Lung Diseases Related to High-Altitude, Diving, and Near-Drowning

Bruce P. Krieger, MD, FCCP

Objectives:

1. To explain the pathophysiology of diving-related and high-altitude pulmonary-related complications by using the ideal gas law and the alveolar gas equation.
2. To contrast he differences in thoracic volume and pressure that occur in breath-hold diving, scuba diving, and descent in a submarine.
3. To predict which patients with pulmonary disease will require supplemental oxygen when flying in commercial aircraft.
4. To discuss preventive methods to avoid acute mountain sickness and high-altitude pulmonary edema.
5. To recognize that noncardiogenic edema occurs in fresh-water and saltwater near-drowning victims and that most immersion accidents are preventable.

Key words: air travel; altitude; barotrauma; decompression; diving; drowning

"It remains for someone to elucidate the evolutionary processes responsible for men being able to reach the highest point on earth while breathing ambient air…"

West, et al. American Medical Research Expedition to Everest, 1981

Pulmonologists have a good background to understand the pathophysiology of diving and high altitude-related disorders since they are related to breathing gases at varying ambient pressures. Therefore, basic gas laws form the physiologic foundation for a discussion of the more common disease states associated with diving and high altitude that may be encountered by pulmonologists.

Diving Physics

The alveolar gas equation defines the changes in alveolar oxygen tension that occurs with changes in barometric pressure.

Alveolar Gas Equation:

$$P_{AO_2} = (P_B)\,F_{IO_2} - \frac{P_{aCO_2}}{R}$$

P_{AO_2} = alveolar oxygen tension

P_B = barometric pressure

R = respiratory exchange ratio (CO_2 production/O_2 consumption)

At sea level (one atmosphere, atm), the barometric pressure is approximately 760 mm Hg resulting in a $P_{AO_2} \approx 100$ mm Hg (assuming $P_{aCO_2} = 40$, R = 0.8, $P_{H_2O} = 47$); at 3 atm pressure while breathing ambient air, the P_{AO_2} would be approximately 420 mm Hg and close to 2200 mm Hg if breathing 100% O_2 (such as in a hyperbaric chamber). At the top of Mt. Everest, the American Medical Research Expedition (1981) measured the $P_B = 253$ mm Hg and the end-tidal $CO_2 \approx 8$ to 10 mm Hg; under simulated conditions, the $P_{AO_2} \approx 40$ mm Hg.

Whereas body tissues are nearly incompressible within the usual normal range of human activity, the physical behavior of gases is affected by three factors - pressure, volume, and temperature. This interrelationship is described by the Ideal Gas Equation:

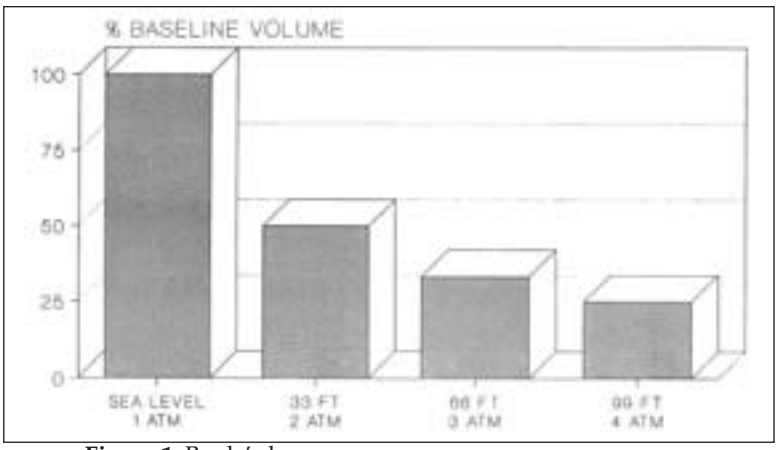

Figure 1. Boyle's law.

Ideal Gas Law Equation:
PV = nRT
P = pressure; V = volume;
n = number of moles of gas;
R = universal gas constant;
T = absolute temperature

The sea of air under which we live is defined as one atmosphere (atm) and at sea level is the equivalent of 33 feet of seawater or 14.7 pounds per square inch (psi). According to Boyle's Law, the volume of a given mass of gas is inversely proportional to its pressure: PV = K (a constant). These physical changes form the basis for understanding diving-related barotrauma (Fig 1).

Henry's Law provides the basic concept for understanding decompression sickness, nitrogen narcosis, and how bubbles form when a champagne bottle is opened. The law states that the amount of a gas that will dissolve in a liquid at a given temperature is directly proportional to the partial pressure of that gas. Dalton's Law forms the physical principle of hyperbaric therapy and also helps to explain decompression sickness. This law states that the total pressure (P_T) expected by a mixture of gases is the sum of each individual gas: Dalton's Law:

$$P_T = P1 + P2 + P3 + ...P_N$$

Table 1 summarizes the effects of these gas laws as they apply to diving (in seawater) and hyperbaric medicine.

