**Chapter 52  
Diving injury**

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**Historical perspective**

Water comprises 70% of the surface of our planet. It is only natural that given this large percentage of our home, human beings would be drawn to explore this environment. It is currently estimated that there are 1.2 million active scuba divers worldwide [1] and that approximately 200,000 new divers are certified every year [2]. Although diving is considered to be a relatively safe sport, operating in an environment with unique hazards where life-supportive breathing gases must be carried leaves little margin for error. Comparing the total hours involved, diving is estimated to be 96 times more dangerous than operating a motor vehicle [3].

**Introduction**

Emergency medical services physicians and medical directors of EMS systems need not be certified scuba divers, but will benefit from developing a fundamental knowledge of dive-related physiology and hazards. There are four main categories of diving injury: injuries on the surface, injuries of descent, injuries at depth, and injuries of ascent. This chapter deals with injuries below the surface.

Due to the high density of water, small changes in depth cause significant changes in the pressure exerted on an object. At the surface, a body is subjected to the weight of the earth’s atmosphere, which is equal to 1 atmosphere absolute (ATA). During descent, for every 33 feet of seawater (fsw) or 34 feet of freshwater (ffw) traveled below the surface, pressure increases by 1 atmosphere (atm). Typical units of measure for pressure include: 33 fsw = 34 ffw = 1 atm = 760 mmHg = 760 torr = 14.7 psi. The majority of recreational diving occurs between 33 and 120 fsw (2 to <5 ATA).

**The gas laws**

To appreciate the physiology of diving injury, EMS physicians must be familiar with Boyle’s law, Dalton's law, and Henry's law.

**Boyle's law**

Governing the physiology of barotrauma and recompression therapy, Boyle’s law states that given a constant temperature, the volume and pressure of an ideal gas are inversely related. It also deals with conditions related to changes in pressure in hollow, air-filled organs and structures in the body.

As an example, during descent the pressure is doubled; on a descent from the surface (1 ATA) to a depth of 33 fsw (2 ATA), the volume of a gas is halved ([Figure 52.1](https://jigsaw.vitalsource.com/books/9781118990827/epub/OPS/c52.xhtml#c52-fig-0001)). The law is typically stated as:

[**Figure 52.1**](https://jigsaw.vitalsource.com/books/9781118990827/epub/OPS/c52.xhtml#R_c52-fig-0001) Boyle’s law.

As a diver is descending in the water column, the volume of air in gas-filled organs will decrease. If the volume of air in the lungs at the surface is V then at 33 fsw the volume will be 1/2 V, at 66 fsw 1/3 V, etc.

If using compressed air, as in scuba diving, when a breath is taken at 66 fsw, lung volume returns to V. If ascent occurs at this point without exhalation, as in an unconscious diver, the lung volume will expand to 1.5 V at 33 fsw and 3 V at the surface, with potential for barotrauma.

Liquids and liquid-filled organs are non-compressible. The body tissues are composed primarily of water and thus there is no change in volume with pressure increases and decreases.

**Dalton's law**

Dalton’s law explains the physiology of conditions such as oxygen toxicity and nitrogen narcosis. The law states that total pressure exerted by a mixture of gases is the sum of the partial pressures of the gases in the mix. Thus for fresh air:

As total pressure is increased, the partial pressures of each gas in the mixture will increase proportionally. Fresh air is composed of 79% nitrogen and 21% oxygen. These ratios remain constant as pressure is increased at depth.

The partial pressure of nitrogen in air at sea level is approximately 600 mmHg or 0.79 ATA (0.79 × 760 mmHg), and of oxygen is 160 mmHg or 0.21 ATA (0.21 × 760 mmHg). At a depth of 66 fsw, the partial pressure of each would be 3 × 600 = 1800 mmHg (2.37 ATA) for nitrogen and 3 × 160 = 480 mmHg (0.63 ATA) for oxygen.

