Diving and Marine Medicine Review
Part II: Diving Diseases
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Diving Diseases

Diving is a high-risk sport. There are approximately between 1 to 3 million recreational scuba divers in the USA (with over a quarter-million learning scuba annually), there are about 1 million in Europe and over 50,000 in the United Kingdom. In this population 3-9 deaths/100,000 occur annually in the US alone, and those surviving diving injuries far exceeds this. Diving morbidity can be from near-drowning, from gas bubbles, from barotrauma or from environmental hazards. In reality, the most common cause of death in divers is drowning (60%), followed by pulmonary-related illnesses. The mean number of annual diving fatalities in the USA from 1970 to 1993 was 103.5 (sd 24.0) and the median was 106.

This article will focus primarily upon pressure effects on the health of a diver. There are two principle ways pressure can affect us: by direct mechanical effects and by changing the partial pressures of inspired gases. Dysbarism is a general term used to describe pathology from altered environmental pressure, and has two main forms: barotrauma from the uncontrolled expansion of gas within gas-filled body compartments and decompression sickness from too rapid a return to atmospheric pressure after breathing air under increased pressures.

Greater than 90% of the human body is either water or bone, which is incompressible; the areas directly affected by pressure changes thus are those that are filled with air or gas. These sites include the middle ear, the eustachian tube, the sinuses, the thorax, and the gastrointestinal tract. Air in these cavities is compressed when the ambient pressure rises because the pressure of inhaled air must equilibrate with the ambient pressure.

Barotrauma

Barotrauma is the damage or injury from a pressure gradient between the environment and air-containing

Figure 1  Primary air-filled spaces of the body (illustration by the author).
body cavities distorting tissues. It is a mechanical problem of pressures not being equal between body spaces and the hydrostatic pressures (or the diving equipment). 

This gradient can be positive during descent underwater with increasing pressure or it may be negative during ascent towards the surface when the pressure within the body cavities is greater than the surrounding environment. It can occur in as little as 4 feet of depth without equalization (90 mm Hg pressure gradient).

**Otic** The middle ear is the most common body part affected from barotrauma, occurring in up to 30–60% of divers. While the inner ear is fluid-filled, the middle and outer ear canal are air-filled. The middle air space is separated from the outer ear by the tympanic membrane (TM); the only way air pressure can be equalized in the middle ear is via the eustachian tube into the oropharynx (with an intact tympanic membrane).

Middle Ear Squeeze occurs during descent when the surrounding water pressure exceeds the air pressure in the middle ear and is dependent upon rate of descent and the state of eustachian tube function. This unequal pressure bows the tympanic membrane inward, causing discomfort or a sensation of fullness; if not relieved, pain follows. Equalizing the pressure is accomplished by opening the eustachian tubes so that pressurized air from the scuba tank present in the mouth can enter the middle ear space. The eustachian tube is normally closed by positive middle ear pressure and can be opened by swallowing or various maneuvers (below). If the eustachian tube is closed or congested or if the pressure change is rapid, the tympanic membrane is deformed into the middle ear space. If middle ear space air pressure does not rise to balance the pressure on the opposite side of the tympanic membrane, then vascular congestion, hemorrhage, pain and membrane rupture may follow. If the TM ruptures and cold water rushes into the middle ear, the vestibular system is disrupted by this cold caloric stimulus; vertigo, nausea and vomiting may ensue and underwater this may be fatal. Hearing loss and tinnitus may also occur.

Common causes for eustachian tube dysfunction are inability to equalize as rapidly as the pressure changes; acute or chronic inflammation; allergy; anatomical deformities; prolonged use of nasal drops; frequent or acute upper respiratory infection, nasal allergies, nasal obstruction, or ear disease; and excessive smoking. Scarring of the tympanic membrane from otitis media during child-

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**Figure 2** Principle parts of the ear in cross section. (Bove & Davis' Diving Medicine, 3 Ed. page 238, 1997. Reprinted with permission, W.B. Saunders Company.)
hood contributes to an inflexible membrane and reduced eardrum flexibility when the pressure gradient changes; this can lead to an inability to equalize and the risk of drum rupture. It is also more difficult to equalize underwater if the diver descends head-first; when descending feet-first, drainage is more effective and easier. Underwater, if equalization is not successful, the diver must ascend until the level when equalization does occur and then redescend slowly.

As the pressure gradient is relatively greatest in the first 33 ft/10m under the surface (especially in the first 6 ft/2 meters), it is most likely to occur near the surface than deeper down. Middle ear barotrauma of descent is diagnostically graded by otoscopy. Grade 0 entails symptoms without signs; Grade I has injection of the tympanic membrane, especially along the handle of the malleus; Grade II has injection plus slight hemorrhage with the tympanic membrane; Grade III has gross hemorrhage within the tympanic membrane; Grade IV has free blood in the middle ear—seen as blueness and bulging; Grade V consists of tympanic membrane perforation.
A reverse squeeze is a situation where the air pressure in the middle ear exceeds that of the ambient water pressure, often from cerumen, stenosis, atresia, or tight-fitting hoods.20 Air in the middle ear will expand by Boyle's Law as one rises to the surface; if not relieved by opening the eustachian tube, the tympanic membrane will bulge outward as the water pressure decreases closer to the surface. This can also occur when a diver has taken decongestants which wear off while underwater and edema returns to the eustachian tube and middle ear before ascending.13

Alternobaric Vertigo is an asymmetric middle ear barotrauma during ascent, where middle ear air on either side cannot exit on ascent, with unequal vestibular stimulation, leading to vertiginous symptoms underwater. This vertigo can stop by halting the ascent or by descending again.17 Very rarely this can lead to facial paralysis.21

External ear squeeze occurs if the external auditory canal is obstructed, usually by cerumen, tight fitting hoods or ear plugs. It may occur on ascent or upon descent. Pain, hemorrhage and possible tympanic membrane rupture may occur.13

Inner ear barotrauma can have severe consequences from round or oval window rupture with possible perilymph fistulae or cochlear hemorrhage with sensorineural hearing loss and vestibular dysfunction.13 Round window rupture from excessively negative middle ear pressure is more likely with overly forceful valsalva maneuvers which raise cerebrospinal fluid (CSF) and inner ear pressures.13 The round window is located between the middle and inner ears, and rupture often leads to tinnitus and decreased hearing. This should be suspected when tinnitus and vertigo are severe, associated with nerve deafness, and occurred after a no-decompression dive.17 It can mimic inner ear decompression sickness (DCS) and recompression therapy may nevertheless be necessary.8

Divers who develop otic barotrauma must be able to clear their ears before continuing a dive; if unable to do so the dive must be aborted. If symptoms do not improve upon surfacing, referral to a otorhinolaryngologist is recommended. If there are no objective tympanic membrane findings then one should not dive until nasal and ear symptoms are gone, and the diver can easily autoinflate both ears. Systemic decongestant-antihistamines can be used as well as nasal drops that are long acting. If neccessary.17 Very rarely this can lead to facial paralysis.21

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Equalization Techniques As one descends under the water, equalization must be repeated often to prevent barotrauma from developing because the pressure continues to rise: every 2 feet (0.61m) raises the pressure by 1 psi. When a squeeze develops during descent, one must first stop the descent and equalize.4 If equalization fails, then ascend a few feet and try again. If not able to equalize at any point, the dive must be aborted.17

The main ways to equalize the pressure in the middle ear are ascending a few feet; swallowing with the nose and mouth closed; yawning; or gentle blowing against a closed mouth and nostrils. Beware that strong valsalva maneuvers can rupture the round window.

