**Dysbarisms, Dive Injuries, and Decompression Illness**

Heather Murphy-Lavoie and Tracy Leigh LeGros

engaging in breath-hold diving, a sharp increase in diving injuries has been seen in EDs (Box 133.2). Deaths from breath-hold diving alone have almost doubled in the last 5 years, thus illustrating the potential for injury associated with this form of diving. With acute DCI, rapid assessment and treatment are the foundation of management. Three keys to successful ED treatment are having a high index of suspicion (DCI may have nonspecific findings), performing a thorough neurologic examination, and obtaining hyperbaric medicine consultation when DCI is suspected.

**PATHOPHYSIOLOGY**

Dysbarism refers to the effects of variations in ambient (surrounding) pressure on the body. Hypobaric (low-pressure) exposure, such as that experienced by climbers, pilots, and astronauts, can result in symptoms and injuries similar to those found in divers with DCI who are exposed to high pressure while at depth. Decompression injuries during high-pressure (hyperbaric) exposure are far more common.

**DIVING PHYSIOLOGY**

Evaluation of a diver with a water-associated injury requires a basic understanding of diving physiology and the physics of pressure and gases. Different gases have different properties at different depths, which allows gases to be used alone or in combination for different types of diving. The gases of most interest are air, oxygen, nitrogen, helium, and occasionally argon. Deep diving (past 180 feet of sea water [fsw]) often requires helium-oxygen combinations (heliox) to mitigate the effects of nitrogen narcosis (discussed later). Moreover, enriched nitrogen-oxygen combinations (nitrox) may be used to reduce obligations for decompression stops, which is the time spent at more shallow depths to help divers offload the nitrogen built up in the body before exiting a dive.

In general, most recreational divers breathe compressed air and use a self-contained underwater breathing apparatus (SCUBA) when diving to depths of less than 135 fsw. Nitrogen represents about 78% of the gas inhaled with compressed air diving. During diving, hydrostatic pressure “pushes” nitrogen into tissues; nitrogen (an inert gas) then becomes dissolved in plasma and permeates tissues. While at depth, gases remain in solution, and most divers experience minimal difficulty. The deeper that divers travel and the longer that they remain at depth, the more saturated the blood and tissues...
Bubbles have damaging effects when they remain within the body tissues, such as the joint spaces, tendon sheaths, periarticular sheaths, and peripheral nerves. Once inside these areas, bubbles can act as emboli and block perfusion of distal tissues or act as foreign bodies with resultant vascular damage through activation of the inflammatory and clotting cascades. Interestingly, scientists are now evaluating a possible biologic marker of DCI. As gas emboli within the circulation induce decompression stress, endothelial cells release microparticles in response to cellular activation or cell death. These microparticles may, in the future, reflect a biologic marker of decompression stress that can be used to gauge the extent of disease, efficacy of treatment, or prophylaxis.

PRINCIPLES OF GAS LAWS AND DYSBARISM

An understanding of the pertinent diving gas laws, units of measurement, abbreviations, and mathematic conversions helps facilitate the treatment and disposition of dive-injured patients. At sea level, the pressure of the atmosphere on the body (ambient pressure) is 760 mm Hg, which equals 1 atm. The term for the absolute pressure on a diver at sea level is called atmospheres absolute (ATA), and it represents the total sum of the pressure on a diver. Therefore, at sea level, a dive computer gauge reads zero, but sea level also represents one surrounding atmosphere of pressure (1 ATA). This knowledge helps the physician better comprehend the circumstances surrounding a dive injury. Although there are a large number of gas laws, the two that are the most important in diving medicine are Boyle’s law and Dalton’s law.

**Boyle’s Law**

Boyle’s law aids in understanding why a diver needs to exhale while ascending from depth. According to Boyle’s law, the volume of a quantity of gas (V) varies inversely with the pressure on that gas (P) if it is kept at a constant temperature. It is often represented by the following formula:

\[
P_1V_1 = P_2V_2
\]

where the subscripts 1 and 2 indicate two different combinations of pressure and volume; in other words, the product of pressure and volume will always remain a constant.

