COLLECTIVE REVIEW
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A Short Course in Diving Medicine

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PRESSURE EFFECTS (BAROTRAUMA)

At sea level, the atmosphere exerts a ubiquitous absolute pressure of approximately 760 mm Hg (29.9 inches Hg = 14.7 pounds per square inch [psi]). This is the standard one atmosphere absolute (1 ATA) pressure. Pressure changes in water are linear; one atmosphere of pressure is added for each 33 feet of sea water (fsw) depth (34 feet for fresh water) so that the pressure at a depth of 66 feet is 3 ATA or 44.1 psi in sea water. This increased pressure is balanced by breathing air delivered at the new ambient pressure and by equalizing the pressure in all gas-containing body cavities to ambient.[1,2]

Following Boyle's law, at a constant temperature, the volume of an enclosed gas varies inversely with the surrounding absolute pressure. The relationship between depth/pressure and volume is shown (Figure 1). The greatest pressure changes occur at shallow depths. Most diving pathology related to pressure effects is a result of this pressure-volume relationship.

THE EAR

Middle Ear Squeeze

The middle ear can be considered a gas-filled structure almost completely surrounded by bone. The tympanic membrane (TM) is the only distensible part, and the eustachian tube is its only avenue for gas venting. Unless additional air is allowed into the middle ear during descent, the tympanic membrane is forced inward by the increasingly unbalanced ambient pressure. If this pressure is not equalized, a feeling of fullness will occur at a pressure differential of 60 mm Hg (3 fsw). Equalization becomes nearly impossible between 90 and 120 mm Hg (4 to 5 fsw) because of a pharyngeal valve effect, and TM rupture will follow at between 100 and 500 mm Hg (4.5 to 21 fsw).[1-4] These effects are most apparent in shallow water because the percent volume change is greatest near the surface.

As the differential pressure increases, the middle ear mucosa changes in a predictable pattern, best observed in the TM as follows:[1] TM capillary dilation, edema of the TM mucosa, hemorrhage into the TM and its mucosa, hemorrhage or serious transudation into the middle ear, and tympanic membrane rupture (usually with acute relief of pain).

When TM rupture occurs in the water, vertigo may result from caloric vestibular stimulation.[5,6] Hemorrhage and decreased TM compliance may produce a conductive hearing loss in severe cases.[7,8] This spectrum of pressure imbalance effects is ear barotrauma or ear "squeeze," and is the most common diving-related injury. The characteristic tympanic membrane changes are used to classify ear squeezes into the five clinical categories (Table 1).[9]

While descending, experienced divers will "keep ahead" of their pressure imbalance, using a gentle Valsalva or Frenzel maneuver[1,2,5,8-10] to clear their ears in advance of the feeling of fullness. When a diver feels pain, he is close to the differential pressure at which he will be unable to clear; the best remedy is to ascend to a depth of relief and gently try to clear again.

Uncommonly, a reverse ear squeeze can occur when air in the middle ear

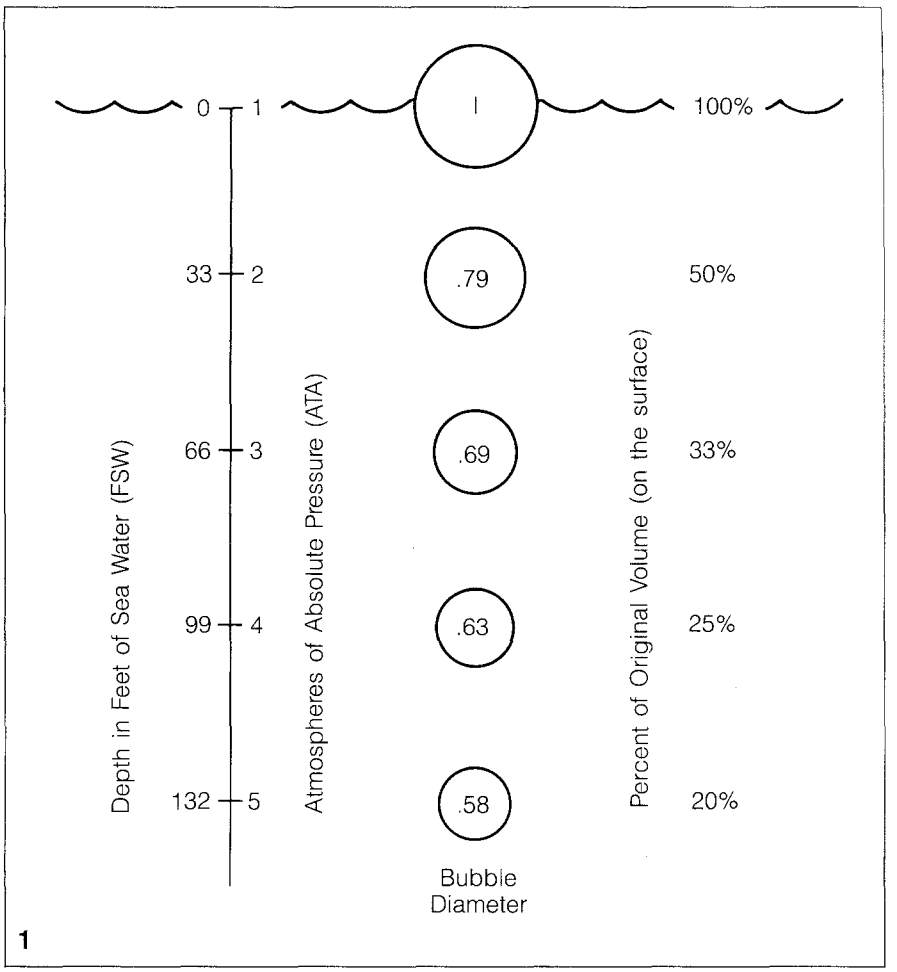

FIGURE 1. *The relationship between pressure (or depth) and gas volume changes following Boyle's law. The greatest relative pressure changes (and, hence, volume changes) occur nearest the surface.*

fails to vent through the eustachian tube during ascent.[1,2,8,10] The diver feels a fullness as the TM bulges outward and may perforate or cause alternobaric vertigo, a transient vertigo due to unequal middle ear pressure equilibration.

Depending on severity, ear squeezes can be treated symptomatically with decongestants or a decongestant/antihistamine combination.[7,8] Introducing compressed air into the nose while the patient loudly repeats "K..K..K..K" can force air into the eustacian tubes and may be useful if a significant middle ear negative pressure persists. Antibiotics (ampicillin or erythromycin for penicillin-allergic patients) should be prescribed if the TM has been ruptured.[7,8] Holes in the superior-posterior aspect may be associated with disruption of ossicular continuity. Further diving should be postponed until the condition has cleared (Table 1). TM perforations are an absolute contraindication to diving because of the risk of calorically-induced vertigo.

