



# Decompression Illness in Scuba Divers

## What You Need to Know

The initial compression and subsequent expansion of air during a dive can have serious adverse effects on a wide range of body systems. As the popularity of the sport has increased in recent times, so has the likelihood that an emergency physician will see novice divers with decompression sickness, arterial gas embolism, and even pulmonary barotrauma. The authors explain how to be prepared.

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**R**ecreational scuba diving has become increasingly popular since the 1980s, with more than 500,000 new certifications awarded each year. As the sport has

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grown, so has the incidence of diving-related injuries—in particular, decompression sickness (DCS) and arterial gas embolism (AGE), which are collectively referred to as decompression illness.

Decompression sickness results from gas bubbles that form in the venous circulation and tissues as the ambient pressure decreases when a diver ascends to the surface. Arterial gas embolism occurs when gas bubbles causing circulatory and inflammatory derangements enter the arterial circulation, obstructing blood flow to various organs. Emergency physicians need to be prepared to recognize and manage the effects that decompression illness can have on a wide range of body systems.

### HAZARDS OF CHANGING PRESSURE

In recreational scuba diving, divers typically do not exceed a depth of 130 feet sea water (fsw). As divers descend, the ambient pressure exerted on them

increases. At a depth of only 33 fsw, the ambient pressure on a diver has already doubled.<sup>1</sup> As stated in Boyle's law, when a gas is subject to increasing pressure, its volume will decrease. Increased pressure is felt not only in the air-filled spaces of a diver's body, mainly the middle ear and sinuses, but also in the lungs and intestines. Proper equalization techniques are stressed in diver education to avoid pain and damage to these spaces.

During ascent, the ambient pressure decreases back toward that of the atmosphere and the volume of gas expands. If air becomes trapped in the lungs from disease or because a diver does not exhale during ascent, expanding air can cause the lungs to overinflate and burst, a condition called *pulmonary barotrauma*.

Scuba divers usually breathe a mixture of oxygen and nitrogen, which becomes compressed due to the increased pressure during descent. Compressed air contains more molecules at depth, and thus a descending diver receives more air molecules with each breath. This causes larger amounts of gas to become dissolved in the blood and tissues. Oxygen is continually used by the body and does not accumulate, but the excess nitrogen molecules are not readily used, and the diver is left with greater residual amounts at increased depths.

The Professional Association of Diving Instructors, one of the largest diver training organizations, recommends that scuba divers ascend slowly, no faster than 60 fsw per minute.<sup>1</sup> Recreational divers are encouraged to perform a safety stop for dives deeper than 60 fsw. A safety stop is merely a recommended precautionary stop performed during ascent at a depth of 15 to 20 fsw for 3 to 5 minutes. This recommended stop is different from a required decompression stop, which is necessary for divers who descend to depths greater than 130 fsw or who exceed the maximum time limit at a given depth. These divers, depending on how deep they go, may be required to make multiple decompression stops.<sup>2</sup>

The purpose of a safety stop is to compensate for variation in ascent rate and individual differences in nitrogen absorption and elimination. A slow ascent time combined with a recommended

**TABLE 1. Symptoms of Decompression Sickness**

Type 1	Type 2
<ul style="list-style-type: none"> <li>• skin rashes</li> <li>• joint pain</li> <li>• musculoskeletal pain</li> </ul>	<p><i>CNS</i></p> <ul style="list-style-type: none"> <li>• headache</li> <li>• paraplegia</li> <li>• ataxia</li> <li>• unconsciousness</li> </ul> <p><i>Pulmonary (chokes)</i></p> <ul style="list-style-type: none"> <li>• hemoptysis</li> <li>• cough</li> <li>• substernal chest pain</li> <li>• dyspnea</li> </ul>

safety stop should allow ample time for the excess nitrogen to leave the tissues, enter the low-pressure venous circulation, move into the pulmonary capillary bed, and be exhaled and eliminated in the normal manner.