**Henry's law**

Henry’s law is the foundation for decompression sickness (”the bends”). The law states that at equilibrium, the concentration of a gas dissolved *in* a liquid is directly proportional to the partial pressure of the gas *above* the liquid ([Figure 52.2](https://jigsaw.vitalsource.com/books/9781118990827/epub/OPS/c52.xhtml#c52-fig-0002)). This is stated as:

[**Figure 52.2**](https://jigsaw.vitalsource.com/books/9781118990827/epub/OPS/c52.xhtml#R_c52-fig-0002) Henry’s law.

where P is the partial pressure of the gas above the liquid, k is a constant, and C is the concentration of the gas in the liquid. The common example is opening a carbonated beverage container. As the pressure is reduced by opening the can, the CO2 dissolved in solution escapes, forming bubbles as the gas equalizes with the atmospheric partial pressure of the gas.

**Injury of descent: barotrauma of descent**

Barotrauma is an injury that occurs due to changes in pressure of an air-filled structure during descent or ascent. Barotrauma is the most common medical problem associated with diving and can involve almost any structure that can have entrapment of gases. Barotrauma causes injury by a change in volume of free gas in an air-filled organ resulting in a pressure disequilibrium. Both increasing and decreasing pressure can cause mechanical injury to body structures. Pain is typically the initial complaint.

**Middle ear barotrauma**

Middle ear barotrauma, also known as barotitis media or “ear squeeze,” is the most common complaint and medical problem of scuba divers. It is experienced by 30–40% or novice scuba divers and 10% of experienced divers [4]. As a diver descends in the water column, water pressure against the tympanic membrane (TM) increases. A diver will employ various methods to force air into the middle ear through the eustachian tube to equalize this pressure across the TM. If the diver is unsuccessful at “clearing” his ears, continued attempts may be futile due to the collapsible nature of the medial third of the eustachian tube. Ascent and reattempts at clearing are the only option for resolution. Further descent may cause TM rupture and result in cold water caloric stimulation and vertigo which may precipitate panic, disorientation, rapid ascent with other associated types of barotraumas, or drowning.

Treatment of middle ear barotrauma is usually with decongestants and analgesics and it will typically resolve over 3–7 days [5]. Refraining from diving with a cold or other symptoms that may cause difficulty with pressure equalization and early recognition of symptoms are common prevention strategies.

**Inner ear barotrauma**

Inner ear barotrauma is much less common than middle ear barotrauma, but has a higher morbidity. Damage to the cochleovestibular apparatus is the result of a large negative pressure gradient in the middle ear that occurs due to a forceful Valsalva maneuver against an occluded eustachian tube. As a result of the increased pressure during the attempted Valsalva, the pressure differential between the cerebrospinal fluid through the vestibular and cochlear structures and the middle ear may result in several injuries, including round or oval window rupture, middle ear hemorrhage, Reissner’s membrane tear, fistualization of the windows, or a combination of these.

A triad of findings are associated with inner ear barotrauma: vertigo, unilateral roaring tinnitus, and hearing loss. In addition, a feeling of ocular fullness, nystagmus, disorientation, ataxia, and nausea and vomiting may be seen. Immediate concerns relate to the occurrence of panic in the underwater environment, uncontrolled ascent, or drowning.

Treatment of inner ear barotrauma includes elevating the head of the bed to 30°, bed rest, avoidance of strenuous physical activity, and symptomatic treatment. Early otolaryngology consultation should be obtained for further treatment recommendations as surgical repair options remain relatively controversial [6].

**External ear barotrauma**

A less common condition than middle ear barotrauma, external squeeze may occur with a tight-fitting wetsuit hood creating a relative negative pressure in the external canal. The TM is pulled outward due to trapped air in the canal. Cerumen, earplugs, and structural abnormalities may also contribute to this condition.

**Sinus barotrauma**

The sinus cavities are susceptible to pressure-volume changes according to Boyle’s law. The ethmoid, maxillary, and frontal sinuses require patent nasal passages for equalization of pressure. Any mechanical abnormalities such as deviated septum, polyps, or physical conditions such as bacterial and viral infection, or upper respiratory infections may predispose a diver to sinus barotrauma and resultant barosinusitis.

The frontal sinus is the most commonly affected sinus cavity due to the long connection to the nasal passage [4]. Common signs and symptoms in barosinusitis include facial pain or fullness during descent or ascent, numbness to the front of the face, upper tooth pain, and epistaxis. Systemic decongestants and topical nasal vasoconstrictors are the mainstay of treatment. Some authors advocate a short corticosteroid burst to hasten recovery and return to diving [5].