External Ear Squeeze

If air cannot freely enter the external canal (because of cerumen impaction or the wearing of a tight-fitting wet suit hood or ear plugs), the resultant negative pressure in the canal will cause the TM to bulge outward. The lining of the canal can become edematous and, eventually, hemorrhagic. The diver will experience pain not ameliorated by middle ear equalization. Treatment is ascent. Snorkeling and scuba diving should never be performed with ear plugs.

Round or Oval Window Rupture and Perilymph Fistula

A serious manifestation of inner ear barotrauma is rupture of the round or, rarely, the oval window.[10] A pressure differential between the inner and middle ear can be created, causing either an implosive or explosive round or oval window rupture. This usually occurs close to the surface during a difficult descent as the inner-middle ear pressure differential is accentuated by a forceful Valsalva-induced increase in inner ear pressure.[11] Explosion can result from overpressurization of the cerebrospinal fluid within the inner ear through the cochlear aqueduct. Implosion occurs when sudden equalization causes exaggerated movement of the ossicular chain.

Round or oval window rupture is marked by the sudden onset of severe vertigo not relieved by ascent, roaring tinnitus, nystagmus, a feeling of fullness in the affected ear, and a sensorineural hearing loss.[10,11] Round or oval window rupture can be confused with alternobaric vertigo, although the latter usually occurs during deep dives and is transitory, and with inner ear decompression sickness that is most often associated with deep dives using a helium and oxygen breathing mixture. A trial of recompression is not indicated as further inner ear damage might result.[10,11]

When a diver presents with vertigo and a sensorineural hearing loss, inner ear barotrauma must be considered.[7,8] The patient should be evaluated for possible early surgical repair. An electronystagmogram and an audiogram are helpful in locating and assessing the extent of the fistula, and antivertiginous drugs may be of benefit.[10,11] Patients whose audiogram shows a flattened curve over all frequency ranges seem to recover more completely than those patients whose initial deficit is confined to the higher frequencies.[10,11]

Round or oval window ruptures may heal spontaneously after five to seven days of bed rest.[10,11] Surgical repair may be necessary.

Round or oval window rupture can be prevented by not diving when there is difficulty equalizing pressure at the surface, by using only gentle Valsalva or Frenzel maneuvers when relieving middle ear pressure, and by ascending when there is difficulty clearing.[10,11] The Frenzel maneuver is performed by increasing pressure only within the nasopharynx while the glottis remains closed and generates less intrathoracic and intracranial pressure than does a

TABLE 1. *Ear squeeze clinical categories*[9]

Category	Description	Restriction From Diving
Teed 0	Normal	
Teed 1	Vascular congestion of pars flaccida, umbo, and anulus; occurs at pressure differential of 100 mm Hg	1 to 3 days
Teed 2	Vascular congestion of entire tympanic membrane; occurs at pressure differential of 100 to 150 mm Hg	1 to 3 days
Teed 3	Hemorrhage within tympanic membrane	1 week
Teed 4	Hemorrhage into middle ear with or without TM rupture*	4 to 6 weeks
Teed 5	Hemorrhage fills middle ear	4 to 6 weeks

*Open TM perforation is an absolute contraindication to diving because of the risk of calorically induced vertigo.

Valsalva maneuver.[2,12]

Alternobaric Vertigo

This condition is a transient, sudden, and overwhelming feeling of disorientation and spinning occurring more frequently on ascent than descent.[5,6,13] It is thought to be caused by a usually unilateral pressure differential greater than 50 cm H_2O between the middle and inner ears.[12,14,15] Usually lasting less than a minute but often persisting for hours, the severe disorientation can be extremely hazardous in the water.

Alternobaric vertigo on ascent is thought to be caused by an inability to equalize one or both ears.[6,14] The diver feels a fullness preceding the onset of vertigo. The vertigo may persist on the surface and be accompanied by nausea, vomiting, and nystagmus without tinnitus. Decongestants hasten clearing, and a myringotomy may be necessary.[2,7,8,12]

Alternobaric vertigo on descent usually follows a difficult descent with many forceful Valsalva maneuvers and sudden equalization.[5,6,12-14] It may be caused by a sudden middle ear overpressurization and/or rapid shift in the positions of the round and oval windows.[12,14] The signs, symptoms, and treatment are the same as for alternobaric vertigo on ascent and can be prevented by descending slowly while gently equalizing middle ear pressure. A purely conductive hearing loss may signal dislocation of the stapes.[2,5,6,14]

Very rarely, if the facial nerve is exposed to prolonged middle ear overpressurization, its blood supply can be compromised with resulting "alternobaric facial palsy."[16] Complete unilateral facial nerve palsy is the most extreme result but is, fortunately, transient and resolves as soon as circulation is restored by equalizing middle ear pressure.[16]

THE SINUSES

Sinus squeezes occur less frequently than do ear squeezes and are usually associated with a preexisting upper respiratory infection that distorts the normal architecture of the ostia.[1,2,8] Prophylactic decongestants administered just before diving have been beneficial.[1,2,8]

Sinus squeezes most often involve the frontal and maxillary sinuses and usually occur on descent as the pressure differential increases between the sinus and ambient pressures. The mucosa becomes hyperemic, edematous, and, eventually, hemorrhagic; hematoma formation and frank bleeding from the nose are common.[1,2,8] The diver experiences acute pain over the area corresponding to the affected sinus, and a dull ache may persist after equalization. Immediate treatment consists of ascending to a depth of relief and gently trying to equalize, discontinuing the dive if symptoms persist. Adjunctive therapy is symptomatic[1,2,8] and antibiotics are recommended if bacterial infection is superimposed.[1,2,8] Hematomas may resolve slowly. Diving should be suspended until symptoms have symptomatically and radiologically cleared.

Uncommonly, a reverse sinus squeeze can occur on ascent if an ostium is ostructed by redundant mucosa, polyp, or mucous plug.[1,2,8] While the symptoms will be the same as for a sinus squeeze on descent, immediate treatment is to descend to a depth of relief and ascend slowly. Occasionally, paresthesias or numbness along the infraorbital nerve distribution may occur with a reverse maxillary sinus squeeze.[17,18]

PULMONARY OVERINFLATION

Air in the lungs also follows Boyle's law. At a depth of 33 fsw, the lung volume of a snorkler who has inhaled to total lung capacity (TLC) at the surface will be one-half of the original volume at the surface. Conversely, the lung volume of a scuba diver who inhales to TLC at 33 fsw and ascends will double by the time he reaches the surface. Constant exhalation is required to vent air from the ascending scuba diver's lungs so the increasing volume won't overpressurize the alveoli. Failure to do so will result in alveolar rupture with escape of air in one of three pathologic directions to produce interstitial emphysema, pneumothorax, or arterial gas embolism (Figure 2).[19] The usual cause of an overinflation accident is inadvertent breathholding during an emergency ascent by an inadequately trained diver.

