues where it is needed. There are two ways to increase oxygen delivery with increased demand: increasing blood flow through the tissue and raising the coefficient of oxygen usage. The former is compromised by the inferior cardiovascular condition of the smoker (consider the number of serious atheletes who smoke). The latter is increased by two things that happen automatically: greater partial pressure of oxygen between blood and tissue (resulting from the increase in oxygen consumption in the tissues) and the rise in carbon dioxide as a byproduct of increased metabolism. This increase in carbon dioxide causes the hemoglobin curve to shift to the right and allow more release of oxygen. This

typically beneficial reaction is countered by the smoker's CO poisoning and the shift back to the left. The really adverse effect of smoking is the 20-30 percent rise in peripheral resistance (closing or restriction of small blood vessels) caused by the presence of nicotine. Small blood vessels are where the exchange of gases takes place and a reduction of circulatory efficiency in this area may be significant. Reduced blood flow and impeded oxygen release prevent efficient oxygenation especially when it is needed most. Therefore, the simple act of smoking initiates circulatory reactions that place divers in harm's way. Whether from decompression illness risk or ineffectual response to stressful environments, the smoker intentionally places himself and his team at greater risk.

Understanding Smoking's Short Term Impact on Diving

Smokers and those who choose to dive with them should consider not only the long-term health impacts, but the immediate implications of smoking and diving. Consider the increase in sudden cardiac death, the reduced ability to absorb and deliver oxygen to the cells, the obvious cognitive impairment, the likely increased risks of decompression illness, the increased likelihood of lung overpressurization injuries and the many other dangerous effects of smoking and diving. With all of the damage and risk associated with smoking and diving, what possible justification (save addiction) can there be to continue? Individuals with drug addictions, which is clearly what smoking is, must be encouraged to seek assistance and be freed from this damaging habit.

Consider that many "diving deaths" are thought to be cardiovascular in nature: cardiac arrhythmias, myocardial infarcts and strokes just to name a few. The smoker's incidence of these maladies is much higher. With this in mind, can a smoker be a responsible diving buddy? Can they help other divers out of trouble or are they merely likely to create problems? With increased anxiety, the heart beats faster and the breathing rate increases. Increased heart rate is the number one cause of increased oxygen use by the heart muscle and the heart of a smoker has a reduced ability to deal with the increased demand for oxygen. As a result, pulmonary exchange is poorer and utilization of breathed gases is compromised, leading to greater gas consumption and reduced ability to assist other divers. All dives are decompression dives. The list is long on how smoking causes decreased gas exchange and potential for decompression sickness. The ability of the lungs to filter bubbles is a major reason that every dive does not result in clinical decompression injury. The lungs are directly damaged by smoking. Ventilation, monitored by FEV1, is decreased, and the Forced Vital Capacity, or FVC, is decrease by at least 10%. With decreased pulmonary function, the lungs' function as a big bubble trap is compromised and the risk of decompression illness is increased.

Nicotine causes significant peripheral constriction, further compromising elimination of gas in the areas most difficult to get the inert gases out — the small vessels and the area they perfuse. It causes increased platelet aggregation and fibrinogen production which only gives the body a head start on the same process that bubbles produce in occluding vessels and damaging vessel walls. One prominent theory of decompression illness suggests that bubbles in the bloodstream cause damage to the endothelium, the lining of the blood vessel walls, setting off a cascade of body reactions to repair itself. With nicotine in the body this process is aggravated and accelerated, causing platelets and blood clots to clog the small blood vessels. This reduces the body's ability to get rid of inert gasses. Nicotine gives the body a head start on the bad things that happen with

bubble formation. The smoker has increased numbers of red blood cells per volume, or increased hematocrit, which sounds good, but actually makes the blood "thicker." Increased atmospheric pressure from diving causes sludging of red blood cells in small vessels and the clogging of these vessels is aggravated by the increased hematocrit of the smoker. This is more bad news for perfusing the small vessels in the decompression part of the dive. Increased hematocrit may be directly involved with the endothelial damage which has been implicated in DCS. Carbon monoxide inhibits the transportation of oxygen mostly in its effect upon the hemoglobin and the hemoglobin disassociation curve. Smoking directly reduces pulmonary blood volume and the number of open capillaries in the lung, causing a ventilation to perfusion impairment with the obvious impairment of gas transfer at a time when every little bit is vital.

Acute nicotine withdrawal causes severe performance degradation, memory impairment, confusion, impulsiveness and slowed reaction time, just to name a few. Any of these are serious problems when simple decisions become life or death decisions under water. In a recent study of "undeserved hits" (a dive where supposedly all decompression limits are met and ascent rates are appropriate, but the diver still suffers from decompression illness), smoking and lung damage from smoking seemed to play a key role. Two groups emerged, those with intra-cardiac shunts and those without. Those with shunts had more brain symptoms and none smoked, while those without shunts, 50 percent smoked, a remarkable number. These divers experienced mostly spinal neurological sequelae and had deficits identical to divers with rapid ascents and pulmonary barotrauma. This implies that the smokers had occult lung disease that precipitated the pulmonary barotrauma giving more evidence of hindrance on the body's bubble filter. This makes perfect sense when considering the damage caused by smoking on the small airways and the alveolar walls which allow bubble to pass though the system instead of being filtered. Please think about these facts before picking up that next cigarette or diving with someone who smokes. If you smoke, see your doctor for help with overcoming the addiction. Make your diving safe and fun.

The effect of hyperbaric oxygen on the oxygen window

BY EDDIE BRIAN, JR., M.D.

The oxygen window. Inherent unsaturation. Partial pressure vacancy. Most divers with an interest in decompression diving have likely encountered one of these terms at some time. All three terms are used to describe the same physical phenomenon. For this article, the term oxygen window will be used, as it appears to be the most commonly applied term. However, the terms "inherent unsaturation" and "partial pressure vacancy" more correctly describe the physical phenomenon.

Current techniques of oxygen-facilitated decompression diving are based on use of the oxygen window. Despite common use of the oxygen window by divers, it appears to be one of the least- appreciated concepts in decompression diving. Understanding the oxygen window requires knowledge of circulatory and gas transport physiology, and the best place to start is with normobaric physiology.

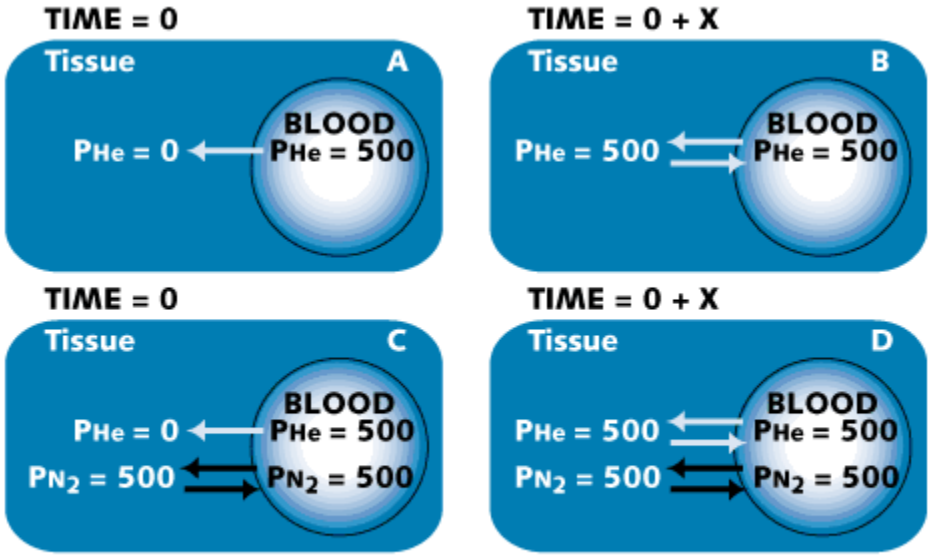
LIFE AT ONE ATMOSPHERE

Physiology is not homogeneous. In healthy individuals under normal conditions, lung blood flow and ventilation, as well as tissue blood flow and metabolism, vary over wide

ranges. Blood flow, ventilation and metabolism can vary both between individual areas and globally over time. These variables affect the precise gas exchange occurring in individual areas of the lungs and body tissues. To make these complex processes somewhat easier to understand, much of the physiology has been reduced to the simplest terms. However, the descriptions correctly reflect the global processes of gas uptake and elimination, and values presented represent average values. Partial pressure values are expressed in millimeters of mercury (mmHg), and 1 ATA equals 760 mmHg. For Systeme International purists, division by 7.5 converts mmHg to kilopascals, the correct SI unit of pressure.

Gas movement from lung to tissue and back is dependent on a partial pressure gradient. The concept of the partial pressure of a gas in solution is sometimes confusing, as gas in a liquid is dissolved in solution. A gas dissolved in liquid does not exert hydrostatic pressure like a gas in the gas phase, because the gas atoms or molecules are no longer free to move about as in the gas phase. This is an extremely important concept to understand or accept. The forces that hold a gas in solution are the same forces that hold any non-ionized solute (gas, liquid or solid) in solution. Tissues are principally liquids, and the partial pressure of a gas dissolved in a liquid is defined as the partial pressure that the gas would exert if the gas phase were in equilibrium with the liquid. Tissue gas partial pressures are commonly expressed as mmHg or atmospheres absolute (ATA). Tissue gas partial pressure is an index of the amount of gas present in the tissue. The total amount of gas present in a tissue is also affected by gas solubility, which can vary between gases and tissues. A tissue will absorb a larger volume of a highly soluble gas as opposed to a lower solubility gas before reaching any given partial pressure. In other words, if a given volume of gas dissolves in a tissue, the tissue partial pressure of a highly soluble gas will be lower than the tissue partial pressure of a low solubility gas.

Gas in solution moves by diffusion from an area of higher partial pressure to an area of lower partial pressure. Although the force for diffusion is a partial pressure gradient, it is not "pressure" per se that drives the movement of gas. When a gas line is pressurized to fill a cylinder, the pressure differential drives bulk movement of gas atoms or molecules. However, diffusion is not bulk movement of gas but rather the movement of individual gas atoms or molecules due to random atomic or molecular movement. Diffusion of an individual gas into or out of a tissue is dependent only on the partial pressure gradient of the gas, and not on other gases present in the tissue. This may seem paradoxical, as divers frequently conceptualize gases present in tissue as exerting a "pressure" that "holds" other gases out of the tissue. This analogy is incorrect. The diffusion of gases is not dependent on the bulk movement due to a pressure differential, but rather movement of individual gas atoms or molecules down the partial pressure gradient. The interaction of individual gases dissolved in solution does not affect diffusion of gases.



All values are partial pressures in mmHg.

Figure 1

As an example, Figure 1 shows gas diffusion from blood into tissue. In Figure 1A, at time 0, blood with a helium (He) partial pressure (PHe) of 500 mmHg flows into a capillary in tissue with a PHe of zero. Due to random movement, He atoms begin to encounter the inner capillary wall. Some He atoms cross the capillary wall into tissue, where the He atoms can either diffuse further into tissue or diffuse back into the capillary. The direction of movement is a random event, but at this point there are many more He atoms encountering the capillary inner wall as opposed to the outer wall, and the overall He diffusion is out of the capillary. At some intermediate time point between Figures 1A and 1B, the tissue PHe would rise to 250 mmHg. At this point, He atoms in tissue would encounter the capillary outer wall with one-half the frequency that He atoms in blood would encounter the capillary inner wall. The overall He diffusion is still out of the capillary but at one-half the rate as in Figure 1A. Eventually at time 0 + X, equilibrium is reached and the PHe is 500 mmHg in both blood and tissue (Figure 1B). At equilibrium, He atoms continue to cross the capillary wall and diffuse into tissue at the same rate as when the tissue PHe was zero. However, the net diffusion of He atoms is now zero, because He atoms in tissue now encounter the capillary outer wall and diffuse back into the capillary at the same rate as He atoms encounter the capillary inner wall and diffuse into tissue (denoted by the arrows of the same length). In Figure 1C, a condition similar to Figure 1A exists except that blood and tissue have first been equilibrated with 500 mmHg partial pressure of nitrogen (PN2). The N2 is in equilibrium, and N2 molecules are diffusing out of and back into the blood at an equal rate. Assuming that ambient pressure is equal to or greater than 1000 mmHg, when blood with a PN2 of 500 mmHg and a PHe of 500 mmHg flows into the capillary, He diffuses into tissue as in Figure 1A. The N2 molecules do not block He diffusion from blood into tissue. The He diffusion gradient is 500 mmHg in both Figures 1A and 1C. In Figure 1D, He would reach equilibrium with the tissue in the same amount of time as it took for He alone to reach equilibrium in Figure 1B.

When a gas diffuses through a liquid, the interactions of the gas molecules with the liquid molecules predominate over any gas-gas interactions. As an example, if water is saturated with N2 at 1 ATA and 37¼ C, the N2 molecules are only 0.01% of the total molecules (water and N2). If the amount of N2 were doubled (2 ATA), then N2 molecules increase to only 0.02% of the total number of molecules. In reality, the chance of interaction between N2 and water molecules is greater than the above percentages would indicate, as the molecular diameter of water is larger than most gas molecules (water is a larger target). Furthermore, because of solvent-solute (water-gas) interactions, the dissolved gas molecules tend to remain surrounded by water molecules. The concept that gas atoms or molecules dissolved in tissue can "push" other gas molecules out of the tissue due to gas-gas interactions is not correct.

GAS TRANSPORT IN BLOOD

Under all conditions, blood that perfuses the lungs gives up CO2 and absorbs O2. Under normal conditions at 1 ATA, we are saturated with N2 and other trace gases, so there is no partial pressure gradient for these gases between lung and tissue. For most normobaric physiology, N2 and other trace gases are ignored, as there is no active exchange of these gases. For our purposes, it is helpful to include nitrogen and trace gases, as it helps illustrate how the oxygen window can be enlarged. In the following, all of the trace gases (principally argon) have been included with N2 to simplify the discussion and figures. Atmospheric CO2 has also been ignored as CO2 represents only a fraction of a percent of the atmosphere.

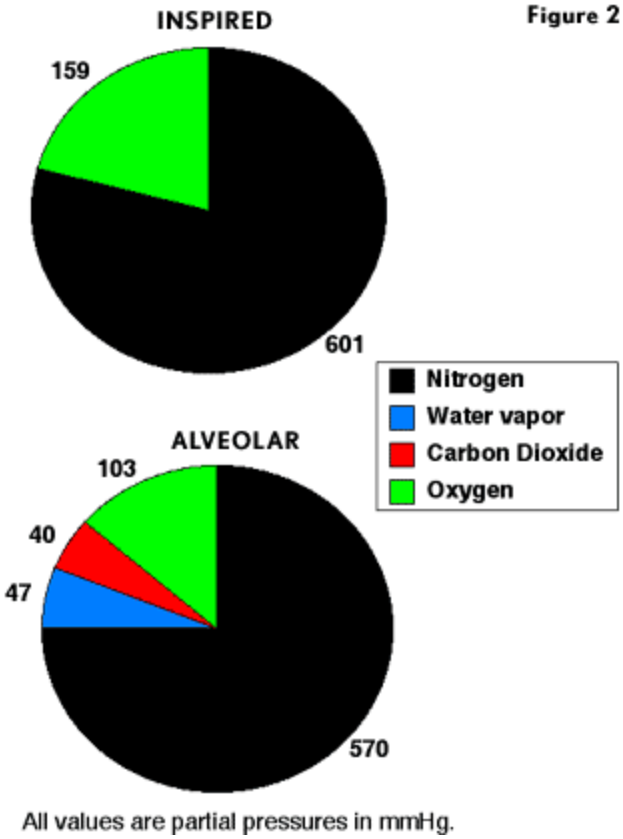


Figure 2

All values are partial pressures in mmHg.

When we breathe air at 1 ATA, the inspired air moves down our respiratory tract where it reaches the alveoli, the gas exchange units of the lungs. As gas moves into our lungs, it becomes saturated with water vapor, diluting the inspired gases. At 37C, the partial pressure of water vapor is 47 mmHg. The membrane of the alveoli does not constitute a barrier to gas diffusion, and alveolar gases rapidly equilibrate with the blood traversing the alveolar capillary. Because alveoli are gas spaces in communication with ambient atmosphere, the sum of gas partial pressures in the alveoli must equal ambient pressure. Oxygen diffuses out and CO2 diffuses into alveoli, both processes lowering the O2 partial pressure (PO2) in alveoli. Figure 2 shows inspired and alveolar gas partial pressures for air. At 1 ATA, dry air has a PO2 of 159 mmHg. However, by the time air reaches an alveolus and equilibrates with blood, the alveolar PO2 (PAO2) has fallen to 103 mmHg. This means that the PO2 in blood perfusing the alveoli capillary cannot be higher than 103 mmHg. If all alveoli in the lungs had perfect ventilation and perfusion, the PO2 of arterial blood would be 103 mmHg. However, ventilation and perfusion in the lungs are not perfect, and under normal conditions in healthy individuals some blood traverses the lungs without undergoing gas exchange. All blood flowing though the lungs eventually mixes together in the left side of the heart. During mixing, the unventilated blood removes some oxygen from blood that underwent gas exchange, resulting in a further lowering of arterial PO2 (PaO2) to 95 mmHg. A PaO2 of 95 mmHg is an optimal value, and actual PaO2 values of healthy individuals may vary between 85 and 95 mmHg.