**Mask squeeze**

A face mask must seal tightly around the face and forehead, and under the nose. Diving masks enclose the nose to allow nasal exhalation when equalizing the pressure between the mask and the outside environment. Equalization failure may result in capillary rupture with facial petechiae, ecchymosis, and scleral/conjunctival hemorrhage.

**Suit barotrauma**

Suit squeeze develops where folds in wet or dry suit material become compressed during descent, causing a partial vacuum and resulting in an impressive area of ecchymosis post dive. Despite the dramatic appearance, the condition is benign and will resolve in days to weeks.

**Dental barotrauma**

Tooth squeeze or barodontalgia is an infrequent but dramatic type of barotrauma. Air trapped below decayed teeth or in other dental structures may cause pain during ascent or descent as air bubbles expand, causing a negative or positive pressure related to ambient pressure. This condition is mostly benign and self-limited, although may be quite painful.

**Injury at depth**

**Nitrogen narcosis**

Nitrogen is an inert gas and does not interact biochemically in the body. Nitrogen narcosis, also known as inert gas narcosis or “rapture of the deep,” develops typically at depths greater than 100–120 fsw. Nitrogen begins to have anesthetic properties at 3.2 ATA. The symptoms result from the intoxicating effects of increased nitrogen tissue concentrations. Divers may become euphoric, have a false sense of well-being, inappropriate laughter, and develop numbness and tingling in the face, lips, or legs. Decreased decision making and judgment combined with loss of fine motor skills and delayed reaction times may result in drowning or contribute to a dive emergency. As in most physiological states, the effects are variable and there are no absolutes as to who is likely to develop this condition and at what depth. The diving “martini rule” describes that every 1 ATA (33 fsw) descended greater than 100 fsw equates to the consumption of one martini.

The condition improves rapidly with ascent, assuming that it is recognized in time. Divers may be unaware that they were affected by the condition. Cold temperature, workload, alcohol, hangovers, and fatigue may contribute to the onset and severity. It is generally recommended that recreational divers not dive with compressed air to depths greater than 120 fsw. Commercial divers use other inert gases, such as helium, neon, and argon, in their compressed gas mix to offset the effects of narcosis. Oxygen itself may become narcotic if left unmetabolized in tissues [7].

**Oxygen toxicity**

Despite the necessity for oxygen to sustain life, increased pressures and lengthy durations of exposure can be damaging to living organisms. The damaging effects are a result of increased partial pressures of oxygen and not necessarily the inspired oxygen percentage in the gas mixture. Although all organs may suffer oxygen’s toxic effects, brain, lung, and eye function are often the first to be disrupted. Oxygen free radicals are believed to be responsible for the deleterious effects of high partial pressures of oxygen. Intermediates such as superoxide anions, hydroxyl radicals, and hydrogen peroxide are potentially toxic to cell membranes [8].

Two types of oxygen toxicity relevant to diving are central nervous system (CNS) and pulmonary (“whole body”). CNS toxicity has a more rapid onset even after short exposures (A). Whole-body toxicities usually follow prolonged exposures to oxygen at lower partial pressures (B).

1. Oxygen is generally considered to become toxic to the CNS when the partial pressure exceeds 1.6 ATA. Partial pressures less than 1.4 are unlikely to produce toxicity. These partial pressures are highly variable among individuals and make planning for dives riskier as high pressures are used for aggressive dive profiles.

The toxicity experienced may range from visual changes to the extreme of convulsive activity. The convulsion experienced is not damaging in itself, but obviously in an aquatic environment can lead to life-threatening sequelae. The mnemonic VENTIDC [4] may be used to recall the range of toxicity.

**V:** Visual changes (tunnel or blurry vision)

**E:** Ear ringing/tinnitus

**N:** Nausea

**T:** Tingling, twitching, or muscle spasms (usually facial muscles or lips)

**I:** Irritability, anxiety, agitation, confusion

**D:** Dyspnea, dizziness, fatigue, problems with coordination

**C:** Convulsions

Any diver experiencing any of these symptoms should ascend from depth at the earliest opportunity to prevent the potential catastrophic consequences of unconsciousness or convulsions.