Sinuses The sinuses present in the head are the second most common sites of barotrauma.20 The frontal sinuses are affected more than the ethmoids and maxillary sinuses with pain and epistaxis being the most common consequences.21 The ostia draining mucus from the various sinuses into the nasal cavity can easily clog with minimal inflammation, and if the pressure between the sinuses and the surrounding water does not balance, barotrauma results.

Upper respiratory infections, sinusitis, nasal polyps, allergies, nasal spray overuse, cigarette smoking and anatomic abnormalities such as deviated septum can all predispose to sinus barotrauma. During descent, when most sinus barotrauma occurs, the negative pressure in the sinus cavity can tear mucous membranes from the sinus walls, which may be both painful and bloody.22 During ascent, the membranes are compressed and the thin skeletal walls may deform.

As with otic barotrauma, equalization is key; enough time should be taken during descent and ascent to allow pressures to balance within the sinuses and the surrounding water. Most cases need no treatment,23 but may include vasoconstrictor agents, both topical and systemic, analgesics and antibiotics for secondary infections if necessary.17

Pulmonary Pulmonary barotrauma encompasses the Pulmonary Overpressurization Syndromes (below) and
also includes thoracic squeezes and diffuse alveolar hemorrhage from rapid or uncontrolled ascents.\(^3,4,6\) A thoracic squeeze occurs when compression of air in the lungs reduces the volume to less than residual volume.\(^6\) In general, pulmonary barotrauma is uncommon, but the consequences are severe as outlined below.

**Gastrointestinal** When a diver swallows air underwater it will expand during ascent, stretching the intestines. This barotrauma of ascent may range from uncomfortable to painful, and may be associated with increased eructation and flatulence post-dive.\(^6\) This is most common in novice divers and those who drink carbonated beverages or eat heavily before diving.\(^6\) It also occurs with steep head-down angles during descent.\(^6\) Rarely, a hollow viscus overdistended by air, such as the stomach, may rupture from barotrauma.\(^7\)

**Dental** Barontalgia is also called Tooth Squeeze. It can arise from caries, defective caps or crowns, temporary fillings, root canal therapy, periodontal abscesses, maxillary sinus congestion or dental lesions.\(^6\) By the gas laws, such teeth may actually implode during descent or explode upon ascent.\(^6\)

The affected tooth is often sensitive if not painful, and pain may be referred to the sinuses. The reverse is also possible; many cases of dental pain originating after flying or diving are actually sinus in origin. All divers should have good dental care and those with temporary crowns should not dive.\(^6\)

**Mask** A mask squeeze develops as a result of not enough air pressure within the mask during descent and as a result of lower pressure, facial tissue is pulled into the mask, resulting in subconjunctival hemorrhage, periorbital petechiae or ecchymoses. While frightful appearing, it is not serious, resolving spontaneously within days. Prevention is simply by exhaling slightly through the nose or mouth during descent.\(^6\) There have been reports of orbital subperiosteal hematomas, but they are rare.\(^6\)

**Pulmonary Overpressurization Syndrome**

Pulmonary Overpressurization Syndrome (POPS) is a dysbaric illness and has four clinically important entities: arterial gas embolism, pneumomediastinum, pneumothorax and subcutaneous emphysema. It is due to pulmonary barotrauma, PBT, and is also called “burst lung.”\(^12,13,30\) These arise from gas expansion during ascent which exceeds the lung's elastic ability, resulting in alveolar tissue rupture. It does not normally occur in snorkel or breathhold diving because there is no additional gas added to decreased lung volumes at depth—as with compressed air diving. The lungs therefore can only reexpand to their original volume upon ascent to the surface and not beyond that volume.

In healthy lungs, POPS is most likely due to a diver breathholding during ascent or shooting to the surface too rapidly for adequate exhalation to compensate for the degree of gas expansion. There are several pathological conditions as well which may predispose to pulmonary barotrauma: chronic obstructive pulmonary disease; acute and chronic bronchitis; asthma with mucous plugging and bronchoconstriction; pulmonary blebs with spontaneous pneumothorax; pulmonary abscesses and Restrictive Lung Diseases.\(^30\)

**Arterial Gas Embolism (AGE)** This is the most important and feared form of pulmonary overpressurization syndrome, ranking second only to drowning as the cause of death in divers.\(^6,10,31\) Bubbles of expanding gas rupture from overdistended alveoli into the blood stream and immediately become emboli, lodging in arterioles and capillaries, where they produce damage.\(^31\) This condition becomes apparent rapidly, usually within 10 minutes of surfacing often with bloody froth at the mouth or chest pain; there is frequent cardiovascular collapse;\(^30,31,32\) rarely presentation may be delayed.\(^32\) It is seen primarily in scuba divers; it is extremely rare in breathhold divers.\(^30\) Emboli may lodge anywhere: in the brain, the primary organ affected by AGE, these bubbles cause unconsciousness, vertigo, paraesthesias, convulsions, paralysis or paresis, nausea, visual disturbances, headache, confusion or even personality disorders, cerebrovascular accidents and seizures or death;\(^14,17,32,35\) in the heart they may occlude coronary vessels leading to myocardial infarction;\(^36\) in the spinal cord paralysis and numbness may develop. Underwater, arterial gas embolism is often fatal. Dyspnea may or may not be present.\(^30\) Surprisingly, 20% of AGE spontaneously resolve.\(^33\)

In 1994 there were 55 cases of AGE documented by Divers Alert Network (DAN);\(^33\) more than half had symptom onset within 2 minutes. Nearly all occur within 10 minutes of surfacing; many cases occurred while still submerged. Uncontrolled or panic ascents, and breathholding during ascent are the most common reasons for the development of AGE. Divers with underlying lung disease, such as congenital emphysematous bulla, bronchial carcinoma, asthma or any air-trapping mechanisms may significantly increase the risk of developing embolisms.\(^37\)

AGE may occur in very shallow dives (a matter of feet) or very brief dives (lasting several seconds).\(^1,3,30\) The laws of physics as explained in Part I of this series explains that the risk of air embolism is actually greatest near the surface. For example, between 99 ft to 66 ft there is a change of 4 to 3 atmospheres whereas between 33 ft to the surface the change is from 2 to 1 ATA—a greater gradient, which means greater gas expansion. Each foot of seawater has a pressure of roughly 25 mm Hg, so a diver who inhales from a tank at a mere 4 feet and holds his breath to the surface has enough of a pressure gradient developed to force air across the alveolar membrane into the blood.\(^17\)
**Pneumothorax** If the expanding gas ruptures through the alveoli and fills the pleural space it will collapse the lung. Pleuritic chest pain and dyspnea are the most common findings. If the escaped air continues to expand as during ascent it may push on the mediastinal structures, forcing them into the opposite hemithorax, leading to a tension pneumothorax. In this situation cardiac blood flow is impaired, resulting in hypotension, venous jugular distension and decreased breath sounds on the opposite hemithorax. Pulmonary hemorrhage is not uncommon.