Table 1—*Effects of Gas Laws on Diving and Hyperbaric Medicine*

Depth, ft	Pressure, (Total), atm	Volume, %	O_2, mm Hg	N_2, mm Hg	P_T mm Hg
0	1	100	160	600	760
33	2	50	320	1,200	1,520
66	3	33.3	480	1,800	2,280
99	4	25	640	2,400	3,040

Table 2 - *High Altitude Physiologic Changes*

Altitude Meters	Feet	P_B, mm Hg	P_{IO_2}, mm Hg
0	0	760	160
1,000	3,280	674	141
2,500	8,200	560	118
4,000	13,120	463	97
6,000	19,680	354	79
8,000	26,240	268	56

High-Altitude Physiology

The physiologic changes which occur at high altitude are the consequence of the reduced barometric pressure which results in low partial pressure of O_2 ($P_{IO_2} = P_B \times F_{IO_2}$). P_{IO_2} decreases 4 to 5 mm Hg for each 1,000-ft elevation (Table 2).

The ventilatory response to hypoxia varies with time. The acute cardiopulmonary responses include increases in ventilation (when $Pa_{O_2} < 60$ mm Hg) and cardiac output in order to maintain adequate oxygen delivery (D_{O_2}):

D_{O_2} = $Ca_{O_2} \times CO$

Ca_{O_2} = arterial O_2 content = Hgb × 1.39 × Sa_{O_2} + 0.0031 (Pa_{O_2})

CO = cardiac output = SV × HR

Sa_{O_2} = arterial O_2 saturation; SV = stroke volume

HR = heart rate; Hgb = hemoglobin

This response was demonstrated by a study in which hypoxemia was induced in normal men by breathing progressively lower F_{IO_2} while changes in Sa_{O_2}, HR, SV and calculated D_{O_2} were monitored noninvasively. Despite progressive hypoxemia, D_{O_2} remained stable (Table 3).

Chronically, some of this ventilatory response may become blunted; this response may be less prevalent in native highlanders.

Pulmonary function and hemodynamics change acutely upon ascent (Table 4).

Adaptation occurs in high-altitude residents, especially natives ("highlanders"). Their vital capacity (VC) and diffusing capacity

Table 3—*Cardiovascular Response to Graded Hypoxemia: Percent Change From Baseline ($F_{IO_2} = 0.21$)*[*]

F_{IO_2}	Sa_{O_2}	HR	SV	CO	D_{O_2}
0.21	96%	100	100	100	100
0.17	90%†	103	101	103	98
0.15	88%†	105†	104	107	99
0.12	76%†	119†	110†	130†	103
0.10	67%†	127†	119†	148†	103

[*] HR=heart rate; SV=stroke volume.
†$p < 0.05$ compared to baseline.

of the lung for carbon monoxide (D_{LCO}) are not as decreased as lowlanders who ascend to altitude. Their response to exercise also differs in that their endurance increases but their ventilatory response diminishes. In addition, it appears that chronic pulmonary hypertension results in permanent restructuring of the pulmonary arterial bed (smooth muscle hypertrophy and fibrosis) which may not reverse with O_2 or descent.

Oxygen delivery is effected by altitude. Hemoglobin concentration increases acutely due to the diuresis that frequently accompanies ascent. Within one day, serum erythropoietin levels rise; after 2 to 4 days, levels decline as acclimatization occurs and red blood cell volume increases. Although the resultant polycythemia may augment D_{O_2}, the increase in blood viscosity may diminish perfusion at the microvascular level, thus impairing exercise performance. The opposing action of these phenomena may be both adaptive and maladaptive; an individual's response is unpredictable. Another factor in D_{O_2} is the rise in 2,3-diphosphoglycerate (2,3-DPG) which accompanies hypoxemia and results in a rightward shift of the oxyhemoglobin curve. Cardiac function remains relatively well preserved. Cardiac output was preserved for a given O_2 uptake in normal volunteers.

Many of these physiologic responses were studied in detail under simulated altitude in a controlled 40-day sojourn in a hypobaric chamber. Termed "Operation Everest" and "Operation Everest II," these studies expanded our knowledge to the peak of Mt. Everest.

Diving-Related Disorders

Pulmonary Barotrauma

The most serious pulmonary complications of scuba diving are referred to by divers as "bubble trouble" (Table 5).