Because the greatest pressure and volume changes occur near the surface this is also the area of greatest danger. An overpressurization of only 90 cm H_2O is necessary for alveolar rupture to occur,[20-22] and near the surface, this can be attained in less than six fsw. Breath holding for only the last six feet of ascent can result in pulmonary overinflation. Individual variation and preexistent pulmonary pathology that causes a decrease in lung compliance has been shown to increase the risk of pulmonary overinflation.[23]

Interstitial Emphysema

Interstitial emphysema is always present with alveolar air escape and may be its only manifestation.[21] Air can dissect into the mediastinal space and, from there, into the pericardium, cephelad into the neck as subcutaneous air, or caudally as retroperitoneal air.[21,22,24,25] Signs and symptoms include supraclavicular crepitus, change in voice timbre, substernal crunching, pericardial air, compromised venous return from the head,

obvious radiograph findings, and dyspnea.[19,20-22,24-26] Unless the pericardial air severely compromises circulation, or the mediastinal or cervical air hinders breathing, interstitial emphysema is not life-threatening and can be treated symptomatically with complete resolution expected in a few days. Breathing 100% oxygen can hasten resolution,[19,20,24] but is not generally recommended,[19,20,24] and intubation may be required.[19,24]

The presence of interstitial emphysema does indicate the escape of alveolar air and the patient must be monitored for the other more serious consequences of pulmonary overinflation.

Pneumothorax

Pneumothorax is a sequela of air extravasation from alveoli adjacent to the pleura, especially in a diver with preexisting blebs.[19-21] The signs and symptoms are the same as for a pneumothorax on the surface and the pneumothorax is usually neither under tension nor life-threatening.[19]

Placement of a chest tube is sometimes indicated[19] and efforts should be made to rule out a concomitant arterial gas embolism. If recompression therapy is initiated for a coexisting arterial gas embolism or decompression sickness in a diver with a pneumothorax, a chest tube must be inserted, usually during recompression, to prevent tension from developing on ascent.[19] A Heimlich valve can be used to permit greater mobility within the normally small recompression chamber.[19]

Arterial Gas Embolism

Arterial gas embolization is the most serious and rapidly fatal of all diving accidents and is second only to drowning as the leading cause of death associated with sport diving.[19,24] Air bubbles that enter the pulmonary capillaries coalesce and travel through the pulmonary veins to the left atrium, where they are directed into the left ventricle. From the left ventricle, some of air may enter either the coronary arteries or the cerebral circulation. The combined effects of air that enters the coronary arteries and air entering the vertebral artery circulation can have a dramatic effect on coronary circulation and myocardial function.[24,25] Cerebral air has essentially the same effects as any embolic disruption of circulation: decreased local blood flow distal to the obstruction, accelerated intravascular coagulation, increased capillary permeability and leakage, and edema of surrounding tissues.[26-35]

Arterial gas embolization occurs on ascent, and the time from alveolar rupture to manifestation of symptoms is nearly always less than ten minutes.[19,24] A complete neurologic examination is essential to pick up the often subtle manifestations of air embolization.[36] Some patients present with neurologic symptoms but without impairment of spontaneous respiration and cardiac function.[19,24] Neurologic symptoms can range from subtle changes in mood or affect to the more usual global effects presenting as immediate unconsciousness. Others present with apnea, loss of consciousness, and cardiac dysrhythmia or arrest.[19,24]

The pathogenesis of most symptoms is a localized obstruction of cerebral blood flow by an embolus of air.[37] Local capillary endothelial damage results in vasogenic edema[38] with prompt extravascular movement of small molecules and somewhat delayed (30 to 60 minutes) leakage of large molecules from cerebral vessels.[29,34,39] The combined effect is an increase in total brain water content beginning approximately 30 minutes after embolization and lasting nearly 24 hours.[28-30] Intracranial pressure has been shown to rise almost immediately, peaking in one hour, and remaining elevated for several hours.[32]

Definitive treatment of any gas embolism requires a recompression facility and expeditious transportation is vital. The following steps are recommended while the patient is in transit:

1. Assure adequate airway, ventilation, and circulation. Intubated patients should be hyperventilated to decrease intracranial pressure. Endotracheal tube and Foley catheter cuffs must be filled with water to avoid volume changes during recompression treatment.

2. Administer 100% oxygen by the most efficient means (rebreathing mask if not intubated).

3. Treat any dysrhythmias or cardiac arrest with the advanced cardiac life support protocol;[19,24,25] dysrhythmias caused by bubble embolization into coronary arteries will tend to be refractory to treatment until the bubble is reabsorbed or reduced in size by recompression.

4. Treat hypotension with pressors such as dopamine in doses of 1 to 10 μg/kg/min to increase coronary artery blood flow, cardiac output, and renal blood flow without increasing myocardial oxygen demand.[24,40]

5. A Foley catheter should be inserted if clinically indicated.

6. Transport the patient in the supine position. Ground transportation is preferred but, if air transportation is used because of the distance to be covered, cabin altitude pressurization should be kept as close to sea level as possible.[41]

7. If transportation is delayed, IV crystalloid with glucose should be started through a large-bore catheter at less than daily maintenance rates.[42]

8. Diazepam may be administered in 5-mg IV boluses to a maximum dose of 30 mg for control of seizure activity as well as for sedation and to increase compliance with an endotracheal tube.[42] Barbiturates are second choice drugs.

9. Hypertonic solutions such as mannitol should *not* be used routinely because of the risk of a rebound increase in intracranial pressure.[42] Their use should be reserved for cases of impending herniation.

