symptomatic embolism. Those with hip fractures are at particularly high risk. Hypercoagulable states, either primary (e.g., factor V Leiden, prothrombin mutations, and antiphospholipid syndrome) or secondary (e.g., obesity, recent surgery, cancer, oral contraceptive use, pregnancy), are important risk factors. Indwelling central venous lines can be a nidus for formation of right atrial thrombi, which can embolize to the lungs. Rarely pulmonary embolism may consist of fat, air, or tumor. Small bone marrow emboli are often seen in patients who die after chest compressions performed during resuscitative efforts.

The pathophysiologic response and clinical significance of pulmonary embolism depend on the extent to which pulmonary artery blood flow is obstructed, the size of the occluded vessels, the number of emboli, and the cardiovascular health of the patient. Emboli have two deleterious pathophysiologic consequences: *respiratory compromise* due to the nonperfused, although ventilated, segment; and *hemodynamic compromise* due to increased resistance to pulmonary blood flow caused by the embolic obstruction. Sudden death often ensues, largely as a result of the blockage of blood flow through the lungs. Death may also be caused by acute right-sided heart failure (*acute cor pulmonale*).

MORPHOLOGY

Large emboli lodge in the main pulmonary artery or its major branches or at the bifurcation as a saddle embolus (Fig. 15-27). Smaller emboli travel out into the more peripheral vessels, where they may cause hemorrhage or infarction. In patients with adequate cardiovascular function, the bronchial arterial supply sustains the lung parenchyma; in this instance, hemorrhage may occur, but there is no infarction. In those in whom the cardiovascular function is already compromised, such as patients with heart or lung disease, infarction may occur. Overall, about 10% of emboli cause infarction. About three fourths of infarcts affect the lower lobes, and in more than half, multiple lesions occur. They vary in size from barely visible to massive lesions involving large parts of a lobe. Typically, they extend to the periphery of the lung as a wedge with the apex pointing toward the hilus of the lung. In many cases, an occluded vessel is identified near the apex of the infarct. Pulmonary embolus can be distinguished from a postmortem clot by the presence of the lines of Zahn in the thrombus (Chapter 4).

The pulmonary infarct is classically hemorrhagic and appears as a raised, red-blue area in the early stages (Fig. 15-28). Often, the apposed pleural surface is covered by a fibrinous exudate. The red cells begin to lyse within 48 hours, and the infarct becomes paler and eventually red-brown as hemosiderin is produced. With the passage of time, fibrous replacement begins at the margins as a gray-white peripheral zone and eventually converts the infarct into a contracted scar. Histologically, the hemorrhagic area shows ischemic necrosis of the alveolar walls, bronchioles, and vessels. If the infarct is caused by an infected embolus, the neutrophilic inflammatory reaction can be intense. Such lesions are referred to as **septic infarcts**, some which turn into abscesses.

Clinical Course. **A large pulmonary embolus is one of the few causes of virtually instantaneous death.** During cardiopulmonary resuscitation in such instances, the patient frequently is said to have *electromechanical dissociation*, in which the electrocardiogram has a rhythm but no pulses are palpated because no blood is entering the pulmonary arterial circulation. If the patient survives after a sizable pulmonary embolus, however, the clinical syndrome may mimic myocardial infarction, with severe chest pain, dyspnea, and shock. *Small emboli* are silent or induce only transient chest pain and cough. Pulmonary infarcts manifest as dyspnea, tachypnea, fever, chest pain, cough, and hemoptysis. An overlying fibrinous pleuritis may produce a pleural friction rub.

Findings on *chest radiograph* are variable and can be normal or disclose a pulmonary infarct, usually 12 to 36 hours after it has occurred, as a *wedge-shaped infiltrate*. The diagnosis of pulmonary embolism is usually made with spiral computed tomographic angiography. Rarely, other diagnostic methods, such as ventilation perfusion scanning or pulmonary angiography are required. Deep vein thrombosis can be diagnosed with duplex ultrasonography. After the initial acute insult, emboli often resolve via contraction and fibrinolysis, particularly in the relatively young. If unresolved, over the course of time multiple small emboli may lead to pulmonary hypertension and chronic cor

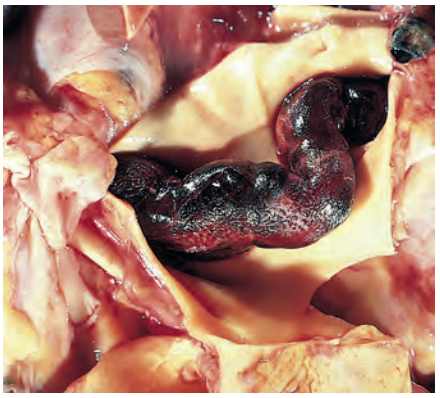

Figure 15-27 Large saddle embolus from the femoral vein lying astride the main left and right pulmonary arteries. (From the teaching collection of the Department of Pathology, University of Texas Southwestern Medical School, Dallas, Tex.)

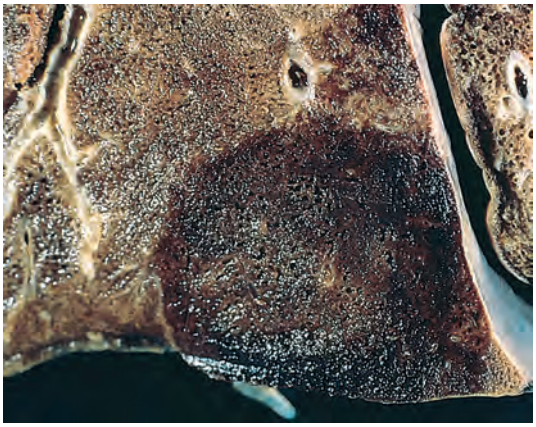

Figure 15-28 Acute hemorrhagic pulmonary infarct.