Table 4 - *Pulmonary Function and Hemodynamic Changes Upon Ascent*

Variable	Response
Vital capacity (VC)	Decreased
Diffusing capacity (D_{LCO})	Decreased
$\dot{V}/\dot{Q}$ match	Decreased
Pulmonary vascular resistance	Increased
Pulmonary artery pressure	Increased
Exercise performance	Decreased
Max O_2 consumption	Decreased

Barotrauma is the second leading cause of death in recreational scuba divers, the leading cause being drowning accidents. It occurs when a gas-filled space, such as an alveolus, sinus, dental-filling, or middle ear fails to equalize its internal pressure relative to changes in ambient pressure. Barotrauma can occur during descent as well as during ascent, although the latter is the more life-threatening process.

Scuba-diving (self-contained-underwater-breathing-apparatus) differs from breath-hold-diving in that the scuba apparatus allows the diver to maintain near-normal lung volumes despite being exposed to increased ambient pressure. When a breath-hold-diver returns to the surface, the volume of gas in his lungs can be no greater than the volume which was present at the beginning of the dive. However, the scuba diver can theoretically double his alveolar volume if he ascends from 33 feet to the surface rapidly and without exhaling; if he ascends in a similar manner from 99 feet, the alveolar volume will approximately quadruple. The shell of a submarine maintains pressure at 1 atm at any depth; therefore, alveolar volume will not change despite being at depth (Fig 2).

A pneumothorax and pneumomediastinum can easily occur in scuba divers even if they have no underlying pulmonary pathology. This danger is increased if there are areas of the lungs with prolonged time constants (resistance × compliance) such as occurs in patients with obstructive lung disease. Barotrauma can even occur when a diver inhales too rapidly from an underwater breathing apparatus that is located deeper than the diver; this maneuver simulates positive pressure ventilation. Stresses in peribronchial alveolar tissues may also be abnormal if pulmonary compliance is decreased

Table 5—*"Bubble Trouble" - Pulmonary Complications of Diving*

Barotrauma of ascent
Pneumothorax
Pneumomediastinum and subcutaneous emphysema
Arterial air embolism
Barotrauma of descent
Middle ear squeeze
Paranasal-sinus and dental squeeze
Decompression sickness (the "bends")
Venous air embolism (the "chokes")
Paresthesias, paralysis, vertigo, seizures
Nitrogen narcosis ("rapture of the depths")
"Dirty air" - CO_2 or CO contamination

which was demonstrated by measuring pressure-volume relationships in 14 divers who had suffered pulmonary barotrauma in whom there was no evidence of obstructive airways disease. When transpulmonary pressure (intratracheal - alveolar) exceeds 100 cm H_2O, gas can escape along perivascular sheaths and rupture into the mediastinum (pneumomediastinum), pleural space (pneumothorax), or the subcutaneous tissues (subcutaneous emphysema). If the air were to rupture into pulmonary veins, an arterial air embolism could result.

Arterial Air Embolism: This is the most serious sequelae of barotrauma since gas bubbles that are released into the systemic circulation may occlude cerebral or coronary arteries. Within minutes of surfacing, an afflicted diver will develop neurological symptoms ranging from focal motor, sensory, or visual deficits to unconsciousness, apnea, seizures, and death. If a coronary vessel is occluded, arrhythmias and/or cardiac arrest will dominate the clinical picture. Physical examination of the affected diver may show characteristic marbling of the skin of the upper torso along with focal pallor of the tongue.

Pneumothorax: This develops if air ruptures into the pleural space. Although this is uncommon, (10% of barotrauma), it may be fatal due to expansion of the pleural air as the diver ascends (Boyle's Law), resulting in a tension pneumothorax. This is manifested by tachypnea, tachycardia, hypotension, cyanosis, diminished ipsilateral breath sounds with hyperresonance to percussion and deviation of the trachea to the contralateral side. A chest radiograph establishes the diagnosis.

Mediastinal Emphysema: This is frequently accompanied by crepitation in the neck due to associated subcutaneous emphysema. A chest radiograph establishes the diagnosis. Symptoms include pleuritic chest pain that may radiate to the shoulders, dysphagia, coughing, and dyspnea.

Pulmonary Barotrauma During Descent: This is caused by air being trapped in a closed space as the ambient pressure increases. This increased pressure can evoke pain which divers refer to as a "squeeze". The most common squeezes involve the middle ear and sinuses due to eustachian tube dysfunction although even dental cavities can cause severe pain.

