Carbon Dioxide, Narcosis and Diving

Johnny E. Brian, Jr., M.D.
Associate Professor
Department of Anesthesia
University of Iowa
Iowa City, IA

Carbon dioxide (CO_2) is the gaseous end product of aerobic metabolism of oxygen. CO_2 is highly soluble in body tissues, and readily diffuses from cells to blood where the 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, so 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 35 to 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 dissolved 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 (P_ACO_2) and blood PCO_2 reach 40 mmHg. The volume of gas breathed per minute (minute ventilation) controls removal of CO_2 from 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, including total loss of consciousness. In humans, acute elevation of arterial PCO_2 above 70 to 75 mmHg reduces the level of awareness (20) and $PaCO_2$ above 100 to 120 mmHg produces unresponsiveness (26). Severe elevation of $PaCO_2$ by inhalation of 30% to 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 produce 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, which indicates a high degree of correlation between lipid solubility and anesthetic potency. Xenon and nitrous oxide have approximately equal lipid solubility as CO_2. If the anesthetic effect of CO_2 was produced only by lipid solubility, then the CO_2 point should lie along the line with the nitrous oxide and xenon points. CO_2, however, falls below the line, which means that anesthesia is produced by a

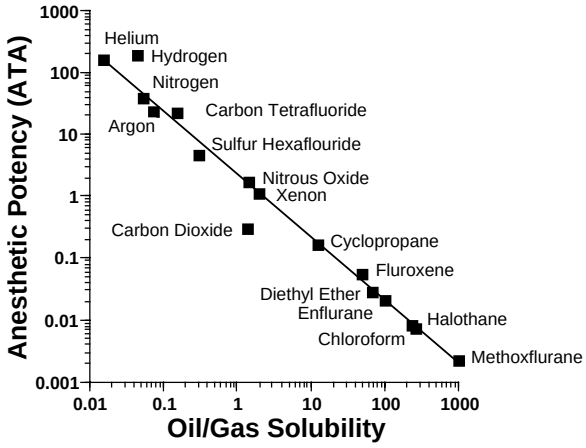


Figure 1

lower partial pressure of CO_2 than would be predicted from the lipid solubility. The anesthetic potency of CO_2 is about 130 times nitrogen, much greater than the ratio of lipid solubilities of CO_2 and nitrogen. This suggests that CO_2 produces an anesthetic effect independent of lipid solubility.

Elevation of CO_2 has been associated with decreased level of consciousness during hyperbaric chamber and wet dives. Case and Haldane used inspired CO_2 to elevate arterial PCO_2 at 1 ATA and during hyperbaric exposures to 300 FSW in human volunteers (1). Most data reported in this study are subjective impressions, and 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/O_2 dive (1). At 1 ATA, 6 to 8% (45 to 60 mmHg) inspired CO_2 produced a marked increase in respiration, but little change in mental or physical skills (1). Although Case and Haldane did not measure arterial PCO_2, 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 CO_2 at 300 FSW, which suggests that the subjects were unable to increase minute ventilation to an equal level as during the 1 ATA exposure. This can only mean that the increase in arterial PCO_2 was more severe at 300 FSW than at 1 ATA. At 300 FSW during inspired CO_2, there was severe impairment of both mental and physical skills. Subjects noted that the “narcosis” was much more severe than exposure to air alone at 300 FSW. When inspired CO_2 was increased to 0.8 to 0.9% at 300 FSW (which equals 8 to 9% at 1 ATA; 60 to 70 mmHg), subjects quickly lost consciousness and some seized. Subjects were described as lapsing into unconsciousness “quietly and easily”. Although PCO_2 was not measured, arterial

PCO_2 was likely greater than 80 to 100 mmHg at 300 FSW. Case and Haldane theorized that the respiratory response to CO_2 was suppressed by nitrogen narcosis.