The proper positioning of an air embolism patient has been controversial. Early studies[43] have shown that embolization while in the head-up position rapidly distributes air primarily to the cerebral circulation; in the head-down position, the coronary arteries receive a greater proportion; when supine as well as in the left lateral decubitus position, air is distributed to both the cerebral and coronary circulations. Although a vital organ will be embolized in any position, survival in the face of repeat *embolization* is probably greatest in the head-down position.[43-47] The head-down position can, however, also cause an increase in cerebral blood flow and a further increase in intracranial pressure.[44-47] In contrast, cerebral circulation may fall if there is systemic hypotension. The best position for transportation, therefore, is probably supine with the head in a neutral position to allow unrestricted arterial and venous blood flow.[47]

Definitive treatment will be rendered by the recompression facility personnel during recompression. The patient is usually recompressed for 30 minutes at a treatment depth of 165 fsw (6 ATA), which mechanically re-

TABLE 2. *Decompression sickness classification and comparison with gas embolism*

	Disorder	Symptom Complex	Pathology	Treatment
Type I	"Pain-only bends"	Pain confined to an extremity, usually peri-articular, aggravated by movement, often relieved by direct pressure (ie, with sphygmomanometer cuff)	Bubble evolution within a non-distensible tissue (ligaments, tendons, bone)	Table V recompression; Observe for serious symptoms
	Skin pruritis	Intense itching, begins during decompression or shortly thereafter, primarily with air dives in chamber, direct pressure may relieve	Gas forced into sweat and sebaceous glands during dive evolves as bubbles on ascent (not true decompression sickness)	No treatment
	Skin marbling	Itching, discoloration, cyanosis over affected area, blanches on pressure	Subcutaneous bubble evolution causing local venous stasis	Table V recompression; Observe for serious symptoms
	Lymphatic symptoms	Lymph node pain and swelling, tissue edema distal to affected nodes	Bubble accumulation within nodes, mechanical obstruction	Table V recompression; Observe for serious symptoms
Type II	All other pain	Pain elsewhere than extremities	May represent referred pain from visceral sites or spinal cord involvement	Table VI recompression; Observe for other symptoms
	Central nervous system	Any hard or soft neurologic symptom; paralysis, sensory deficit, seizures, extreme fatigue, change in personality, headache, visual symptoms, etc	Bubble evolution or accumulation anywhere in brain, spinal cord, or cranial nerves	Table VI recompression
	Labyrinthine	A CNS symptom also known as the "staggers": any 8th nerve symptom, ie, unsteadiness, vertigo, tinnitus, deafness, nystagmus	Bubbles obstructing labyrinth nutrient arteries, ? endolymph bubbles	Table VI recompression
	Pulmonary (chokes)	Increasing substernal burning pain begins within minutes of surfacing, cough and progressive dyspnea follow; often associated with respiratory failure and shock	Accumulation of bubbles within pulmonary arterial tree	Table VI recompression
	Gas Embolism	Any CNS symptom on ascent or within ten minutes after surfacing, can be rapidly progressive to circulatory collapse	Alveolar overpressurization with resultant free arterial air in cerebral and coronary circulation	Table VIA recompression; Life support measures as required

duces the size of the offending bubble to enhance passage of the bubble through the capillary circulation.[48] After 30 minutes at 165 fsw, the pressure is reduced to 60 fsw (2.8 ATA), and the patient breathes 100% oxygen alternating with short air breathing periods to prevent oxygen toxicity. Hyperbaric oxygen improves tissue oxygenation, reduces intracranial pressure,[49] and provides a large diffusion gradient to aid bubble absorption.[48,49]

After treatment, the patient should receive thorough neurologic, cardiology, and pulmonary examinations. Divers who suffer an air embolism should be advised not to return to diving. The risk of another embolic episode with its high mortality should preclude further sport diving.[31]

MISCELLANEOUS PRESSURE EFFECTS

Air within a tooth, either in an area of decay or forced under a filling during descent, also will obey Boyle's law. Extreme pain is the usual manifestation of a tooth squeeze that occurs on ascent when the trapped air expands and causes an increased pressure within the tooth.[2,31] The pressure occasionally is relieved when the filling becomes dislodged; otherwise, recompression in a chamber is indicated.[31] Occasionally, direct mechanical decompression is attempted by drilling[31] but is not universally successful. Tooth squeeze can be prevented through careful dental hygiene and a program of regular professional dental care.

On descent, a negative pressure, a mask squeeze, will be created within the diving mask if the diver fails to adequately ventilate the mask. Sub-

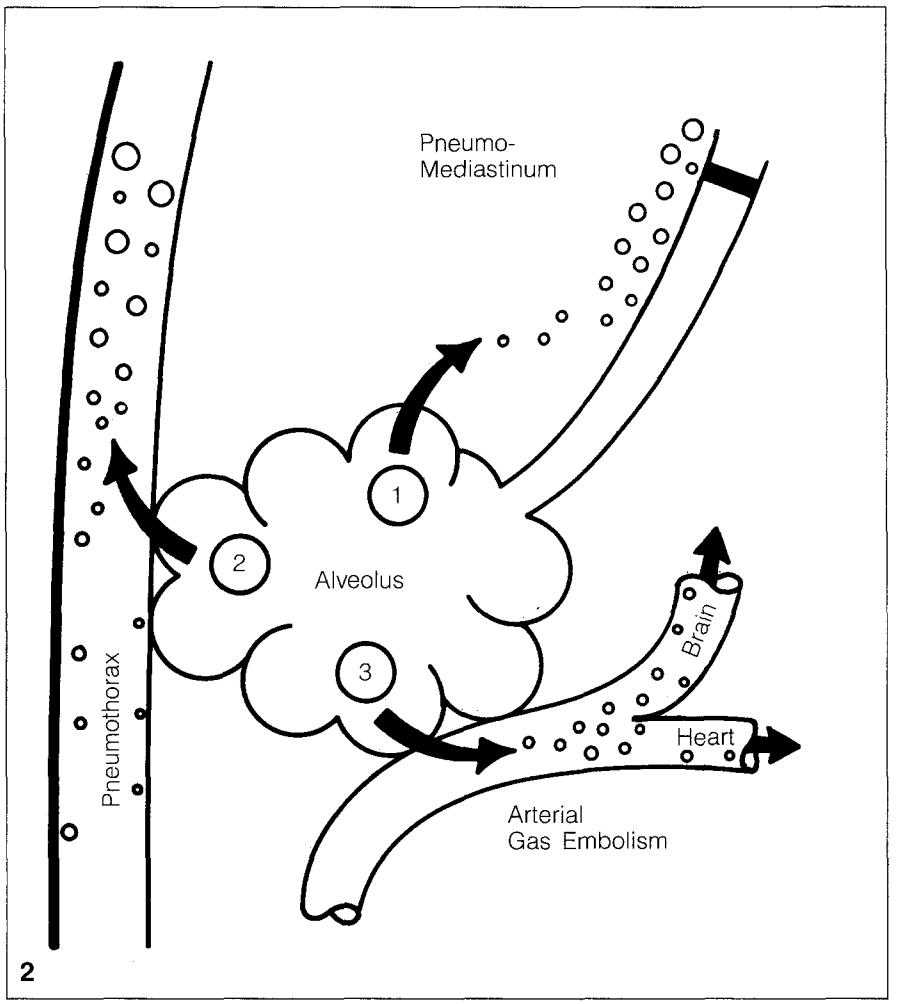

FIGURE 2. *Air released from an overpressurized alveolus can extravasate in one of three pathologic directions to cause (1) interstitial emphysema and pneumomediastinum, (2) pneumothorax, or (3) arterial gas embolism.*

cutaneous capillary flushing and small hemorrhages are common but benign effects.