Decompression Sickness or Caisson Disease: This was first recognized in tunnel workers laboring in the compressed environment of caissons used for building bridge foundations. The term "bends" has been associated with Caisson Disease. This was linked to a contortionist posture of walking by woman socialites during the era in which the Brooklyn Bridge was constructed. Prolonged exposure at depth causes N_2 and O_2 to "supersaturate" the tissues as predicted by the gas laws. Oxygen is readily released during ascent but nitrogen release may be delayed, forming the nidus for bubbles to develop and circulate. There are other physical factors which influence the formation of bubbles during the rapid pressure changes of diving. The consequences of these bubbles are diverse, ranging from local joint pain (approximately 75% of cases), to paresthesias (4%), skin rash (4%), vertigo (2.5%), visual changes (1%), paralysis (<1%), fatigue, agitation, headache, seizure, and cranial nerve involvement.

Pulmonary Decompression Sickness: This is relatively rare (<1% of reported decompression

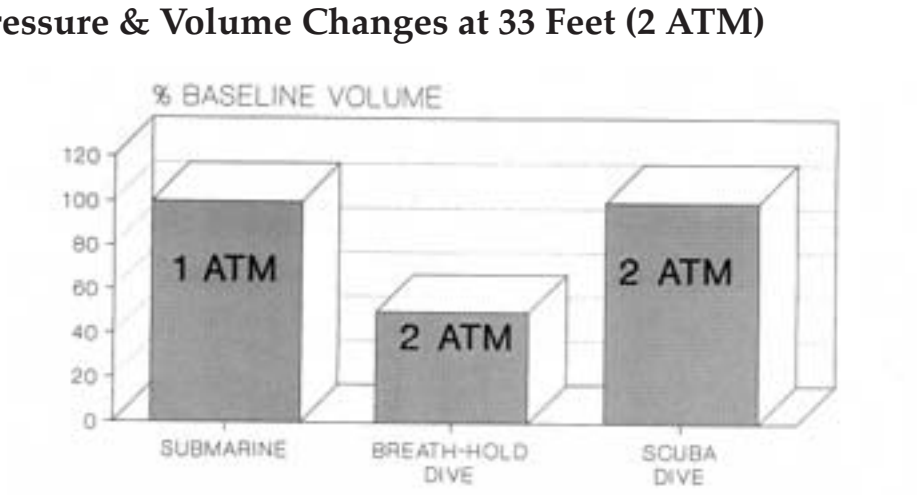

Figure 2. Pressure and volume changes at 33 feet (2 atm).

cases) but is frequently fatal. It is more common in military aviators than divers. Illness is characterized by substernal discomfort and cough that has been termed "the chokes". It occurs when venous air bubbles lodge within the pulmonary vasculature, causing partial obstruction. This triggers a cascade of vasoactive substances and proteolytic enzymes, resulting in leukocyte adherence and release of toxic oxygen radicals. In an *in vivo* sheep study, the pulmonary artery pressures and pulmonary vascular resistance increased acutely after bubbles were injected into the venous circulation. This was followed by a fall in cardiac output without a significant change in pulmonary ventilation or perfusion as measured by nuclear imaging techniques. This led the authors to hypothesize that the changes were not due solely to the mechanical obstruction of the pulmonary vasculature but rather to a cascade of vasoactive substances.

The affected diver or aviator presents with signs and symptoms of noncardiogenic pulmonary edema, including diffuse infiltrates on the chest radiograph, rapid shallow breathing, cyanosis, hemoptysis, acute right-sided heart failure, and hypotension. The appearance of clinical symptoms is usually avoided if rates of ascent are controlled as specified in decompression tables used and published by the US and the Royal Navy. However, silent (asymptomatic bubbles) have been detected by ultrasound imaging in the pulmonary circulation of divers during decompression from depths that are not clinically felt to require stops during ascent. The long-term effect of silent bubbles has not been determined, although magnetic resonance imaging (MRI) scans show subtle changes in the cortical matter of scuba divers. Risk factors that may enhance the accumulation of inert gases in tissues include increasing age, obesity, poor physical fitness, and sex (females). Repetitive dives are major problems since a considerable quantity of nitrogen may remain in the tissues after the first dive and be compounded during subsequent descent to depths.

Nitrogen Narcosis: This has been termed "rapture of the depths" because its presentation is similar to that of an alcohol or benzodiazepine intoxication in which there is impairment in neuromuscular performance, intellectual capabilities, and normal behavior. The condition is caused by raised partial pressure of nitrogen in the tissue of the nervous system that can occur at depths usually greater than 100 feet as predicted by the Ideal Gas Law. It is exacerbated by cold water, hypercarbia, fatigue, and alcohol. By itself, it does not cause adverse effects as long as a diver ascends; however, it is a precipitant of impaired judgment and therefore drowning accidents. Carbon dioxide toxicity can also occur if a diver's rebreathing apparatus is faulty which results in loss of consciousness and drowning. Similar consequences result if carbon monoxide contaminates a diver's source of air.