1. In pulmonary or “whole-body” toxicities, the lung is the primary organ affected, but many other parts of the body can be affected as well. The term whole-body toxicity is used to include any organ systems other than the CNS. Pulmonary irritation due to prolonged exposure of oxygen at lower partial pressures is an example of whole-body toxicity. Other symptoms that may be experienced in the whole-body category are itching, skin numbness, nausea, dizziness, and headache.

### Immersion pulmonary edema

Cases of immersion pulmonary edema (IPE) have become widely recognized since 1989 [9]. IPE, typically occurring in divers with no underlying medical diseases, presents as a rapid onset of dyspnea at the bottom and continued dyspnea on the surface associated with cough and blood-tinged, frothy sputum. Because the fluid builds up in the air-containing spaces of the lungs and interrupts gas exchange, IPE resembles drowning. The important difference is that the obstructing fluid comes from within the body rather than from inhalation of surrounding water.

The cause of IPE has yet to be determined. Aggressive hydration prior to diving may be a contributing factor. It has been seen in triathletes and Navy SEALs doing high-intensity surface swims. Divers get IPE when swimming on the bottom without clear evidence of stress. In some cases, the diver mentions a tight-breathing regulator, and in others no evident stress or equipment problems are noted.

Immersion pulmonary edema is not a manifestation of decompression sickness and does not require recompression. The treatment is oxygen and diuretics to remove water from the lungs.

## Injury of ascent: barotrauma of ascent

During ascent, the volume of all gases in air-containing structures will increase as the pressure decreases (Boyle’s law). This type of barotrauma is the result of expansion of gases as pressure is decreased during ascent.

### Reverse sinus or ear barotrauma (reverse squeeze)

Reverse sinus or ear barotrauma is less common than its counterparts during descent. The process of equalization on ascending is normally easier. As the ambient pressure is reduced, the pressure in the middle ear passively diffuses through the eustachian tube or through the sinus cavities. Initial descent problems may lead to inflammation and subsequent swelling of the passages, disrupting the ability to easily equate these pressures in these structures. Complications can range from blood from the ear or nose to tympanic membrane rupture, sinus fracture, or pneumocephalus.

### Alternobaric vertigo

Alternobaric vertigo may occur during ascent as the middle ear pressures become unequal. Onset is sudden and usually preceded by a full feeling in the ears. The difference in pressures causing asymmetric stimulation of the vestibular system may result in significant vertigo. Nausea and vomiting may accompany the vertigo. Typically a self-limited condition, the real danger of alternobaric vertigo is a possibility of diver panic with rapid ascent and resultant pulmonary barotrauma or arterial gas embolism. Near drowning may also occur. Descent of a few feet should result in improvement of symptoms.

### Gastrointestinal (GI) barotrauma

Aerogastralgia is a rare type of ascent barotrauma in divers, usually novice divers. Expansion of bowel gas during ascent may cause GI discomfort and abdominal pain. Causes include eating large meals or gas-producing foods (legumes) prior to a dive or drinking carbonated beverages. Swallowing air or Valsalva maneuver with the head down may also contribute to the condition. The standard cure is evacuation of gas through the two anatomical venting orifices with the resultant decrease in pressure.

### Pulmonary barotrauma

Pulmonary barotrauma (PBT) is the most serious type of barotrauma. Overexpansion of gas trapped in the lungs can result in pulmonary overpressurization. Sudden rapid and uncontrolled ascent in sport divers breathing compressed air at depth is the most common cause for PBT. Situations involving panic such as breath-holding out-of-air scenarios, buoyancy compensator malfunctions, loss of regulator, or accidental loss of weight belt can all contribute to these events.

During ascent without exhaling, the volume of gas in the lungs will double from a depth of 33 fsw to the surface. The greatest risk for pulmonary barotrauma occurs in less than 10 feet of water [4]. A pressure differential of 80 mmHg (alveolar air) above ambient water pressure on the chest wall, equivalent to 3–4 feet of seawater depth, is all that is necessary to force air bubbles across the alveolar–capillary membrane [10]. Conditions that may result from this physiology are alveolar hemorrhage, pneumothorax, pneumomediastinum, subcutaneous emphysema, and the most feared complication – arterial gas embolism (AGE).