Mild pneumothoraces need no treatment as the body will resorb the air. More serious pneumothoraces require drainage of the air with a chest tube or a Heimlich valve; a tension pneumothorax is a surgical emergency, requiring immediate removal of the trapped air with a midline needle thoracostomy in the second intercostal space of the affected side and subsequent chest tube placement. Recompression can actually worsen the situation—by converting a simple pneumothorax into a tension pneumothorax if a tube thoracostomy had not yet been performed.16,17

Anyone with a history of spontaneous pneumothorax is precluded from ever diving.

**Pneumomediastinum** If the expanding gas ruptures into the mediastinal spaces it can track up around the heart and the great vessels and even compress the trachea. Hammon’s Crunch is the sound of air in the precordium which is best heard during systole. Chest pain or dyspnea may result. Without neurological signs or symptoms, recompression is not necessarily indicated for treatment.20

**Subcutaneous Emphysema** If the expanding gas ruptures under the visceral pleura it may track under the skin with the presentation of soft-tissue crepitus, a crunching sensation when the skin is palpated: the compression of tiny air bubbles under the skin feels like the popping of a bubble packing sheet. In severe cases, which may occur concurrently with pneumomediastinum, air can track up along the trachea, where swelling of the neck and face may occur, along with dysphonia or cyanosis.40

**Decompression Sickness (DCS)**

Dive trips to remote and exotic locations are extremely common and easy today; after flying home from such a trip a diver with decompression sickness can present to any physician, anywhere on the planet.33

Recall that air is 21% oxygen; the rest primarily consists of metabolically inert nitrogen, which is not metabolized, accumulating in body tissues. The air a scuba diver inhales under pressure increases the amount of nitrogen absorbed into tissues; when this pressure is released, as during ascent from a scuba dive, nitrogen comes out of solution, and if it does so too rapidly, it forms bubbles in extravascular or intravascular tissues, which was first proven in the last century41 from work done on caisson workers (caissons are specialized units used for constructing underwater tunnels and bridge abutments).42,43 This disease is also known as the “bends,” from the
Unlike air embolisms, decompression sickness does not occur in shallow water, having a very different etiology. Bubbles may form from too much time spent at depth breathing air so that the amount of nitrogen supersaturates tissues or it may be from ascending at a rate where the pressure on the gas is relieved quicker than the body can handle. Unlike arterial gas emboli, these bubbles are primarily venous. Factors that lead to decompression sickness include too rapid an ascent rate, diving for too long or at too great a depth beyond the limits of no-decompression diving limits; exercise; dehydration; age; obesity; previous injury; alcohol; and cold. Decompression sickness is a very complicated pathological process: gas uptake by tissues, gas elimination, and numerous independent factors contribute to poorly predictable bubble formation. The equilibrium rate for $N_2$ varies between different tissues. If an ascent rate is too rapid, supersaturated dissolved gas will coalesce or nucleate and form bubbles in either the bloodstream or in tissues, such as the skin, joints, muscles and nerves. Bubble formation is possible even by boarding an airliner after a dive—as standard cabin pressures are kept at pressures varying between 5,000–8,000 feet aboard commercial craft, bubbles continue to expand as the aircraft climbs to cruising altitude. It is estimated that up to 10% of inert gas absorbed by tissues is released as bubbles upon ascent. Bubbles may cause vascular obstruction, distort tissues, denature proteins, aggregate red blood cells and platelets and activate the coagulation cascade with disseminated intravascular coagulopathy. There are several tables which serve as guidelines for safe depth and time exposures to avoid decompression illness. These are not infallible, however: for example, one can stay within the limits of the United States Navy Dive Tables and still have a finite chance of being "bent." Please refer back to Part I of this series for further details regarding the diving tables.

Decompression sickness may also be called Decompression Illness (DCI) when arterial gas emboli are included in the definition. DCS syndromes include pain syndromes, spinal cord syndrome, cerebral syndrome, peripheral nerve syndrome and dysbaric osteonecrosis. Generally, the sooner the onset of symptoms after a dive, the more severe the case and often the more rapid the progression. Cases involving the brain, spinal cord, heart or lungs are medical emergencies.

The overall statistical risk of developing decompression sickness is between 0.004% and 0.001% or an incidence according to DAN statistics for decompression illness of 1–2 divers per 10,000. Sixty percent of all DCS symptoms occur within 30 minutes after completing a dive; 95% are seen within 24 hours after a dive. Joint pain is seen in 80% of all DCS cases, making it the most common symptom of decompression sickness. In 41% of cases it is the very first presenting symptom. In 1994, DAN recorded 1,163 treated cases of decompression illness, nearly double that of the 678 cases 5 years previously. While 40% of divers have neurological symptoms as a presenting symptom, it ultimately develops in 80% of all decompression sickness cases. It is advisable to view any symptom after a dive as decompression sickness and not wait for signs to develop. In the 1996 DAN Report on Diving Accidents & Fatalities, there were 566 reported cases of decompression sickness or arterial gas embolism treated in 1994. Over 60% of those occurred in the USA, with the states of Florida, California, and Washington having the highest incidence of diving accidents. The ratio of male to female accident victims was 28.6:71.4. There is a clear association between fewer accidents occurring with increasing diving experience. The average number of scuba fatalities in American citizens over the past 25 years has been 103 deaths per year.

DCS is a continuum of bubble-induced injury: for convenience it is often divided into the milder DCS I and the more serious DCS II. DCS I is defined as having skin rash or muscle/joint pains only. Dermatologic findings usually include pruritis and diffusely mottled erythematous patchy rashes as well as lividity or marbling or formation. Patches may have central cyanosis and blanch. It is often seen on the shoulders and upper thorax. Bubbles blocking the lymphatics may cause local pitting edema. Musculoskeletal symptoms are the most common feature of decompression sickness: the pain is often vague and diffuse, ranging from superficial to deep and is often near a synovial joint. It is usually asymmetrical, may shift in both character and in its temporal pattern. The upper extremities are the most commonly affected body parts—the shoulder is the common site, followed by the elbows and arms in turn by other body parts. The pain is often characterized as dull and difficult to localize. Redness, swelling, tenderness and increased pain with movement are not typical of DCS. There is no change with movement of a joint, but applying and inflating a blood pressure cuff over the affected area often reduces pain when due to bubbles, but not when due to old sports or traumatic injuries.