DECOMPRESSION SICKNESS

The nitrogen and oxygen in the compressed air breathed by scuba divers manifest effects because of their increased tissue partial pressures as well as direct effects of bubble evolution caused by liberation of nitrogen from tissues that have become supersaturated during a dive.[50,51]

Decompression sickness (also known as Caisson's disease and the "bends") is the result of a series of pathophysiologic responses to the evolution of dissolved tissue gases, precipitated by changes in ambient pressure. Bubbles released from solution by a too-rapid reduction in ambient pressure either obstruct blood flow, cause blood chemistry changes, or stretch and damage tissue. The symptoms can range from innocuous skin itching to central nervous system or pulmonary compromise. Prompt diagnosis and recompression treatment are essential.

On-Gassing and Off-Gassing

Following Henry's law, the rapidity and amount of gas dissolved (or released from solution) in tissues is proportional to the change in ambient pressure. When a mixture of gases is inspired, the amount of each gas that becomes dissolved in tissues is proportional to the partial pressure of each gas. Further, the eventual tissue concentration of inspired gases also depends on the rate at which the gases are either removed or metabolized within the tissues. When breathing air, the dissolved tissue gas of concern is nitrogen because the oxygen component, unlike nitrogen, is rapidly consumed by tissue metabolism.[51] The major factors that govern dissolved gas accumulation due to changes in ambient pressure are as follows:

1. Ambient pressure governs the rate and total amount of gas dissolved in tissues. The two most important factors that determine the amount of gas dissolved in the tissues are the depth of the dive and the amount of time spent at depth.

2. Partial pressure governs the amount of each gas (in a mixture) that is dissolved in tissues.

3. Tissue metabolism removes oxygen; inert gases and carbon dioxide remain.

Exercise increases gas uptake and elimination by increasing the respiratory rate, cardiac output, and tissue perfusion, increasing the rate of tissue gas delivery and removal.[50-53] Gas temperature and the diver's state of hydration also affect the rates of gas uptake and elimination.[54,55] Smoking and alcohol consumption may increase susceptibility to decompression sickness by a variety of poorly defined mechanisms.[51] Age and sex also have been mentioned as factors influencing the incidence of decompression sickness, but their association probably has limited practical value in sport diving.[56-59]

Under increased ambient pressure, gases become dissolved in different tissues at varying rates because each tissue has its characteristic rate of gas uptake known as the "tissue half time."[51] Gases become rapidly dissolved in muscle but are dissolved very slowly in bone and fat.[51] Fat can take on a large amount of nitrogen because nitrogen is five times more soluble in fat than in water.[60,61] Because gas elimination follows a similar time course, nitrogen is eliminated from fat tissues very slowly, and supersaturation (with subsequent bubble formation) may occur if the ambient pressure is reduced rapidly.[51] Thus obese people seem to experience decompression sickness more frequently than do lean divers.[56,57,62-64] This is usually only a factor in longer and deeper dives than those attempted by sport divers.

At sea level, gases (mainly nitrogen) are dissolved in tissues at a steady state *minimum* level determined by

the three major factors discussed previously. There is also a *maximum* amount of gas that can be dissolved in tissues without evolution of the gas as bubbles. The gradual uptake of nitrogen during a dive is shown (Figure 3). Gas uptake, or "on-gassing," occurs at a faster rate during a deep dive than on a shallow dive and continues from the start of the dive until the ambient pressure is reduced by ascent. If ascent is delayed, tissue gas levels will continue to increase until the tissue concentration reaches the steady state level consistent with the new, higher ambient pressure. This takes approximately 12 hours at the higher ambient pressure[51,60,61] and is only of concern when performing saturation dives in which divers intentionally stay underwater for many hours or days.[51,60,61]

If the diver ascends before the level of dissolved nitrogen reaches the maximum permissible at sea level (supersaturation), gas elimination, or "off-gassing," will occur gradually and without bubble formation (curve [A]). If the diver were to remain at the increased ambient pressure until the amount of dissolved tissue nitrogen exceeded the supersaturation level, nitrogen probably would be liberated as bubbles if the diver surfaced directly. The increased ambient pressure at depth keeps the additional tissue nitrogen dissolved, whereas the lower ambient pressure at the surface would promote gas evolution at a rapid date. In this case, the diver should ascend to a safe depth for a "decompression stop," at which the ambient pressure is sufficient enough to allow gradual off-gassing while preventing bubble formation, yet is not great enough to cause additional gas to become dissolved in the tissues (curve [B]). After sufficient time at the decompression stop to bring the tissue nitrogen concentration below the supersaturation level, the diver can ascend directly to the surface (curve [C]). Some dives require more than one decompression stop, in which the first stop is made to allow enough off-gassing for the diver to safely ascend to the next stop.

Sport divers should always follow non-decompression diving tables and ascend before the surface supersaturation level is reached. The US Navy diving tables[65] are used as a guide to safe diving time/depth constraints.

Because the amount of dissolved tissue gas gradually returns to the normal sea level amount over the 12 hours following a dive, the times for repetitive dives on the same day must be adjusted. The additional tissue nitrogen remaining at the beginning of a subsequent dive is called the residual nitrogen.[51,60] Because the second dive starts with some residual nitrogen, the time allowed for the second dive should be shortened by the time that would have been required (if it were a single dive) to obtain the tissue nitrogen equivalent to the amount left over from the first dive. This is called the residual nitrogen time and is the time between points (A) and (B) (Figure 4). Deviations from established depth/time constraints can easily result in the diver exceeding a tissue supersaturation level and increase the risk of decompression sickness.

Clinical Manifestations

Symptoms are evident within an hour of surfacing in approximately 80% of patients and within four hours in more than 95%;[66] symptoms of decompression sickness arising more than 12 hours after a dive are very unusual. The occurrence of a gas embolism is presumed when the symptoms arise within ten minutes after surfacing.[25,26,28] The most frequent error in treatment of decompression sickness is failure or delay in making the diagnosis.[51,52,65,66] This can lead to delayed treatment, which often results in permanent sequelae instead of prompt resolution.[67]

Decompression sickness has customarily been divided into two categories (Table 2), Type I (musculoskeletal), which manifests as limb pain or with skin or lymphatic involvement; and Type II (neurologic), also known as "serious" decompression sickness, which encompasses all other symptoms.[51,65,66,68] Decompression sickness is dynamic and a patient initially presenting with Type I symptoms may progress to more serious manifestations. The diagnosis of any type mandates immediate transport to a recompression facility.

