resorption and exhalation of the gases, which prevents the obstructive bubbles from reforming.

Amniotic Fluid Embolism

Amniotic fluid embolism is the fifth most common cause of maternal mortality worldwide; it accounts for roughly 10% of maternal deaths in the United States and results in permanent neurologic deficit in as many as 85% of survivors. Amniotic fluid embolism is an ominous complication of labor and the immediate postpartum period. Although the incidence is only approximately 1 in 40,000 deliveries, the mortality rate is up to 80%. The onset is characterized by sudden severe dyspnea, cyanosis, and shock, followed by neurologic impairment ranging from headache to seizures and coma. If the patient survives the initial crisis, pulmonary edema typically develops, frequently accompanied by disseminated intravascular coagulation. Note that these features differ from those observed with pulmonary embolism from a deep venous thrombosis; in fact, much of the morbidity and mortality in amniotic fluid embolism may stem from the biochemical activation of coagulation factors and components of the innate immune system by substances in the amniotic fluid, rather than the mechanical obstruction of pulmonary vessels by amniotic debris.

The underlying cause is the infusion of amniotic fluid or fetal tissue into the maternal circulation via a tear in the placental membranes or rupture of uterine veins. Classic findings at autopsy include the presence of squamous cells shed from fetal skin, lanugo hair, fat from vernix caseosa, and mucin derived from the fetal respiratory or gastrointestinal tract in the maternal pulmonary microvasculature (Fig. 4-17). Other findings include marked pulmonary edema, *diffuse alveolar damage* (Chapter 15), and the presence of fibrin thrombi in many vascular beds due to disseminated intravascular coagulation.

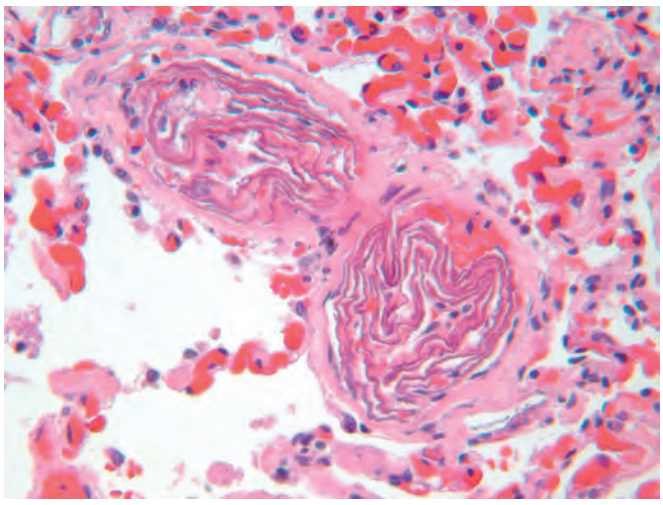

Figure 4-17 Amniotic fluid embolism. Two small pulmonary arterioles are packed with laminated swirls of fetal squamous cells. There is marked edema and congestion. Elsewhere the lung contained small organizing thrombi consistent with disseminated intravascular coagulation. (Courtesy Dr. Beth Schwartz, Baltimore, Md.)

KEY CONCEPTS

Embolism

- An embolus is a solid, liquid, or gaseous mass carried by the blood to a site distant from its origin; most are dislodged thrombi.
- Pulmonary emboli derive primarily from lower extremity deep vein thrombi; their effects depend mainly on the size of the embolus and the location in which it lodges. Consequences may include right-sided heart failure, pulmonary hemorrhage, pulmonary infarction, or sudden death.
- Systemic emboli derive primarily from cardiac mural or valvular thrombi, aortic aneurysms, or atherosclerotic plaques; whether an embolus causes tissue infarction depends on the site of embolization and the presence or absence of collateral circulation.

Infarction

An infarct is an area of ischemic necrosis caused by occlusion of either the arterial supply or the venous drainage. Tissue infarction is a common and extremely important cause of clinical illness. Roughly 40% of all deaths in the United States are caused by cardiovascular disease, and most of these are attributable to myocardial or cerebral infarction. Pulmonary infarction is also a common complication in many clinical settings, bowel infarction is frequently fatal, and ischemic necrosis of the extremities *(gangrene)* is a serious problem in the diabetic population.

Arterial thrombosis or arterial embolism underlies the vast majority of infarctions. Less common causes of arterial obstruction leading to infarction include local vasospasm, hemorrhage into an atheromatous plaque, or extrinsic vessel compression (e.g., by tumor). Other uncommon causes of tissue infarction include torsion of a vessel (e.g., in testicular torsion or bowel volvulus), traumatic vascular rupture, or vascular compromise by edema (e.g., *anterior compartment syndrome*) or by entrapment in a hernia sac. Although venous thrombosis can cause infarction, the more common outcome is just congestion; in this setting, bypass channels rapidly open and permit vascular outflow, which then improves arterial inflow. Infarcts caused by venous thrombosis are thus more likely in organs with a single efferent vein (e.g., testis and ovary).

MORPHOLOGY

Infarcts are classified according to color and the presence or absence of infection; they are either red (hemorrhagic) or white (anemic) and may be septic or bland.

- **Red infarcts** (Fig. 4-18*A*) occur (1) with venous occlusions (e.g., testicular torsion, Chapter 19), (2) in loose, spongy tissues (e.g., lung) where blood can collect in the infarcted zone, (3) in tissues with dual circulations (e.g., lung and small intestine) that allow blood to flow from an unobstructed parallel supply into a necrotic zone, (4) in tissues previously