For practical purposes, liquids are incompressible and do not respond to changes in ambient pressure. Because of this, the sum of gas partial pressures in a liquid can be less than ambient pressure. Liquids such as blood and other body tissues will equilibrate only with the gas partial pressures to which they are exposed. On the alveolar side of the alveolar membrane, the total partial pressures must equal ambient pressure. However, on the liquid side of the membrane, the total partial pressures can be less, and in some areas may be quite a bit less than ambient pressure. The partial pressure that a gas exerts in a liquid depends on the temperature, the solubility of the gas in the liquid and the amount of gas present. Thus, if the amount of gas present and the temperature remain constant, the partial pressure of the gas in a tissue is fixed. If one gas is removed from a tissue, the remaining gases do not expand to fill the partial pressure vacated by the gas that was removed. Figure 3 shows total partial pressures for air breathing at 1 ATA from inspired gas to venous blood. Because of the decline in PO2 from alveoli to arterial blood, the total gas partial pressure in arterial blood during air breathing at 1 ATA is 752 mmHg, less than ambient pressure (760 mmHg). If PaO2 is lower than 95 mmHg (assumed in this example), then the total partial pressure in arterial blood will be less.

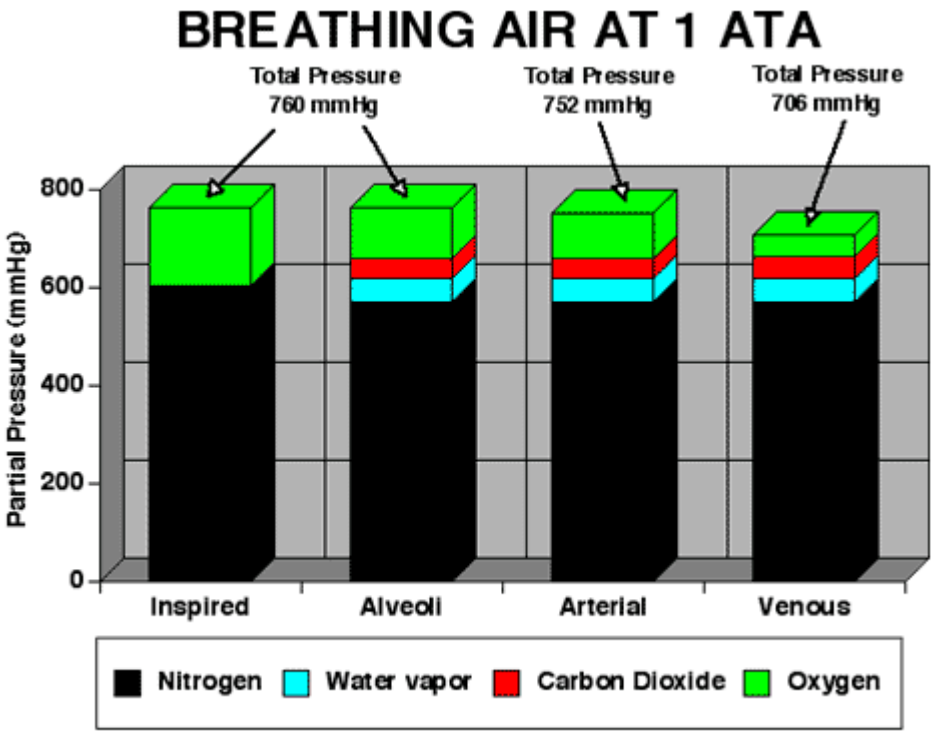


Figure 3

Metabolically inactive gases such as He and N2 are transported only in the dissolved phase in blood and the amount of gas present in blood is directly related to the gas partial pressure. In contrast, the metabolic gases O2 and CO2 have highly specialized transport systems. At 1 ATA, most O2 is transported in blood bound to hemoglobin. Hemoglobin is a specialized protein in red blood cells (RBCs) that reversibly bind O2. When O2 is bound to hemoglobin, it is no longer dissolved in solution and no longer contributes to the PO2. Because of O2 binding to hemoglobin, the relationship between the O2 content of blood (CO2) and PO2 is very non-linear due to the non-linear O2-hemoglobin disassociation curve (Figure 4). The vertical axis is the hemoglobin percent saturation, which represents the fraction of hemoglobin molecules that have O2 bound. The horizontal axis is the PO2. The left side of the curve is relatively steep, and as PO2 increases, the percent saturation rapidly increases as O2 is bound to hemoglobin. As hemoglobin saturation approaches 90%, the curve begins to flatten, and the increase in saturation becomes less for any increase in PO2. Under normal conditions, hemoglobin binds 1.39 mL O2 per gram of hemoglobin. In the following examples, a normal hemoglobin concentration of 15 grams of hemoglobin per deciliter (dL) of blood was assumed. The amount of O2 bound to hemoglobin can be calculated by multiplication of the hemoglobin concentration by 1.39 then multiplication by the hemoglobin saturation. For example, if the hemoglobin is 15 g/dL and saturation is 97.25%, then the O2 bound to hemoglobin is (1.39)(15)(0.9725) = 20.28 mL O2/dL blood. Oxygen also dissolves in blood, but the amount of dissolved O2 is small compared to the amount bound to hemoglobin. Only 0.003 mL O2/deciliter blood/mmHg PO2 will dissolve in blood. With 97.25% hemoglobin saturation, the PO2 is 95 mmHg, so the dissolved O2 is (0.003)(95) = 0.29 mL O2/dL blood. The CO2 of blood is the sum of the hemoglobin-bound fraction and the dissolved fraction. Thus, the CO2 is related to the PO2, but it is not a simple linear relationship as with gases that are purely dissolved. Carbon dioxide is also

transported in states other than dissolved, being converted to bicarbonate ions inside of RBCs and by binding to hemoglobin. However, these mechanisms are much less efficient than the binding of O2 to hemoglobin; the relationship between the PCO2 and CO2 content (CCO2) is almost linear.

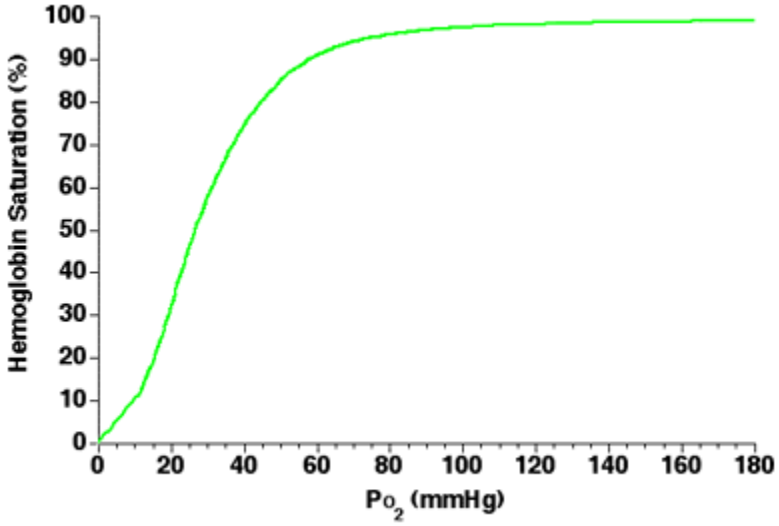


Figure 4

As blood perfuses tissue, O2 moves into tissue and CO2 moves into blood. If the PO2 absorbed from blood were replaced by an equal PCO2 from tissue, there would be no change in total partial pressure from the arterial to the venous blood. However, as blood traverses tissue, the increase in PCO2 is much less marked than the decrease in PO2. This is the genesis of the oxygen window. Breathing air under normal conditions at 1 ATA, the average arterial-venous (a-v) difference in PO2 is about 50 mmHg, meaning that venous PO2 (PvO2) is about 50 mmHg less than PaO2. As PO2 is reduced 50 mmHg, PCO2 increases only 5 mmHg from the arterial to venous blood. PCO2 increases much less than the decrease in PO2 due to two reasons. First, not all O2 consumed is converted to CO2. Under normal conditions, only 80% of O2 is converted to CO2. The second and more important reason is that CO2 is 20 times more soluble in blood than O2. Gases that are more soluble produce a lower partial pressure when a given volume of gas is absorbed into a liquid.

Figure 3 shows total gas partial pressures during air breathing at 1 ATA. The sum of the partial pressures declines slightly from the alveoli to arterial blood, where the total partial pressure is 752 mmHg. The total partial pressure present on the venous side of the circulation is 706 mmHg due to the consumption of O2 by tissue. In this example, O2 declines from 95 to 44 mmHg while CO2 increases from 40 to 45 mmHg. Nitrogen and water vapor partial pressures remain constant from the alveoli to arterial to venous circulation. Under normal conditions breathing air at 1 ATA, venous blood is undersaturated by 54 mmHg. This value was calculated by subtraction of the sum of the partial pressures in venous blood from ambient pressure. The oxygen window is opened when O2 is removed from arterial blood but only partially replaced by CO2 in venous blood. The principle factor in formation of the oxygen window is the a-v PO2 difference. The total gas partial pressure in tissue is less than venous blood due to the diffusion

gradients between tissue and blood. PO2 decreases as the distance from a capillary increases, but PCO2 increases only slightly due to solubility of CO2.

Undersaturation of blood and tissues has been documented in several studies. In 1910, Krogh demonstrated that the total gas partial pressure in arterial blood was less than ambient pressure, although Krogh could not measure individual gas partial pressures (5). Since Kroghs time, inherent unsaturation of the venous blood and tissues has been confirmed by direct measurement of gas partial pressures in venous blood and tissue (1, 6). The findings of the experimental studies are consistent with calculated values presented in the figures in this article. The arterial-to-venous reduction in total gas partial pressure was later termed partial pressure vacancy by Momsen, inherent unsaturation by Hills, and the oxygen window by Behnke (7).

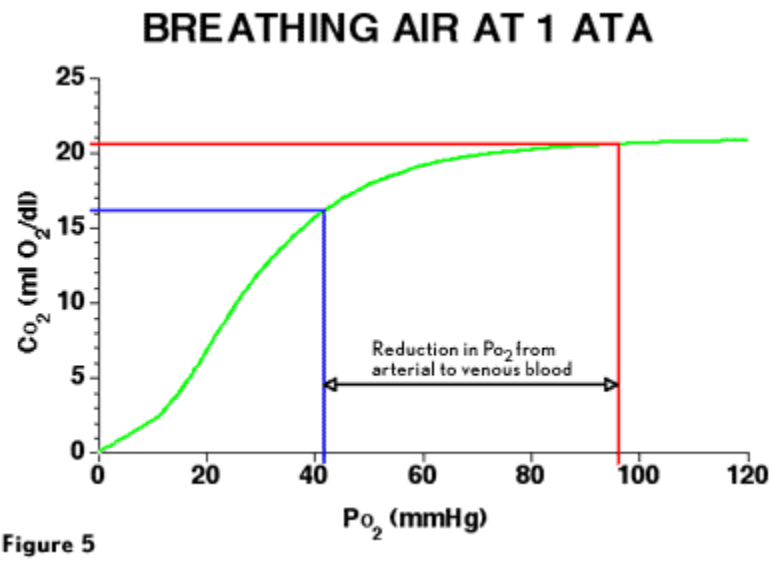


Figure 5

The precise size of the oxygen window depends upon the CaO2 and tissue oxygen consumption. Figure 5 shows the O2-hemoglobin disassociation curve, but the vertical axis is CO2 rather than percent hemoglobin saturation shown in Figure 4. The lines labeled arterial represent PaO2 and CaO2 and the lines labeled venous represent PvO2 and CvO2 during air breathing at 1 ATA. For this graph, an average a-v CO2 difference of 4.5 mL O2/dL blood and a hemoglobin concentration of 15 g /dL blood has been assumed. For any given PaO2, the CaO2 can be calculated, and CvO2 determined by subtraction of 4.5 mL O2/dL from the CaO2 value. PvO2 can then be determined from the curve by finding the PvO2 value that corresponds to the calculated CvO2. The total venous partial pressures can then be summed and subtracted from ambient pressure to determine the oxygen window. To determine the corresponding partial pressure and content values plotted on the O2-hemoglobin disassociation curves in these examples, a more precise O2-hemoglobin nomogram was used. The O2-hemoglobin disassociation curve flattens at higher PO2 values because hemoglobin is approaching 100% saturation, and any additional O2 is carried principally in the dissolved phase. Because the amount of O2 that will dissolve is much less than will bind to hemoglobin, when hemoglobin is fully saturated the increase in CO2 is much less for any increase in PO2. When O2 is transferred from blood into tissue, dissolved O2 diffuses from blood into tissue, and then is replaced by O2 released from hemoglobin.

As blood passes through tissue, a fixed volume (content) of O2 is removed. The change in PO2 required to supply the volume of O2 depends on where the CO2 values lie on the O2-hemoglobin disassociation curve. Because the slope of the O2-hemoglobin disassociation curve flattens on the right of the curve, as PaO2 moves to the right, the reduction in PO2 becomes greater when a fixed volume of O2 is removed. This is because a greater amount of dissolved O2 is being removed as you move to the right on the curve, meaning that the average change on the vertical axis is always 4.5 mL O2/dL, but the change on the horizontal axis varies depending on where the CO2 values lie along the curve. This is how the oxygen window is enlarged when PO2 is increased. Assuming that the volume of O2 removed is constant (4.5 mL O2/dL), then the amount of CO2 produced is constant, and the increase in PvCO2 is also constant. This means that the a-v increase in PCO2 is always about 5 mmHg, while the a-v PO2 reduction can vary from 50 to greater than 1000 mmHg.

LIFE BREATHING OXYGEN AT ONE ATMOSPHERE

Before moving into hyperbaric physiology, understanding how the oxygen window can be enlarged at 1 ATA is helpful in understanding the transition from surface to depth.

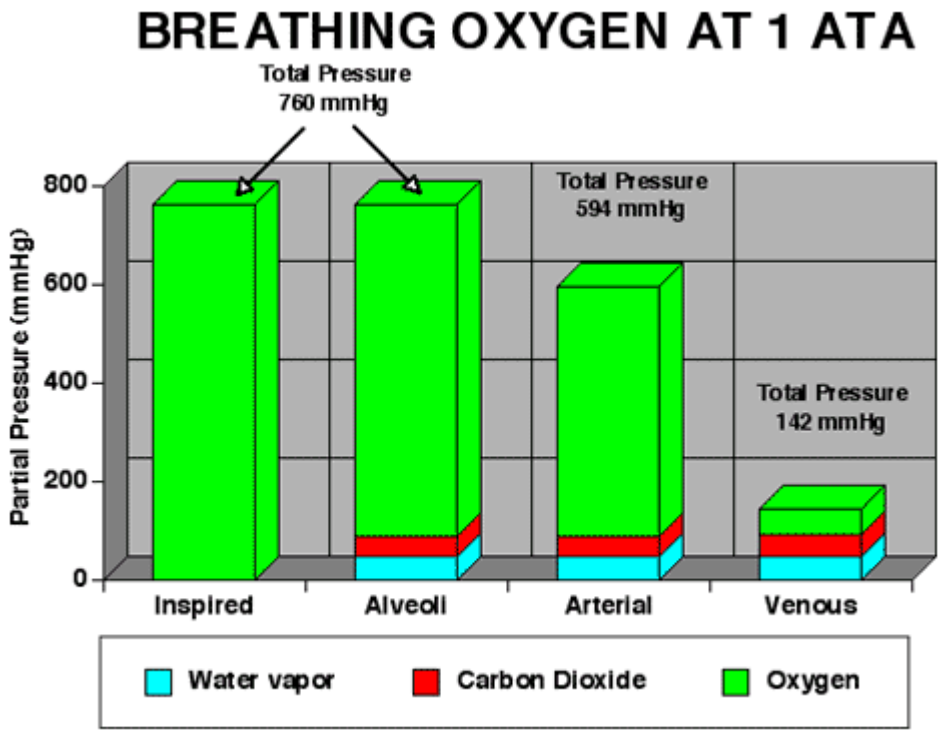


Figure 6

Figure 6 shows inspired to venous blood partial pressures during O2 breathing at 1 ATA. In this example, it is assumed that all nitrogen, argon and other trace gasses have been washed out of the system. During O2 breathing, the ventilation/perfusion inequalities in lung have a much greater impact on PaO2 than during air breathing. Under optimal conditions during O2 breathing at 1 ATA, PaO2 would be about 500 mmHg. Because of the greater difference between alveolar and arterial PO2, arterial blood is undersaturated by 166 mmHg. As blood moves through tissue, the same 4.5 mL O2/dL blood is

extracted, and PO2 falls to 57 mmHg in venous blood. Thus, venous blood is unsaturated by 518 mmHg during O2 breathing at 1 ATA.

