may be bruised or even ruptured as it is pushed inward. Negative pressure in the middle ear results in engorgement of blood vessels in the mucous membranes and leads to effusion or bleeding, which can be associated with a *conductive* hearing loss. MEBT is much less common during ascent because expanding gas in the middle-ear space tends to open the eustachian tube easily and "automatically." Barotrauma may also affect the respiratory sinuses, although the sinus ostia are usually widely patent and allow automatic pressure equalization without the need for specific maneuvers. If equalization fails, pain usually results in termination of the dive. Difficulty with equalizing ears or sinuses may respond to oral or nasal decongestants.

Much less commonly the inner ear may suffer barotrauma (IEBT). Several explanations have been proposed, of which the most favored holds that forceful attempts to insufflate the middle-ear space by Valsalva maneuvers during descent cause sudden transmission of pressure to the perilymph via the cochlear aqueduct and outward rupture of the round window already under tension because of negative middle ear pressure. The clinician should be alerted to possible IEBT after diving by a *sensorineural* hearing loss or true vertigo (which is often accompanied by nausea, vomiting, nystagmus, and ataxia). These manifestations can also occur in vestibulocochlear DCS (see below) but should never be attributed to MEBT. Immediate review by an expert diving physician is recommended, and urgent referral to an otologist will often follow.

The lungs are also vulnerable to barotrauma but are at most risk during ascent. If expanding gas becomes trapped in the lungs as P_{amb} falls, this may rupture alveoli and associated vascular tissue. Gas trapping may occur if divers intentionally or involuntarily hold their breath during ascent or if there are bullae. The extent to which asthma predisposes to pulmonary barotrauma is debated, but the presence of active bronchoconstriction must increase risk. For this reason, asthmatics who regularly require bronchodilator medications or whose airways are sensitive to exercise or cold air are usually discouraged from diving. While possible consequences of pulmonary barotrauma include pneumothorax and mediastinal emphysema, the most feared is the introduction of gas into the pulmonary veins leading to cerebral arterial gas embolism (CAGE). Manifestations of CAGE include loss of consciousness, confusion, hemiplegia, visual disturbances, and speech difficulties, appearing immediately or within minutes after surfacing. The management is the same as for DCS described below. It is notable that the natural history of CAGE often includes substantial or complete resolution of symptoms early after the event. This is probably the clinical correlate of bubble involution and redistribution with consequent restoration of flow. Patients exhibiting such remissions should still be reviewed at specialist diving medical centers because secondary deterioration or reembolization can occur. Unsurprisingly, these events can be misdiagnosed as typical strokes or transient ischemic attacks (TIAs) (Chap. 455) when patients are seen by those unfamiliar with diving medicine. *All patients presenting with neurologic symptoms after diving should have their symptoms discussed with a specialist in diving medicine and be considered for recompression therapy.*

DECOMPRESSION SICKNESS

DCS is caused by the formation of bubbles from dissolved inert gas (usually nitrogen) during or after ascent (decompression) from a compressed gas dive. Bubble formation is also possible following decompression for extravehicular activity during space flight and with ascent to altitude in unpressurized aircraft. DCS in the latter scenarios is probably rare in comparison with diving, where the incidence is approximately 1:10,000 recreational dives.

Breathing at elevated P_{amb} results in increased uptake of inert gas into blood and then into tissues. The rate at which tissue inert gas equilibrates with the inspired inert gas pressure is proportional to tissue blood flow and the blood-tissue partition coefficient for the gas. Similar factors dictate the kinetics of inert gas washout during ascent. If the rate of gas washout from tissues does not match the rate of decline in P_{amb}, then the sum of dissolved gas pressures in the tissue will exceed P_{amb}, a condition referred to as "supersaturation." This is the prerequisite for bubbles to form during decompression, although other less well-understood factors are also involved. Deeper and longer dives result in greater inert gas absorption and greater likelihood of tissue supersaturation during ascent. Divers control their ascent for a given depth and time exposure using algorithms that often include periods where ascent is halted for a prescribed period at different depths to allow time for gas washout ("decompression stops"). Although a breach of these protocols increases the risk of DCS, adherence does not guarantee against it. DCS should be considered in any diver manifesting symptoms not readily explained by an alternative mechanism.

Bubbles may form within tissues themselves, where they cause symptoms by mechanical distraction of pain-sensitive or functionally important structures. They also appear in the venous circulation as blood passes through supersaturated tissues. Some venous bubbles are tolerated without symptoms and are filtered from the circulation in the pulmonary capillaries. However, in sufficiently large numbers, these bubbles are capable of inciting inflammatory and coagulation cascades, damaging endothelium, activating formed elements of blood such as platelets, and causing symptomatic pulmonary vascular obstruction. Moreover, if there is a right-to-left shunt such as through a patent foramen ovale (PFO) or an intrapulmonary shunt, then venous bubbles may enter the arterial circulation (25% of adults have a probe-patent PFO). The risk of cerebral, spinal cord, inner ear, and skin manifestations appears higher in the presence of significant shunts, suggesting that these "arterialized" venous bubbles can cause harm, perhaps by disrupting flow in the microcirculation of target organs. Circulating endothelial microparticles, which are elevated in number and size after diving, are currently under investigation as indicators of decompression stress or possibly as injurious agents in their own right. How they arise, and their role in DCS, remain unclear.

Table 477e-3 lists manifestations of DCS grouped according to organ system. The majority of cases present with mild symptoms, including musculoskeletal pain, fatigue, and minor neurologic manifestations such as patchy paresthesias. Serious presentations are much less common. Pulmonary and cardiovascular manifestations can be

TABLE 477e-3 MANIFESTATIONS OF DECOMPRESSION SICKNESS

Organ System	Manifestations
Musculoskeletal	Limb pain
Neurologic	
Cerebral	Confusion
	Visual disturbances
	Speech disturbances
Spinal	Muscular weakness
	Paralysis
	Upper motor neuron signs
	Bladder and sphincter dysfunction
	Dermatomal sensory disturbances
	Abdominal pain
	Girdle pain
Vestibulocochlear	Hearing loss
	Vertigo and ataxia
	Nausea and vomiting
Peripheral	Patchy nondermatomal sensory disturbance
Pulmonary	Cough
	Dyspnea
	Pulmonary edema (rare)
Cardiovascular	Chest pain
	Arrhythmia
	Hemoconcentration
	Coagulopathy
	Hypotension
Cutaneous	Rash, itch
Lymphatic	Soft tissue edema, often relatively localized
Constitutional	Fatigue and malaise