Type I decompression sickness is the more common variety and is the classic presentation of "pain only" bends.[51,65,66] This includes pain confined to the arms or legs that may be aggravated by movement and relieved by direct pressure on the area, ie, with a sphygmomanometer cuff.[51,66,68,69] There should be no association with systemic symptoms and the pain should not be referred or confused with paresthesias or hypesthesias. The pain is usually peri-articular, involves the upper extremities three times more frequently than the lower extremities in divers,[66,69] and ranges from mild discomfort (often called the "niggles") to severe pain.

There are many forms of skin bends: pruritis alone, various rashes (most commonly an erythematous, serpiginous rash), and skin marbling (cutis marmorata).[68-70] Pruritis usually is encountered only after deep chamber dives in which the diver is surrounded by compressed air, and itching usually begins during decompression or shortly after reaching the surface.[70] Gas entering the sweat and sebaceous glands during compression is thought to form pruritic bubbles as the diver surfaces.[70] The itching of skin bends must be differentiated from the tingling of paresthesias or hypesthesias. The pruritis may be relieved somewhat by direct pressure and, although usually localized, may involve a large area such as an extremity or the torso, but does not follow dermatomes. Pruritis is not considered a true form of decompression sickness and resolves rapidly without treatment.[51,65,66]

Marbling of the skin occurs when subcutaneous bubbles cause venous stasis. The symptoms commonly occur over the torso and shoulders and commonly begin as intense itching quickly followed by discoloration.[70] Erythema progresses to cyanotic mottling that may be linear or patchy, is non-tender, characteristically blanches on direct pressure, and resolves rapidly when treated with recompression. Marbling is a true form of decompression sickness, indicating that tissue supersaturation has occurred, and may be a harbinger of systemic involvement and impending serious symptoms.

Although rare, isolated lymphatic symptoms occasionally occur as a result of lymphatic obstruction by bubbles.[51,66,70] There is usually pain and swelling of lymph nodes or groups of lymph nodes with variable edema of the tissues normally drained by the affected nodes. Although recompression usually produces rapid relief of the pain, swelling may persist for days.[51,66]

Type II decompression sickness includes all other manifestations of evolved gas pathology. These are pain

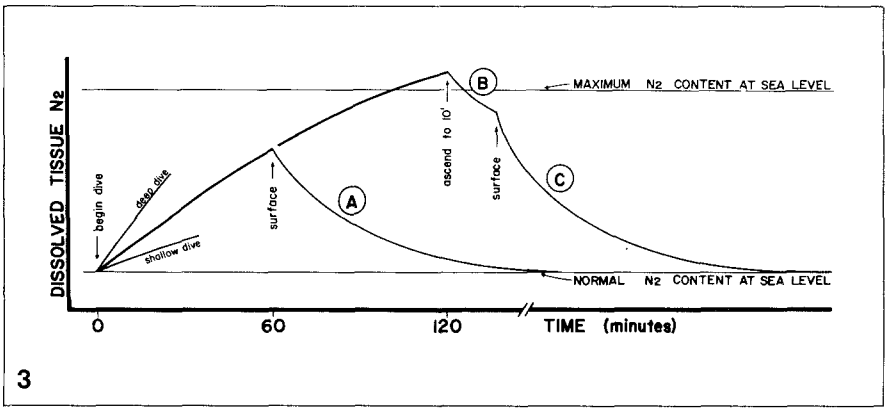

FIGURE 3. *The effect of depth on dissolved tissue nitrogen levels. A diver can safely surface directly if the tissue level of nitrogen at depth has not exceeded the maximum level that the tissues can hold at sea level (curve A). If this maximum level is exceeded, the diver must ascend to a depth at which off-gassing can occur without bubbling (10 feet in this case) and remain there until the tissue level falls below the surface supersaturation level (curve B); he can then safely ascend to the surface (curve C).*

elsewhere than the extremities, any central nervous system sign or symptom, and pulmonary manifestations (chokes). Pain other than in the extremities, although not a serious symptom *per se*, may represent referred pain from visceral sites or spinal cord involvement.[35,51,67-69] Therefore, pain in the head, neck, and torso should be classified as a Type II symptom and treated accordingly while observing for other serious symptoms.

No clear central nervous system symptom complex is characteristic for decompression sickness.[35] *Any* neurologic symptom can be associated with the intra- and extra-vascular evolution of bubbles anywhere in the nervous system. Spinal cord symptoms are most common in divers, while cerebral symptoms predominate in aviation personnel.[51,59] Paresthesia is a very common presenting symptom of a spinal cord "hit" and may progress to ascending numbness, a dermatomal distribution of pain at approximately the level where bubbles have accumulated, or paraplegia.[35] Cord lesions are most common in the lumbosacral region and can be associated with bladder paralysis, urinary retention, fecal incontinence, and, occasionally, priapism.

Cerebral decompression sickness can present with signs such as seizures, hemiplegia, or the common visual symptoms of scotomata, diplopia, tunnel vision, or blurring.[35,51,65-69] Headaches are also common and may mimic migraine headaches. Any hard neurologic sign can result from evolution or accumulation of bubbles in any area or near any cranial nerve. Unconsciousness and shock represent global and fulminant manifestations and are rare.[35,51,65-69] Soft signs and symptoms are common and include unusual fatigue, a sense of detachment from the surroundings (being within one's self or viewing the activities from a point removed from one's body), inappropriate or uncharacteristic behavior, or any number of other variable presentations.[35,51,68,69]

Although the cranial nerves are not usually affected, if they are, labyrinthine or inner ear decompression sickness (the "staggers") is a common presentation.[35] Either the cochlea or the vestibule can be involved, and the patient often presents with any combination of vertigo, nausea, vomiting, deafness, tinnitus, and nystagmus. Immediate treatment is important because prolonged obstruction of the small nutrient arteries supplying the inner ear structures can result in permanent damage.[35,68]

Pulmonary decompression sickness (the "chokes") usually occurs within minutes of surfacing and presents as a triad of substernal pain, cough, and dyspnea.[51,65,66,68,69] Substernal pain, usually the first symptom, is described as "burning" in nature, and is aggravated by deep inspiration. The cough is initially occasional but is easily evoked by cigarettes and may progress to uncontrollable paroxysms.[51,68,69] The substernal pain increases as the frequency of cough increases and dyspnea becomes superimposed. Although the "chokes" is uncommon, its severity and prognosis make immediate treatment mandatory. Respiratory failure and shock are often the terminal events in pulmonary decompression sickness.[51,66,68,69]

Pathophysiology of Decompression Sickness

The fundamental cause is evolution of bubbles within tissues and vessels. Tissues with an increased nitrogen content must release nitrogen to the blood, which transports it to the lungs for elimination. When the ambient pressure is reduced too much or too rapidly, some tissues cannot diffuse their nitrogen into the blood fast enough and they become supersaturated, resulting in some nitrogen coming out of solution as bubbles. These bubbles can be interstitial, intralymphatic, or intravascular and have their effect either mechanically (as they obstruct blood flow or distort tissue) or indirectly (through endothelial damage, protein denaturation, and altered blood coagulation).[51,67,68,71,72]