Usually, as a diver descends deeper, the surrounding pressure increases in linear fashion. Water pressure at the surface is referenced as 1 ATA. In general, for every 33 ft of water, the pressure increases by half the original amount of gas at the surface. Therefore, a descent to 66 ft would equal 2 ATA, a descent to 99 ft would equal 3 ATA, and so on. Furthermore, for the first 33 ft descended, the volume of gas present is reduced by half the original amount of gas at the surface. At 66 ft, the volume is one third the original volume. The greatest changes in volume occur closest to the water surface and represent a significant vulnerability to injury (Fig. 133.1).

**Dalton’s Law**

Dalton’s law of partial pressures is critical to understanding the mechanisms for DCS, nitrogen narcosis, and oxygen toxicity. It is represented by the formula

\[
P_{\text{total}} = P_1 + P_2 + P_3
\]
DYSBARISMS, DIVE INJURIES, AND DECOMPRESSION ILLNESS

1155

PRESENTING SIGNS AND SYMPTOMS

BAROTRAUMA

Barotrauma is sustained from failure to equalize the pressure of an air-containing space with that of the surrounding environment. The most common examples of barotrauma occur during air travel and scuba diving. Barotrauma occurs only in gas-containing (compressible) body spaces. More than 95% of the body is composed of water (incompressible). Typical gas-filled spaces include the sinuses, middle and inner ears, air-filled areas within carious or filled teeth, and hollow viscous organs such as the intestines and lungs. Barotrauma incurred during descent is called a “squeeze.”

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incurred during ascent is called a “reverse squeeze,” “reverse block,” or expansion injury.

**DIFFERENTIAL DIAGNOSIS AND MEDICAL DECISION MAKING**

Table 133.1 lists the differential diagnosis for dive injuries based on the time of onset of symptoms.

**EAR BAROTRAUMA**

With an intact tympanic membrane (TM), the only communication for equilibration of pressure between the middle ear and the ambient atmosphere is through the eustachian tube (ET). Divers typically perform Valsalva maneuvers during descent to equalize pressure in the middle ear. Failure to equalize leads to pain and damage from injury to the middle or inner ear and results in TM edema, rupture, or hemorrhage, as well as rupture of the oval or round window (may lead to a perilymphatic fistula).

**Table 133.1** Differential Diagnosis of Dive Injuries Based on the Onset of Symptoms

<table>
<thead>
<tr>
<th>SYMPTOM ONSET</th>
<th>INJURIES TO CONSIDER</th>
</tr>
</thead>
<tbody>
<tr>
<td>Descent</td>
<td>Ear or sinus barotrauma</td>
</tr>
<tr>
<td></td>
<td>Inner ear barotrauma</td>
</tr>
<tr>
<td>Bottom</td>
<td>Nitrogen narcosis</td>
</tr>
<tr>
<td></td>
<td>Oxygen toxicity</td>
</tr>
<tr>
<td></td>
<td>Trauma</td>
</tr>
<tr>
<td>Ascent</td>
<td>Arterial gas embolism</td>
</tr>
<tr>
<td></td>
<td>Pneumothorax</td>
</tr>
<tr>
<td></td>
<td>Pneumomediastinum</td>
</tr>
<tr>
<td></td>
<td>Subcutaneous emphysema</td>
</tr>
<tr>
<td></td>
<td>Severe decompression sickness</td>
</tr>
<tr>
<td></td>
<td>Ear or sinus barotrauma</td>
</tr>
<tr>
<td></td>
<td>Barodontalgia</td>
</tr>
<tr>
<td></td>
<td>Gastrointestinal barotrauma</td>
</tr>
<tr>
<td></td>
<td>Trauma</td>
</tr>
<tr>
<td></td>
<td>Alternobaric vertigo</td>
</tr>
<tr>
<td></td>
<td>Alternobaric facial palsy</td>
</tr>
<tr>
<td>15 min after resurfacing</td>
<td>Arterial gas embolism</td>
</tr>
<tr>
<td>15 min to 24 hr after resurfacing</td>
<td>Decompression sickness</td>
</tr>
</tbody>
</table>