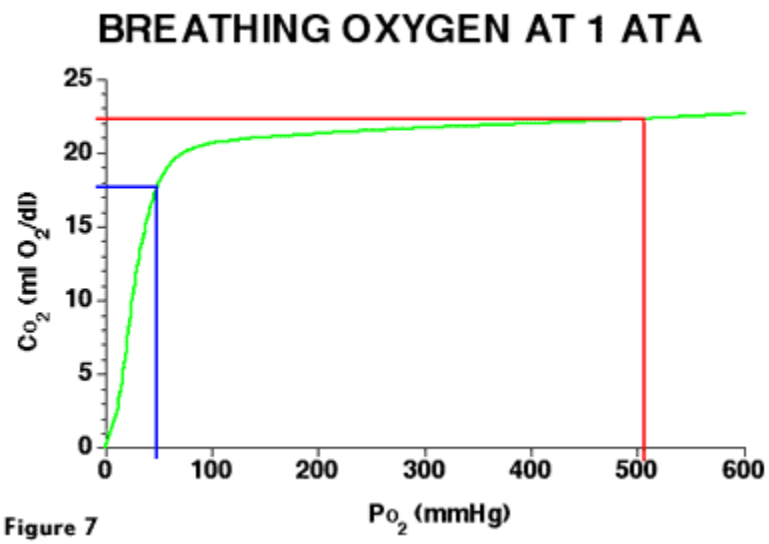


Figure 7

Figure 7 shows the a-v CO2 and PO2 differences during O2 breathing at 1 ATA. This is the same oxygen-hemoglobin disassociation curve shown in Figure 5, but the right side of the graph has been extended to greater PO2 values. Note that the amount of O2 removed is 4.5 mL O2/dL blood (on the vertical axis), the same as in Figure 5. However, because the PaO2 is moved far to the right where the slope of the curve is flat, the change in PO2 is much greater than air breathing at 1 ATA. In this example, the 518 mmHg unsaturation in venous blood is the oxygen window. If a non-respiratory gas were being evolved from tissue, it could occupy some or all of the window.

LIFE UNDER WATER

So finally we arrive at the effects of hyperbaric conditions on the oxygen window. Breathing O2 at 20 FSW results in an inspired PO2 of 1.6 ATA (1216 mmHg). As in the previous example of O2 breathing at 1 ATA, only respiratory gases were assumed to be present. Figure 8 shows inspired to venous gas partial pressures for O2 breathing at 1.6 ATA. As in previous Figures 3 and 6, there is a stair step decline in total partial pressures from the alveoli to venous blood.

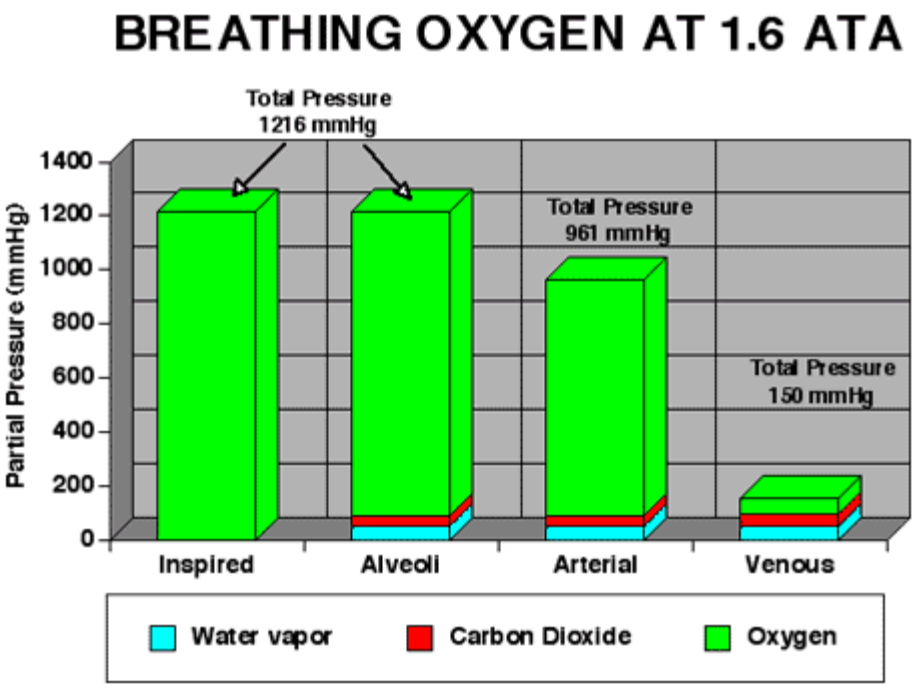


Figure 8

Figure 9 shows the relationship between CO2 and PO2 values for O2 breathing at 1.6 ATA.

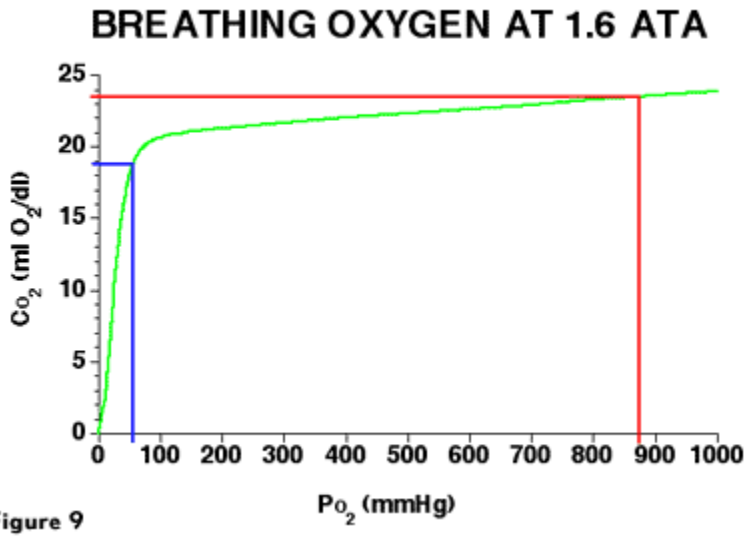


Figure 9

The a-v CO2 difference (vertical axis) remains constant at 4.5 mL O2/ dL blood. However, because dissolved oxygen content has been significantly increased, the CO2 values are shifted upward on the vertical axis, and the PO2 values are shifted to the right on the horizontal axis. Inspection of the curve in Figure 9 indicates that as CaO2 and CvO2 continue to move to the right, the oxygen window will continue to enlarge until

CvO2 is shifted above the knee of the curve. This occurs when venous hemoglobin is fully saturated with O2, and only dissolved O2 is removed to supply tissue O2 demand. To achieve full saturation of venous hemoglobin requires an inspired PO2 of approximately 3 ATA.

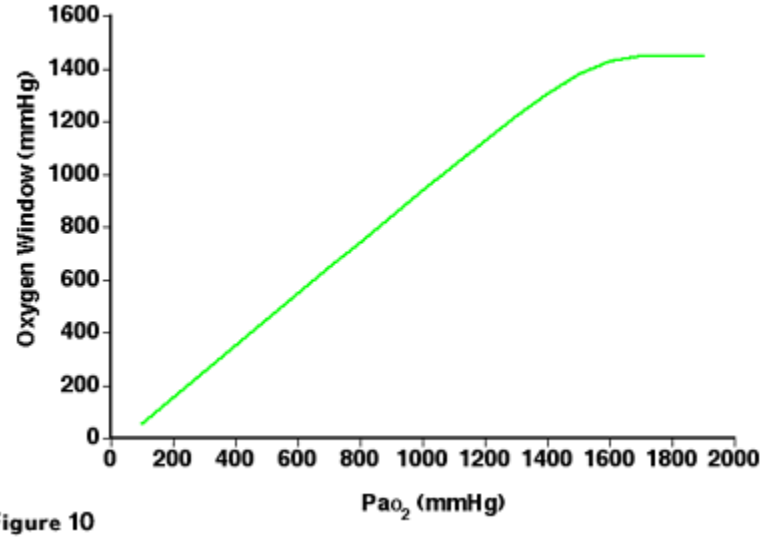


Figure 10

Figure 10 shows a plot of the oxygen window versus PaO2. When PaO2 exceeds 1600 mmHg, the oxygen window has reached a maximum value of 1400 mmHg. Above, this point, further increase in inspired PO2 will not increase the oxygen window. Oxygen toxicity clearly limits the oxygen window to much lower values during in water diving operations.

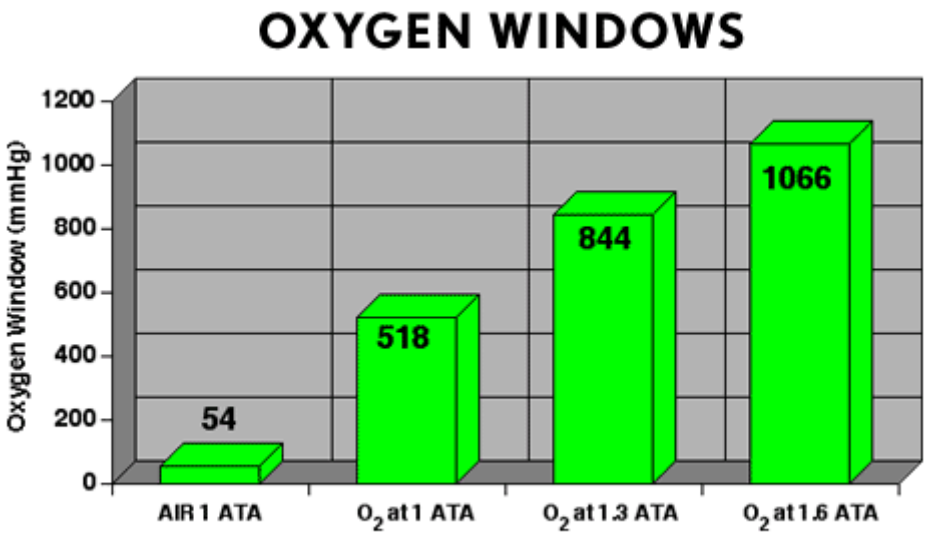


Figure 11

It is useful to consider what happens to the oxygen window when O2 is breathed at 10 and 20 FSW. Figure 11 shows oxygen windows for various circumstances. When O2 is

breathed at 20 FSW (1.6 ATA), the oxygen window is 1066 mmHg. If O2 is breathed at 10 FSW (1.3 ATA), the oxygen window is reduced to 844 mmHg, a decrease of 222 mmHg. This means that there is less partial pressure vacancy in venous blood for non-metabolic gas to occupy during O2 breathing at 10 FSW as opposed to 20 FSW. Furthermore, inert gas elimination is independent of depth during oxygen breathing. The gas partial pressure gradient for movement from tissue into blood is not controlled by ambient pressure; it is controlled by the gas partial pressure in the tissue and in arterial blood. As long as the arterial gas partial pressure is zero, the gradient for gas removal from tissue is maximal. Breathing oxygen at a deeper depth has the advantage of a greater hydrostatic pressure to hold dissolved gas in solution.

If a gas mixture with less than 100% O2 were breathed, then some of the oxygen window would be occupied by the inert gas. For example, if a 50% or 80% Nitro mixture were breathed at 20 FSW, then N2 would occupy some of the partial pressure of the oxygen window. How much of the window would be occupied depends on the tissue N2 partial pressure, which will determine how much N2 would diffuse from blood into tissue and the resulting PvN2. The oxygen window would be reduced by an amount equal to the PvN2.

IS THE OXYGEN WINDOW IMPORTANT?

It should be intrinsically obvious that removal of a gas from tissue can be speeded by elimination of the gas from the inspired mixture. If the arterial partial pressure of a gas is zero, then no gas will diffuse into tissue while the gas is diffusing out of the tissue. As discussed above, diffusion of one gas in solution is not affected by the presence of other gases. Despite all of the above discussion of gas diffusion, most decompression models in common use, including Buhlmanns models, are perfusion-limited models. In such a model, diffusion is assumed to be infinite and thus cannot limit tissue gas uptake or removal. Tissue half-times for He and N2 are independent of each other, so the presence or absence of N2 does not change the rate of He on- or off-gassing and vise versa. In theory, He off-gassing should not be altered by breathing air, 50% Nitrox or 100% O2 during decompression from a He dive. He elimination during air or O2 decompression from a He-based dive has been measured, and the decompression gas did not affect the rate or volume of expired He (4). In another study at 1 ATA, tissue N2 washout is not different during O2 or Heliox breathing (3). Both studies are consistent with the physics of gas diffusion in solution, where the presence of a second non-metabolic gas does not slow diffusion of the first non-metabolic gas. The reality is that at any given ambient pressure, regardless of the size of the oxygen window, as long as there is no inspired He, the rate of He off-gassing will be unchanged.

Decompression from an N2-based dive is longer with N2 containing deco mixes because some N2 is continuously diffusing into tissue during deco. Decompression from a He-based dive can be longer with N2 containing deco mixes because N2 is diffusing into tissue as He is diffusing out of tissue. The decompression obligation of a tissue compartment is based on the sum of gas partial pressures in the compartment. This means that if a tissue is loaded with N2 as He is being removed, it tissue has a greater decompression obligation than when no N2 is added to tissue during He off-gassing. Enlarging the oxygen window can only occur when PaO2 is increased to a maximum tolerated value, either by increasing depth or increasing FiO2 of the gas mix, or both. Although enlarging the oxygen window may not directly affect tissue gas removal, it does

directly affect tissue on-gassing during decompression, which affects the amount of time required to decompress the tissue.

Enlarging the oxygen window may have another effect, which is subtler than tissue on- or off-gassing. The following discussion is conjecture based on data available in the literature, and has not been directly studied. During decompression of animals from air dives, venous blood becomes supersaturated with N2 during the early stages of decompression, and venous blood supersaturation appeared related to venous bubble formation (2). Venous blood N2 supersaturation occurred following a relatively mild decompression stress of ascent from 33 FSW to surface. Once bubble formation had occurred, gas removal was slowed, possibly by bubbles in the venous circulation (2). By limiting the speed with which ambient pressure is changed, deep stops may function to limit venous blood supersaturation and limit bubble formation related to the supersaturation. Increasing the oxygen window during decompression will also limit venous blood supersaturation by limiting the amount of non-metabolic gas in blood. In essence, the presence or absence of a second non-metabolic gas will not alter the amount of gas evolved from tissue. However, the presence of an inspired non-metabolic gas could increase the severity of venous blood supersaturation.

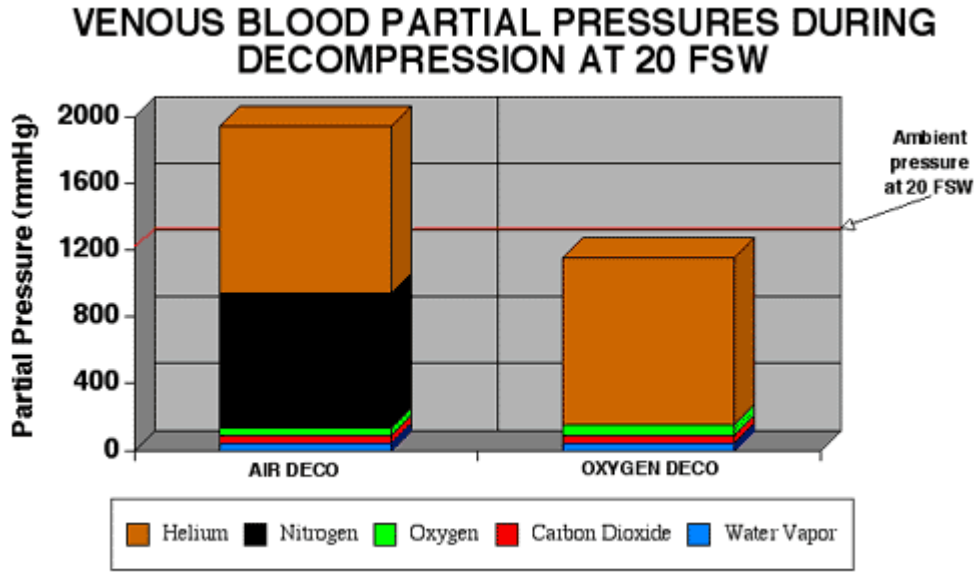


Figure 12

Figure 12 demonstrates hypothetical venous partial pressures during decompression from a He dive with either O2 or air at 20 FSW. In this example, the partial pressure of He in venous blood is assumed to be 1000 mmHg in both conditions. During air breathing at 20 FSW, PaN2 would be approximately 1140 mmHg, so an assumed PvN2 value of 800 mmHg allows some tissue N2 uptake. Ambient pressure at 20 FSW is 1216 mmHg. Due to the oxygen window, the total partial pressure in venous blood during O2 breathing would be 1150 mmHg, less than ambient. Total venous partial pressure during air breathing at 20 FSW would be 1937 mmHg, above ambient pressure. Although no

direct experimental data exists on this topic, oxygen breathing may limit venous blood supersaturation, prevent venous bubble formation, and thus speed tissue gas removal.