Any neurological, cardiopulmonary or vestibular findings place the disease into "Type II." Type II DCS may be considered a diffuse, multifocal central nervous system disease, recognized for over a century. This can include paraesthesia, hypesthesia, paresis, paraplegia, hemiplegia, urinary retention, impaired consciousness to life-threatening coma, ataxia, seizures, and death. Among the visual disturbances seen from decompression sickness are blurred vision, scotomata, visual field defects and blindness, which can be confused with cerebral arterial gas embolism. Chest pain...
and cough may also occur with intrathoracic intravascular bubbling ("the chokes"). Hemoconcentration is also seen in serious cases of DCS. It has recently been suggested that repeated subclinical DCS from cerebral bubbles may also lead to a condition analogous to multi-infarct dementia. Lacunae and white matter small arteriolar hyalinization has been shown to occur in divers with DCS. Adkisson scanned patients with confirmed cases of DCS I, DCS II and AGE: he found Type I patients to have normal scans whereas cerebral perfusion deficits were present in every patient with Type II or AGE.

Types I and II DCS may appear simultaneously as the disease is actually a spectrum of bubble-induced injury; there is an overlap between them, as it is a spectrum of pathology and not truly separate categories of disease. The traditional classification scheme of DCS I and DCS II appears to be incorrectly used frequently: 48% of divers classified as Type I reported neurological symptoms to DAN.

Spinal DCS cases are often due to bubbles in the myelin sheath causing vascular edema and focal ischemia with resulting neuropathy which may well resolve; other causes such as venous plexus bubbles and hemorrhage in contrast have a poor prognosis. Spinal cord involvement is the site of the most frequent neurological DCS involvement, and symptoms often include low back pain, abdominal pain, lower extremity weakness or paraesthesias; signs include paralysis and urinary retention. Paraplegia is more common than quadriplegia. Symptomatic involvement is the site of the most frequent neurological DCS involvement, and symptoms often include low back pain, abdominal pain, lower extremity weakness or paraesthesias; signs include paralysis and urinary retention. Paraplegia is more common than quadriplegia. Symptoms tend to progressively wax and wane and there may be residual symptoms even after treatment.

The "chokes" is an acute pulmonary hypertension secondary to massive venous bubbling in the pulmonary vasculature. It is uncommon but serious as respiratory failure and shock ensue and too often is fatal. This risk also exists in an airplane if the cabin were to suddenly decompress to 32,000 feet when it had been pressurized to 8,000 feet. Increasing age and obesity are contributing factors. It is usually reversible by prompt recompression.

Inner Ear DCS is also called vestibular decompression sickness (aka the "Staggers") and presents with vertigo, tinnitus, hearing loss, or nausea. If these symptoms occur upon descent then it actually due to inner ear barotrauma and the dive must be aborted; oval and round window ruptures must be included in the differential diagnosis. However, if the symptoms develop during or shortly after decompression from a dive, it must be considered to be inner ear DCS. It is more common with decompression from deep heliox dives but rarely can present from shallower recreational diving depths. Treatment includes recompression but the use of any medications with anticoagulatory effects such as aspirin should be avoided for fear of inner ear hemorrhage. Individuals who suffered permanent ear injury from this condition in the past have been advised that they cannot return to diving but recent evidence shows that in some cases after proper evaluation it may cautiously be permitted.

An anatomic predisposition to symptomatic decompression illness is patent foramen ovale (PFO). Approximately 20% of the population is estimated to have PFO while nearly 40% of DCS cases have PFO. The lungs filter most of the small nitrogen bubbles formed in the venous system during a dive, which limits tissue damage. However if blood passes from right atrium to left atrium bypassing lung filtration then bubbles may enter the arterial system where they can do great damage as emboli. This is not simply a theoretical exercise: in a study of diving clubs around Heidelberg it was discovered that multiple brain lesions were associated with a patent foramen ovale despite the absence of symptoms. These lesions are undoubtedly due to arterial gas emboli. Wilmshurst, the medical advisor to the British Sub-Aqua Club, has stated that enough studies have been published to eliminate any controversy that PFO has a significant role in decompression illness. This begs the question: should everyone have cardiac echocardiography prior to taking scuba classes and should the presence of PFO preclude diving?

It may be that dehydration contributes to DCS, although the exact role is unclear. Dehydration while diving may occur from breathing dry compressed air; perspiration; immersion diuresis; hypothermia; alcohol and caffeine; emesis from motion sickness or diarrhea from traveler's diarrhea.

The psychological profile of a diver may also contribute to the morbidity of DCS vis-a-vis delaying treatment: denial is the most common factor; others include anxiety, panic, embarrassment and depression which may retard or prevent a diver from presenting for therapy. DAN statistics reveal that 20% of DCS divers had symptoms prior to their last dive. DAN statistics also show divers have an average delay of approximately 32 hours before calling for assistance in cases of DCS. Fifty percent of injured divers with mild or pain-only DCS wait over 12 hours before seeking assistance; 20% wait greater than 96 hours. Rationalization is also a common denominator in postponing evaluation; other divers perversely view being "bent" as a measure of their experience or machismo. Many such divers refuse to seek medical care or if they do, refuse recompression. They may return to
diving prematurely which can lead to further decompression illness or permanent damage.

**Flying after Diving** Flying puts a diver at risk for decompression illness if the flight occurs right after diving.\(^5\) Most commercial aircraft keep the cabin pressure only between 5,000–8,000 feet, not at sea level, so the pressure gradient holding supersaturated nitrogen in tissues is lowered even more\(^6\) and so by Boyle's Law, gas bubbles expand. Various authorities such as the US Navy, the US Air Force and DAN have recommended intervals of 12–24 hours between one's last dive and flying.\(^6\) Studies at Duke University suggest that the probability of DCS drops asymptotically with time after diving, and suggests that a 24 hour guideline is conservative.\(^7\) While the number of volunteers in the study was not large, they have preliminary results that show that the probability of definite DCS drops from 8.3% with a preflight surface interval of 3 hours to 1% at 12 hours; over the same time interval, the probability of "ambiguous" DCS drops from 19% to 2%.\(^5\)

**Dive Computers** Dive computers have become commonplace diving equipment. Yet the jury is still out on whether they are more accurate or more reliable than the traditional dive tables.\(^7\) Dive computers, which are compact, are either worn or carried by the diver along during the dive. They constantly monitor time and depth, and calculate the diver's nitrogen exposure. They are meant to minimize DCS, prolong bottom times and enhance diving by allowing multilevel dives, in contrast to the standard dive tables which were devised for fixed time and depth dives, as with military missions.