**Table 133.2** Barotrauma of the Ear

<table>
<thead>
<tr>
<th>MIDDLE EAR BAROTRAUMA</th>
<th>INNER EAR BAROTRAUMA</th>
<th>ALTENOBARIC VERTIGO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptoms</td>
<td>Ear pain on descent</td>
<td>Ear pain on ascent</td>
</tr>
<tr>
<td></td>
<td>Hearing loss</td>
<td>Transient hearing loss</td>
</tr>
<tr>
<td></td>
<td>Possible transient vertigo</td>
<td>Nausea</td>
</tr>
<tr>
<td>Signs</td>
<td>Conductive hearing loss</td>
<td>Nystagmus</td>
</tr>
<tr>
<td></td>
<td>TM injury</td>
<td>Vomiting</td>
</tr>
<tr>
<td></td>
<td>Unilateral facial paralysis (rare)</td>
<td>Ataxia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Romberg sign</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Neural hearing loss</td>
</tr>
<tr>
<td>Treatment</td>
<td>Decongestants</td>
<td>Referral to an otorhinolaryngologist</td>
</tr>
<tr>
<td></td>
<td></td>
<td>None if resolved</td>
</tr>
</tbody>
</table>

**External Ear Barotrauma (“Squeeze”)**

During diving, water replaces the air in the external ear canal. Obstructions such as wax, a bony growth, or earplugs can create an unvented air space that changes in volume in response to changes in ambient pressure. During descent, the increased pressure “squeezes” this space and causes the TM to bulge outward toward the canal with resultant pain, small hemorrhages, or TM blebs. Prevention consists of cleaning the external canal and removing any foreign bodies.

**Middle Ear Barotrauma (“Squeeze”)**

Middle ear barotrauma (middle ear squeeze, barotitis media) is the most common disorder in divers and hyperbaric medicine patients. It usually occurs during descent as a result of an inability to equalize pressure across the TM. It occurs in 30% of novice divers and 10% of experienced divers. When water exerts pressure on the external TM and pushes it inward, a diver can usually equalize the pressure in the ears by swallowing, yawning, performing a Valsalva maneuver, or blowing against closed nostrils. Divers are often unable to clear their ears because of anatomic variability of the ET, inflammation, a viral infection, or upper aerodigestive dysfunction. Without equalization of the pressure, the TM ruptures. As little as 100 mm Hg (5 fsw) can create a pressure differential large enough to rupture the TM. Symptoms of middle ear barotrauma are ear pain, pressure, and muffled hearing. If the TM is ruptured, vertigo may occur because of the effects of cold water on the middle ear or TM. Treatment consists of decongestants, rest from diving, and follow-up with an otorhinolaryngologist (refractory cases). In general, these injuries are self-limited. However, if a patient needs hyperbaric medicine treatments, pressure equalization tubes may be placed to prevent middle ear barotrauma. Any form of TM rupture, placement of a pressure equalization tube, or

(TM, Tympanic membrane.)
myringotomy would be a contraindication to wet water diving because water comes in direct contact with the middle ear.

**Alternobaric Facial Palsy** Alternobaric facial palsy, a complication of middle ear barotrauma after diving, is a syndrome consisting of unilateral facial nerve palsy, ataxia, vertigo, nausea, and vomiting. The symptoms can be confused with those of AGE or DCS; however, the mechanism is elevated middle ear pressure pressing against the facial nerve and causing ischemic neurapraxia.  Alternobaric palsy is also observed in those who fly after diving, fly at high altitude in unpressurized airplanes, and experience explosive decompression in flight. Though uncomfortable, the symptoms usually resolve within minutes once middle ear pressures equilibrate.

**Reverse Middle Ear Barotrauma** Reverse middle ear barotrauma, or reverse squeeze, is similar to middle ear squeeze but involves increased pressure in the middle ear pushing outward on the TM. It is often due to ET obstruction and occurs on ascent. The symptoms are similar to those of middle ear squeeze; however, reverse squeeze can rupture the round window as well.

**Inner Ear Barotrauma**
The inner ear is a complicated and delicate organ, and any diver complaining of hearing loss, vertigo, or ear pain might have inner ear barotrauma (IEBT). Though less common than middle ear squeeze, IEBT is associated with significantly higher morbidity because it often involves damage to the cochleovestibular organs. Separating the middle and inner ear are the thin membranes of the round and oval windows. Damage to these structures may cause leakage of fluid from the inner to the middle ear. IEBT can result from difficulty equalizing pressure during a dive. Performing a vigorous Val-salva maneuver or forcefully attempting to clear the ears can cause damage to the round window. The symptoms can be quite significant and include extreme dizziness, vertigo, nausea, and vomiting. The patient might also have nystagmus, ataxia, and hearing loss. IEBT is considered an emergency and mandates immediate evaluation by an otolaryngologist.