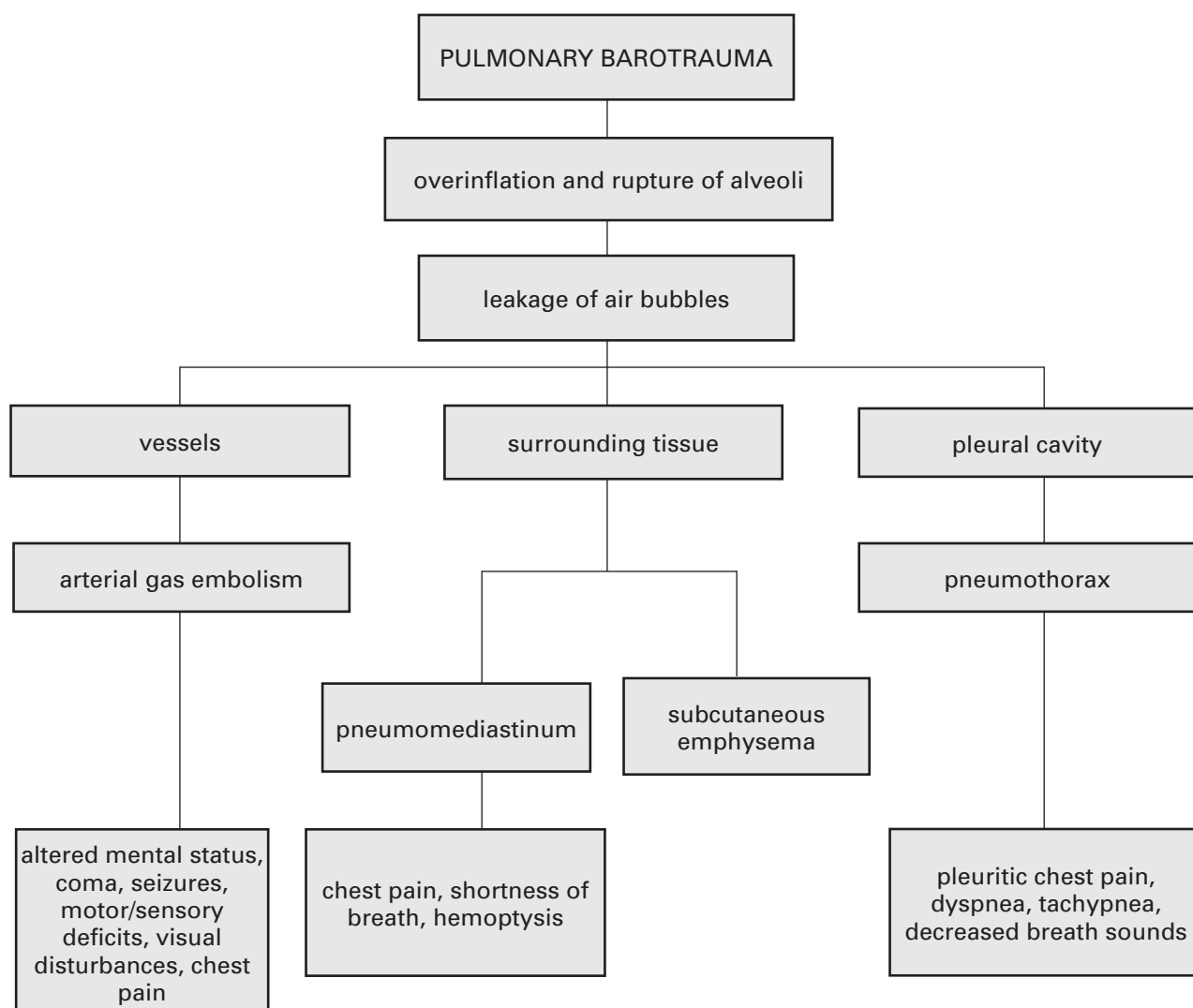
### SEVERAL TYPES OF ILLNESS

If divers ascend too rapidly, they may not allow enough time for excess gas to leave the tissues and blood. Rapid ascent can cause gas to come out of solution and form bubbles, which remain in tissues or in the blood. With continued ascent, the gas bubbles will expand as pressure decreases. The expanded bubbles can obstruct blood flow directly or cause tissue ischemia indirectly by activating platelets and the coagulation cascade. Gas bubbles can also lead to vascular endothelial dysfunction with capillary leakage, resulting in complement activation and pathogenic interaction between leukocytes and endothelial tissues. These changes provoke swelling that leads to pain in muscles, joints, and tendons.<sup>3</sup>

**DCS types 1 and 2.** Two distinct forms of DCS are recognized (Table 1). Type 1 DCS, also called *the bends*, results from the rapid formation and expansion of gas bubbles in the musculoskeletal, cutaneous, and lymphatic systems. Possible symptoms

### >>FAST TRACK<<

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**FIGURE. Pulmonary Barotrauma and Subsequent Complications**

include arthralgia, myalgia, and skin rash. Type 1 DCS usually presents within hours of surfacing but can occur up to 48 hours later.