Diving and Preexisting Disease

Conditions that affect the lungs, upper airways, or cardiovascular system can predispose subjects to diving complications. Specialists in diving medicine should be consulted if there are any doubts as to whether a patient is medically fit to scuba dive (Table 6).

Pulmonary Disease: Because of the drastic pressure and volume changes that occur during scuba diving, any condition which predisposes to hyperinflation is a contraindication to diving. Asthma is a classic example of this and is a vexing problem when an afflicted individual desires to dive. Even though pulmonary function tests may be normal, this does not insure safety since these tests are insensitive to individual lung units that may have prolonged time constants. Hyperreactivity of the airways can be present even in the face of normal lung function and would require bronchoprovocation tests to discern. In addition, exercise or hyperventilation of dry air (such as is respired from scuba tanks) may provoke airway hyperreactivity. Therefore, any history of significant active asthma is considered a contraindication to diving. However, the absolute prohibition of diving with stable asthma has recently been lifted.

Table 6 — *Cardiopulmonary Conditions That Disqualify Patients From Scuba Diving*

Obstructive lung disease
Asthma
COPD, emphysema
Bullous disease
Cystic fibrosis, bronchiectasis
Previous barotrauma
Pneumothorax, subpleural blebs, previous thoracic surgery
Cardiac disease
Exercise-induced tachyarrhythmias or ventricular arrhythmias
Coronary artery disease
Unrepaired intracardiac shunts

Similarly, a history of COPD, previous pneumothorax from any cause, cystic fibrosis, bronchiectasis, or thoracic surgery, should be viewed as an absolute contraindication to scuba diving. In addition, interstitial lung disease may also predispose divers to barotrauma.

Preexisting Cardiovascular Disease: The effects of diving on the cardiovascular system are mainly related to the work and exercise involved. Therefore, any individual with known heart disease or previous cardiac surgery would require a cardiac stress test before being allowed to dive. The presence of ventricular arrhythmias also would require stress testing before diving is allowed, although controlled supraventricular arrhythmias should not pose a problem. A right to left shunt, such as a patent foramen ovale, can pose a problem since venous gas emboli due to decompression sickness could pass through the shunt and cause arterial embolization. Recently, patent foramina ovale were diagnosed by using a Valsalva maneuver during echocardiographic imaging of the heart with venous bubble contrast in 60% of divers who had experienced arterial gas emboli.

Pregnancy: Women may incur difficulties because of physiologic changes that occur during pregnancy. Congested mucous membranes may cause difficulties in equilibrating the middle ear and sinuses, while abnormal temperature regulation may predispose to decompression sickness. Another concern of diving while pregnant is the effect on the fetus. A survey of women who scuba dived during pregnancy showed a small but significant increased risk of birth defects compared to divers who refrained from diving although this complication rate was no higher than that in the general population. Most experts advise against taking any risks during pregnancy and therefore discourage scuba diving. However, this is not an absolute contraindication and many pregnant divers remain active, albeit at a less intense level.

Treatment of Diving-Related Disorders: Hyperbaric Chamber

The mainstay of therapy for serious pulmonary barotrauma or decompression sickness is hyperbaric chamber treatment. While arrangements are being made to transfer the injured diver to a recompression chamber, basic life support measures should be instituted, including the delivery of 100% oxygen, intravenous electrolyte solution, positioning of the injured diver in the left lateral recumbent position either supine or in a mild Trendelenburg in an effort to trap any venous air bubbles in the right atrial chamber, and placement of a chest tube or a catheter if a pneumothorax is suspected. Some experts question the advisability of specific positioning of these patients. Evacuation may be expedited by contacting the divers alert network hotline (919-684-8111).

Patients with air embolism or decompression sickness should be transported to the nearest hyperbaric facility. Treatment with oxygen under hyperbaric condition results in the mechanical reduction of the volume of gas emboli according to Boyle's Law as well as providing oxygenation to hypoxic tissue. In addition, the nitrogen pressure gradient between emboli and blood increases, thus accelerating the absorption of emboli. There are various specialized protocols that have been developed by the US Navy to accomplish this, and it requires a qualified physician trained in hyperbaric medicine to administer it. Hyperbaric medicine therapy has been used for a multitude of diseases. Recently, it has been termed a "therapy in search of disease". Bubble troubles (gas emboli and decompression sickness) are diseases that all authorities agree are appropriate to treat with hyperbaric oxygen therapy (Category I). Other uses range from promising to anecdotal (Categories II - IV).

Respiratory mechanics are altered by hyperbaric conditions. The gas laws predict that increased pressure will cause gas density to increase. Maximal voluntary ventilation breathing room air is halved as pressure increases from 1 to 4 atm. Exercise capabilities decline due to this increased gas density as well as to dynamic airway compression that simulates obstructive lung disease. If 100% O_2 is used under hyperbaric conditions, there is a progressive decline in lung volumes (especially VC) associated with an increased alveolar-arterial O_2 gradient. The mechanism for this decline appears to be related to early O_2 toxicity and possibly resorption atelectasis.