CONCLUSIONS

It should be obvious from the above discussion that much decompression physiology is poorly understood, and models used at best approximate in vivo physiology. Clearly, not all decompression illness can be predicted or prevented. However, thoughtful application of available models coupled with careful diving technique can minimize risk of decompression illness. By reducing non-metabolic gas to a minimum and reducing tissue on-gassing, the oxygen window can be utilized to increase tissue off-gassing during decompression. Real life experience indicates that use of O2-enriched deco mixes can function to limit decompression time and possible the incidence of decompression illness. Use of high O2-mixes requires careful attention to dive planning and execution. As always, the careful, thoughtful diver will be the safer diver.

Decompression Experimentation

From the logistics of underwater exploration to the strange malady that became known as decompression illness, divers and scientists have been struggling for decades to successfully investigate the underwater world. As early as 1670 Boyle described, with detailed accuracy, bubbles in the blood and body fluids of small animals subjected to low pressures. These bubbles, first located in the eye of a snake, marked more than 300 years of debate about the meaning, repercussions, and logistics of exposure to elevated ambient pressure. The quest for procedures that allowed effective immersion and safe retreat from hyperbaric exposures is dotted with a medley of evolving theories, best-guess practices, and hopeful global conceptualizations.

Despite decades of research, many aspects of decompression are very poorly understood. For example, even the principle com-ponent(s) of hyperbaric illness remain(s) contested. Consider that, originally, painful symptoms were seen as the indication of bubbles impinging on nerves in the body, representations of an unsuccessful decompression. Yet, later studies indicated that gas bubbles (thought to be the precursor and eventual cause of painful DCS) were often present in the body when painful symptoms were not evident. These asymptotic bubbles were predicted as early as 1951 by Bateman and Behnke and termed "silent" due to their lack of symptomatic pain. The advent of ultrasonic monitoring techniques showed that these "silent" bubbles could be detected, often without any DCS symptoms being present. There did seem to be a very loose correlation between the size and frequency of Doppler detected bubbles and the onset of DCS. Yet, should the existence of these bubbles (separate from pain or other symptoms) be considered a form of DCS and, if so, what role should they play in modifying decompression profiles? Some individuals complain of DCS symptoms without detectable bubbles while some people with no pain have a very high presence of bubbles in their blood. Furthermore, some research indicates that microbubbles might

permanently exist in the body, acting as seeds that may be fed by surrounding tissues or blood. Still other studies postulate the occurrence of bubbles that are formed in the arterial side, perhaps in the turbulent blood flow around the nodes of the heart. These and other widely divergent attempts to understand the nature of DCS demonstrate that we are far from any precise scientific model, one that can accurately describe the phenomenon and its associated symptoms. For individuals engaging in dives that require very limited decompression, these issues are arguably less problematic. These divers are, to some extent, insulated by the high number of diving profiles that establish a low statistical risk in a particular time/depth range. It is debatable to what extent this verifies any scientific model; instead, it may be principally an indicator of profiles with a risk well established through trial and error. Divers that extend beyond the recreational range into more aggressive diving profiles are more at risk; these divers have far fewer dives by which to gauge low statistical risk. While greater numbers of successful dives can reduce the statistical risk of certain profiles, several factors call into question the best dive profile. These issues include: the way one classifies decompression sickness (i.e., pain or bubbles), hyperbaric damage not immediately obvious to the diver (e.g., bone necrosis), the safest profile for aggressive or necessarily abbreviated decompression times, and the cascade of problems and/or inefficiencies associated with bubbles created at various places during the decompression profile (i.e., reduced efficiency and/or damage). The above complications are a sample of the problems confronting divers in general and technical divers in particular. Technical divers, due to their expanded depth and bottom times, have to be savvier about the particulars of safe decompression; this is particularly true for divers that engage in very aggressive dives. Aggressive diving profiles (i.e., long and/or deep) immerse the diver for long periods. Therefore, maximum decompression efficiency is essential, particularly in times where long immersions can expose the diver to additional risk, such as from bad ocean conditions or oxygen toxicity. Furthermore, it seems likely that bubble formation has an impact on the symptoms of decompression illness and that especially large collections of bubbles can be very dangerous. It is therefore prudent to enact procedures that are likely to limit the formation of bubbles. Procedures such as slowed ascent rates, limiting multi-ascent dives, and the use of safety stops are prudent well beyond the scope of theoretical decompression discussions. It will quickly become obvious to most seasoned technical divers that there is a great deal of variability in decompression profiles. For example, divers may follow vastly different profiles on the same dive, often with the very liberal diver experiencing the same success as divers with much greater conservatism. Conversely, some divers seem to experience problems with deep dives and/or long bottom times almost regardless of their decompression profile. Poor fitness, high fat content, and dehydration often appear when evaluating these decompression episodes, yet when trying to explain the variations in unsuccessful decompression, these factors paint an incomplete picture. To be sure, increased fitness, reduced fat content, and improved hydration have helped dozens of problem divers. However, decompression success remains a mystery to many divers and those undertaking very aggressive diving profiles have very little solid research information to rely upon.

Studying bubble sonograms at Duke University. Photo ©Anthony Rue

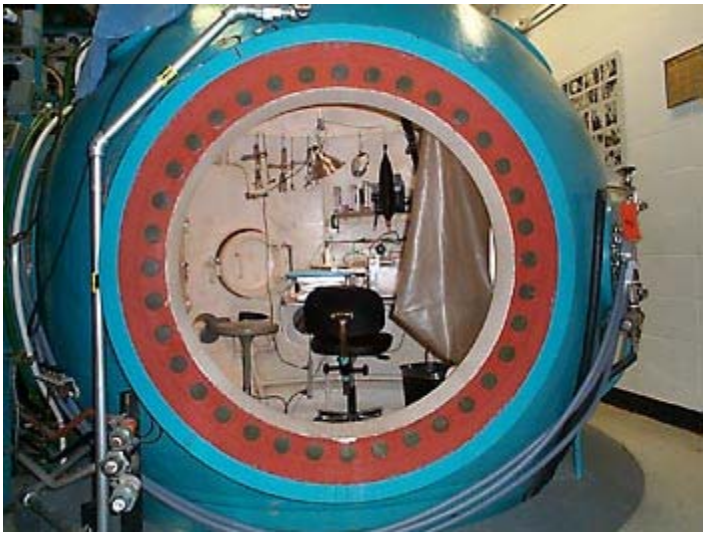
It has often been said that necessity is the mother of invention. This motivation clearly ignited my passion for resolving decompression mysteries. The early years of technical diving found the mysteries of decompression consistently represented, but it was early involvement in the diving of the WKPP that heavily challenged inconsistencies in decompression knowledge and practice. As one of the early members of the WKPP, this challenge was amplified with diving that rapidly pushed the established decompression envelope. These early challenges are usually unknown or de-emphasized by most in technical diving, leaving many divers to imagine that technical diving has been popular for many years. In fact, very little deep and mixed gas diving occurred outside commercial/military operations. Furthermore, the diving done within these groups generally consisted of large support operations and often involved saturation diving.

During the late 1980s, technical diving started to become more common among a very small group of divers. Not until the mid 1990s did the practice of deep and mixed gas diving become solidly entrenched within "recreational" (i.e., non-paid) circles and become acknowledged as technical diving. The lack of popularity experienced by technical diving during its early growth period led to a great deal of confusion and misinformation. Both diving leaders and individuals could be found promoting incorrect and dangerous misconceptions about the risks of Helium and the safety of deep air. Furthermore, there was a general confusion over proper decompression schedules and little solid information to guide one's efforts. For most early technical divers, obtaining deep, mixed gas decompression tables constituted one of many roadblocks to safe deep diving. Available tables tended to come from a hodgepodge of locations, and often relied on extreme conservatism as insulation against lack of under-standing. It was during these initial years that George Irvine and I began the ongoing task of actively refining practical decompression practices. As time progresses we continue to strive for "scientific validation"; yet, our earliest efforts and, to a lesser extent, our current focus centers on maximizing decompression efficiency and repeatability.

We initially found that common decompression assumptions subjected divers to extremely long decompression obligations, ones that, regardless of their length, seemed inefficient. In other words, divers noted that very long decompressions seemed inefficient, and that by including a greater proportion of deeper stops than were commonly accepted they were able to reduce the length of shallower stops, thereby

reducing overall decompression time. The result of this was that decompression became more effective, effectiveness being represented by subjective physical health.

The earliest technical diving uses of deep stops were based almost solely upon personal experience. Several individuals, many within our group, toyed excessively with various ranges of deep stops, employing progressively deeper stops for varying times and then shortening the times in the pursuit of maximum efficiency. George Irvine and I pushed these and other assumptions to their limits, consistently striving to develop a means of maximizing decompression efficiency; George has been especially aggressive in this regard, helping to establish a true minimum range for decompression times. Divers from the WKPP established several working rules of thumb for deep stops, such as the convention of a first stop that should start one atmosphere shallower than one's maximum depth during the dive. I later worked with Erik Baker to study these techniques as part of a more global approach. Together with Simon Traemer and indispensable assistance from Gas Diving UK (Graeme Davison, Sue Davison, and Andy Kerslake) the group developed the first of several versions of DecoPlanner dive calculator. Here, deep stops are specifically defined by the point in which ambient pressure and leading compartment pressure (theoretical pressure within body tissues) are equal. Deeper than this point would actually result in on-gassing, while too shallow could promote bubble formation.



Decompression chamber at the DukeCenter for Hyperbaric Medicine.
Photo ©Anthony Rue

Ironically, the use of deep stops has a measure of scientific support, dating back to the 1950s, and is largely unknown to most technical divers. Physicist David Yount originally postulated that deep stops could reduce bubble formation. His studies initially focused on the first stop, evaluating the point at which bubbles would first form. The initial trials reported good success but divers were so entrenched in the conventional profiles that they resisted the idea of spending so much time at increased depth. These later studies, taken together with Yount's early work, became the basis for several theories, including VPM and RGBM.

Assuming that there is good reason to believe that deep stops are done reasonably efficiently, one must then reflect on the next and perhaps more problematic phase of the decompression--that of middle or intermediate stops. Realistically, the concerns that guide the middle portion of decompression are similar to those that limit decompression in general. For example, there exists a fair degree of debate over exactly what causes DCS symptoms. Most people would agree that bubbles are a negative part in a chain of events that results in DCS symptoms. Consequently, we strive to reduce and/or eliminate bubble formation. Several theories strive to manage bubbling, and it may be theoretically possible to have a "no bubble" dive. However, the profiles often result in exceedingly long decompressions. Therefore, as divers we often accept the risk (or perhaps reality) that some bubbling is likely a consequence of diving. Assuming this is true one might conclude that a choice could be made as to where in the decompression profile bubbling creates the most trouble. Consider that bubbles formed at depth could grow and become dangerously large, impeding circulation more directly and/or for a longer time. The longer divers wait to accept a greater bubbling risk, the longer the decompression profile could become (due to necessarily longer stops and their resulting decompression obligations).

To this end George and I worked to reduce the time in this mid-range of decompression stops, remaining convinced (particularly on long dives) that intermediate stops in conventional decompressions caused a large accumulation of gas that would then have to be eliminated. While this process has been successful, one should be aware that these times result in very high theoretical compartment pressures with M-values well above anything conceived under conventional "wisdom." Shortening the middle section of one's decompression might theoretically allow shorter shallow decompression stops. Personal experience indicates the truth of this assumption. However, divers should be cautious in their own personal experimentation.

Duke Center for Hyperbaric Medicine.

Photo ©Anthony Rue

During the early years of "recreational" deep diving (late eighties and early nineties), the use of Helium was often discouraged and an immense amount of fear was associated with its use. This fear stemmed largely from ignorance and a few early problems well known to the early deep divers (i.e., Hans Keller's fateful success and Hal Watts' DCS-laden Mystery Sink dive). Most early cave and wreck diving during this time was filled with discouragement for the use of Helium; many divers were told that the use of Helium led to much greater risk of central nervous system damage and that there was no safe way to decompress from Helium dives. For many years it was widely believed that if Helium must be used, divers should switch from it as soon as possible. Of course we later realized that these switches were unnecessary and dangerous, and that the emergency "need" for breathing air was irrelevant. Actually, there is no need for air anywhere in the diving profile. Instead, we realized years ago that safety and decompression efficiency were improved by using Helium in conventional air regions (i.e., 190-120 feet).

Overcoming the misconceptions of deep air freed us to look at Helium in a variety of areas. George Irvine and I started using progressively higher Helium mixtures in different regions and began using Helium in areas shallower than 100 feet. The much longer decompressions we endured while pushing the far reaches of Wakulla led us to see Nitrogen as the real enemy. Analyzing our profiles led to the conviction that removing Nitrogen from the slow compartments was the true difficulty. Nitrogen was particularly troublesome with extensive intermediate and shallow decompression stops. I later began playing with Helium enriched gasses in the 120-150 foot range during decompression to assist in eliminating Nitrogen from the breathing mixes and reducing accumulation in the tissues. Helium use in the 120-150 foot range actually provides shorter calculated decompressions when doing very long dives such as those conducted in Wakulla, even when calculations are based upon conservative dissolved gas models. However, shorter dives (i.e., in the 20-30 minute range) are more problematic. For example, calculated decompression for 30 minutes at 300 feet with no Helium results in about 10 minutes less decompression than with Helium in both nitrox mixes, and about 5 minutes less than with Helium in the 120 bottle when calculated with standard Buhlmann algorithms. However, this additional time is not in keeping with personal experience and evolving decompression concepts (such as those demonstrated by bubble theory represented in VPM and RGBM). It seems, for example, that our own trials actually demonstrate that the body can tolerate greater quantities of Helium than Nitrogen. It may be that Helium is traditionally treated too conservatively, and that it is either able to off gas more rapidly and/or is tolerated at higher pressure. Alternatively, this may be individual physiology or misconception; yet time and continued trials seem to hint that Helium is useful in a wide variety of diving depths. GUE's VPM version of DecoPlan promises to allow further careful study of the interesting relationship between conventional dissolved gas conceptualizations and bubble-based algorithms. EXPERIMENTATION

The previous discussion has been a brief, personal account representing more than a decade of individual and group experience. It is only fair to conclude this discussion with a note about the nature of decompression experimentation. The long-term effects resulting from hyperbaric exposure are difficult to quantify; even more problematic are the developments of sound decompression practices. Decompression diving (particularly

aggressive technical diving) is very much an experiment in progress. Generally speaking, tables are, in my opinion, more a growing statistical reality justified by some (hopefully) relevant theory. In other words, modelers come up with new theories to describe what is going on. For Haldane it was the basics like different compartments, gas loading and removal, and the role of gradients in gas elimination/absorption. Haldane's efforts drastically reduced DCS risk, allowing divers to forge deeper for longer periods of time. More aggressive diving was permissible because divers were suffering far fewer cases of DCS. Eventually these new schedules did not satisfactorily protect the progressively more aggressive diving; DCS risk again became more prominent and new theories were developed. Each of these "new models" redefined the parameters based on theoretical assumptions. Each "success" with a new theory leaves us feeling justified in our result; in truth, numbers may be sufficiently tweaked to provide a better statistical fit with minimal representation of "reality." The point here is to demonstrate that all tables are largely a best statistical guess; our assumptions may be entirely incorrect. This analysis leaves one wondering what constitutes success. Should profiles be based upon limiting overt DCS symptoms (such as pain), reducing bubble presence (as measured by Doppler), or some combination? It is very likely that we do not fully appreciate the long-term repercussions of our diving or of our decompression experimentation. Yet, reasonable measures of success include limited painful DCS symptoms, successful Doppler tests, and favorable long bone x-rays, indicating that overt damage is at least limited while drastically reducing decompression time. Of course, only time will tell where these representations fall in the long string of decompression theories. I am willing to bet that they will prove to be a valuable and substantial push toward a more appropriate global conceptualization.