Dive computers calculate the nitrogen saturation through algorithms. Using biophysical models incorporating gas laws and bubble formation to simulate uptake and release of N\(_2\), they predict the time and pressure schedule that would prevent DCS from occurring. Few have been tested and confirmed by human models—unlike the dive tables—so are thus based on theories. These algorithms are corporate technical secrets and not generally available to scientific scrutiny. They cannot take into account individual variations, current, water temperature, hydration status, etc. Validation of computer algorithms is usually done by extrapolating tests done on other models.\(^7\) Dive computers are not safer than the USN dive tables when the tables are properly followed.\(^7\)

Dive computers are simply tools; as such they can be misused and abused. Many divers use computers to extract every possible second out of their dives, ignoring the fact that developing DCS is not a black and white issue, with variability and unpredictability. Some divers exceed the depth limits of their computers, thus making their analyses invalid. Other divers try to artificially reset the nitrogen exposure; cases have been documented where divers dunked computer models in buckets of water or overboard from diving boats to trick the computer into resetting itself by "adding" decompression time to the device. Some divers turn the computers off in an attempt to clear the nitrogen from the units—a clear lack of reality cognition and a challenge to Darwinian evolution. In reality, computers can be affected by nitrogen, and may indeed be "bent" themselves. Another problem is the human nature attribute of having blind faith in data produced by a machine, leading to a false sense of security. Some divers turn their brains off when they turn their computers on, and rely completely on the electronic device. Some of those will ignore information the computer provides if they don't like what they see. Other divers rely on information that comes from computers worn by other divers, not themselves. A computer must initialize on the first dive; if it does not, then subsequent dive information is invalid, yet divers have persisted in continuing to dive under these circumstances. In an Australian survey 29% of divers using a computer reported experiencing a computer failure while diving,\(^7\) a possibility with any mechanical or electronic device that must be planned for.

When compared to the U.S. Navy dive tables, computers often omit decompression;\(^7\) if used in multilevel dives this may be acceptable as the computer calculates the reduction in nitrogen that occurs while swimming at less depth; however, studies have shown that instead, divers often used the computer to extend dive time at deeper depths, not the reason they were designed.\(^7\)

In the DAN 1996 Report on Diving Accidents and Fatalities, 55% of DCS cases were computer users; comparable information from the Royal Navy revealed similar numbers but only 16% of their subjects had kept their dives within the limits of their computers.\(^7\) This suggests that the computers may have offered the correct advice and that the divers simply ignored or violated it. Frighteningly, 8% of the accident cases recorded were by divers who used neither tables nor computer, throwing self-responsibility to the wind.\(^5\)

The American Academy of Underwater Sciences has the following recommendations for diving with computers:

Each diver relying on a dive computer to plan dives and indicate or determine decompression status must have his own unit; on any given dive, both divers in the buddy pair must follow the most conservative dive computer; if the computer fails at any time during the dive, the dive must be terminated and appropriate surfacing procedures should be initiated immediately; once a dive computer is in use, it must not be switched off until it indicates complete outgassing has occurred or 24 hours have elapsed, whichever
comes first; when ever practical, divers using a dive computer should make a stop between 10 and 30 feet for 5 minutes, especially for dives below 60 fsw; repetitive and multilevel diving procedures should start the dive, or series of dives, at the maximum planned depth, followed by subsequent dives of shallower exposures; dive computers should not be pushed to their limits; divers need to realize that they have to take responsibility for their actions.74,75,76

Evaluation and Treatment

Few physicians have training in the diagnosis and treatment of diving diseases; yet most cases of such diving-related injuries present to emergency departments or primary care practitioners initially.77 As delays in initiation of proper therapy correlates with worse outcome and the ease of modern long distance travel, it is important for physicians around the world to be familiar with the recognition and treatment of decompression sickness and arterial gas emboli.

A history must include the dive location, level of diving experience, dive profile, the rate of ascent, the time of onset of symptoms and variations in symptoms, and which type of gas was in the tank.1-4,14 A fundamental question is whether the injured diver was using scuba or simply snorkeling, and thus more likely a near-drowning victim.14 While most neurological abnormalities associated with diving are from DCS and AGE, do not forget to rule out head injury, carbon monoxide or carbon dioxide poisoning, or marine toxins. Cerebral bleeding such as subarachnoid or intracerebral hemorrhages should be included in the differential diagnosis and even internal artery dissection may occur from strenuous exercise.78 Other pertinent information includes respiratory disease history, heart conditions, CNS diseases, alcohol or drug abuse.79 Signs and symptoms of AGE are usually immediate or within 10 minutes of surfacing while decompression sickness may present with a delay of minutes to hours.1 If such information is unavailable from the diver, it can be obtained from the diving buddy or the diver's computer if such was used.

The physical examination should include vital signs and evidence of pneumothorax, pneumomediastinum (such as "Hamman's Crunch" or a brassy sounding voice), subcutaneous emphysema and otic barotrauma.31 The exam must also include a very thorough neurological examination including mental status, cranial nerves examination; sensory testing for pinprick, fine touch and proprioception; motor strength and symmetry; deep tendon reflexes; and coordination with cerebellar and spinal function testing. A proper cardiac exam should include looking for PFO which will likely require echocardiography to detect. Do not forget to perform direct ophthalmoscopy and otoscopy.

Laboratory tests are usually not needed as they do not help in diagnosis or guiding therapy. A near drowning may complicate or be the true cause of the condition, a chest x-ray and arterial blood gas should be obtained, but should not delay nor compromise recompression therapy.80 Radiographic tests may be of utility, but should not delay treatment.31 Up to 42% of pulmonary barotrauma cases may have radiographic evidence of ectopic air although subtle cases may be easily missed.80

Observe the fundamental ABCs—airway, breathing, and circulation. Transport patients to emergency departments or hyperbaric units at altitudes less than 300m/1,000 ft. which often requires aircraft with the capability to completely pressurize the interior. Patients should be laid in the left lateral decubitus position, not head down as taught in the past (to avoid increased intracranial pressure) and given 100% oxygen by face mask at high flow rates.14,31,48,81 Nasal cannulae are usually inadequate for this condition.17 For patients whose symptoms resolve while breathing oxygen only observation may be needed; for all others more aggressive therapy is advisable. Intravenous fluids to maintain urine output and a Foley catheter are advisable and as CNS injury is exacerbated by glucose, glucose-free isotonic fluids are preferred.53,8 Oral fluids can be given to fully alert patients whose airways are not in danger.4 If intubated, then the cuffs on both the endotracheal tube and the Foley catheter should be inflated, with water, which is incompressible, so as to avoid problems in the hyperbaric chamber.52 Glass IV bottles if used must be vented.14 Aspirin, lidocaine and the use of steroids are of questionable value in treating either DCS or AGE.19,46,48