Warkander et al. studied CO_2 accumulation during exercise at 6.8 ATA, and reported 2 subjects that required rescue from a wet pot due to severe CO_2-induced incapacitation (24). Both subjects had elevation of arterial PCO_2 above 80 to 90 mmHg, and both were unaware of their incapacitation. In the same study, other subjects continued to function with similar elevation of arterial PCO_2. This suggests that CO_2-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 CO_2 in volunteers under normobaric and hyperbaric conditions (6, 7). They found that a modest increase of PCO_2 to 50 to 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 CO_2 was additive, but not synergistic with, nitrogen narcosis. Fothergill et al. also studied the effect of PCO_2 elevation to 50 to 60 mmHg on a battery of mental tests in volunteers, reporting that the modest increase in PCO_2 reduced the number of correct responses principally by reducing the number of attempts at the tests (4). This suggests that increased PCO_2 slows comprehension of presented information. These data also suggest that modest elevation of PCO_2, which may occur during diving, may contribute to “narcosis”, independent of elevation of PN_2.

Although Case and Haldane theorized that narcosis limited the respiratory response to CO_2, it is the increase in gas density, and not 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 and 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.

A common method to measure the respiratory response to CO_2 is to allow a subject to breathe CO_2 and measure the increase in lung ventilation. Nitrox at 4 ATA attenuates the increase in lung ventilation with inspired CO_2, and reducing gas density with He/O_2 restores the CO_2 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 CO_2 during exertion may be significantly limited by increased gas density, and maintenance of a normal $PaCO_2$ may not be possible when breathing dense gas.

Elevated CO_2 is a normally a potent respiratory stimulus and under normobaric conditions, causes increased respiratory rate (hyperventilation) and the sensation of shortness of breath. Further elevation of PCO_2 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 reported that the sensations of hyperventilation and shortness of breath may not be noted (24). It is possible during diving that CO_2-induced dizziness could be mistaken for nitrogen narcosis. Although increased CO_2 is normally a potent respiratory stimulus, elevation of PCO_2 to levels associated with a decreased level of consciousness (100 to 200 mmHg or greater) progressively depresses respiration (18, 19). Thus, severe elevation of $PaCO_2$ will cause further CO_2 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, and table 2 lists the composite densities of diving mixes. The density of other mixes can be calculated by summing the fractional gas densities of the mix, and multiplying by the depth in ATA. 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 CO_2 should be very similar. Oxygen is slightly denser than nitrogen, so substitution of oxygen for nitrogen slightly increases mix density relative to air.

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

Table 1. Gas densities at 37°C

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

Table 2. Mix densities at 37°C

At rest while breathing nitrox at 4 ATA, PCO_2 is normal indicating adequate ventilation to eliminate CO_2 (10). During exertion, however, the increase in lung ventilation is less than occurs at 1 ATA, and PCO_2 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, PCO_2 must increase. The response of ventilation and PCO_2 to exercise during 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 breath from demand valve regulators, which may impose additional work of breathing. During air breathing 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 ft^3/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 ft^3/min with a low resistance breathing circuit. Real life in water diving is usually conducted under less than optimal conditions and it should be expected that the maximal possible minute ventilation will be less. Divers swimming at 50 to 60 ft/min require a ventilation rate of about 0.6 ft^3/min or less (13). Experience indicates that breathing less than 1 ft^3/min of gas during technical and cave diving is tolerated without symptoms of CO_2 accumulation. However, gas consumption increasing above 1 ft^3/min, especially as gas consumption approaches 2 ft^3/min, there is increased likelihood of CO_2 accumulation and resultant deleterious effects.