**Alternobaric Vertigo**
Alternobaric vertigo occurs on ascent as a result of differences in pressure between the two middle ear spaces. This difference causes asymmetric stimulation of each vestibular organ, which leads to vertigo. Symptoms include severe nausea and vomiting, as well as transient hearing loss. The patient will report abrupt relief with clearing of the ears or return to pressure.

**Sinus Barotrauma**
Sinus barotrauma (“sinus squeeze”) is the second most common disorder in divers, but it is significantly less common than middle ear barotrauma, with only 1% of divers affected. Symptoms include sinus pain on descent and bloody nasal discharge on ascent. Treatment consists of decongestants, antinfiammatory agents, and rest from diving.

**Pulmonary Barotrauma and Pulmonary Overpressurization Syndromes**
Pulmonary overpressurization syndromes can occur during rapid ascent with breath-holding in which the pulmonary parenchyma is ruptured. Ascending too fast without exhaling allows the rapidly expanding gases in the lungs to enlarge and stretch the lung parenchyma, followed by overdistention and ultimately parenchymal rupture. Gas then enters the perilung spaces, which creates a pathway for bubbles to embolize to the brain. As little as an 80–mm Hg pressure differential is sufficient to rupture the alveolar lining. It can occur with breath-holding during the last 3 to 4 fsw of ascent.

**Pneumomediastinum and Mediastinal Emphysema** Pneumomediastinum occurs when alveolar rupture allows gas to enter the mediastinum through the perivascular sheath; it causes chest pain and sometimes crepitus that is appreciated with auscultation (Hamman sign). Treatment consists of supplemental oxygen and observation; the diagnosis is made by chest radiographic evaluation and physical examination. Recompression is usually unnecessary, although a thorough neurologic examination to screen for AGE is recommended. Approximately 30% of divers with AGE will be found to have pneumomediastinum on a chest radiograph.

**Subcutaneous Emphysema** Similar to pneumomediastinum, subcutaneous emphysema is due to alveolar rupture causing release of gas into tissues that can track up into the neck and under the skin. Symptoms include palpable crepitus, a sensation of fullness in the chest, possible alteration of voice, and occasionally dysphagia. Treatment is similar to that for pneumomediastinum; recompression is not generally necessary for isolated subcutaneous emphysema.

**Pneumothorax** Pneumothorax is a severe, potentially life-threatening pulmonary overpressure syndrome. Rupture allows bubbles and gas to enter the pleural space, which can place pressure on the lung itself. Symptoms vary greatly, depending the volume of air entering the pleural space and the patient’s baseline lung function. Patients may have simple dyspnea or exhibit shock or life-threatening cardiac arrest (tension pneumothorax). The most common symptoms are sharp chest pain, shortness of breath, and occasionally a sudden, dry, hacking cough. Pain may also be felt in the shoulder, neck, or abdomen. Treatment is immediate administration of high-flow oxygen and possible needle decompression or tube thoracostomy (or both). If pneumothorax occurs during hyperbaric treatment or surface decompression table treatment, immediate venting of the pneumothorax (Heimlich valve) may be necessary to prevent the development of tension pneumothorax.

**Arterial Gas Embolism** AGE, the most lethal result of pulmonary barotrauma, is a common cause of death in recreational divers. Its incidence is underestimated because many in-water deaths are classified as drowning. When rupture of the lung parenchyma leads to intravascular bubbles, these bubbles can embolize and cause end-organ damage. Sadly, this disorder is often seen in inexperienced divers who panic at depth and shoot to the surface without exhaling slowly. The symptoms are dramatic, usually LOC immediately or within minutes of the diver reaching the surface. Death is common. The arterial emboli are most deadly when they travel to the coronary or cerebral circulation. Cerebral AGE is manifested
very much like a stroke and results in headache, confusion, agitation, hemiplegia, or sudden LOC. Air embolism can also block blood flow through the coronary circulation and lead to cardiac ischemia, dysrhythmias, shock, and death.