Type 2 DCS is diagnosed when gas bubbles have induced local ischemia in the central nervous, cardiovascular, pulmonary, and digestive systems.<sup>4</sup> The central nervous system is especially susceptible to decompression illness because nitrogen dissolves so readily in its fatty tissues. Spinal cord involvement is usually the result of gas bubbles in the venous plexus system that eventually occlude venous

outflow from the cord. This prevents the dissolved nitrogen already in the cord from leaving, which can result in the formation of in-situ bubbles that cause severe damage to the spinal cord. Type 2 DCS symptoms include severe headache, altered mental status, paraplegia, motor weakness, ataxia, and loss of consciousness.<sup>4</sup>

Pulmonary DCS (the chokes) is caused by a large number of gas bubbles entering the pulmonary artery. Symptoms range from cough, hemoptysis, dyspnea, and substernal chest pain to cardiovascular collapse.

**Pulmonary barotrauma.** A very serious possible consequence when a diver holds his or her breath on ascent, causing air-trapping in the lung, is pulmonary barotrauma. As pressure decreases, the trapped air expands, which can lead to rupture of the alveolar lining, allowing air to leak into the interstitium of the lungs. The intermediate consequence of this is pulmonary interstitial emphysema, usually asymptomatic unless further damage and air leakage occur. From the interstitium, air can travel and cause three main complications (Figure): mediastinal and subcutaneous emphysema from accumulated air in the mediastinum, AGE from air bubbles in the arterial system, and pneumothorax from accumulated air between the lung and chest wall. Continuing, unrelieved accumulation of air may result in tension pneumothorax.<sup>5</sup>

**Arterial gas embolism.** In AGE secondary to DCS, the pulmonary capillary beds become overwhelmed with bubbles. If this occurs, some bubbles may pass into the left side of the heart and subsequently into the arterial circulation. Gas bubbles can also directly enter the left side of the heart if the patient has a septal defect or patent foramen ovale. Bubble distribution in the arterial system mainly depends on blood flow but most commonly occludes the cerebral and coronary circulations.<sup>4,5</sup>

Cerebral AGE occurs when gas bubbles lodge in the smaller cerebral arteries. It can cause a variety of neurologic and stroke-like symptoms, including altered mental status, loss of consciousness, hemiplegia, and seizures. Symptoms of cerebral AGE secondary to pulmonary barotrauma usually present during ascent or immediately upon surfacing.<sup>4</sup>

Diagnosis of AGE is clinical and relies on the diver's history and symptoms. Three main criteria are used to diagnose AGE: onset of symptoms less than 15 minutes after surfacing, presence of cerebral neurologic signs and symptoms, and symptom duration greater than 15 minutes. An absence of neurologic manifestations does not rule out AGE, however. The most common of these, a general stupor or altered mental status, is observed in only 24% of patients with AGE, followed by coma without seizures (22%), coma with seizures (18%), unilateral motor deficits (14%), visual disturbances (9%), and vertigo, unilateral sensory deficits, and bilateral motor deficits (all 8%).<sup>6</sup>

The diagnosis is strengthened when a diver's history includes a rapid ascent to the surface, out-of-

air emergency ascent, or cardiopulmonary or other symptoms of pulmonary barotrauma, including pneumothorax and subcutaneous emphysema. In the case of suspected cerebral AGE, brain imaging can show intravascular cerebral air bubbles. Usually, however, imaging is normal even in the face of severe neurologic abnormalities.

### WHAT TIPS THE SCALES?

Deep and repetitive dives, rapid ascent, cold water dives, and air travel after diving are all significant risk factors for decompression illness. The deeper the dive, the more excess nitrogen is dissolved into the body tissues and the greater the likelihood that bubbles will form. Repetitive dives (two or more dives in one day) also result in excess nitrogen in the body, because residual nitrogen remains dissolved in body tissues for at least 12 hours after every dive. Air travel also heightens risk because of the reduced ambient pressure inside the aircraft, which increases the possibility of bubble formation. It is recommended that divers wait a minimum of 12 hours after a single shallow dive or 24 hours after deep or repetitive dives before they fly.

Individual risk factors include dehydration, fatigue, obesity, smoking, diabetes, patent foramen ovale, cardiovascular disease, and lung disease. Patients with pulmonary diseases such as emphysema already have air-trapping in their lungs, which presents a significantly high risk of pulmonary barotrauma and decompression illness. Divers with a patent foramen ovale have a much higher risk of developing decompression illness, especially type 2 DCS, because the defect allows gas bubbles to be shunted directly from the right side of the heart to the left, bypassing the lungs and entering the arterial circulation. These patients have a high risk of neurologic DCS and AGE.