The patient should be rapidly transferred to a hyperbaric unit for recompression therapy—delay being the main factor in not obtaining full recovery.31 As Boyle's law, recompression will reduce bubble size, resulting in a decrease or elimination of vascular obstruction and tissue distortion. Recompression may be with the US Navy dive tables using 100% oxygen or the Comex Tables which use heliox (50% oxygen/50% helium) in addition to oxygen and air.8,44 Recompression with oxygen is the primary treatment for AGE and decompression illness.8,43,52,80 J.S. Haldane at the turn of the century devised air decompression stages to avoid and treat decompression sickness, and his method is still the basis for decompression schedules today.84,85 The USN diving tables 1-4 are air recompression schedules and are rarely used; tables 5 and 6 use oxygen although table 5 is not commonly used.8,85 For AGE, recompression may be with the US Navy dive table 6A using 100% oxygen or the Comex Tables.83,84 The USN table 6A recompresses initially to 6 ATA—a "depth" of 165 fsw.8,31,85
After the initial bounce dive down to 165 fsw breathing 100% oxygen to compress any bubbles, the pressure is reduced to 60 fsw and the diver continues breathing oxygen except for "air breaks" to reduce the incidence of oxygen toxicity.8

Thorough neurological and cardiopulmonary exams are important both before and after hyperbaric recompression treatments for gas emboli. With prompt therapy, the majority of pulmonary overpressurization syndrome patients have full recovery. All doubtful or unclear cases should be presumptively treated.1 While hyperbaric therapy should be sought immediately, delayed recompression may still be beneficial.86

Table 5 is used to treat DCS I—a shortened version of table 6 (by 150 minutes) and is effective under naval conditions where a diving medical officer is present to do a proper neurological evaluation (ruling out any neurological DCS) at the time of occurrence. If DCS recurs, the diver is conveniently close-by the chamber. It is efficient with military conditions and needs—divers being under shorter treatments and thus available sooner for more missions/assignments. With sports scuba, there are time delay factors that change these considerations— including denial, the length of transport and delays in getting a chamber team in once called in. With the possibility of a neurological symptom being present at onset but not evident to the diver, discretion is the better part of valor and so the more intensive and definitive table 6 is used. This also reduces the risk of recurrence.82 DAN statistics reveal that from 1989-1994 73.3% of DCS cases were initially treated with a table 6.82 As DCS is often a vague presentation, a "trial of pressure" may be attempted in the hyperbaric chamber. Response to recompression can be diagnostic.82 It is an error not to treat doubtful cases, as omitting a treatment may result in irreversible damage while treating another condition is not likely to produce irreversible harm.

Table 5 may be used if there are pain-only bends, with a completely normal neurological exam, with an onset within 6 hours of reaching surface and resolution of symptoms at 60 fsw (19msw) within ten minutes of recompression therapy.8.48

Table 6 (see graph), the preferred treatment table, may range from 4 hours 45 minutes to 8 hours. Initially, one is "dived" (treated) to a depth of 60 fsw (18 msw or 2.8 ATA) for a specified time, then is brought up to 30 fsw (9 msw) for longer periods. The diver breathes 100% FiO2 throughout the dive except for "air breaks" to minimize the possibility of oxygen toxicity.31.52 The use of helium-oxygen mixtures for treating emboli under pressures greater than 2.8 ATA is becoming quite common around the world, and may become the standard of care at the beginning of the millennium: however, there is no evidence that they are superior to 100% oxygen.48 If symptoms in DCS Type I cases resolve within 10 minutes on O2, then modifying the course to treatment table 5 is permitted8 although it is quite common to complete the standard table 6 to avoid recurrence of symptoms during decompression or after the treatment is finished.

DCS cases may be retreated until symptoms resolve or their improvement plateaus and there is no further improvement.87

In water recompression is inadvisable, particularly when there are hyperbaric chambers nearby.31 Aside from the inability to administer proper medical care,
hypothermia is a serious complication when left underwater for prolonged periods of time—even in tropical waters. Still, in remote areas where careful supervision is present, it may be on rare occasion life-saving. With the development of portable hyperbaric units, it would be preferable to evacuate a diver in a portable unit to a proper hyperbaric chamber from a remote site.

With recompression barotrauma is possible, particularly to the ears, if attempted too rapidly. When breathing 100% oxygen under pressure, there is a risk of oxygen seizures which are none the less not a contraindication for continuing the treatment. The seizures stop after stopping the flow of oxygen and having the patient resume breathing compressed air for 15 minutes. Oxygen-induced seizures are not an indication of underlying epileptic disease.

The success of hyperbaric treatment for decompression illness is inversely related to the time from symptom onset to chamber treatment: the more delay between symptoms and treatment, the less resolution. Only 28.8% DCS cases seek medical care within 4 hours of symptom onset and 6% presenting over 96 hours after onset. Up to a 20% failure rate occurs in cases of delayed treatment. On occasion, symptoms may recur after recompression therapy. DCS can have significant long-term morbidity—even with treatment, some divers have persistent or recurrent symptoms. Cases of spinal cord DCS are particularly resistant to treatment. The later symptoms present, the better the outcome. The DAN Report on Diving Injuries revealed that on average, 16% of DCS cases will have symptoms for up to 3 months after treatment. Thirty-five percent of cases

Table 2: List of Primary Recompression Tables and their Applications

<table>
<thead>
<tr>
<th>Treatment Table</th>
<th>Type of Table</th>
<th>Application</th>
</tr>
</thead>
<tbody>
<tr>
<td>USN 5</td>
<td>Oxygen treatment of pain-only (type I) decompression sickness (DCS)</td>
<td>Treatment of pain-only DCS in cases where symptoms are relieved within 10 minutes at a pressure (depth) of 60 ft/18.3 msw</td>
</tr>
<tr>
<td>USN 6</td>
<td>Oxygen treatment of serious (type II) DCS</td>
<td>Treatment of serious DCS or of pain-only DCS in cases where symptoms are not relieved within 10 minutes at a pressure (depth) of 60 ft/18.3 msw</td>
</tr>
<tr>
<td>USN 6A</td>
<td>Air and oxygen treatment of arterial gas embolism (AGE)</td>
<td>Treatment of gas embolism. This table is to be used only in cases where it is not possible to determine whether the symptoms are caused by AGE or serious DCS</td>
</tr>
<tr>
<td>USN 7</td>
<td>Oxygen/air treatment of unresolved or worsening symptoms of DCS/AGE</td>
<td>This table is to be used only in cases that are life threatening and have not resolved after treatment on USN tables 4.6.5 or 6A.</td>
</tr>
<tr>
<td>USN 1A</td>
<td>Air treatment of pain-only type I DCS (100 ftsw/30 msw)</td>
<td>Treatment of type I DCS in cases where oxygen is unavailable and pain is relieved a pressure (depth) &lt; 66 ftsw/20 msw</td>
</tr>
<tr>
<td>USN 2A</td>
<td>Air treatment of pain-only type I DCS (165 ftsw/50 msw)</td>
<td>Treatment of type I DCS in cases where oxygen is unavailable and pain is relieved a pressure (depth) &gt; 66 ftsw/20 msw</td>
</tr>
<tr>
<td>USN 3</td>
<td>Air treatment of serious (type II) DCS or AGE</td>
<td>Treatment of type II DCS or AGE in cases where oxygen is unavailable and symptoms are relieved &lt;30 minutes at a pressure (depth) &gt; 165 ftsw/50 msw</td>
</tr>
<tr>
<td>USN 4</td>
<td>Air treatment of serious (type II) DCS or AGE</td>
<td>Treatment of type II DCS or AGE that have worsened during the first 20 minute oxygen breathing period at a pressure (depth) of 60 ftsw/18.3 msw on table 6, or for treatment in cases where symptoms are not relieved in &lt;30 minutes at a pressure (depth) &gt;165 ftsw/50 msw when table 3 is used</td>
</tr>
<tr>
<td>COMEX CX 30</td>
<td>Helium-oxygen or nitrogen-treatment of vestibular or neurological DCS</td>
<td>Treatment of vestibular or type II DCS that occurs after or a normal or a shortened decompression. To be used in cases where the patient shows deterioration at a pressure (depth) of 60 ftsw/16.3 msw on USN Table 6A but shows good improvement when brought to 100 ftsw/30 msw</td>
</tr>
<tr>
<td>COMEX CX 30A</td>
<td>Air treatment of type I DCS when oxygen poisoning has occurred</td>
<td>Treatment of type I DCS in cases where the stricken diver shows signs of oxygen toxicity. To be used in cases where the patient shows deterioration at 60 ftsw/18.3 msw on USN Table 6A but shows good improvement when brought to 100 ftsw/30 msw</td>
</tr>
<tr>
<td>ROYAL NAVY</td>
<td>Air treatment of DCS or AGE in cases where decompression depths greater than 165 ftsw/50 msw are needed and mixed gas is not available</td>
<td>Treatment of DCS or AGE to be used in cases where the patient remains in poor condition after 2 hours at 165 ftsw/30 msw and slow decompression is desired or in cases where a pressure (depth) &gt;165 ftsw/50 msw is needed and mixed gas is not available</td>
</tr>
</tbody>
</table>