The mechanical bubble effects are most prominent in spinal cord, brain, pulmonary, and possibly inner ear as well as peri-articular (pain only) decompression sickness.[51,66,71,72] Analysis of spinal cord decompression sickness in dogs has revealed the following sequence:[71-73]

1. Accentuated by slowed venous return, bubbles accumulate and coalesce in the epidural venous plexus.
2. Aided by an increase in blood coagulability, these bubbles progressively obstruct epidural vertebral veins.
3. Accumulation of bubbles in the pulmonary vasculature causes an increase in pulmonary artery pressure, increased mean central venous pressure, and increased central venous pressure fluctuations in response to varying intrathoracic pressure. When transmitted to the epidural veins, these pressure changes slow blood flow and enhance obstruction of the epidural veins by bubbles.
4. Venous cord infarct eventually occurs. Hemorrhagic white matter infarcts often occur at multiple levels with relative sparing of grey matter.[74,75]

The mechanical bubble effects are attributed to accumulation of gas bub-

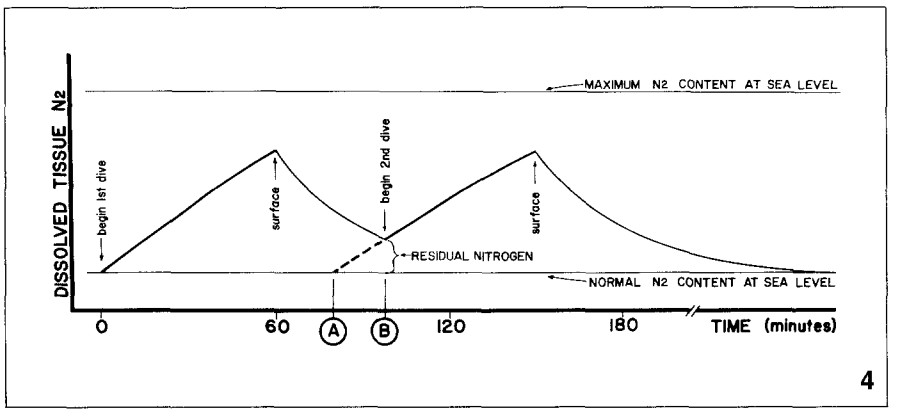

FIGURE 4. *Nitrogen on-gassing during repetitive dives. If a second dive is begun before the tissue nitrogen accumulated on the first dive has totally off-gassed (approximately 12 hours), then the allowable time for a second dive must be shortened by the amount of time that would have been required to have accumulated the amount of "residual nitrogen" in only one dive. This time is known as the "residual nitrogen time" and is the time between points A and B.*

bles in the pulmonary arterial tree. Doppler studies have demonstrated the presence of pulmonary bubbles in asymptomatic men and in decompressed dogs who, although asymptomatic, have increases in pulmonary artery and pulmonary wedge pressures, increased pulmonary vascular resistance, and decreased cardiac output.[75,76] With further bubble accumulation, tachypnea occurs early and is closely followed by increases in pulmonary artery systolic and diastolic, right ventricular systolic, and right ventricular end diastolic pressures, as well as decreases in cardiac output and arterial pO_2.[77-79] Decreased dynamic compliance and increased pulmonary vascular resistance have been reported.[80] Although adult respiratory distress syndrome has been associated with pulmonary decompression sickness, it is rare because patients with such serious pathology die before reaching a treatment facility.[81]

Type I (pain only) decompression sickness also has been attributed to the mechanical effect of gas bubble evolution within non-distensible tissue such as tendons and ligaments.[82]

There also are many indirect effects caused by the blood-bubble surface activity interaction. Globular plasma protein denaturation has been reported,[83,84] which results in release of free phospholipids, cholesterol, triglycerides, and free fatty acids. Because coalescence of these lipids into globules has been observed,[84,85] they may contribute to vascular obstruction.

There is an increase in the number of free circulating endothelial cells[85,86] and endothelial cells involved in platelet clumps[85] as well as histologic evidence of focal endothelial cell loss[86] in swine decompression sickness. The denuded areas may be sites for platelet adhesion and fibrin deposition, contributing to the vascular obstruction.

Bubbles accelerate clotting in whole blood and cell-free plasma *in vitro* as a suspected result of Hageman factor activation.[27] A fall in circulating platelets, alterations in the PTT and factors V and VIII, and an increase in fibrin split products have been demonstrated after decompression in rats.[87] Platelet and fibrinogen consumption increase even in asymptomatic divers.[88-91] An intravascular bubble acts as a nidus for platelet adherence and aggregation, further obstructing blood flow.[88] Coating of red blood cells with denatured protein can cause red blood cell clumping, thereby increasing blood viscosity and promoting stasis.[92] Because venous blood flow is already slow, any further slowing could result in clotting with an increase in capillary filtration pressure leading to interstitial edema and loss of plasma volume — a vicious cycle.[92,93]

Most cases of decompression sickness that are treated with recompression relatively early show rapid and complete improvement of all symptoms and recover without residua.[51,65,66,69,94] These indirect pathophysiologic effects are important in patients left with permanent sequelae, either because treatment was delayed or the pathologic insult was great. There is also a middle spectrum of patients who have slowly resolving symptoms.[69,94,95] In these, the indirect effects probably had begun prior to treatment and their pathologic course requires time to reverse.

Decompression Sickness Treatment

Early recognition and treatment of decompression sickness is essential for resolution without sequelae.[94,95] Even divers stricken with severe effects of decompression sickness can recover totally with prompt recompression, while delay can result in lifelong disability. Patients whose recompression has been delayed must, nonetheless, be referred expeditiously because even delayed treatment can be beneficial.[95]

Patients with Type I decompression sickness will require only reassurance, oral fluids, and immediate transport to a recompression facility, but patients with Type II decompression sickness usually will require early, more vigorous supportive and therapeutic measures. The initial treatment guidelines for Type II decompression sickness should follow those listed for arterial gas embolism.[96,97]

Recompression is the mainstay of decompression sickness therapy.[98] The details of recompression therapy will be handled by a specially trained treatment team at the recompression facility. The vast majority of treatments are performed using US Navy treatment tables[69] in dry chambers. These tables describe recompression chamber operations that bring the patient to an initial treatment depth of 60 fsw (2.8 ATA), where 100% oxygen is breathed (usually for 20-minute periods separated by five-minute air breathing breaks to prevent pulmonary oxygen toxicity). After a variable time, a slow transition is made to 30 fsw where oxygen and air breathing are again alternated. After a variable time at 30 fsw, the patient is slowly brought to sea level pressure. The difference between the tables is the length of time the patient remains at the 60 and 30 fsw depths. The total treatment time varies from 140 to 270 minutes for uncomplicated treatments.