High-Altitude-Related Disorders

Acute Mountain Sickness

Acute mountain sickness (AMS) occurs in 25% to 50% of travelers who ascend rapidly to altitudes > 7,000 to 15,000 feet. It is manifested by headaches, malaise, nausea, vomiting, anorexia, dyspnea and insomnia that occur within 6 to 48

hours after arrival at high altitude. AMS can be exacerbated by alcohol or sedative consumption and overexertion during the first few days. Although the exact pathophysiology has not been determined, the most accepted theory is that AMS represents a continuum of hypoxia-induced cerebral vasodilation resulting in mild brain edema. The time course of hypoxia-induced increased retinal blood flow (which is thought to reflect cerebral blood flow) corresponds to the appearance of AMS and resolves after measures directed at reducing cerebral blood flow (such as forced hyperventilation or steroid use).

AMS can be prevented by a 2- to 4-day acclimatization stay (including sleep) at lower altitudes with gradual ascent to higher elevations. Acetazolamide is a carbonic anhydrase inhibitor that causes a sodium and bicarbonate diuresis. This results in a metabolic acidosis that stimulates ventilation, thus decreasing nocturnal hypoxemia and the associated periodic breathing pattern. When taken prophylactically 1 to 2 days before ascent and continued for 48 hours after ascent, the symptoms and duration of AMS are lessened. Dexamethasone is effective in alleviating symptoms, although this may be due more to its euphoric and anti-emetic properties than to a measurable reduction in cerebral edema. Because dexamethasone has potentially more serious side effects, acetazolamide is considered the drug of choice for prophylaxis. In two controlled trials, ginko biloba also reduced the symptoms and incidence of AMS.

Treatment involves descent to lower altitude or the administration of supplemental oxygen, especially during sleep. Both dexamethasone and acetazolamide have been used successfully as treatment, although the symptoms of AMS often subside within seven days just by avoiding overexertion.

High-Altitude Pulmonary Edema

High-altitude pulmonary edema (HAPE) is a form of noncardiogenic pulmonary edema that occurs after rapid ascent to altitudes > 14,000 feet. The reported incidence in the general mountaineering population ranges from 0.5% to 10%, although susceptible individuals have a 60% incidence upon re-exposure to high altitude. The pathophysiology of HAPE has been hypothesized to be due to hypoxic pulmonary vasoconstriction resulting in acute pulmonary hypertension that may be associated with sympathetic overactivity. Regional overperfusion due to inhomogeneous vasoconstriction is thought to cause stress failure of the pulmonary capillaries. This theory is supported by studies performed at simulated altitude and by the finding that those individuals with the highest predilection for HAPE have an exaggerated pulmonary vasoconstriction response to hypoxia. Healthy subjects who were abruptly exposed to an altitude of approximately 12,000 feet had mean pulmonary artery pressures (measured at catheterization) of 21 mm Hg, while patients with HAPE had recorded pressures of 60 mm Hg (range, 33 to 117). Other contributing factors include hypoxia-induced impairment of sodium and water clearance from the alveolar space that can be prevented by the prophylactic inhalation of salmeterol. Cytokine activation as a consequence of capillary stress failure has recently been shown to be a consequence and not a cause of HAPE.

Symptoms usually commence between the second and fourth day after ascent and include: dyspnea, cough, fatigue, altered mental states, and somnolence. These symptoms correlate with hypoxemia and a variable radiographic appearance which includes a normal-sized heart, enlarged pulmonary arteries, patchy infiltrates most commonly in the right middle and lower lobes but bilateral in more severe cases.

Basic treatment depends on the delivery of high-flow O_2 and descent. Some climbers carry a portable (15 pound) individual hyperbaric chamber (Gamow bag) which can be pressurized with a foot pump. A double-blind study of nifedipine in climbers who had previously experienced HAPE showed a significant decrease in the development of HAPE when compared to placebo therapy. The nifedipine-treated group had Doppler-echocardiographic estimated mean pulmonary artery pressures which were 20% to 25% lower than the placebo group (53 mm Hg). Gradual acclimatization and avoidance of overexertion may help prevent HAPE, while acetazolamide and good physical conditioning did not.

High-Altitude Cerebral Edema

Cerebral edema frequently accompanies HAPE and is thought to represent a more severe form of AMS. Prominent symptoms include headache, mental status changes, and signs of cerebellar dysfunction. Immediate descent and oxygen therapy are recommended, although parenteral dexamethasone has been reported to be helpful. Unless treated, cerebral edema and HAPE are potentially fatal.