Patent Foramen Ovale: Background and Impact on Divers

Common anatomical defects of the heart were identified as risk factors for decompression sickness in the 1980s. Between 17% and 35% of the normal population are found to have an anatomical atrial septal defect, or patent foramen ovale (PFO), beyond infancy (1). The incidence among divers who have suffered from serious neurological decompression sickness symptoms was reported to be as high as 61% (2) or 66% (3). This article will present an overview of the condition with emphasis on the implications for the diving population.

FETAL CIRCULATION

Fetal humans rely on placental circulation and the maternal pulmonary system for gas exchange. Since the growing lungs are not yet functional, they require only enough blood delivery to support their own development. The fetal cardiovascular system has two primary shunts to allow blood to bypass the lungs. It is the interatrial septum that allows blood in the right atrium to flow directly into the left atrium that is the concern of this discussion. Placental circulation is lost at birth and the infant must start breathing independently. After one or more gasps, the previously unused lungs will start to expand. Upon expansion, a redistribution of pressure causes a tissue flap within the left atrium of the heart to be pushed against the septal wall to functionally close the foramen. In the

normal course of events, the septal tissues will fuse within the first year after birth to make the closure permanent. However, in a post-mortem review of 965 normal hearts, patent interatrial openings were observed in 27% of the non-infant population (4).

EVALUATION OF INTERATRIAL SHUNTS

Post-mortem autopsy examination is the most reliable means of identifying anatomical foramen ovale. However, anatomical (or probe) patency does not equate to functional (or physiological or hemodynamic) patency. Being able to work a probe through the cardiac septum at autopsy does not indicate the degree of lateral shunting that was present in the living system. This is why in vivo studies usually report lower incidence rates than post-mortem studies. Functional patency was reported in 9% of 1,000 consecutive patients being scanned by transesophageal echocardiography (5). Functional patency is reliably evaluated using two-dimensional bubble contrast echocardiography (6,7). This technique involves the venous injection of small amounts (5-10 ml) of normal sterile saline solution that has been agitated in the presence of air to ensure the presence of large numbers of microbubbles. Bubbles are highly reflective targets easily detected by the ultrasonic pulses used in echocardiographic instruments. Injections of bubble contrast are made repeatedly during resting breathing while the echocardiograph captures a cross-sectional view of either all four chambers of the heart or the two atria. The microbubbles are clearly visible as they enter the right side of the heart. Functional patency is confirmed if bubbles are seen to cross the septum, and no further testing is conducted. If bubbles are not seen to cross the septum during resting trials, further injections are made during or just prior to the release of the strain phase of a Valsalva maneuver. Injections are repeated until crossover is seen or a pre-established maximum number of test cycles has been reached. As a control, the pressure generated by the Valsalva maneuver will usually be standardized, generally within the range of 40-60 mm Hg above ambient.

The dominant pattern of interatrial shunting is left-to-right since the left heart pressure is significantly greater than the right. The Valsalva maneuver is useful in assessing foramen ovale patency by augmenting or prolonging a transient pressure gradient reversal that encourages right-to-left shunting. Septal crossover is usually evident following the release of the strain phase.

The presence of a functional patent foramen ovale may have no adverse effect under normal conditions. A minor left-to-right flow would reduce cardiac efficiency, since blood would be sent to the lungs repeatedly, but this may not be problematic. Right-to-left shunting is a greater concern since blood bypasses the lungs and is sent directly through the body (systemic circulation).

Minor right-to-left shunts may not affect oxygen content appreciably, but major shunts can limit physical performance. A more insidious problem is the introduction of potentially embolitic materials (e.g., gas bubbles, blood clots) to the systemic circulation that would normally be filtered out in the pulmonary bed. Right-to-left shunting is the suspected agent in a significant number of unexplained or "paradoxical" stroke cases (8). A number of factors may increase right-to-left shunting. These include coughing, the Valsalva maneuver, pulmonary hypertension, chronic obstructive pulmonary disease, and the use of positive pressure ventilation (5). It has also been suggested that a spontaneous

reversal of the normal left-to-right pressure gradient may occur during the early phase of ventricular contraction (9). IMPACT OF INTERATRIAL SHUNTS ON THE DIVING POPULATION

Divers face risks from right-to-left shunting beyond those experienced by the non-diving population. The first is a direct result of the action of Boyle's Law. Any bubble present in the systemic circulation is subject to expansion during ascent. Initially non-problematic microbubbles that are shunted systemically could potentially become large enough to cause circulatory blockages. Right-to-left shunting may explain some cases of paradoxical arterial gas embolism (referred to as "undeserved embolism") when classic signs and symptoms are not accompanied by appropriate history or clinical indications of pulmonary barotrauma (10). Interatrial shunting may also alter bubble formation. While the mechanisms of bubble formation and decompression sickness have not been completely resolved, there is strong evidence that micronuclei "seeds" of some nature will initiate or exacerbate bubble formation. Materials shunted right-to-left (that would normally have been filtered out at the pulmonary bed) could serve as "seeds" and increase an individual's susceptibility to bubble formation (2). A substantial bubble load (number and size) may trigger the cascade of events resulting in decompression sickness.

The pattern of right-to-left shunting may also be influenced by the activity of diving. It has been demonstrated that the hydrostatic compression of the legs during immersion will increase the cardiac volume prior to contraction (the end diastolic volume or Ôpreload') such that stroke volume may increase up to 30% (11). Right atrial pressure can also be increased by 13 mm Hg (12). The reduced difference between right and left heart pressures may make gradient reversals easier to achieve. Cardiac tissue distension arising from preload increases could also cause tissue distortions that might transiently increase the size and/or patency of the foramen (2).

The first major review of foramen ovale patency in divers found that 11 out of 30 divers (37%) treated for decompression sickness had right-to-left interatrial shunts demonstrable by contrast echocardiography. More importantly, the authors reported that 11 out of 18 (61%) who presented with serious signs and symptoms demonstrated shunting during resting respiration (2).

An independent group of investigators reported patency in 15 out of 63 divers (24%) in a control group with no history of DCS. This contrasted a patency of 66% (19/29) in divers who had experienced neurological symptoms within 30 minutes of surfacing (3). More recent analyses have employed logistic regression to estimate the relative risk associated with patent foramen ovale. Bove (13) computed a 2.5-fold increase in the odds ratio for developing serious decompression sickness. Schwerzmann et al. (14) suggested a 4.5-fold increase in the odds ratio for developing decompression sickness, but this was a relatively weak study based on retrospective self-reports.

One of the issues raised but not resolved in the available literature is the relationship between patent foramen ovale and brain lesions. While it has been suggested that patent foramen ovale is associated with greater numbers of brain lesions (14,15), there is no evidence that this is related to functional deficits.

Reef dive, Ft. Lauderdale, Florida. Photo ©David Rhea

IMPLICATIONS FOR THE DIVING POPULATION

The presence of a functional patent foramen ovale does appear to increase the risk of decompression sickness. The issue of how to proceed, however, remains contentious.

Some investigators encourage the broader use of contrast echocardiography to screen potential divers (16), while others maintain that routine screening is not warranted (13). There is risk associated with introducing contrast bubbles into the bloodstream (17), although the established morbidity rate of bubble contrast echocardiography (0.07% in 41,000 studies) is lower than that of other accepted diagnostic techniques (for example, exercise testing for ischemic heart disease at 0.09% in 518,448 tests) (16). While the debate regarding screening continues, the overshadowing question is how to counsel the diver with a patent foramen ovale. It is probably safe to say that functional patency does represent a relative contraindication for diving. This does not imply that it should be an absolute contraindication. There are several arguments against using patent foramen ovale as a disqualifying factor. Even if the relative risk is higher, the absolute risk is still very small. Tripling a very small risk still results in a very small risk. And unlike many disqualifying conditions, the risk from foramen ovale patency may be moderated if divers adhere to dive profiles that minimize bubble development. Finally, interatrial crossover represents only one of several potential pathways for the arterialisation of bubbles. Extra-alveolar shunts and pulmonary crossover may occur independent of patent foramen ovale. The frequency and import of these pathways has not been evaluated. Further investigation is required and it is likely that prophylactic evaluation of divers will increase in the future. Evaluation may be most useful for professional divers who may be obligated to more severe exposures. The standard of care for decompression research studies is also evolving. Doppler ultrasonic monitoring for venous bubbles has served as the standard for almost 20 years. Laboratory procedures are now beginning to add two-dimensional echocardiographic imaging to identify any bubbles that may be arterialised.

GUIDELINES FOR DIVING PRACTICE

All divers or potential divers should be made aware of the hazards of patent foramen

ovale and the availability of testing options. Dive profiles should be selected to minimize bubble formation. Equalizing techniques employing the least Valsalva strain should be used.

SUMMARY

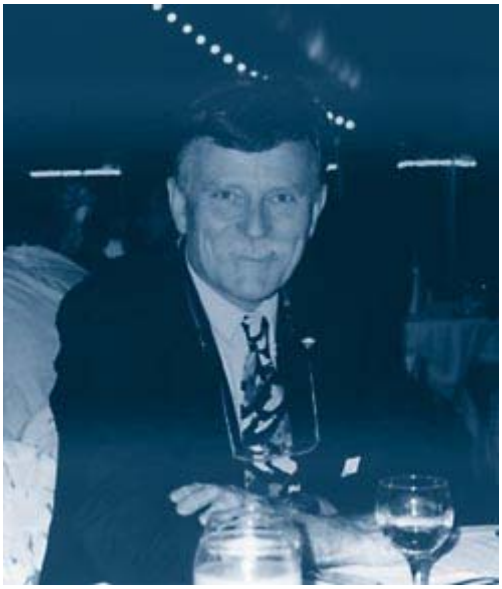
The association of serious decompression sickness and foramen ovale patency indicates a need for continued investigation. The true risk and optimal course of management remain to be determined. Current opinion is divided and standards are vague; but divers should be informed of the potential problems and testing options. In addition, divers should also be encouraged to adhere to dive profiles that can limit bubble formation.

During the last 15 or so years the practice of decompression has changed dramatically, after a long period of what might be considered as steady evolution dating from the original work of Haldane. This is a brief review of that changing landscape. This is not to say that there have not been many creative developments on the way; but to me the thing that is new, the really unique change, is the do-it-yourself capability of divers to generate their own decompression tables. Let us look at a bit of the history.

The Changing Landscape of Decompression

THIS IS THE WAY WE'VE ALWAYS DONE IT

Building on Haldane's original methods, starting in the 1930s and carrying on through the 1950s, the US Navy developed its decompression tables. This included, of course, the venerable Standard Air Tables with no-stop and repetitive diving capability, and also the technique of surface decompression where the diver leaves the water and finishes the decompression in a chamber. The partial pressure heliox tables were also developed. Commercial diving, both in the US and elsewhere, used the USN tables. Although they existed at the laboratory level, there were no operationally ready tables for use with oxygen-enriched air or O2-N2. In the early 1960s, commercial divers modified the USN heliox tables to eliminate the use of oxygen in the water (it took the Navy another 35 or so years to get around to doing that). This is all there was. In America the prevailing commercial practice was to use surface decompression with oxygen, sur-d/O2. Most commercial companies followed Navy practice, and because of peculiarities in the legal climate they were often subject to devastating litigation when divers got the bends. If the companies deviated from Navy procedures, they were likely to lose in court. SERIOUS DEVELOPMENT



Dr. Bill Hamilton

In the 1960s, the oil industry began moving offshore, and they needed diving services. Some companies and laboratories began to develop their own tables for deep heliox bounce diving using deep diving systems. A deep diving system consisted of a diving bell and a deck chamber. The divers could go to the worksite at atmospheric pressure, and then pressurize and lock out to do the work; decompression was in the bell and deck chamber, and it usually involved breathing a lot of oxygen-rich mixtures and oxygen. This system, operating in the range extending deeper than 400fsw, resulted in a lot of bends, and many companies did not have bounce tables (here a "bounce" dive is one followed by direct decompression, i.e., not saturation). In due course they were able to convince the oil companies that saturation was a better way to go, and saturation techniques took over most of the deep diving; saturation is used today for many kinds of offshore diving. Since I was involved in some of this deep bounce table development, I've been known to refer to saturation as "the magnificent cop-out." Interestingly, the saturation diver had the most status, because the job was easier and the pay was better, and the more senior divers got those assignments. But it took much more skill on the part of the divers to do the deep bell bounce diving. These were the elite. Commercial diving has remained relatively stable since the 1970s. Major surveys of the DCS outcome of North Sea offshore air diving operations showed that the highest incidences of DCS appeared to be from dives done with surface decompression, sur-d/O2. Many, many thousands of dives were surveyed, but only the outcome was looked at, not the profiles. The fact that the more stressful dives were the ones done with sur-d was lost on the authorities (so one would expect more bends from those dives), and rather than go for better tables, they ultimately shortened allowable dive times and required stressful dives to be done using "transfer under pressure" techniques. Better tables were in fact developed, for example, a set of sur-d tables done by astronaut Dr. Michael Gernhardt and Dr. Chris Lambertsen; Mike used a dynamic bubble growth algorithm for his computations. DECOMPRESSION IS EMPIRICAL

One fundamental thing about decompression development that is often overlooked is the fact that decompression tables are empirical. That is to say, they are based on experience. The outcome of yesterday's dive leads to tomorrow's table. A rather short feedback loop was used during the development of the US Navy tables, with results for certain time-depth exposures validated by perhaps six man-dives. During commercial deep heliox development, changes in the algorithm were often made based on an exposure of only two divers. Later projects used thousands of dives. The important thing is that experience is a big part of table development. This begs the question of how this experience is incorporated. This leads to the concept of a "decompression model."

What is a "decompression model?" Quantitative scientists in many fields often use mathematical "models" to describe the behavior of, say, a biological system. Such a model might, for example, describe the relationship in numerical terms of the various factors involved in control of the cardiovascular system, incorporating heart rate, exercise level, temperature, oxygen capacity of the blood, and a myriad of other factors that might allow the prediction of blood pressure under given conditions. Some readers might spot the fact that models are not limited to physical and biological science, since they are valuable in other areas such as social science or economics. The point about a model is that it can be used to predict behavior of the system when certain elements are changed. Models, incidentally, have been around a lot longer than computers, but computers have greatly expanded what can be done with them.

Decompression tables are usually generated by some sort of mathematical computation. Drawing on modeling concepts and terminology, many of those who do this sort of thing refer to the computational algorithms used to calculate tables as "models." Although they share many characteristics with true models, it is more realistic to refer to the equations solved in generating a table as something like "computational algorithms" or just "formulas" or "equations." This is not meant to lay to rest the use of the term "model" in decompression. Many models start out trying to imitate some physiological function, but in due course enough changes have to be made that it makes the resulting algorithm more mathematical than physiological, and often more practical than elegant. Experience is incorporated by the judgment of the designers. In fact, an extremely effective set of parameters dedicated to improved air tables were based on the judgment of a group of senior diving medical officers in the Swedish Navy. The values that fit this model were also found, serendipitously, to work for technical dives. In the meantime, at DCIEM in Canada, the Kidd-Stubbs pneumatic analogue device was programmed electronically (thus becoming a model), which yielded the highly regarded DCIEM tables. The Haldane type of computation can be called "deterministic." That is to say, the calculations provide a dive profile for the diver to follow, but without much in the way of a quantitative indication of the reliability, or conversely, the risk, of using such a table. A more systematic method of dealing with the probability of decompression sickness is covered below. With my long-time colleague, Dave Kenyon, I have been calculating tables for 25 or so years, using our program DCAP (Decompression Computation and Analysis Program). We tend to favor a deterministic Haldane-Workman-Schreiner algorithm, but DCAP can do many others. Bob Workman, with the US Navy, showed how to convert Haldane's ratios to M-values, making the algorithm work better for longer and deeper dives. Heinz Schreiner (who was our boss at the Union Carbide-Ocean Systems lab where Dave and I worked) devised a method for handling multiple gases. With adequate dive experience for reference, we learned to make reliable tables (most of the time!).