#### Arterial gas embolism

The most striking and dramatic condition associated with PBT is AGE. The second most common cause of mortality, among sport divers, following drowning, AGE accounts for roughly 30% of diving-related deaths [4]. Victims of AGE manifest symptoms during ascent or within 10 minutes of reaching the surface.

Patients suffering AGE can be classified into three groups. The first group is composed of those patients who suffer immediate loss of consciousness, apnea, and cardiac arrest on reaching the surface. This group represents 4% of AGE patients and in these cases, recompression, cardiopulmonary resuscitation, and advanced life support are unlikely to be successful [10]. These patients are suspected to have suffered a large bolus of air to the central vascular bed, particularly the pulmonary arteries and right ventricle. The resulting vascular obstruction leads to pulseless electrical activity and death.

A second group of patients accounts for an additional 5% of deaths; these patients reach the hospital and will die as a result of the AGE or severe near-drowning accompanying AGE. Fifty percent of the remaining patients will have a complete functional recovery [10].

Victims suffering from AGE present with a variety of systemic and neurological findings. The severity and location of AGE depend on the amount and distribution of the air embolus. The most common initial findings are neurological in nature and include loss of consciousness, confusion, or stupor. AGE may involve multiple organ systems and the presentation is variable ([Table 52.1](https://jigsaw.vitalsource.com/books/9781118990827/epub/OPS/c52.xhtml#c52-tbl-0001)).

[**Table 52.1**](https://jigsaw.vitalsource.com/books/9781118990827/epub/OPS/c52.xhtml#R_c52-tbl-0001) Presenting signs and symptoms of patients with arterial gas embolism

| **Neurological** | **Pulmonary** | **Visual** | **Other** |
| --- | --- | --- | --- |
| Loss of consciousness Focal paralysis Confusion Coma Convulsions Vertigo Ataxia Unilateral motor/sensory deficit Bilateral motor/sensory deficit Dizziness Headache Memory difficulty | Chest pain Hemoptysis Crepitance Dyspnea | Blindness Nystagmus Gaze preference | Nausea Vomiting Cardiac arrest |

### Decompression sickness: “the bends”

Decompression sickness (DCS) occurs after a reduction in ambient pressure usually due to decompression back to ambient pressure from either a dive or hyperbaric chamber exposure. The pathophysiology of DCS results from the inflammatory and obstructive effects of inert gas bubbles in the vascular system and tissues. DCS represents a spectrum of clinical illnesses previously classified as DCS types I, II, III and now more commonly referred to by the affected organ system ([Table 52.2](https://jigsaw.vitalsource.com/books/9781118990827/epub/OPS/c52.xhtml#c52-tbl-0002)). The incidence of DCS is 2.8 cases per 10,000 dives [5].

[**Table 52.2**](https://jigsaw.vitalsource.com/books/9781118990827/epub/OPS/c52.xhtml#R_c52-tbl-0002) Organ systems affected by decompression sickness (DCS)

| **DCS type** | **Location** | **Symptoms** | **Other names/cautions** | **Incidence** |
| --- | --- | --- | --- | --- |
| Musculoskeletal – “the bends” | Major joints | Pain – boring, deep ache, may be sharp or throbbing | Pain only bends, joint or limb bends | Most common 70% of patients |
| Skin or cutaneous | Skin | Rash, pruritus, formication | Cutis marmorata, mottling or marbling of skin may indicate more severe decompression sickness | Relatively uncommon, usually benign |
| Pulmonary or “the chokes” | Substernal on inhalation | Non-productive cough, dyspnea, cyanosis | Represents massive pulmonary gas embolism | Unusual but serious |
| Neurological | Any level of central nervous system; typically spinal cord (low thoracic/lumbar) | Random and may be diffuse: paresis, paraplegia, paresthesia, dysesthesia, bowel or bladder dysfunction | Decompression sickness of the brain produces symptoms that may be indistinguishable from arterial gas embolism | Found in 50–60% of scuba diving casualties [5] |
| Inner ear or vestibular, “the staggers” | Inner ear | Vertigo, dizziness, nausea, vomiting, nystagmus | High incidence of residual inner ear damage as opposed to inner ear barotrauma | Usually seen in saturation divers or very deep heliox dives |
| Vasomotor | Vascular system | Hypotension unresponsive to fluids | Decompression shock | Rare, rapidly life threatening, most do not survive |
| Dysbaric osteonecrosis | Major joints | Avascular or aseptic necrosis of bone | Long-term sequela to inadequate decompression | <1% to more than 80% based on age and type of diving performed in lifetime |
| Dysbaric retinopathy | Eye | Variable | Suspected to be related to small bubble microembolization | Uncommon |