A number of studies have reported that divers have an abnormal respiratory response to CO_2 (8, 9, 12, 17). Lanphier reported that US Navy divers swimming at about 75 ft/min exhibited abnormal elevation of PCO_2 that averaged 55 mmHg (12, 13). The study subjects were hard had divers, in whom inadequate helmet ventilation often causes

CO_2 rebreathing. These divers were later exercised at 1 ATA, where they also exhibited marked and abnormal elevation of PCO_2 (12). Lanphier theorized that chronic CO_2 rebreathing in these divers led to CO_2 insensitivity. However, Kerem et al. studied open circuit scuba divers, and also reported a reduction of the respiratory response to CO_2 (8). Similar findings were reported by Sherman et al. (21). These findings suggest that chronic CO_2 rebreathing is not required for a diver to develop a depressed respiratory response to CO_2. The depressed respiratory response to elevation of CO_2 appears to vary greatly between individuals, with some divers being normal and other having a very depressed CO_2 response (12, 16). Divers may consciously reduce their rate of ventilation to conserve gas, which would lead to CO_2 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 CO_2, but only testing under hyperbaric conditions was successful (13). The existence and prevalence of impaired CO_2 response in cave and technical divers is not known.

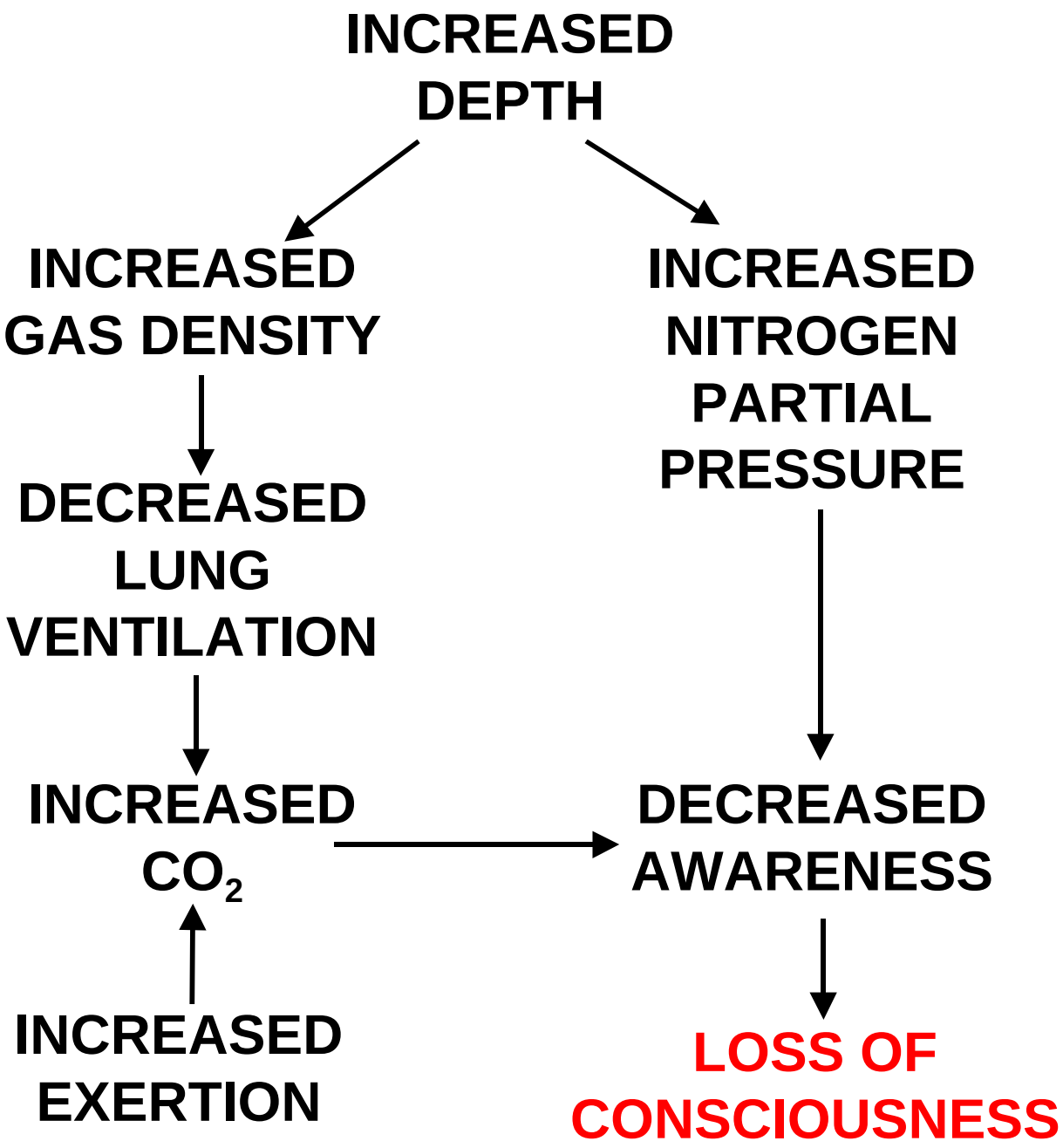
Scuba regulators can add additional resistance to breathing, limiting the ability to eliminate CO_2. Almost all studies of respiratory dynamics at depth are conducted under optimal respiratory conditions. There has been very limited study of the effect of demand valve regulators on the ability to breathe at depth. Breathing air at 4 ATA significantly reduces the maximum possible minute ventilation, and 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 PCO_2 during He/O_2 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 CO_2 retention (12). The overall impact of breathing through a modern, well-maintained scuba regulator on the response of PCO_2 to exercise is unknown. However, breathing resistance should be kept to a minimum to reduce the possibility of CO_2 retention.