PROBABILISTIC DECOMPRESSION

Perhaps the ultimate incorporation of the "empirical" concept, and likely the most significant contribution to decompression science since Haldane, is the method of maximum likelihood introduced by Paul Weathersby and colleagues of the US Navy. This allows the developer to analyze a diverse set of dive profiles, to determine their basic probability of decompression sickness, PDCS, and to be able to estimate the PDCS of independent dives using the original data set as a basis. How good the estimate is

depends on many things, including how uniform the data set is, how much DCS it includes, and how closely the new dives match the data set. This method was used by the Navy to generate a comprehensive set of air tables with an even more comprehensive repetitive capability; these were intended to have a uniform PDCS throughout, which proved to be difficult. They found that to make the longer and deeper--the more stressful--dives have the same PDCS of around 2%, they had to make their decompression times unreasonably long. They ended up having to live with higher PDCS for these. Even so, because of their complexity (a book 3" thick to replace a few pages in the USN manual), and because some of the shallow dives ended up with shorter allowable no-stop times, the Fleet did not accept the new tables. So the Navy dug out an algorithm developed and tested by Dr. Ed Thalmann at the Navy Experimental Diving Unit with over 3000 man dives. This is the "Mk15/16 Real Time Algorithm" or VVAL18. It has been programmed into a dive computer for the Special Forces, and it will also be used to develop a new set of air tables; it is manufactured by Cochran. VALIDATION AND THE VALIDATION WORKSHOP

Closely linked to the generation of new tables is the need to validate them. At one time, to be "legal" and have even a slight degree of immunity to unreasonable litigation, this had to be done with laboratory simulations. This is an expensive process at best. To investigate alternatives, the UHMS held a workshop sponsored by NOAA. The Validation Workshop (Schreiner and Hamilton, UHMS, 1989) concluded that tables that were "interpolative" and referenced to known limits could be introduced as provisional under special supervision and circumstances. The judgmental decisions (such as which tables are interpolative, when are they operationally ready, etc.) are the responsibility of the developing organization, which may charge a person or small group with that duty. The Validation Workshop principles have been implemented a number of times.

FLIRTING WITH OXYGEN-ENRICHED AIR

The 1980s saw the introduction to scientific diving of a method of improving decompression by adding extra oxygen to the breathing mix. This had been working well within NOAA's diving program, but it did cause controversy when introduced into recreational diving, for reasons more political than physiological. Little new decompression work was needed for diving with OEA, since one merely finds an established air table and determines the dive depth with the same partial pressure of nitrogen, PN2, as the mix being breathed and uses that table for decompression. One important implication of this is that it started people thinking about using gases other than air for diving.

In my opinion the practice we call technical diving began when Parker Turner and his WKPP colleagues began to add helium to their breathing gas in order to dive to 240fsw or so without excessive narcosis (as would be the case with air). This became possible because special custom decompression tables could be obtained. True, people had been diving with the USN Exceptional Exposure Tables and breathing oxygen in the shallow stops, with good success. This did not eliminate the narcosis of deep air, but it met the definition of "technical diving" in that more than one breathing mix was used on a dive. The British Navy had called diving with rebreathers technical diving for half a century, but the name stuck here.

Left to Right: Bill Gavin, Parker Turner, Bill Main, and Lamar English proudly display the University of Florida's certificates of accomplishment presented by Milledge Murphy after completing the world record Sullivan traverse.
Photo ©David Rhea

Working with Parker, we learned to design tables having bottom mixes with nearly optimal oxygen and helium, and to use one or sometimes two OEA mixes as intermediate gases with oxygen at 20 and 10fsw. This is the basic pattern for technical trimix diving that is still in use today.

Just as soon as these dives became known--things in this field became known quickly through Michael Menduno's aquaCorps journal--many "gurus" began to compute tables, enabling the main principles to be exercised and tested under diverse circumstances. Virtually all the decompression experts based their calculations on the work of Prof. AA BŸhlmann, first because Prof. BŸhlmann's neo-Haldanian algorithms were reliable, but mainly because they were available in published form in his book. Not only did the gurus do tables, several of them prepared and distributed computer programs that others could use to generate (they say "cut") tables. Some are free; others have a significant cost. As far as I can see they all do the basic calculations correctly. What vary are two main things: ease of use, and how to judge the conservatism. The first is obvious to the user; the second defies easy assessment. Unless one is willing to do dozens of tables and compare them, the conservatism is hard to pin down. And even with a bunch of profiles, it often seems like comparing apples and pineapples. Such a program should keep records of what has been done and make it possible to retrieve any table. They should show what calculations were made, including what parameters were used, "J-factors," and any other relevant variables. Few if any of these programs give any hint as to the PDCS of the table. Even so, these programs represent a new wave, a new landscape, in decompression. These programs put a lot of "decompression power" in the hands of the user. In most cases, however, users are the ones who dive the tables, so they use the degree of conservatism they feel comfortable with. I just hope that the people using these programs have enough experience to know what they are doing. DEEP STOPS AND LOW SUPERSATURATIONS
Another aspect of this changing landscape is a plethora of new or resurrected

computational models. They are dealing with a somewhat anecdotal but important finding that "deeper stops" produce better profiles. Brian Hills tried to tell us about this in the 1970s, along with his "zero supersaturation" approach. Divers have been changing their ascent profiles, often arbitrarily, to include deeper stops. These in many cases have worked well, and there is now what might be called a scramble to come up with models that reflect this and permit the technique to be used universally. Many of the new approaches use one or more hypothetical bubbles (hypothetical just like Haldane's compartments) as the monitor or regulator of the ascent profile. My approach is to watch these efforts and try to take advantage of the ones that work. CONCLUSION
We are in the midst of a revolution in decompression. The tried and true methods are still working for navies and other major players, but technical divers on the cutting edge are learning how to calculate custom tables and to improve the outcome of their decompressions, and are able in many cases to do this with their own computations. New models are being used and evaluated, and it all seems to be working. This is in keeping with one of my old principles of decompression science: What works, works.

The Varying Permeability Model: A Decompression Razor

BY DR. ERIC MAIKEN

Decompression prep at Wakulla Springs. Photo ©Ron DeAmorim

The Varying Permeability Model (VPM) was originally developed by researchers at the University of Hawaii to describe laboratory observations of bubble formation and growth during depressurization of systems ranging from gelatin to aquatic animals. During the late 1980s, David Yount and Don Hoffman applied this first-principles model to calculate diving decompression tables suitable for human use (Reference 1). Nonetheless, the mathematical and scientific bases of the VPM bubble models have kept them inaccessible to all without strong technical backgrounds. So, apart from reports filtering back from the front lines, few have understood, seen or used these decompression methods. David Yount, Erik Baker and I recently collaborated to extend the VPM to repetitive and mixed gas diving as practiced by modern recreational divers (References 2, 3). An essential principle of our collaborations was that our work should be open in calculations, algorithms and code. This so that those on the line can fully access and understand our methods and assumptions--and critically assess the applicability of the methods to their personal

diving needs. Erik Baker's recently released open source code FORTRAN program incorporates the results of our work. These latest VPM methods now underpin a number of freeware decompression programs. DETAILS OF THE VPM Microscopic bubble nuclei have been observed in many types of aqueous (water-containing) media under equilibrium conditions. The VPM therefore presumes that even prior to beginning a dive, with the non-equilibrium cycle of compression and decompression, these nuclei also exist in a diver's tissues. VPM decompression procedures are designed to eliminate bubbles that grow from these seed nuclei as well as gas that is dissolved in the diver's tissues. The term "Variable Permeability" refers to the different responses of bubble nuclei to pressurizations encountered on dives deeper than approximately 9 ATA, compared to shallower dives. On deep dives, nuclei are thought to become impermeable to the flow of gas, and the VPM generates more conservative tables for these deeper dives. The VPM postulates that as a diver ascends, nuclei larger than a specific "critical" size, which is related to the maximum dive depth, descent rate, and breathing mix, will grow upon decompression. The VPM aims to minimize the total volume of these growing bubbles by keeping the external pressure large (through deep stops), and by keeping the inspired inert gas partial pressures low during decompression. The VPM uses an iterative procedure to refine decompression schedules. In each iteration step, a new ascent schedule is calculated. The total decompression time is fed back into the calculation to revise the critical gradients, and a more liberal schedule is produced at each step. This process repeats until the decompression time converges to a length that corresponds to the formation of the maximal allowable amount of free gas bubbles. The total decompression time depends on the contributions of the magnitude of the growth gradient and the time that the gradient acts to drive bubble growth. After a short dive, the tissues will off-gas rapidly to circulation. Hence, because the time that the gradient G acts is small, the magnitude of G can be increased in each VPM iteration to allow shorter and shallower stops. So, for short or no deco dives, the first and last iterations produce substantially different ascent schedules.

A divergence of the VPM from conventional calculations is in the details of how a diver's ascent is controlled. Rather than setting predefined limits (like M-values) on the maximum pressure ratio between gas dissolved in tissues and ambient pressure, ascents are limited by gradients that depend on specific details of a particular dive, which include factors such as depth, gas mix, and descent rate. The objective is to control the volume of gas that evolves in the body due to the inevitable formation of bubbles. As long as this volume is kept smaller than a certain "critical volume," it is presumed that a diver's body has the ability to tolerate the bubbles. If the volume of bubbles exceeds the critical volume, then the diver is at risk of developing DCI.

VPM decompression computations handle the in- and out-gassing of dissolved gas in a set of compartments the same way as standard dissolved gas algorithms. However, the VPM does not associate individual compartments with specific organs or tissues in the body. Parallel compartments with exponential half-times ranging from minutes to hours are used to model the body's range of time scales governing the uptake and elimination of dissolved inert gas. This is simply a method for replacing the complex reality of human metabolism under pressure with a tractable set of metrics that span the space of the body's

response time scales to pressurization and depressurization. There has been a tendency for vendors of commercial decompression software to market a large set (say, more than 16) of compartments as an enhancement to their products. This dubious exercise is counter to the responsible modeler's objective of minimizing the free parameters that relate a system's responses to measurable inputs. Whether you abide by Ocam's razor, Hogarth, or K.I.S.S., a deco modeler's guiding principle should be to eliminate arbitrary and unnecessary parameters. Towards these ends, the VPM differs from conventional dissolved gas algorithms by replacing the ascent-limiting matrix of M, or a-b values, with only five constants for mixed gas single dives, and seven for repetitive dives. These quantities correspond to measurable physical and physiological quantities, and are used to limit ascents by specifying critical over pressure gradients. In contrast to conventional M-values, which have no direct physical interpretation, gradients are related to linear gas transport and to the rate of bubble-growth via the diffusion equation.

BENCHMARKING THE VPM

It is important to note that the total decompression times generated by the VPM were forced to be similar to the (old) US Navy Standard and Exceptional Exposure Air deco times in Reference 1. However, much of the decompression time is deeper than the USN-specified depths. Presumably, a diver would evolve fewer bubbles using a VPM schedule than on the Navy table. This is, perhaps, not too comforting once you consider the risky nature of the old USN exceptional exposure tables from both bends-threshold and DCI incidence standpoints. Yount and Hoffman might just as well have calibrated the times to be similar to Bühlmann's for conservatism. Nonetheless, as detailed in Reference 3, decompression times can be increased by adjusting the VPM parameters. As an example, a moderately conservative choice for the five single-dive VPM parameters are lambda = 7180fsw min, gamma = 17.9 dyne/cm, gamma_crush = 257 dyne/cm, roN2 = 1 microns, and roHe = 0.9 microns. No-stop time limits for air diving that are generated by these parameters are tabulated below.

Wakulla Springs deco. Photo ©Jarrod Jablonski

If an ascent made with a closed oxygen window is compared to an ascent made with high ppO2 deco mixes, then not only are the stops with high ppO2 mix shortened, but so also are the preceding deeper stop times for the low ppO2 ascent. There is no analogy to this effect in a supersaturation calculation (where only the stop times when the mix is breathed are reduced). This is an artifact of the iterative procedure used in the VPM model. Because the dissolved inert gas will wash out faster when high ppO2 mixes are used in shallow stops, the total deco time is reduced-- thereby reducing all of the stop times. Practically, it is prudent to plan deco only for gas supplies that can be guaranteed to be with the diver during ascent.

Oxygen toxicity must be preeminent in dive planning. A DCI incident is inconvenient, whereas a toxicity-hit is final. Within toxicity limits, it's best to open the O2 window as wide as possible and as early as possible in the ascent (keep ppO2 high). However, given a limited number of stage gases, and short deep stop times, remember that because the volume of bubbles depends on growth time, one shouldn't sacrifice high ppO2 on the longer shallow stops for an early switch. Inert gas management is also important, withÊlong decompression dives favoring high helium content in decompression mixes to take advantage of the fast desaturation rate and deep no deco saturation depth. MIXED GAS AND THE VPM

Extension of the VPM algorithm to Trimix (or multi-inert-gas) diving was done by fractioning the in- and out-gassing of inert gases in compartments as originally described by H. Keller, who was responsible for the algorithms employed by A. Bühlmann. Weighted critical ascent gradients are formed from the partial pressures of the inert gases, and a numerical method is employed to keep the sum of the helium and nitrogen partial pressures less than the critical gradient. Although this is a conventional procedure for modeling multiple inert gasses, the physiological and physical basis of this technique is somewhat dubious because both helium and nitrogen diffuse into bubbles independently-- yet the rates of transfer of each gas are coupled through the dependence of bubble pressure on bubble volume. YOU ARE THE FUTURE OF THE VPM

With the ready availability of technical details of VPM algorithms, open source code, and freeware decompression programs, divers have essential information available to them to make judgments and modifications on this new set of decompression procedures. The VPM model is considered by many diving practitioners of deep-stop-driven ascents to be a more robust and accurate analog to physiological processes occurring within the body compared to conventional methods. After all, if you can't decompress Jello™, how can you expect to ascend from a frontier level dive? Further benefits, including greatly shortened ascent times, as well as cleaner decompressions, potentially arise from VPM-based methods. This holds true especially in cases where dives are made far beyond the tested limits of conventional procedures, where the grounding of the VPM in basic scientific observations provides leverage for extrapolation. Deep stops and the related VPM modeling continue to peak the interest of technical divers everywhere. As individuals continue their quest to obtain a reasonable approximation of decompression procedures, one can hope that continued explorations offer a sound, repeatable, and trouble-free calculation scheme. Nonetheless, in this early stage of deployment of the VPM, caution is the word. It is advisable to plan dives for ample deco and only rely on the enhanced safety of initial deep stops or theoretically shorter ascent times for insurance. For example, in the case that a direct ascent to the surface is required due to a hazardous operational situation or other emergency, you have a built-in safety backup through making an optimal ascent every step of the way home. REFERENCES

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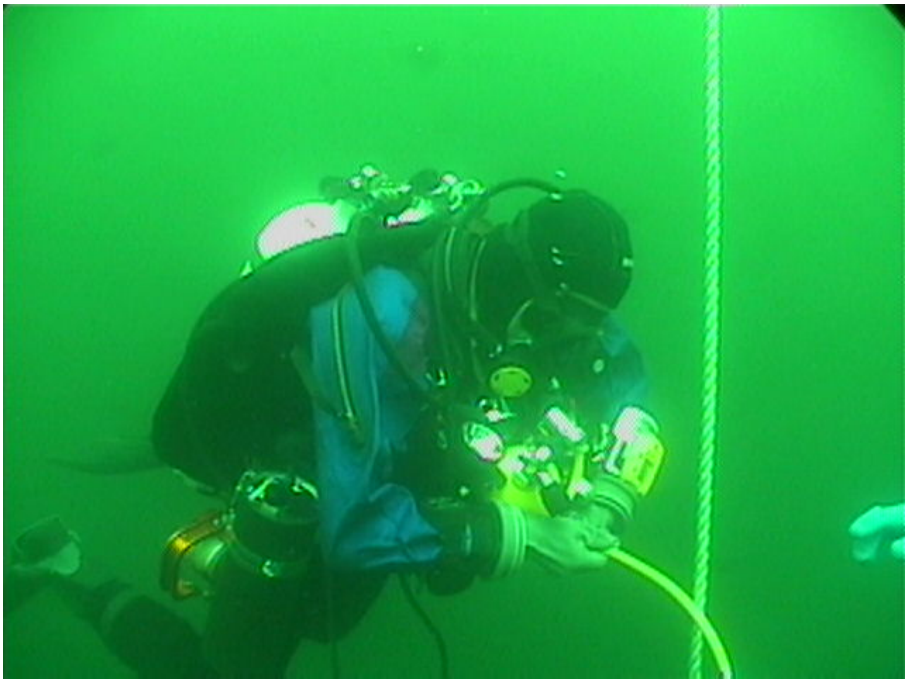
Cold Stress Complicates Decompression Risks

By Neal W. Pollock, Ph.D.



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Most scuba divers would probably recognize thermal stress as a risk factor in diving. However, due to the way thermal stress is portrayed in diving texts, many probably think that hypothermia is the only hazard. Hypothermia, however, is only part of the story.