Risk of DCS is increased by the length and depth of a dive, and DCS may result despite strict adherence to appropriate dive tables. Contributing factors include age, obesity, dehydration, exercise prior to diving, fever, cold ambient temperatures post dive, exertion, and flying after diving. Men are 2.6 times more likely to experience DCS than women, perhaps due to variable risk-taking behaviors. A patent foramen ovale may also increase risk of DCS, having been found in 65% of divers with serious DCS [5].

The clinical diagnosis of DCS is suspected based on a history of exposure to increased atmospheric pressure and development of characteristic signs and symptoms. Most patients are symptomatic within 1 hour of reaching the surface. The remainder of patients will develop symptoms within 3 hours. Cases have been reported days following a dive although this amounts to less than 2% of cases [5].

### Shallow water blackout

Shallow water blackout is a loss of consciousness caused by cerebral hypoxia towards the end of a breath-hold dive, when the alveolar PCO2 is lowered to 20–30 mmHg without a significant increase in PO2. During the dive or swim, exercise-induced hypoxia sufficient to cause loss of consciousness may occur before CO2 reaccumulates to provide stimulation to breathe. Victims are often established practitioners of breath-hold diving (sport free divers), are fit, strong swimmers, and have not experienced problems before.

## Transport and destination hospital considerations

Patients suspected of suffering from AGE or DCS should be transported as rapidly as possible to a facility with resources for evaluation by a diving physician and possible hyperbaric oxygen therapy. Early treatment is more efficacious than delayed care, but there are numerous cases reported to have benefited even with delays of greater than 6 hours [10]. Recompression is the essential and primary treatment for these disorders.

Prehospital care should consist of supplemental oxygen at a flow rate of at least 10 L/min by non-rebreather mask, or appropriate airway management in patients suffering near drowning. Maintenance of intravascular volume is also important to support capillary perfusion and assist with elimination of bubbles from the arteriolar-capillary level. Intravenous isotonic fluids should be administered to maintain urine output of 1–2 cc/kg/hour.

Historically, it was recommended to position a patient in Trendelenburg position for transport. Current recommendations are to position AGE and DCS patients throughout their evaluation and treatment in a manner that allows the greatest access to and care of the patient [5,10].

The Divers Alert Network (DAN: 919.684.9111) is a 24 hour a day, 7 day a week international resource for dive-related injury management and referral. On-call diving physicians, paramedics, and emergency medical technicians are available to provide medical information, referrals, and evacuation assistance as needed.

## Conclusion

In general, diving is considered to be a relatively safe sport. Most diving operations will be free from major medical problems provided that divers pay attention to some general rules and have been properly trained.

Barotrauma of the ears and sinuses is the most common dive injury experienced. Pulmonary barotrauma is an infrequent complication but should be suspected when neurological or pulmonary symptoms are present.

The two main dive conditions that may benefit from recompression treatment and hyperbaric oxygen therapy are arterial gas embolism and decompression sickness. Decompression sickness occurs in deep long dives and in about 1% of divers. Arterial gas embolism occurs rapidly upon resurfacing. Rapid diagnosis and appropriate referral to definitive care may prevent additional decline in condition, further injury, and long-term sequelae of dive-related injuries.

Emergency medical services physicians and medical directors should have a fundamental knowledge of dive-related physiology and hazards. An understanding of the basic conditions encountered during a dive, combined with the knowledge of the dive phase at which an injury occurred and chief complaint, will help to diagnose and treat an injured diver.

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