Smoking elevates plasma fibrinogen and factor XIII levels, which are associated with increased blood coagulation activity. This puts divers at risk for decompression illness and has been a noted risk factor for pulmonary embolism in the general population.<sup>4,5</sup>

### >>FAST TRACK<<

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**TABLE 2. US Navy Treatment Table\***

Depth (feet)	Time (min)	Breathing media	Total time elapsed (hr:min)
60 <sup>a</sup>	20	O <sub>2</sub> <sup>b</sup>	0:20 <sup>c</sup>
60	5	Air	0:25
60	20	O <sub>2</sub>	0:45
60	5	Air	0:50
60	20	O <sub>2</sub>	1:10
60	5	Air	1:15
60 to 30	30	O <sub>2</sub>	1:45
30	15	Air	2:00
30	60	O <sub>2</sub>	3:00
30	15	Air	3:15
30	60	O <sub>2</sub>	4:15
30 to 0	30	O <sub>2</sub>	4:45

\*Treatment of type II or type I decompression sickness when symptoms are not relieved within 10 min at 60 ft.

<sup>a</sup>Descent rate=25 ft/min. Ascent rate=1 ft/min. Do not compensate for slower ascent rates. Compensate for faster rates by halting the ascent.

<sup>b</sup>If oxygen must be interrupted because of adverse reaction, allow 15 min after the reaction has entirely subsided and resume schedule at point of interruption.

<sup>c</sup>Time at 60 ft begins on arrival.

Adapted from US Department of the Navy Treatment Table 6.<sup>10</sup>

dient into tissues. Oxygen will also show enhanced movement into tissues, thus increasing perfusion. The reduction of bubble size accelerates as the volume of gas declines with increasing pressure in the chamber.

The US Navy developed the most commonly used treatment regimen for decompression illness, including initial treatment of AGE, (Table 2)<sup>10</sup>; it recommends recompression to 2.8 atmospheres absolute or 60 fsw while breathing 100% oxygen. The session lasts anywhere from 60 minutes to 4 hours and 45 minutes, depending on the severity of illness. Additional recompression treatments are recommended until there is no further improvement of symptoms. The number of repeated treatments will be determined by the patient's symptoms, but most divers with mild to moderate decompression illness need two to three repetitions.<sup>5,6</sup> The Divers Alert Network's statistical analysis of 3000 decompression illness cases supports the effectiveness of no more than five to 10 repetitive treatments for most people.<sup>11</sup> Transitioning to other US Navy treatment tables<sup>12,13</sup> is an option, based on clinical response.

Recompression therapy should be initiated promptly upon clinical diagnosis of decompression illness. Long delays to treatment are associated with incomplete resolution of symptoms; however, no cutoff point for therapeutic efficacy has been established.

Certain adjunctive treatments have been suggested in addition to the gold standard of hyperbaric recompression therapy.<sup>14</sup> The addition of nonsteroidal anti-inflammatory drugs (NSAIDs) to routine recompression therapy has not been shown to improve the effectiveness of treatment<sup>15</sup>; however, it did demonstrate a reduction in the number of repeat recompressions needed. Likewise, a helium and oxygen mixture used in the recompression chamber, instead of pure oxygen, also correlates with a reduction in the number of recompressions needed for symptom reduction.<sup>16,17</sup> Neither the addition of NSAIDs nor the replacement of oxygen with the helium-oxygen product appears to improve the patient's odds of recovery. *continued on page 48*

## THE ROAD TO RECOVERY

Emergency management for a scuba diver experiencing signs and symptoms of decompression illness includes intravenous fluids and administration of 100% oxygen via a non-rebreather mask. The patient should be kept as motionless as possible in the supine position; the Trendelenburg position is no longer recommended because it merely increases intracranial pressure and decreases cerebral perfusion.<sup>7-9</sup>

For any form of decompression illness or injury, immediate transport to a hospital with a recompression chamber is recommended. Recompression therapy reduces the size of air bubbles, allowing for easier reabsorption into tissues and subsequent dissipation. The patient is placed in an airtight hyperbaric chamber and 100% oxygen is administered while the pressure in the chamber is increased.<sup>7,8</sup> The increased pressure enhances the movement of nitrogen out of bubbles and down its diffusion gra-

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Neurologic deficits, cardiovascular instability, or both indicate treatment of AGE. If pulmonary barotrauma is the suspected cause of AGE, a chest x-ray should be obtained before hyperbaric recompression to check for a pneumothorax. If one is found, a chest tube should be placed to prevent the development of a tension pneumothorax during recompression therapy.

If a patient with decompression illness develops severe hypoxemia and dyspnea, suspicion of pulmonary embolism should be aroused. Prompt treatment in a hyperbaric chamber, anticoagulation, and, if indicated, administration of fibrinolytics are necessary.<sup>18</sup> □

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