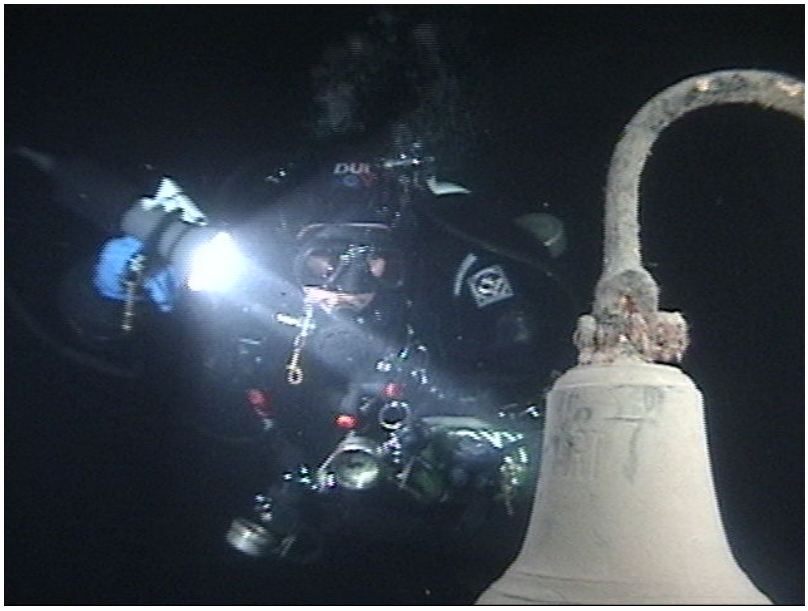
The diving environment holds many conditions that can lead to the development of hypothermia, particularly in less-than-tropical waters.

Here are a few scenarios: A lost diver could become separated from a dive boat and spend a long time floating on the surface, awaiting pick-up. An uncovered dive boat breaks down while returning from a dive site under less than ideal conditions. An extremely long-duration scientific or technical dive is conducted.

While the possibility of developing hypothermia exists in diving, it would be an exceptionally bad day for the average diver—recreational, scientific or commercial—to encounter a substantial risk. The available protective equipment effectively copes with normal circumstances. Generally, dives end by choice or due to air supply limitations before hypothermia becomes a real problem.

More subtle, and potentially more important to general diving safety, is the way in which a diver's thermal status can influence decompression risk.

The first important clarification is the difference between thermal status and environmental conditions. Divers operating in an extreme environment may be more likely to experience greater thermal stress, but they can avoid such stress if they are adequately protected.



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The thermal status (and thermally induced risk) of the diver will be influenced by protective clothing, pre-dive, during-dive and post-dive activity, and an individual's nature—i.e., body shape, body composition. These factors must be kept in mind when evaluating individual cases.

Every diver learns that inert gases—e.g., nitrogen and helium—are absorbed at increasing rates with increasing depth. Every diver is also familiar with some form of dive table and/or dive computer developed to ensure safe dive profiles.

What is often less clearly understood is that a diver's thermal status can substantially alter inert gas exchange, and that changing the inert gas uptake and elimination ultimately affects the decompression requirement for any given exposure. A dive that is safe under one set of conditions may lead to decompression illness in another.

Common misconceptions about thermal status include an expectation of the power of dive computers. First, even though dive computers may display temperature, they do not incorporate temperature measurements into the mathematical model computing decompression. Second, even if they did, they would be basing the input on the surrounding temperature and not the critical element—the thermal status of the diver.

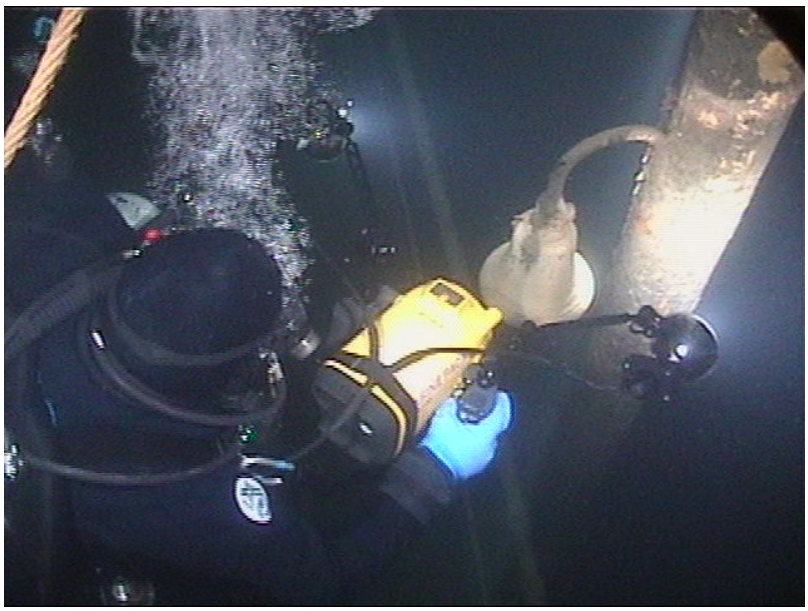
A classic field study demonstrated the ability of thermal stress to affect inert gas uptake. Participants repeated dives in both properly fitting, and in extremely ill fitting, wetsuits in the 50? F / 10? C ocean waters off Vancouver Island, British Columbia (1). Using Doppler ultrasound following the dives, researchers measured the presence and concentration of gas bubbles circulating in the blood. They observed fewer bubbles following cold dives. They concluded that when a diver is cold from the start of a dive, vasoconstriction restricts blood flow to the extremities and total gas uptake is reduced. A lowered inert gas uptake reduces the amount present at the end of the dive.

The theoretical benefits of reducing inert gas loading will be appreciated by most divers. Few, however, will be willing to spend a dive being miserable to gain the benefit. Practically speaking, many cold-water divers actively try to store extra heat before a dive to postpone the chill. This may include pre-heating in a warm room while suiting up or pouring hot water in their dive mitts before they enter the water. These practices cause an increase in tissue temperature. Warmer tissue will be better perfused (i.e., receive more blood). With maximal inert-gas uptake generally occurring during the earliest (and usually the deepest) portion of the dive, such behaviors can substantially increase gas uptake. Ultimately, this may affect the decompression requirement of a given dive.

Despite pre-dive warming strategies, a growing chill will develop as a cold-water dive continues. In many cases, the cold stress may be substantial toward the end of the dive. Under these conditions, extremity circulation may be dramatically reduced. The hands that were so well perfused at the start of the dive may be effectively isolated by the time the diver returns to the surface. Without adequate perfusion, inert gases could not be removed from the tissues of the body's extremities to the blood, and then to the lungs.

If inert gas is not removed from a region, a greater risk of local supersaturation and bubble formation exists. Even if bubbles do not initially form spontaneously, the "risk window"—the period of time when the risk of experiencing complications from decompression increases—is prolonged. This was observed somewhat accidentally in a study of cold stress following diving exposure. Following a cold exposure coupled with a dive, three of four participants developed symptoms of skin bends when taking showers long after the normal risk period expected from the dive had passed (2).

Getting into a shower or hot tub after a cold dive may sound like a wonderful method of warming up, but it can be problematic. The rapid warming of cool tissue may create problems before the extremity blood flow is increased and excess inert gas can be removed. The additional complication is that gas solubility is inversely related to temperature. Warming a nearly saturated tissue may be enough to induce bubble formation and increase the risk of developing decompression illness.



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While we made the point that it is the diver's thermal status that is critical, not the environmental conditions, the ambient conditions will generally influence what the diver experiences. A study correlating climatic and environmental factors coinciding with decompression treatment records in Great Britain demonstrated this in a novel manner (3). Controlling for as many factors as possible, the investigator documented an increased rate of decompression illness associated with days on record with lower air temperatures and higher wind chill factors.

We want to emphasize that this article does not seek to discourage cold-water diving. Its intent is to help divers appreciate some of the more subtle factors of inert gas kinetics, and evaluate diving safety in light of these effects. Eliminating the risk of injury is generally not practical, but making it as low as possible is a worthwhile goal.

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Practically speaking, several strategies may reduce decompression risk when you're diving in conditions of elevated cold stress:

• Plan dives to be more conservative. Reducing in-water times will decrease the cold stress experienced. Shorter and/or shallower dives will reduce the decompression stress for any exposure. Remember that the standard U.S. Navy dive table rule for cold dives is to calculate repetitive groups and time limits with the next greater depth AND the next greater bottom time.

• Implement longer safety stops as an inexpensive form of insurance. Light exercise during the ascent and safety-stop phases may assist in maintaining peripheral blood flow and increasing breathing rate and inert gas removal. It may also keep you from becoming more chilled. Do limit the activity to light exercise. Vigorous exercise could work against your goal by stimulating bubble formation.

• To reduce the risk of complications, minimize post-dive exertion. Remember that this caution period should be extended relative to that following a neutral or warm dive.

• Be conservative with practices for pre-dive warming. Start dives warm but try to not exaggerate peripheral circulation.

• Be conservative and delay active efforts at post-dive warming. Think about that warm shower, but choose the merits of anticipation over instant gratification.

Divers should appreciate the influence of thermal status on decompression safety. Understanding the risks and implications improves your ability to make the best choices regarding your diving health.

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Dissolved and Free Phase Gas Dynamics

BY DR. JOHNNY E. BRIAN, JR.

Photo by David Rhea

The idea that decompression stops deeper in the water column can allow for more efficient inert gas removal seems very counter intuitive. Traditional Haldane-based decompression theory emphasizes the need to move as shallow as possible to maximize gas removal from tissue. Haldane-based models assume that all gas remains in dissolved phase, where the gradient for inert gas removal is the partial pressure of the inert gas in the tissue (determined by the breathing mix and the time/depth profile) and the partial pressure of inert gas in blood (determined by the breathing mix and the current depth). In a dissolved gas model, formation of bubbles is assumed to indicate a violation of allowed supersaturation ratios. Today we know that bubbles are very common, and that phase transition (gas moving from dissolved phase to free phase in bubbles) should be considered in decompression theory. The gradients for gas movement are very different once gas leaves the dissolved phase and enters the free phase, which leads to the need for stops much deeper than predicted based on dissolved gas theory.

LeMessurier and Hills were the first to propose that free phase gas elimination might be an important component of decompression (LeMessurier and Hills, 1965). They based their theory on observations of Japanese shellfish divers who made repetitive dives to 200-300 FSW, and developed decompression procedures without knowledge of pre-existing theory. The divers used decompression stops that were much deeper than any tables in use at the time (1965), and the divers would surface directly from 30 to 40 FSW. The explanation that LeMessurier and Hills developed for why such decompression procedures worked was that gas was being removed from bubbles, and if the bubbles were efficiently eliminated, the need for the shallow stops was greatly reduced or eliminated.

DISSOLVED PHASE GAS DYNAMICS

When gas remains in the dissolved phase, tissue offgassing is controlled by the dissolved gas partial pressure difference between the tissue and blood. The amount of dissolved gas in tissue or blood is expressed as a "partial pressure" value in units of pressure (mmHg, FSW, etc.). This terminology is somewhat unfortunate, as it can mislead divers to think that gas in solution exerts pressure as does gas in cylinders. This

is not true, as gas that is dissolved in liquid (tissue or blood) is dissolved as salt dissolves in water, and does not exert a pneumatic "pressure." Partial pressure of a gas in liquid means that to dissolve a given amount of a gas in a liquid will require exposure of the liquid to a specific pressure of the gas in the gas phase. The amount of gas that dissolves in the liquid depends on the intrinsic properties of the gas and the liquid (i.e., how soluble the gas is in the liquid) as well as the temperature. For example, if we expose one liter of water at 37°C (body temperature) to 1 ATA of nitrogen, 14.4 milliliters of nitrogen will dissolve in the water. The dissolved nitrogen would also be described as having a partial pressure of 1 ATA, which is one way of expressing the amount of nitrogen dissolved in the water. Movement of dissolved gas is driven by diffusion, and not the pressure gradients that we are familiar with that drive bulk gas flow, as in fill whips and cylinders. Diffusion is the random movements of atoms and molecules, and transfer of a species from one place to another is driven by the probability that more molecules will move from an area of higher concentration to an area of lower concentration than visa versa. It is the movement of independent gas molecules during diffusion that causes gas transfer, rather than the bulk movement of many gas molecules driven by pressure differentials.

Following any given dive, the amount of gas (partial pressure) dissolved in a tissue is determined by the tissue half-time, the concentration of inert gas in the breathing mix and the depth/time profile. The gradient for dissolved gas to move out of the tissue is determined by the partial pressure of inert gas in the tissue and the partial pressure of the inert gas in blood. The partial pressure of the inert gas in blood is determined by the partial pressure of the inert gas in the breathing mix and the current depth (ambient pressure). Because depth controls the partial pressure of inert gas in blood, depth controls the gradient for dissolved gas to move from tissue to blood. Haldane's theory was to reduce depth to a minimum to maximize the gradient between tissue and blood short of bubble formation. Gas dissolved in solution is not governed by the pressure-volume gas law for gas in free phase. Dissolved gas can exist at less than ambient pressure (undersaturation) or greater than ambient pressure (supersaturation). Haldane's theory evolved to the M-values developed by Workman which define the dissolved gas supersaturation allowed for each of the tissue half-times (Workman, 1965).

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Figure 1A shows an example of dissolved gas gradients after a dive to 132 FSW on air where there has been saturation of a tissue with nitrogen. The line depicted with squares is the nitrogen partial pressure gradient between a tissue saturated with the nitrogen fraction in air at 132 FSW and nitrogen in blood during ascent to the surface while breathing air. As a diver ascends in the water column, the gradient becomes larger as the partial pressure of nitrogen in the lung is reduced, which reduces the partial pressure of nitrogen in blood. Nitrogen partial pressure in blood is determined by the fraction of nitrogen in the breathing mix and ambient pressure. If the diver continues to breath air to the surface, the only factor that decreases the amount of dissolved nitrogen in blood is the reduction of ambient pressure. This is the basis for dissolved gas models where the underlying idea is to maximize the dissolved gas partial pressure differential between tissue and blood by maximizing the pressure reduction.

BUBBLE PHASE GAS DYNAMICS

When gas moves from the dissolved phase into the gas phase (phase change), gradients for gas movement become quite different. Unlike dissolved gas, gas partial pressure in bubbles is governed by ambient pressure. Because bubbles can expand and contract, they will change size as ambient pressure is altered, which also alters the partial pressure of the gases in the bubble. The movement of gas from the bubble into the dissolved phase is determined by the partial pressure of the gas in the bubble and the partial pressure of the gas in the surrounding tissue (remember, the surrounding tissue could be blood). During ascent to the surface, a bubble will expand, reducing the pressure in the bubble, which also reduces the nitrogen partial pressure in the bubble. Bubbles exist at somewhat above ambient pressure because of the surface tension of the bubble. Hills derived the formula for the gradient between a bubble and surrounding tissue (in mmHg pressure) (Hills, 1970):

$$\text{Gradient} = (\text{Ambient Pressure } (1\text{-}X)) + (47\ X) - 133)$$

where X is the fraction of inert gas in the breathing mix. Similar analysis of gradients can be found in the work of Van Liew and Wienke (Van Liew et al., 1965; Wienke, 1987). The line depicted with circles in Figure 1A is the gradient for gas absorption from a bubble that forms on ascent to the surface. If the diver were to ascend from the bottom at 132 FSW and stop at 99 FSW (80% of the maximum pressure absolute), the gradient from a bubble would be approximately equal to the dissolved gas gradient, shown by the line with squares (24-26 FSW). As the diver ascends from 99 FSW to a shallower depth, however, the gradients diverge with the gradient between the bubble and tissue being reduced as the bubble expands during ascent to the surface. The bubble expands as ambient pressure is reduced, also reducing the partial pressure of gases in the bubble.

The line depicted with triangles in Figure 1A is the oxygen window, which is formed by the metabolic consumption of dissolved oxygen that is incompletely replaced by carbon dioxide. Because more oxygen is transported in the dissolved phase as depth increases, the oxygen window also increases. The gradient for inert gas absorption from a bubble parallels the oxygen window (Figure 1A), because the oxygen window is the primary determinant of the nitrogen gradient between the bubble and tissue. A bubble gradient equation derived by Van Liew better illustrates the importance of the oxygen window in determining the absorption of inert gas from a bubble (Van Liew et al., 1965). The partial pressures of nitrogen inside of a bubble can be defined by subtraction of all of the gas partial pressures other than nitrogen from ambient pressure:

$$P_{bubble}N_2 = P_{ambient} - P_{tissue}O_2 - P_{tissue}CO_2 - P_{tissue}H_2O$$

It is assumed that the tissue and bubble partial pressures are in equilibrium for oxygen, carbon dioxide and water. In a similar fashion, the pressure of nitrogen in the arterial blood is defined by:

$$P_{blood}N_2 = P_{ambient} - P_{blood}O_2 - P_{blood}CO_2 - P_{blood}H_2O$$

With the gradient between bubble and blood as:

$$P_{bubble}N_2 - P_{blood}N_2 = (P_{ambient} - P_{tissue}O_2 - P_{tissue}CO_2 - P_{tissue}H_2O) - (P_{ambient} - P_{blood}O_2 - P_{blood}CO_2 - P_{blood}H_2O)$$

Which reduces to:

$$P_{bubble}N_2 - P_{blood}N_2 = (P_{blood}O_2 - P_{tissue}O_{2)} + (P_{blood}CO_2O - P_{tissue}CO_2)$$

This is the equation for the oxygen window. Values and gradient calculations from the above Hills and Van Liew formulas can be found in Table

The oxygen window determines the gradient for nitrogen across the bubble because the window determines the amount of nitrogen in blood - i.e., the larger the window, the less nitrogen there will be in blood and the larger the gradient will be from bubble to tissue. The bubble must exist at ambient pressure, and the additional partial pressure not occupied by oxygen, carbon dioxide or water vapor inside of a bubble, is composed of nitrogen. Because tissue partial pressure of oxygen is significantly below the partial pressure of oxygen in arterial blood, this means that the nitrogen partial pressure inside of a bubble will always be greater than nitrogen partial pressure in tissue by the value of the oxygen window - i.e., the space in the bubble not occupied by oxygen will be filled with nitrogen. As the oxygen window increases or decreases, so will the nitrogen gradient between the bubble and tissue.