Chronic Mountain Sickness (Monge's Disease)

A minority of highland residents develop a disease characterized by chronic fatigue, headache, dizziness, and poor memory. Compared to highlanders without these symptoms, they have a blunted ventilatory response to hypoxia and an increased hematocrit. Symptoms can be lessened by O_2 administration and phlebotomy.

Altitude and Air Travel in Hypoxemic Patients

Most of the responses to hypoxemia which were outlined earlier in this manuscript were derived from healthy subjects who would have no difficulties flying in pressurized commercial aircraft. Commercial craft usually cruise between 10,000 and 60,000 feet, resulting in cabin P_B of 5,000 to 8,000 ft. (P_{IO_2} of 106 to130 mm Hg). Patients with underlying pulmonary disease, especially COPD, may not tolerate such stresses. For example, 13 patients with COPD (FEV_1/FVC < 50%) were studied at sea level and while flying in an unpressurized aircraft at 5,400 feet (1650 meters) and 7,400 feet (2250 meters) (Table 7).

The incorporation of FEV_1 into the regression equation for COPD patients strengthens the prediction of Pa_{O_2} at 8,000 feet (2438 meters):

$$Pa_{O_2}\ alt = 0.453\ (Pa_{O_2}\ sea\ level) + 0.386\ (FEV_1\ \%\ predicted) + 2.440$$

Although these equations are helpful as a screening test, the most accurate predictions were derived when the sea level Pa_{O_2} was measured on the same day as Pa_{O_2} alt, which is impractical in clinical practice. In addition, the equations do not predict the response to exercise (such as walking to the cabin's bathroom) nor do they allow for patients who are hypercapnic or those who require supplemental O_2 at sea level. Therefore, direct measurement of Pa_{O_2} or Sa_{O_2} under hypobaric conditions or while breathing hypoxic gas mixtures is the most direct method of assessing an individual's response to commercial air travel. The high-altitude simulation test (HAST) simulates in-flight hypoxemia by modifying the F_{IO_2}. It can be performed in any pulmonary laboratory and does not require a hypobaric chamber (Table 8).

If the simulation Pa_{O_2} is < 50 mm Hg (Sa_{O_2} < 85%), supplemental O_2 is suggested in-flight. This usually requires that the airline be notified 48 to 96 hours prior to flight. During a HAST, the appropriate amount of supplemental O_2 can be determined; however, most carriers provide limited options (2, 4, or 8 L/min) and forbid utilization of the patient's own equipment. Other helpful hints are to have the patient's seat location close to the bathroom and to have them void prior to boarding the aircraft.

Table 7 — *Effect of Air Travel on Hypoxemic Patients*[*]

	Sea Level	1,650 m	2,250 m
P_{AO_2} mean (SD); range	68 (7) (55-79)	51 (9) (32-61)	45 (9) (25-66)
P_{AO_2}-P_{AO_2} mean (SD)	32 (7)	25 (7)	24 (8)

[*] All values in mm Hg.

Regression equations have been developed from studies utilizing the level of Pa_{O_2}at sea level to predict the Pa_{O_2} at altitude (Pa_{O_2} alt):

COPD*

Pa_{O_2} alt = 22.8 - 2.74x + 0.68y

Restrictive lung disease*

Pa_{O_2} alt = 25.0 - 3.12x + 0.62y

* x = altitude in thousands of feet; y = Pa_{O_2} at sea level.

Near-Drowning

Drowning is defined as "to die from suffocation by submersion in water" while near-drowning is "to survive, at least temporarily, after suffocation by submersion in water". In the United States, drowning is the fourth leading cause of accidental deaths. It is more prevalent in young children (< 5 years) and young adults (15 to 29 years), males, blacks, and in southern states. Two thirds of drownings occur in fresh water sources (pools, lakes, rivers, bathtubs, toilets, etc). Risk factors include: inadequate adult supervision, seizures, boating accidents, aquatic sporting accidents, alcohol consumption (37% to 48% of adult victims), arrhythmias, spinal cord injuries, and voluntary hyperventilation. This last condition, which has been termed shallow-water-blackout,

Table 8— *High-Altitude Simulation Test*

Simulated Altitude	F_{IO_2}
Sea level	0.21
5,000 ft	0.17
8,000 ft	0.15
10,000 ft	0.14

occurs when a swimmer (usually an athletic young man) hyperventilates prior to submersion to such a degree that hypoxemia-induced unconsciousness occurs before the $Paco_2$ reaches a "break point" value which normally would signal the need to surface.