The primary cause for CO_2 elevation during diving is exertion coupled with increased gas density. Stress increases the metabolic rate and can contribute to increased CO_2 production. Rebreathing expired gas containing CO_2 will also elevate PCO_2. 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 CO_2 by rebreathing, as can rebreathers due to malfunction of the one way valves or exhaustion of the CO_2 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 PCO_2. Increased CO_2 impairs mental and physical skills and may hamper self-rescue. Severe elevation of CO_2 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 CO_2 retention without being aware of their life-threatening situation. Elevated CO_2 also increased the likelihood of hyperoxic seizures.

If a diver experiences symptoms of elevated CO_2, they should stop their exertion and relax, if possible. This will reduce CO_2 production, and should allow time for the ventilation to eliminate the excess CO_2. 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 CO_2. Incapacitated but breathing divers should also be taken to a shallower depth for the same reason. Elimination of excess CO_2 and recovery of consciousness may be possible once gas density is reduced.



References

1. Case, E. M., and J. B. S. Haldane. Human physiology under high pressure I. effects of nitrogen, carbon dioxide and cold. *J. Hyg.* 41: 225-249, 1941.

2. Eisele, J. H., E. I. Eger, II, and M. Muallem. Narcotic properties of carbon dioxide in the dog. *Anesthesiology* 28: 856-865, 1967.

3. Fagraeus, L. Maximal work performance at raised air and helium-oxygen pressures. *Acta Physiol. Scand.* 91: 545-556, 1974.

4. Fothergill, D. M., D. Hedges, and J. B. Morrison. Effects of CO_2 and N_2 partial pressure on cognitive and psychomotor performance. *Undersea Biomed. Res.* 18: 1-19, 1991.

5. Gelfand, R., C. J. Lambertsen, and R. E. Peterson. Human respiratory control at high ambient pressures and inspired gas densities. *J. Appl. Physiol.* 48: 528-539, 1980.

6. Hesser, C. M., J. Adolfson, and L. Fagraeus. Role of CO_2 in compressed-air narcosis. *Aerosp. Med.* 42: 163-168, 1971.

7. Hesser, C. M., L. Fagraeus, and J. Adolfson. Roles of nitrogen, oxygen, and carbon dioxide in compressed-air narcosis. *Undersea Biomed. Res.* 5: 391-400, 1978.

8. Kerem, D., Y. Melamed, and A. Moran. Alveolar PCO_2 during rest and exercise in divers and non-divers breathing O_2 at 1 ATA. *Undersea Biomed. Res.* 7: 17-26, 1980.

9. Lally, D. A., F. W. Zechman, and R. A. Tracy. Ventilatory responses to exercise in divers and non-divers. *Respir. Physiol.* 20: 117-129, 1974.