Figure 1B shows the same gradients as 1A, but instead of breathing air to the surface, in this example the diver switches to 40% nitrox at 99 FSW. The oxygen window, shown by the line with triangles, increases at 99 FSW due to the gas switch, and then declines as ambient pressure is reduced. Both the dissolved gas gradient (squares) and the bubble gradient (circles) are shifted upward by the increase in the oxygen window.

One factor that may not be fully evident in calculation of inert gas gradients from bubbles to tissue is that the tissue surrounding a bubble cannot exist in a state of supersaturation as with dissolved gases. When a bubble forms, dissolved gas in supersaturation will move from the dissolved phase into the bubble. The dissolved gas gradients presented in Figures 1A and 1B are large because of the assumption that the tissue remains supersaturated with the nitrogen fraction of air at 132 FSW. The gradients for free phase gas (bubbles) are based on the tissue surrounding the bubble having a dissolved

nitrogen fraction determined by the inspired fraction of nitrogen and the current ambient pressure. Thus, the partial pressure of nitrogen in the bubble and the surrounding tissue is reduced during ascent to the surface. Any nitrogen present in the tissue above ambient pressure (supersaturation) simply moves into the bubble, and the bubble expands.

Figure 2A. Bubble and tissue gas partial pressures during air breathing at 99 FSW.

Figure 2A shows gas partial pressures in a tissue with a bubble and the surrounding tissue saturated with air at 99 FSW. This example ignores the increase in pressure inside of the bubble due to surface tension for simplicity. The nitrogen partial pressure in the bubble (125.8 FSW) exceeds the nitrogen partial pressure in the surrounding tissue (102.6 FSW) by 23.2 FSW. The oxygen window is the difference between the oxygen partial pressure in arterial blood (25.2 FSW) and the oxygen partial pressure in tissue (2.2 FSW), or 23 FSW, equal to the gradient for nitrogen from bubble to tissue. In Figure 2B, the oxygen window is increased by breathing 40% oxygen at 99 FSW. By increasing the partial pressure of oxygen in arterial blood, there will be less nitrogen in arterial blood, and less nitrogen in tissue. However, the oxygen in tissue changes only slightly, so that the amount of nitrogen in the bubble is unchanged, leading to a larger gradient for nitrogen from the bubble to tissue.

Inspection of Figures 1A and 1B should emphasize why increasing the oxygen window is important for both removal of dissolved and free phase inert gas during decompression. The above examples were applied to nitrogen-based dives for simplicity of presentation, but are equally applicable for other inert gases and combinations of inert gases.

Breathing, Aerobic Conditioning and Gas Consumption

BY JOHNNY E. BRIAN, JR., M.D.

Breathing is an essential part of life. For divers, breathing is of special interest, as it determines the duration of our gas supply. The majority of lung ventilation is regulated without conscious input, being governed by the need for oxygen and the elimination of carbon dioxide (CO_2). However, with aerobic conditioning, divers can influence lung ventilation and reduce their gas consumption. Diving is a sport focused on gas consumption. Understanding how aerobic conditioning affects the respiratory system allows us to become better, more informed divers. Aerobic conditioning is a complex, multifactoral process, of which the respiratory system is only a part. For the purposes of this article, I will address only the respiratory component of aerobic conditioning. To understand these complex events, the best place to start is with basic respiratory physiology.

RESPIRATORY PHYSIOLOGY At rest and during exercise, we breathe to deliver oxygen to and eliminate CO_2 from our bodies. Tidal volume (TV) is the amount of gas inhaled and exhaled with each breath, and averages 6 to 8 milliliters (ml) per kilogram (kg) of body weight (see Table 1). In a typical 70 kg diver, this results in a TV of approximately 500 ml. (Divers more familiar with English units should consult the appendix to convert English to metric units.) At rest, we breathe 12 to 16 times a minute, which results in a minute ventilation (MV) of 6 to 7 liters (L) per minute (TV x rate). At rest, when oxygen consumption is minimal, the need to eliminate carbon dioxide (CO_2)

controls ventilation. When CO2 dissolves in water, it forms carbonic acid (H2O + CO2 = H2CO2), which reduces the pH (a measure of acidity and alkalinity) of the solution. Since our bodies are principally composed of water, changes in CO2 profoundly influence the pH of our bodies. Under resting conditions, what drives our ventilation is CO2-mediated change in brain pH. Normal arterial partial pressure of CO2 is 35 to 45 mmHg. The respiratory control system in the brain is very sensitive to CO2-mediated change in brain pH. In healthy individuals at rest, arterial partial pressure of oxygen averages 95 mmHg. To produce significant respiratory drive, arterial partial pressure of oxygen must be less than 60 mmHg, rare in healthy individuals. Although the arterial CO2 directly controls ventilation, this does not imply that ventilation is independent from oxygen consumption. On an average, 80% of oxygen consumed is converted to CO2 (the remaining 20% is converted to water), so that as oxygen consumption increases, CO2 production also increases, which drives ventilation to supply more oxygen and eliminate more CO2. At rest, our body's metabolism is minimal, and the cells of our body carry on basic metabolic processes to sustain themselves. The actual type of cell is less important than the number of cells. Most studies of aerobically fit individuals indicate that, at rest, oxygen consumption is not different than in unconditioned individuals when body mass is considered. Not surprisingly, minute ventilation at rest also is not different between conditioned an unconditioned individuals. Thus, at rest, the total amount of gas we breathe is primarily determined by body mass. At rest, any given gas supply will last longer for a smaller individual.

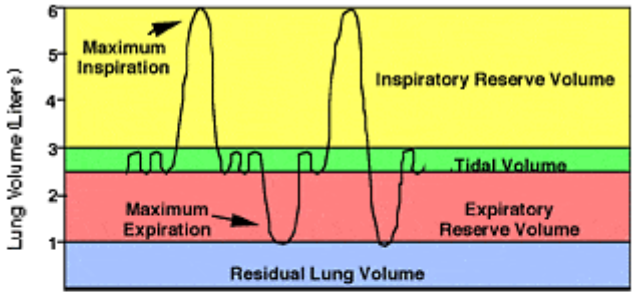

Figure 1. Respiratory volumes during normal breathing and with maximal inspiration and expiration. The black line depicts gas flow in and out of the lungs. Gas volume moved during normal breathing is the tidal volume shown in the green area. The inspiratory reserve volume in yellow is the additional gas volume that can be inhaled after a normal inhalation. The expiration reserve volume is shown in red and is the additional volume of gas that can be exhaled after a normal exhalation. The vital capacity is the volume of gas from maximal inhalation to maximum exhalation. The residual volume in blue is the gas that remains in the lungs ever after maximal exhalation.

Figure 1

When we breathe under resting conditions, we use only a small part of our total lung volume. Our lungs have significant inspiratory and expiratory reserve volumes; so, not only can we take deeper breaths (inspire), we can also exhale (expire) more gas than normal. Figure 1 diagrams these volumes for an average 70 kg person, who would have a one-half liter of tidal volume, 3 liters of inspiratory reserve volume and 1.5 liters of expiratory reserve volume. The total amount of gas that can be moved with a maximal inhalation followed by maximal exhalation is the vital capacity, which is 5 liters in Figure 1. The residual volume is the gas that cannot be expelled even with maximal exhalation, and is 1 liter in Figure 1.

Ventilation increases with exercise, but factors responsible for regulating ventilation during exercise are poorly understood. With the onset of exercise, ventilation increases before arterial CO2 can increase; in general, arterial O2 does not decrease with exercise. The overall regulation of ventilation during exercise likely results from

integration of increased input from peripheral receptors (movement of joints, muscles), and central input (we know we are exercising, and must increase our ventilation). During light and moderate levels of exercise, arterial partial pressures of O2 and CO2 are maintained at resting values. During exercise approaching maximal values, arterial CO2 decreases. During extreme exercise in elite athletes, arterial O2 partial pressure may also decrease.

In an aerobically conditioned individual, light and moderate levels of exercise result in an increase in tidal volume and little to no increase in the respiratory rate. We increase our tidal volume by using some of the lung reserve volume, principally the inspiratory reserve volume. Taking deeper breaths is advantageous, as deeper breaths increase the efficiency of respiration. With each breath, some gas always remains in the airways that conduct gas to the gas exchange portion of the lungs. The gas in the conducting airways does not undergo gas exchange, and thus is "wasted" or "dead space" ventilation. In essence, the oxygen in dead space gas is unavailable for gas exchange, and is thus "wasted" ventilation. On an average, dead space ventilation averages 2.2 ml/kg, which would be 150 ml in our example 70 kg person. This means at rest, 150 ml of each 500 ml breath, or 30% of inhaled gas, does not undergo gas exchange. For a 7 L minute ventilation, this means that 2.1 L of gas breathed each minute does not contribute to gas exchange.

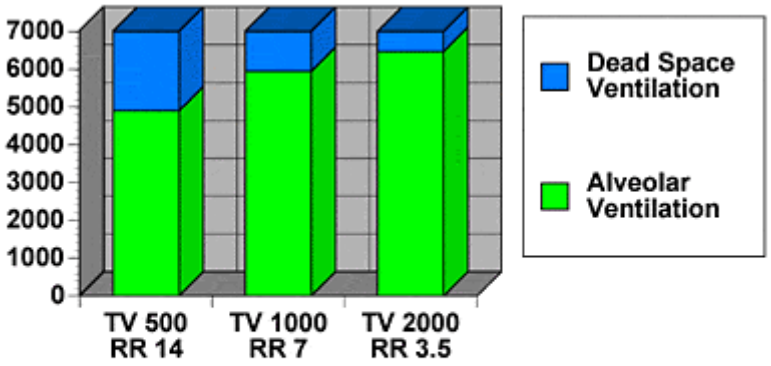

Figure 2. Relative distribution of alveolar and dead space ventilation during minute ventilation of 7 L/min with various tidal volumes (TV) and respiratory rates (RR). As TV increases and RR decreases, the relative amount of "wasted" dead space ventilation declines.

Figure 2

Dead space ventilation is relatively fixed and does not change when tidal volume changes. Larger breaths, however, increase breathing efficiency by reducing the percentage of dead space ventilation. For a 1000 ml breath, the dead space ventilation would remain 150 ml, but the percent dead space ventilation would decrease to 15%. In this case, the amount of gas breathed per minute that does not undergo gas exchange is reduced to 1.05 L. In Figure 2, a minute ventilation of 7 L/min is divided into increasing tidal volumes with decreasing respiratory rates, so that the total ventilation per minute remains constant. With larger and larger tidal volumes, the percentage of dead space ventilation progressively decreases. The important effect of reducing dead space ventilation is to allow more inhaled gas to undergo exchange with the blood, and

increase the oxygen available to blood. Taking fewer, larger breaths is more efficient than fewer, smaller breaths, as oxygen used by the respiratory muscles is only very slightly increased for larger breaths. The increase in breathing efficiency from larger and larger breaths is eventually offset by the increasing requirement for oxygen to support the work of breathing. At some point, the metabolic cost of taking a larger breath is not offset by increased oxygen delivery into the system.

Almost everyone will arrive at the most efficient combination of respiratory rate and tidal volume during the course of aerobic training. During maximal exercise, elite athletes usually have tidal volumes of approximately 60% of vital capacity. The important point for divers is that taking deeper breaths is key for increased ventilatory efficiency during exercise. Restriction of our ability to take deeper breaths, as from restrictive regulators, suits, or harnesses, will reduce our ability to maximize ventilatory efficiency.

One of the important respiratory adaptations of aerobic cond-itioning is that less minute ventilation is required at any given exercise intensity (oxygen consumption). In other words, fit people will always breathe less gas to support a given level of exercise compared to unconditioned people. Resting oxygen consumption is approximately 3.5 ml/kg/min, or about 250 ml/min in a 70 kg individual. The ventilatory equivalent is the volume of gas breathed to extract a given amount of oxygen. At rest, and during submaximal exercise, the ventilatory equivalent averages 20 to 25 L of gas for each 1 L of oxygen consumed. One of the hallmarks of aerobic conditioning is that the ventilatory equivalent is reduced over the course of training. With progressive aerobic training, less gas is breathed to support a given level of exercise. This means that less respiratory work is required to maintain a given level of exercise, and less oxygen will be consumed by the respiratory muscles. With aerobic conditioning, there are other important cardiac and circulatory adaptations that also affect the efficiency of oxygen use. (The cardiovascular component of aerobic conditioning will be addressed in a separate article.)

Aerobic conditioning is obviously much more than learning to take deeper breaths. There are other adaptive changes in the respiratory system that influence gas utilization. We are dependent on respiratory muscles to move gas in and out of the respiratory system. Our ability to sustain exercise is determined in part by the ability of the respiratory muscles to sustain work and gas movement. Aerobic conditioning increases the aerobic capacity of respiratory muscles, just as it does for skeletal muscles. During aerobic conditioning, the oxidative enzyme capacity of respiratory muscles increases, which increases the ability of the muscles to metabolize oxygen. This means that the point at which respiratory muscles convert from aerobic to anaerobic metabolism is moved to a higher work level. Thus, aerobic conditioning of respiratory muscles allows greater gas movement per minute without accumulation of blood lactate derived from the respiratory muscles. Aerobic conditioning of respiratory muscles also increases the strength of the muscles, which allows generation of larger, more sustained pressure gradients to move gas in and out of the airway. These changes together allow more sustained aerobic activity of the respiratory muscles, which helps protect them against fatigue. Swimming offers a unique advantage in enhancing respiratory muscle function. Because the respiratory muscles must work against the added load of water compressing the chest, the respiratory muscles must generate more force with each breath. Thus, the respiratory muscles are strengthened to an added degree during swim training.

Lung volume is related to body size, with larger individuals having larger lungs. Lung volume also determines the surface area for gas exchange. One might think that having a larger lung volume and surface area for gas exchange would be advantageous during aerobic exercise. However, this does not appear to be the case, as marathon runners have lung volumes no different than size-matched sedentary individuals. The greater lung surface area found in larger individuals is likely necessary to support the needed gas exchange for a larger body mass. Swimmers, however, do have larger lung volumes than size-matched non-swimmers. Because of the restriction imposed on ventilation by swimming, it is advantageous to have a larger lung volume, which allows more oxygen to be held in the lungs for exchange during the periods between breaths. The larger lung volumes found in swimmers may be an adaptive change to breath-holding. Measurement of lung volumes in individuals who participate in breath-hold diving demonstrates larger lung volumes after one year of repetitive breath-hold diving. In compressed gas diving, if off-gassing during decompression is diffusion-limited, then a larger surface area for gas exchange might be advantageous during decompression. This has not been tested experimentally, and would likely be difficult to evaluate due to the multifactorial nature of decompression. However, most decompression models are perfusion limited, and not diffusion limited. The respiratory, cardiovascular and muscular systems function as an integrated system during exercise. It should not be surprising that the reduced respiratory requirements that occur with aerobic training appear to be specific to the type of training. This means that the reduced ventilatory requirements that occur with aerobic training by running, for example, do not fully transfer to other types of exercise. Thus, to obtain the maximal benefits from aerobic conditioning, one should include a variety of exercise types in their training program. To train for diving, swimming has obvious advantages, as it conditions the muscle groups used during diving, and has added benefits in conditioning of the respiratory muscles. SUMMARY

The respiratory effect of aerobic conditioning includes both a "learned" and a physiologic component. We innately learn to breath more efficiently during aerobic conditioning, and through continued aerobic exercise arrive at a breathing pattern that is most efficient. With conditioning, our respiratory muscles develop increased aerobic capacity, which increases the threshold for the onset of anaerobic metabolism and lactate accumulation in the muscles. There is no magic to aerobic conditioning, just the time and effort.