(From National Oceanic and Atmospheric Administration Diving Manual, October, 1991.)
have been reported to remain symptomatic even 2 years after recompression treatment. Recurrent symptoms most often were reported when deprived of sleep, with long work hours, with alcohol or with prolonged immobilization and had psychiatric morbidity. Some residual symptoms remain life-long.

Despite the clear relationship between delay from onset to presentation for treatment, delayed cases deserve full attention. Kizer reviewed a series of delayed treatments between 12 and 168 hours and found 90% of such cases had either complete (66%) or substantial (24%) recovery. There are numerous cases which have had mild to significant improvement of DCS weeks to months later.

Follow-up hyperbaric oxygen treatments are used if residual signs and symptoms remain after the initial treatment: they are continued until no further improvements occur. Most individuals with persistent symptoms will not require more than 5-10 repeat treatments.

The traditional rate of a diver’s ascent in the water has been recommended at 60 feet/minute or no quicker than bubbles ascending from the regulator. Recent research has suggested that DCS can be reduced by halving the ascent rate to 30 feet/minute or even less.

How Recompression and Hyperbaric Oxygenation Work

Hyperbaric oxygenation is an intermittent, high dose but short term, oxygen inhalation therapy used to treat decompression sickness and air embolisms in divers. Hyperbaric or Recompression Chambers are artificially pressurized vessels which can accommodate one or more individuals and subject them to an increased pressure environment. This is usually accomplished by using external air compressors which pump this compressed air into the vessels. Breathing oxygen under these conditions creates a high oxygen gradient to perfuse ischemic tissues, increases inert gas elimination and decreases bubble size as well as decreases CNS edema. Under higher pressures nitrogen bubbles are smaller—at 2.8 atm bubble volume is reduced by nearly two-thirds, diffusion increases and equilibration is accelerated. The solubility of oxygen in 0.003 vol%/mmHg PaO2—with hyperbaric oxygen venous hemoglobin will be completely saturated and dissolved plasma oxygen will be enough to sustain life without any hemoglobin—at 2.8 atm bubble volume is reduced by nearly two-thirds, diffusion increases and equilibration is accelerated. The solubility of oxygen in 0.003 vol%/mmHg PaO2—with hyperbaric oxygen venous hemoglobin will be completely saturated and dissolved plasma oxygen will be enough to sustain life without any hemoglobin.
While the pressure induced mimics underwater pressure, the inhabitants remain dry during a "dive" and the outside of the chamber remains at ambient pressure. In engineering parlance, the proper term for such a unit is a PVHO—Pressure Vessel for Human Occupancy. Chambers are usually steel cylinders designed to withstand an internal pressure of at least 6 ATA (165 ftw).

When one breathes 100% FiO under pressure, the oxygen content of the blood and tissues rises, entering areas of low perfusion and injury; the pressure also compresses gas bubbles, allowing perfusion to resume and increases gas resorption.

Besides air, there are a multiplicity of different gas treatment tables including heliox and nitrox. Chambers may be of the monoplace or multiplace variety, the designations simply indicating the ability to operate with one or more occupants. The chamber environment is controlled from the outside by a chamber operator, and levels of oxygen, CO	extsubscript{2}, and humidity must be carefully monitored. In a monoplace chamber, the whole chamber is filled with compressed air and then the chamber is filled with O	extsubscript{2} which the occupant breathes; in a multiplace chamber the whole chamber is also filled with compressed air, but each occupant breathes pure oxygen through a mask or head tent. Monoplace chambers cannot accommodate all USN diving treatment tables, such as the air embolism treatment table 6A (165 feet).

In multiplace chambers, a tender accompanies the patient. Tenders, usually medical personnel, monitor patients during treatments and also manage any crises while recompression is underway. An example is that of a tender removing the oxygen hood in cases of O	extsubscript{2}-related seizures during recompression therapy. There are also portable hyperbaric devices, such as the Gamow Bag, which can be used at high altitude to temporarily treat Acute Mountain Sickness and for treating decompression sickness in remote locales. However, the use of such devices is not common, and usually restricted to expeditions and military operations.

There are special risks and considerations when operating inside a hyperbaric unit. Among the more significant ones are barotrauma (although the rate is controlled and adjusted to the occupants by the chamber operator); oxygen toxicity seizures; fires; and claustrophobia. Reversible myopia is the most common side effect of hyperbaric therapy.

Because of the risk of high concentrations of oxygen, the air in the chamber must be regularly vented and the humidity maintained at above 50%; inflammable materials, such as make-up, hair gels, etc are not allowed in the chamber and must be washed off prior to entry. Electrical systems need to be carefully insulated and grounded to prevent sparks from igniting the inner environment; fire suppression systems are extensive and often redundant within recompression chambers for these reasons. Needless to say, smoking prohibitions must be strictly enforced.