10. Lambertsen, C. J., R. Gelfand, M. J. Lever, G. Bodammer, N. Takano, T. A. Reed, J. G. Dickson, and P. T. Watson. Respiration and gas exchange during a 14-day

continuous exposure to 5.2% O_2 in N_2 at pressure equivalent to 100 FSW (4 ata). *Aerosp. Med.* 44: 844-849, 1973.

11. Lambertsen, C. J., R. Gelfand, R. Peterson, R. Strauss, W. B. Wright, J. G. Dickson, Jr., C. Puglia, and R. W. Hamilton, Jr. Human tolerance to He, Ne, and N_2 at respiratory gas densities equivalent to He-O_2 breathing at depths to 1200, 2000, 3000, 4000, and 5000 feet of sea water (predictive studies III). *Aviat. Space Environ. Med.* 48: 843-855, 1977.
12. Lanphier, E. H. Influence of increased ambient pressure upon alveolar ventilation. In:C. J. Lambertsen and L. J. Greenbaum, Jr. Second Symposium on Underwater Physiology. Washington, D.C.: National Academy of Sciences, 1963: 124-133.
13. Lanphier, E. H., and E. M. Camporesi: Respiration and exertion In P. B. Bennett and D. H. Elliott (eds): *The Physiology and Medicine of Diving*. London, W. B. Saunders Company Ltd., 1993, pp 77-120
14. Leake, C. D., and R. M. Waters. The anesthetic properties of carbon dioxide. *J. Pharmacol. Exp. Ther.* 33: 280-281, 1928.
15. McArdle, W. D., F. I. Katch, and V. L. Katch: *Exercise Physiology*. Baltimore, Williams and Wilkins, 1996, pp
16. Morrison, J. B., J. T. Florio, and W. S. Butt. Observations after loss of consciousness under water. *Undersea Biomed. Res.* 5: 179-187, 1978.
17. Morrison, J. B., J. T. Florio, and W. S. Butt. Effects of CO_2 insensitivity and respiratory pattern on respiration in divers. *Undersea Biomed. Res.* 8: 209-217, 1981.
18. Nunn, J. F.: *Nunn's Applied Respiratory Physiology*. Oxford, Butterworth-Heinemann, Ltd., 1993, pp 90-114

19. Raymond, L. W., and F. G. Standaert. The respiratory effects of carbon dioxide in the cat. *Anesthesiology* 28: 974-980, 1967.

20. Refsum, H. E. Relationship between state of consciousness and arterial hypoxaemia and hypercapnia in patients with pulmonary insufficiency, breathing air. *Clin. Sci.* 25: 361-367, 1963.

21. Sherman, D., E. Eilender, A. Shefer, and D. Derem. Ventilatory and occlusion-pressure responses to hypercapnia in divers and non-divers. *Undersea Biomed. Res.* 7: 61-74, 1980.

22. Varene, P., H. Vieillefond, C. Lemaire, and G. Saumon. Expiratory flow volume curves and ventilation limitation of muscular exercise at depth. *Aerosp. Med.* 45: 161-166, 1974.

23. Vorosmarti, J., Jr. Influence of increased gas density and external resistance on maximum expiratory flow. *Undersea Biomed. Res.* 6: 339-346, 1979.

24. Warkander, D. E., W. T. Norfleet, G. K. Nagasawa, and C. E. G. Lundgren. CO_2 retention with minimal symptoms but severe dysfunction during wet simulated dives to 6.8 ata abs. *Undersea Biomed. Res.* 17: 515-523, 1990.

25. Waters, R. M. Toxic effects of carbon dioxide. *New Orleans Med. Surg. J.* 90: 219-224, 1937.

26. Westlake, E. K., T. Simpson, and M. Kaye. Carbon dioxide narcosis in emphysema. *Q. J. Med.* 24: 155-173, 1955.

27. Wood, L. D. H., and A. C. Bryan. Exercise ventilatory mechanics at increased ambient pressure. *J. Appl. Physiol.* 44: 231-237, 1978.