Pathophysiology

Studies of animal drownings have recorded a sequence of events during drowning: loss of normal breathing pattern, usually an element of panic (except when drowning is secondary to another cause, such as an acute myocardial infarction), breath-holding, and aspiration (fluid, debris, vomitus). Surviving animals aspirate <22 mL/kg of fluid, although laryngospasm is seen ≈10% of the time during which hypoxemia results from apnea and not from aspiration. Hypoxemia, which is the most deleterious consequence of submersion in water, is due to different mechanisms depending on whether fresh or salt water is aspirated. Fresh water alters the surface tension properties of surfactant that allows alveolar collapse. Salt water, being hypertonic, attracts fluid into alveoli. Both mechanisms cause significant shunt physiology, manifested by a non-cardiogenic pulmonary edema picture. Other physiologic consequences include the following: acid-base: acidosis (metabolic and respiratory); cardiovascular: secondary arrhythmias; neurological: edema; rule out spinal cord injury; renal: shock-induced ATN; rare hemoglobinuria; and temperature: hypothermia (which may be protective).

Animal experiments suggested that fresh water drowning causes hemodilution with resulting hyponatremia and anemia while salt water resulting in hemoconcentration. Human near-drowning victims do not aspirate enough liquid for these effects to be clinically significant.

Clinical Picture

Usually, the clinical picture is dominated by noncardiogenic pulmonary edema as manifested by hypoxemia, acidemia (<7.30 in 50% of patients), and chest radiographic abnormalities (aspiration, 30%; edema, 26%; atelectasis, 14%: pneumothorax, 8%; normal, 22%). Resuscitative efforts should be directed at restoring ventilation and circulation as promptly as possible by using standard CPR. There is no evidence to support attempts at emptying fluid from the lungs. Because pulmonary deterioration may be delayed by 12 to 24 hours, all victims should be closely observed or admitted. Prophylactic steroids and/or antibiotics have not been shown to be helpful. Continuous positive airway pressure or positive end-expiratory pressure frequently and promptly restores adequate oxygenation.

Prevention and Outcome

Prevention includes water and drug safety training, public education, proper supervision, protective fences, and appropriate "house-proofing" against toddler accidents. With sophisticated respiratory care, 75% of 711 near-drowning victims survived; 6% of the victims had residual neurological abnormalities (Fig 3). Since respiratory salvage has improved, neurological outcome has become a pragmatic as well as a moral issue, especially in the emergency department when

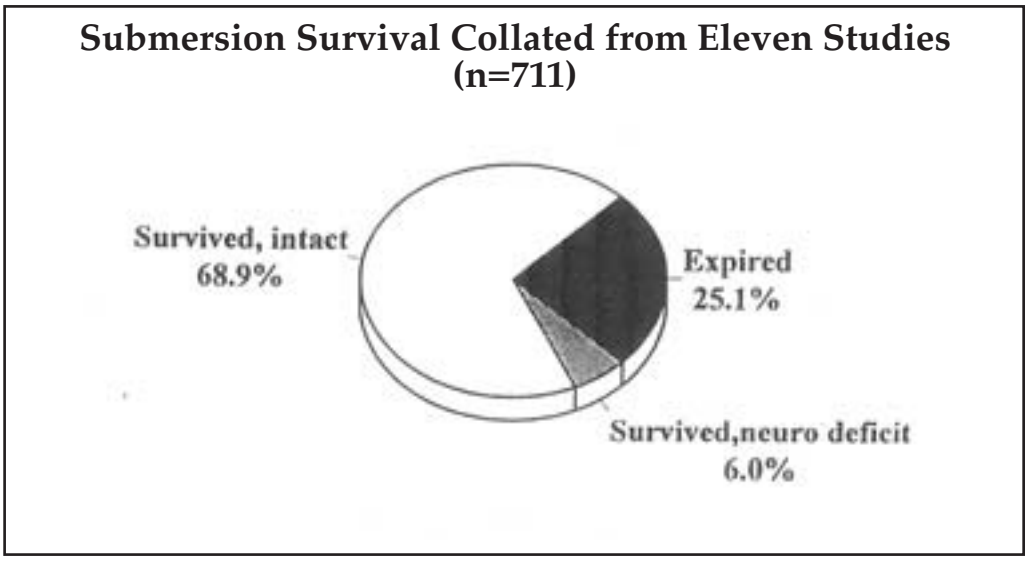

Figure 3. Submersion survival collated from eleven studies (n = 711).

a decision to cease or continue resuscitative efforts needs to be made. Efforts directed specifically at reducing brain edema and metabolism have not been shown to significantly affect outcome. In view of estimates that half of pediatric swimming pool accidents could be prevented by pool fencing and that alcohol use is involved in 40% to 50% of teenage accidents, a greater emphasis on prevention and education clearly is indicated.

Annotated Bibliography

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Notes