The three objectives in recompression therapy are to reduce the size of the bubble, promote bubble reabsorption, and prevent further bubble evolution.[98] Reduction in bubble size is important initially and is accomplished purely by the increase in ambient (chamber) pressure and results in relief of vascular obstruction and tissue distortion, enhancing reperfusion and oxygenation. Intravascular gas is compressed into a spherical shape and reduced in size, promoting distal migration and reduction in the size of the ischemic area.[99-101] Bubble reabsorption also is enhanced by the increase in ambient pressure as the partial pressure of the nitrogen within the bubble exceeds the partial pressure of nitrogen in the surrounding tissue, increasing the diffusion drive.[102,103] Breathing 100% oxygen washes out tissue nitrogen, enhancing diffusion by further widening the nitrogen partial pressure difference between the tissue and the bubble.

Based on the patient's response to recompression treatment, the treatment tables are modified, usually lengthening them to provide added treatment time. Occasionally, treatment must be extended so long to prevent symptom recurrence that movement from 60 fsw to 30 fsw or from 30 fsw to the surface cannot be done without risking further bubble evolution. In this case, the dive may become a "saturation dive," which can be extended almost indefinitely at any depth, terminating with a very slow ascent accomplished over many hours or days. Multiple treatments are often required if symptoms persist; therapeutic benefit is primarily derived from tissue hyperoxygenation rather than the hyperbaric effects.[104-106]

Adjuvant drugs are used during recompression treatment.[107,108] These should never substitute for recompression and are not useful in each case. Oxygen enhances tissue oxygenation, reduces cerebral edema, and washes nitrogen out of tissues to increase the bubble/tissue diffusion gradient.

IV fluid therapy is a necessary adjunct in all cases of Type II decompression sickness because fluid loss, hemoconcentration, and increased blood viscosity promote vascular occlusion.[92,93,107-111] A crystalloid such as Ringer's lactate or normal saline with glucose can be used initially; the fluid for volume and glucose is a metabolic substrate. Fluid replacement in normotensive patients should be delivered at 250 to 500 mL/hr while monitoring blood pressure, hematocrit, and urinary output. Fluid replacement should not be so vigorous that pulmonary edema results. If hypotension arises, colloid replacement should be considered based on the clinical picture, hematocrit, and urinary output, which should be at least 0.5 mL/kg/hr.[51,69,94]

Dextran has been successfully used as a volume expander,[110,111] and it also has an anticoagulant effect. Both dextran 40 (40,000 molecular weight) and dextran 70 (70,000 molecular weight) have similar volume-expanding properties; dextran 40 acts slightly more rapidly than does dextran 70 but is more rapidly cleared by the kidneys.[112-115] Both dextran 40 and dextran 70 cause decreased platelet adhesiveness, decreased levels of platelet factor 3, and decreased vascular sludging, and act to coat platelets, red blood cells, and vascular endothelium.[112-115] Use of dextran is no longer recommended because of the risk of side effects such as fluid overload, renal failure, and anaphylaxis.[114,115]

Shock should be managed with fluids because the underlying pathology is intravascular fluid loss and loss of spinal cord control of vasomotor tone.[113] Dopamine can be used in the 1 to 10 μg/kg/min dosage range to increase coronary artery blood flow, cardiac output, and renal blood flow without increasing myocardial oxygen demand. If shock persists despite dopamine therapy, epinephrine, neosynephrine, or levophed also may be used.[110,113]

Diazepam is useful in controlling vertigo, nausea, and vomiting associated with labyrinthine decompression sickness.[108,116] It also will help control seizures, decrease patient agitation, and increase compliance with intubation. Diazepam should be used with caution because it may delay treatment by masking a progression of labyrinthine symptoms.[116]

Antiplatelet agents such as aspirin and dypridamole (and their analogs) may be effective in prevention of decompression sickness because of their actions on platelet kinetics, but their efficacy in treatment has not been confirmed.[89,116-119] The use of heparin and other anticoagulants has been examined by many investigators[87,108,116,120-125] with inconclusive results. In cases of labyrinthine decompression sickness, anticoagulants (including dextran) are contraindicated because hemorrhage is thought to be important in its pathogenesis.[124]

After Treatment

Patients should be evaluated for admission to the hospital for overnight observation following any recompression treatment. Any recurrent or new symptom following treatment is automatically classified as Type II, a serious symptom, and the treatment is recompression.[107,108,110] The patient should not dive for at least four to six weeks after a Type I hit and at least three to six months following a Type II hit. Repeated Type I hits should not preclude reexposure, but a second Type II episode should warrant critical evaluation of the patient's fitness for further diving.

NITROGEN NARCOSIS

Nitrogen narcosis ("rapture of the deep") is the progressive intoxicating or anesthetic effect of nitrogen that increases with depth.[51,65,66,69,126] Each 50 fsw has informally been equated to having one martini, hence, the "martini rule."[51,69] With considerable individual variation, at 100 fsw, a diver may feel lightheaded and euphoric, lose dexterity[126] and reasoning ability,[127] and have increased reaction times.[127] A diver can become disabled by the disorienting and euphoric effects of nitrogen narcosis, can lose his mouthpiece and be unable to find it, and not be able to determine which direction is up. Hypercapnia can accentuate the effects.[128] Treatment is to ascend to a depth of relief. Divers who perform frequent deep dives breathing air can partially acclimatize to the narcotic effects of nitrogen. If deep dives are required, a better solution is to change the diluent to helium instead of nitrogen.

SHALLOW WATER BLACKOUT

A technique commonly used by free divers (snorklers who are not using compressed air or an accessory breathing device) to stay submerged for prolonged periods is to hyperventilate just prior to the dive. This eliminates a large amount of CO_2, decreasing arterial pCO_2 — the primary respiratory driving force.[129] Decreasing the pCO_2 also results in cerebral vasoconstriction. As the diver descends, the increased ambient pressure results in a concomitant increase in arterial pO_2.

Because the respiratory drive has been suppressed, the diver tends to remain at depth until either the hypoxic drive or the eventual accumulation of CO_2 urges him to surface. The diver remains conscious because the increased pO_2 at depth has maintained cerebral function but, as the ambient pressure rapidly decreases as he surfaces, the arterial pO_2 falls precipitously and, coupled with the cerebral vasoconstriction, can result in unconsciousness prior to reaching the surface. Shallow water blackout is most common among experienced divers who have become accustomed to holding their breath and tolerating excess arterial pCO_2 beyond normal limits.[51,73] This can be prevented by not hyperventilating prior to a dive.

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