Definitive treatment of AGE consists of high-flow oxygen on site and immediate hyperbaric recompression. The sooner patients are recompressed, the less likely they are to have permanent neurologic injury. Full recovery is common when recompression is available immediately. It is important to remember that AGE can occur in shallow water while breathing compressed gas or during breath-hold diving. This is in contrast to DCS, which usually occurs following a deep or prolonged dive when high nitrogen partial pressure develops. AGE also occurs in the hospital setting as a result of iatrogenic errors (central line manipulation, hemodialysis), but it can occur with any procedure or trauma that can entrain gas into the bloodstream.

Gastrointestinal Barotrauma
The stomach and intestine are both air-filled organs and, though rarely affected, are susceptible to barotrauma. Specifically, gas expansion occurring on ascent can cause nausea, belching, flatulence, mild stomach pain, and reflux. These symptoms resolve fairly quickly after the diver resurfaces because these spaces are typically easily vented.

Tooth Barotrauma
The small air pockets that exist in teeth and under fillings are susceptible to barotrauma (barodontalgia). Most people experience pain only on ascent, but tooth fractures have occurred (odontocrisis). Decompressions as mild as those experienced during commercial air flight pressurization (8000 feet or 0.75 ATA) are sufficient to cause barodontalgia. Such patients should be referred for dental evaluation, and divers should make their dentists aware of their diving hobby so that air pockets can be avoided.

DECOMPRESSION SICKNESS
DCS, or “the bends,” is a type of DCI that usually occurs after diving at deeper depths. In accordance with Dalton’s law, tissues become highly permeated with inspired inert gases with increasing depth. DCS can also occur after long shallow dives if significant tissue saturation has occurred. It can cause a spectrum of symptoms. Diagnosis can be difficult because divers may complain of only mild to moderate symptoms, which they tend to ignore or attribute to other causes. Frequently, a diver complains, “I just don’t feel right,” or has limb pain without trauma that is assumed to be muscular in origin. DCS symptoms rarely develop while the diver is in the water. The key to diagnosing dive-related injuries is to (1) elicit the timing of the onset of symptoms (before, during, or after a dive), (2) determine the presence of any dive-related DCS risk factors (dehydration, alcohol use, inexperience, failure to follow the decompression tables, flying after diving, reverse-profile diving, multiple dives per day, decompression diving, smoking, advanced age, cold water, patent foramen ovale, and obesity), and (3) have a low threshold for suspicion of DCS when symptoms develop. DCS is classically divided into three types according to the severity of illness and the location of symptoms. In reality, DCS symptoms overlap and the basic treatment is usually the same for all types—recompression with hyperbaric oxygen.

Type I (“Mild” Symptoms)
Type I DCS describes mild symptoms such as joint pain (most common), dermatologic manifestations, and lymphatic-associated swelling and edema as a result of the effects of gas bubbles in the tissues (Box 133.3). Bubble formation in joints is due to the greater negative pressure that exists in the joint spaces. Pain is most often felt in the shoulders or knees but can appear in any joint. The pain is gradual and aching and varies in intensity, usually worsening with time. Limb pain in divers affects the upper extremities three times more often than the lower extremities, and its distribution is often asymmetric. Caisson workers, however, are affected more often in their lower limbs. Merritt makes the point that type I DCS usually involves pain in the extremities whereas type II DCS usually involves central structures.

Dermatologic DCI (“skin bends”) can have several manifestations. A diver can experience itching alone (without a rash) in localized or generalized areas of the arms, legs, face, or trunk (“fleas”). This form is commonly thought to follow dry dives, appears shortly after resurfacing, and lasts only a few minutes to a few hours. Other dermatologic manifestations of DCS are mottling (cutis marmorata) and rindlike skin (peau d’orange).

