left atrial dilation and fibrillation. The remainder originates from aortic aneurysms, atherosclerotic plaques, valvular vegetations, or venous thrombi (paradoxical emboli); 10% to 15% are of unknown origin. In contrast to venous emboli, the vast majority of which lodge in the lung, arterial emboli can travel to a wide variety of sites; the point of arrest depends on the source and the relative amount of blood flow that downstream tissues receive. Most come to rest in the lower extremities (75%) or the brain (10%), but other tissues, including the intestines, kidneys, spleen, and upper extremities, may be involved on occasion. The consequences of systemic emboli depend on the vulnerability of the affected tissues to ischemia, the caliber of the occluded vessel, and whether a collateral blood supply exists; in general, however, the outcome is tissue infarction.

Fat and Marrow Embolism

Microscopic fat globules—sometimes with associated hematopoietic bone marrow—can be found in the pulmonary vasculature after fractures of long bones or, rarely, in the setting of soft tissue trauma and burns. Presumably these injuries rupture vascular sinusoids in the marrow or small venules, allowing marrow or adipose tissue to herniate into the vascular space and travel to the lung. Fat and marrow emboli are very common incidental findings after vigorous cardiopulmonary resuscitation and are probably of no clinical consequence. Indeed, fat embolism occurs in some 90% of individuals with severe skeletal injuries (Fig. 4-16), but less than 10% of such patients have any clinical findings.

Fat embolism syndrome is the term applied to the minority of patients who become symptomatic. It is characterized by pulmonary insufficiency, neurologic symptoms, anemia, and thrombocytopenia, and is fatal in about 5% to 15% of cases. Typically, 1 to 3 days after injury there is a sudden onset of tachypnea, dyspnea, and tachycardia; irritability and restlessness can progress to delirium or coma. Thrombocytopenia is attributed to platelet adhesion to fat globules and subsequent aggregation or splenic sequestration; anemia can result from similar red cell aggregation and/or hemolysis. A diffuse petechial rash (seen in 20% to 50% of cases) is related to rapid onset of thrombocytopenia and can be a useful diagnostic feature.

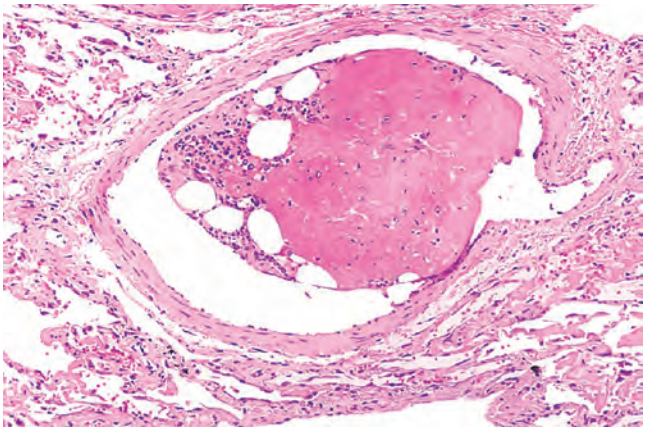

Figure 4-16 Bone marrow embolus in the pulmonary circulation. The cellular elements on the left side of the embolus are hematopoietic cells, while the cleared vacuoles represent marrow fat. The relatively uniform red area on the right of the embolus is an early organizing thrombus.

The pathogenesis of fat emboli syndrome probably involves both mechanical obstruction and biochemical injury. Fat microemboli and associated red cell and platelet aggregates can occlude the pulmonary and cerebral microvasculature. Release of free fatty acids from the fat globules exacerbates the situation by causing local toxic injury to endothelium, and platelet activation and granulocyte recruitment (with free radical, protease, and eicosanoid release) complete the vascular assault. Because lipids are dissolved out of tissue preparations by the solvents routinely used in paraffin embedding, the microscopic demonstration of fat microglobules typically requires specialized techniques, including frozen sections and stains for fat.

Air Embolism

Gas bubbles within the circulation can coalesce to form frothy masses that obstruct vascular flow and cause distal ischemic injury. For example, a very small volume of air trapped in a coronary artery during bypass surgery, or introduced into the cerebral circulation by neurosurgery in the "sitting position," can occlude flow with dire consequences. A larger volume of air, generally more than 100 cc, is necessary to produce a clinical effect in the pulmonary circulation; unless care is taken, this volume of air can be inadvertently introduced during obstetric or laparoscopic procedures, or as a consequence of chest wall injury.

A particular form of gas embolism, called *decompression sickness*, occurs when individuals experience sudden decreases in atmospheric pressure. Scuba and deep sea divers, underwater construction workers, and individuals in unpressurized aircraft in rapid ascent are all at risk. When air is breathed at high pressure (e.g., during a deep sea dive), increased amounts of gas (particularly nitrogen) are dissolved in the blood and tissues. If the diver then ascends (depressurizes) too rapidly, the nitrogen comes out of solution in the tissues and the blood.

The rapid formation of gas bubbles within skeletal muscles and supporting tissues in and about joints is responsible for the painful condition called *the bends* (so named in the 1880s because it was noted that those afflicted characteristically arched their backs in a manner reminiscent of a then-popular women's fashion pose called the Grecian bend). In the lungs, gas bubbles in the vasculature cause edema, hemorrhage, and focal atelectasis or emphysema, leading to a form of respiratory distress called the *chokes*. A more chronic form of decompression sickness is called *caisson disease* (named for the pressurized vessels used in bridge construction; workers in these vessels suffered both acute and chronic forms of decompression sickness). In caisson disease, persistence of gas emboli in the skeletal system leads to multiple foci of ischemic necrosis; the more common sites are the femoral heads, tibia, and humeri.

Individuals affected by acute decompression sickness are treated by being placed in a chamber under sufficiently high pressure to force the gas bubbles back into solution. Subsequent slow decompression permits gradual