During the actual act of recompression, one is exposed to the same changes in barometric pressure as in underwater diving and equalization of the middle ear is the usual rate-limiting step to attaining treatment depth. In addition, as the air entering the chamber is compressed, the molecules are close together and the chamber heats up as a result. Once at the desired depth, which varies with the disease being treated, the temperature and barometric pressure stabilize. Usually at this point one begins breathing 100% FiO with intermittent "Air Breaks" to decrease the incidence of oxygen toxicity. The maximal pressure at which one can safely breath 100% O	extsubscript{2} for short periods is 3ATA or 66 ftw which happens to yield a PaO	extsubscript{2} of 2,000 mm Hg. Some treatments require multiple depths, and if so, the deepest depth is obtained first. Treatment protocols are guided by treatment...
ment tables" (US Navy Handbook in North America, COMEX in France or the Royal Navy Guidelines in the British Commonwealth). With ascents to less depth equivalents, or to the surface, the air in the chamber cools considerably, and may even form mist.

Some patients will require multiple treatment for their condition; this is determined by the type of disease and the clinical progression with recompression therapy. Residual treatment may be treated as per the USN diving manual.

Returning to Diving

In order to return to diving, there should be no increased risk of recurrence or of worsening tissue damage.

Otic Barotrauma

After middle ear barotrauma, once all signs of abnormality have resolved, the hearing is normal and the eustachian tubes can function, one may return to diving. However if one suffered inner ear damage, then specialized evaluation and treatment is recommended before returning to diving, but generally is not recommended.

Pulmonary Barotrauma

Previously anyone who had suffered an AGE was proscribed from ever diving again because of the possibility of an undetected local air trapping portion in the lungs. Now it may be that one can distinguish a deserved versus undeserved episode and if barotrauma was deserved because of violating physical laws, then once healed, one should be able to dive again without incidence. The US Navy dive manual recommends not diving for 4 weeks after arterial gas emboli with complete resolution when treated on a short oxygen table.* Complete resolution when treated on a short table is usually recommended.

Nitrogen Narcosis

Nitrogen under pressure acts as a narcotic. N₂ has a high affinity for lipid, as with gaseous anaesthetics. Its effect is related to depth and rapidity of descent and tolerance develops with repeated deep dives. Jacques Cousteau called it the "rapture of the deep." Martini's Law of diving equates the effect of nitrogen as being equal to a single martini for every 50 feet of depth. Symptoms increase with exercise, cold, alcohol and fear. It can be exacerbated, even accelerated by the ingestion of alcohol. As quickly as symptoms appear they disappear upon ascending.

Musculoskeletal Effects

Dysbaric Osteonecrosis is a necrosis of bones from nitrogen gas expanding in bone marrow adipose tissue that releases liquid fat and triggers local intravascular coagulation. It occurs when there is abundant fatty marrow, prolong exposure to compressed air and inadequate recompression. It never occurs in free divers. In sports scuba it usually occurs with high risk diving—with multiple exposures and symptomatic decompression sickness, but the relationship is not predictable. It has been seen in Miskito Indian lobster divers in Honduras who have extensive repetitive exposures.

It is not advisable for those recovering from a fracture to dive until the injury has completely healed. Not enough information exists regarding bubble formation in damaged bone tissue.

Dental

Temporomandibular joint disorder (TMJ) Temporomandibular joint disorder is often related to tooth alignment and bite, as well as trauma or arthritis. Scuba divers clench regulator mouthpieces which have poor ergonomic design, often requiring thrusting the mandible forward, straining and irritating the temporomandibular joint. Regulator mouthpieces can aggravate a pre-existing case of TMJ disorder or create a new one. This may result in headaches, neckaches, preauricular pain and tenderness. Complications include swelling from inflammation in the eustachian tube which can lead to otic barotrauma and rarely, arthroplasty. There are commercially available devices to keep the temporomandibular joint in proper alignment and reduce the incidence of TMJ disorder in divers but some may need specialized dentures.

Herpes Labialis A case has recently been reported in the literature of a scuba student who became infected with
CO and CO₂ Poisoning from Contaminated Tanks

Though rarer, carbon monoxide and carbon dioxide may rarely contaminate scuba tanks. Carbon monoxide may enter a tank if using a compressor that pulls in air contaminated with exhaust fumes or if hydrocarbon buildup inside a compressor system catches fire. Breathing carbon monoxide can easily be fatal as it binds hemoglobin over 200 times more avidly that oxygen and it also blocks the cytochrome oxidase system in cells. The resultant dizziness, altered mental status, or arrhythmias underwater could easily lead to drowning and death. The Compressed Gas Association allows a maximum of 10 ppm CO and 500 ppm CO₂ while OSHA permits 20 ppm of CO.¹¹⁴

Hypothermia

To be addressed in a future article.

Other Pulmonary Conditions

Cold-induced pulmonary edema has been recognized for over a decade,¹¹⁵ however there are recent reports that such a syndrome is possible in tropical waters.¹¹⁶ This syndrome is quite rare.¹¹⁷,¹¹⁸ Dyspnea can develop at depth; other signs and symptoms include cough, weakness, chest discomfort, wheezing, hemoptysis and dizziness. Mild cases spontaneously resolve and most significant ones lead to hypotension but appears to be independent of cardiovascular disease history.¹¹⁶,¹¹⁹

There have also been suggestions that the silicon used to lubricate scuba regulators may also induce pathology, specifically that of pulmonary fibrosis.¹²⁰ It has also been shown that hyperpnea with dry air causes mucosal injury, inflammation and microvascular leakage in the lungs which may persist for 24 hours or longer.¹²¹

Psychological

The psychological profile of a diver is intimately related to the number of injuries and fatalities in sport scuba. Panic is the primary culprit in diving emergency complications, and divers with increased anxiety traits are at higher risk for morbidity and mortality from scuba diving.²

Commercialization of scuba as a sport has led to an unrealistic appraisal of the dangers present and instructors may gloss over the risks of diving.¹²² This leads to inadequate screening of potential diving candidates, casual weekend resort certification courses, and subsequent increased cost of medical care.

In approximately 40% of drowning deaths the equipment is functional with air remaining in the tank or no signs of trauma or medical problem found.¹ The most likely explanation is that of panic. There are numerous documented cases of divers removing their regulator or making uncontrolled ascents to the surface in cases of panic. Between 19-41% cases of diving fatalities may involve panic.¹²³ Yet despite the obvious significance of panic and anxiety states in the underwater environment, very little mention is made of it in most training manuals.²

Psychological factors are well-known to induce physiological changes. Anxiety may be induced by internal factors such as DCS, hyperventilation; external factors can include difficult swimming conditions, entanglement in nets or kelp, visual loss (as in a cave or in a wreck), shark encounters, equipment malfunction, other divers panicking underwater or even from simply wearing a wet suit.¹²⁴ In addition, anxiety, neurosis, depression and extroversion have all been shown to be associated with ventilatory abnormalities.² Hyperventilation and hypocapnia theoretically may lead to unconsciousness underwater, a lethal situation, but some studies have shown that divers with higher anxiety trait actually breathed less than low anxiety divers.¹²⁵ This has the potential for hypopcapnia, which in the end may very well yield the same result.

Denial is a common problem in cases of decompression sickness as discussed earlier.

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