Type II (“Severe” Symptoms)
Type II DCS causes more severe symptoms that have a high risk of leading to major disability or death. Cardiopulmonary,

<table>
<thead>
<tr>
<th>BOX 133.3 Type I Decompression Sickness</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Musculoskeletal Pain (“Limb Bends”)</strong></td>
</tr>
<tr>
<td>Most common manifestation of DCS</td>
</tr>
<tr>
<td>Dull, aching pain often in the shoulder or knee</td>
</tr>
<tr>
<td>Pain occurring both at rest and with movement</td>
</tr>
<tr>
<td>No evidence of joint inflammation on examination</td>
</tr>
<tr>
<td>Occurs within 24 hours of resurfacing but almost never at depth and rarely within the first few minutes after surfacing</td>
</tr>
<tr>
<td><strong>Cutaneous DCS (“Skin Bends”)</strong></td>
</tr>
<tr>
<td>Itching and pruritus most common symptoms (self-resolving)</td>
</tr>
<tr>
<td>Cutis marmorata (mottled appearance of the skin)</td>
</tr>
<tr>
<td>Peau d’orange (rindlike skin seen in the truncal area)</td>
</tr>
<tr>
<td>“Fleas” (formication-like symptoms of insects on the skin)</td>
</tr>
<tr>
<td><strong>Lymphatic Effects and Edema</strong></td>
</tr>
<tr>
<td>Swelling in the soft tissues or in areas of lymph nodes</td>
</tr>
<tr>
<td>Usually localized</td>
</tr>
<tr>
<td>Very uncommon</td>
</tr>
</tbody>
</table>

**Definitive Treatment**
Definitive treatment is recompression
All patients require oxygen. Remember to consult a hyperbaric medicine specialist early if the patient is being brought from a dive accident

neurologic, and inner ear manifestations predominate (Box 133.4). All patients with type II DCS symptoms require emergency recompression in a hyperbaric chamber. The sooner they are recompressed, the more likely they are to have a full recovery. However, even if treatment is delayed several days, recompression can still be helpful. Multiple treatments may be necessary to maximize recovery.

**Type III Decompression Sickness**
Type III includes both type II symptoms and AGE.

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**TIPS AND TRICKS**

Limb pain as a result of DCS usually persists even at rest, whereas the pain of a traumatic injury often improves with rest or nonuse.

Resolution of the pain at a joint when a blood pressure cuff is inflated is a strong indicator of joint DCS.

Neurologic examination of an injured diver is best performed with the patient out of the stretcher or bed to unmask any neurologic deficits.

Serious diving injuries are much more common in recreational divers than in commercial or military divers.

Abdominal pain may be an early signal of spinal cord injury in divers with DCS.

**BOX 133.4 Type II Decompression Sickness**

Type II is associated with a higher risk for permanent disability or death than type I.

**Pulmonary DCS (“Chokes”)**
Persistent, dry, nonproductive cough
Substernal, pleuritic chest pain
Seen more with high-altitude DCS and in tunnel and caisson workers

**Neurologic DCS**
Represents 60% to 70% of DCS injuries
Spinal cord affected three times more often than the cerebrum
Seen more often in recreational divers.

Symptoms usually occur hours after ascent:
- Tingling in the trunk
- Progressive numbness and paresthesias
- Ascending motor weakness
- Bowel, bladder incontinence
- Paralysis
- Memory impairment, aphasias, visual disturbances, personality changes

**Vestibular DCS (“Staggers”)**
Dizziness, nausea, vomiting, nystagmus, hearing loss, tinnitus
Uncommon in recreational divers
Can be confused with middle ear barotrauma

**Definitive Treatment**
Recompression

**DCS, Decompression sickness.**

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**NEUROLOGIC EXAMINATION**

The importance of performing a thorough neurologic examination cannot be understated. Unless an obvious neurologic deficit is noted, many dive-injured patients do not realize that they have an injury. However, if “hard” neurologic symptoms are observed, the patient requires immediate recompression with hyperbaric oxygen therapy without delay (Box 133.5). The neurologic examination should include a diver’s mental status, cranial nerves, balance, coordination, sensory testing, deep tendon reflexes, pathologic reflexes, and both fine and gross motor skills. Preprinted neurologic examination forms enable the physician to maximize diagnosis and treatment, as well as to standardize findings.

**TREATMENT**

**PREHOSPITAL MANAGEMENT AND AIR EVACUATION FROM THE SITE**
Prehospital treatment of a dive injury is similar to that given in the ED. However, the hyperbaric team should be notified early that the patient is incoming to minimize delays (Box 133.6).

**EMERGENCY DEPARTMENT EVALUATION AND TREATMENT**
ED treatment of dive-injured patients must follow a standard approach. In addition to ordering routine laboratory tests, oxygen, and intravenous fluids, obtaining a thorough history is critical to treatment (Boxes 133.7 and 133.8).
**BOX 133.6 Prehospital Treatment and Air Evacuation Instructions for Dive-Injured Patients**

1. Maintain the ABCs of resuscitation and evaluate the patient’s serum glucose level.
2. Use ACLS protocols to stabilize the patient.
3. Administer high-flow oxygen, establish intravenous lines, and begin fluid administration.
4. Keep the patient flat; avoid the Trendelenburg position.
5. If air evacuation is being performed, ensure that the cabin of the airplane is pressurized or have the pilot fly at altitudes below 1000 feet.
6. Transport the patient along with all gear (it will have to be examined later).
7. Remember that the other members of the diving party might also need transport and evaluation for decompression sickness; they should accompany the patient to the ED.
8. Alert the consultant dive physician and hyperbaric center ahead of time that a dive injury has occurred and the patient is being brought to the ED.

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**FOLLOW-UP, NEXT STEPS IN CARE, AND PATIENT EDUCATION**

Although each patient is unique, many patients with DCI can be safely discharged from the hospital. However, any diver with serious DCS symptoms should be admitted. All patients who have experienced DCS or embolic dive injuries should be transferred immediately to the closest emergency hyperbaric facility. The physician should err on the side of caution; any patient with concerning symptoms should not be discharged until a hyperbaric physician has been consulted (Boxes 133.9 and 133.10).

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**BOX 133.7 Emergency Department Diagnostic Work-up for Dive-Injured Patients**

**Laboratory Tests**

- Complete blood count
- Measurement of serum electrolytes
- Consider markers of injury LDH, CPK
- Oxygen saturation measurement

Add these tests for altered mental status:

- Urine and blood toxicologic tests with acetylsalicylic acid or acetaminophen and ethanol levels
- Arterial blood gas measurements, including a carboxyhemoglobin level

**Imaging**

Consider the following evaluations for suspicion of pulmonary barotrauma:

- Chest radiograph
- Computed tomography of the chest (evaluation for blebs, small pneumothoraces, and other pulmonary problems)

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**BOX 133.8 Dive History Interview: Questions to Ask Dive-Injured Patients**

1. Where did the dive occur (e.g., ocean, river, pool)?
2. When was the onset of the patient’s symptoms (on resurfacing, during descent or ascent, at the bottom)?
3. How deep did the patient go? Was a dive computer being used?
4. Was the patient intoxicated or dehydrated?
5. What type of diving equipment was used? What type of gas was used (compressed air, mixed gas, enriched air)? What was the source of the gas?
6. Did the patient perform heavy exertion or work during the dive?
7. Did the dive approach or exceed decompression limits?
8. How many dives did the patient perform and what were the depths, bottom times, total times, and resurface intervals for all dives in the previous days preceding symptoms (the dive “profiles”)?
9. Were decompression stops missed? Was in-water recompression attempted?
10. What was the time delay from the last dive to the time of air travel or travel to altitude such as a mountain range?
11. Did the patient experience ear or sinus problems on this dive or in the past?
12. Does the patient have any other medical problems? What medications does the patient take?
13. Was oxygen given at the scene?

**FLYING AFTER DIVING**

One of the most important factors in the disposition of divers is how long the diver should wait to fly after diving or a diving-related injury. Forgetting to counsel a patient about
flying limitations has the potential for serious injury. A number of guidelines are available. For the most part, it is unwise to fly within 12 to 24 hours after diving. A diver should delay flying for at least 12 hours after a single no-decompression obligation dive. A diver who participates in multiple-day, unlimited diving should delay flying for at least 24 hours after the last dive. A diver who has experienced DCS should be advised to not fly for 3 to 7 days after treatment of type I DCS or for 4 weeks after recompression therapy for type II DCS.

**SUGGESTED READINGS**


**REFERENCES**

References can be found on Expert Consult @ www.expertconsult.com.
REFERENCES

12. London Diving Chamber and Hyperbarics website. Available at www.londondivingchamber.co.uk/.