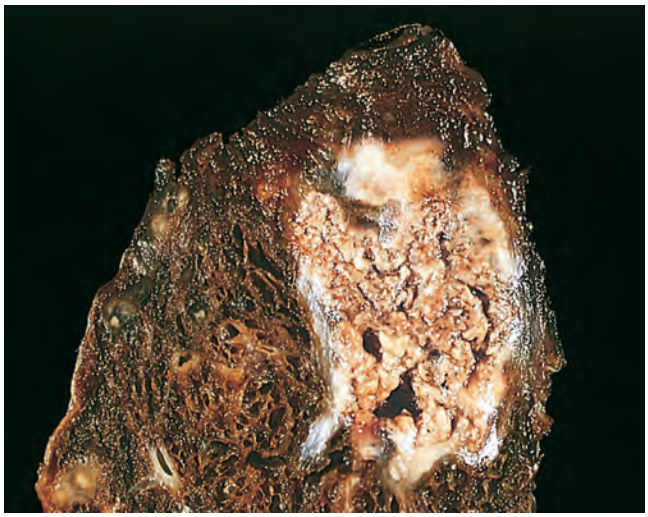

Figure 2-13 Caseous necrosis. Tuberculosis of the lung, with a large area of caseous necrosis containing yellow-white and cheesy debris.

resulting from release of activated pancreatic lipases into the substance of the pancreas and the peritoneal cavity. This occurs in the calamitous abdominal emergency known as acute pancreatitis (Chapter 19). In this disorder pancreatic enzymes leak out of acinar cells and liquefy the membranes of fat cells in the peritoneum. The released lipases split the triglyceride esters contained within fat cells. The fatty acids, so derived, combine with calcium to produce grossly visible chalky-white areas (fat saponification), which enable the surgeon and the pathologist to identify the lesions (Fig. 2-14). On histologic examination the necrosis takes the form of foci of shadowy outlines of necrotic fat cells, with basophilic calcium deposits, surrounded by an inflammatory reaction.

Fibrinoid necrosis is a special form of necrosis usually seen in immune reactions involving blood vessels. This pattern of necrosis typically occurs when complexes of antigens and antibodies are deposited in the walls of arteries. Deposits of these "immune complexes," together with fibrin that has leaked out of vessels, result in a bright pink and amorphous appearance in H&E stains, called "fibrinoid" (fibrin-like) by pathologists (Fig. 2-15). The immunologically mediated vasculitis syndromes in which this type of necrosis is seen are described in Chapter 11.

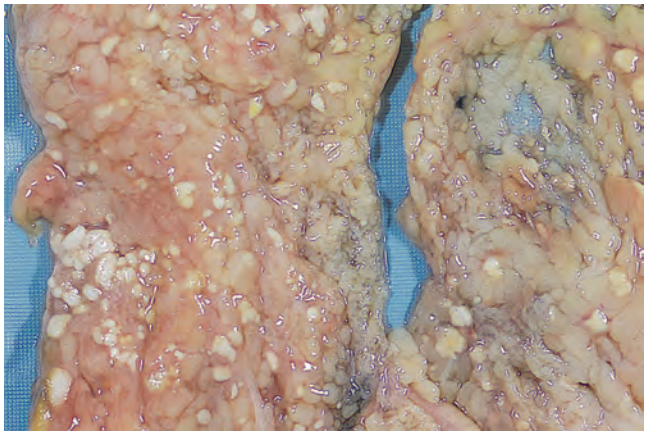

Figure 2-14 Fat necrosis. The areas of white chalky deposits represent foci of fat necrosis with calcium soap formation (saponification) at sites of lipid breakdown in the mesentery.

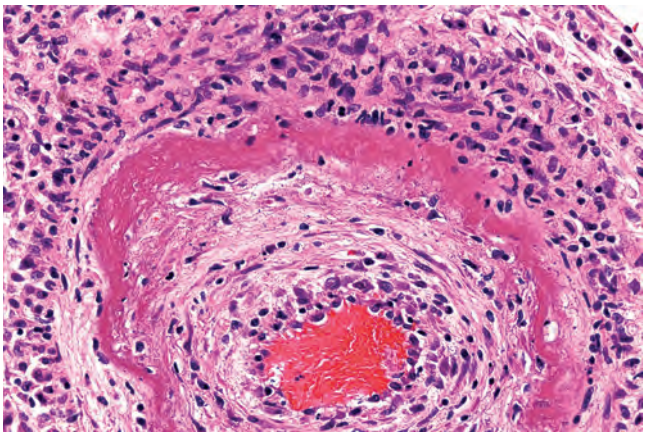

Figure 2-15 Fibrinoid necrosis in an artery. The wall of the artery shows a circumferential bright pink area of necrosis with inflammation (neutrophils with dark nuclei).

Ultimately, in the living patient most necrotic cells and their contents disappear due to enzymatic digestion and phagocytosis of the debris by leukocytes. If necrotic cells and cellular debris are not promptly destroyed and reabsorbed, they provide a nidus for the deposition of calcium salts and other minerals and thus tend to become calcified. This phenomenon, called *dystrophic calcification*, is considered later in the chapter.

KEY CONCEPTS

Morphologic Alterations in Injured Cells and Tissues

- **Reversible cell injury:** Cellular swelling, fatty change, plasma membrane blebbing and loss of microvilli, mitochondrial swelling, dilation of the ER, eosinophilia (due to decreased cytoplasmic RNA)
- **Necrosis:** Increased eosinophilia; nuclear shrinkage, fragmentation, and dissolution; breakdown of plasma membrane and organellar membranes; abundant myelin figures; leakage and enzymatic digestion of cellular contents
- **Patterns of tissue necrosis:** Under different conditions, necrosis in tissues may assume specific patterns: coagulative, liquefactive, gangrenous, caseous, fat, and fibrinoid

Mechanisms of Cell Injury

The discussion of the cellular pathology of cell injury and necrosis sets the stage for a consideration of the mechanisms and biochemical pathways of cell injury. The molecular pathways that lead to cell injury are complex and are best understood in the context of normal cell biology (Chapter 1). There are, however, several principles that are relevant to most forms of cell injury:

- **The cellular response to injurious stimuli depends on the nature of the injury, its duration, and its severity**. Small doses of a chemical toxin or brief periods of ischemia may induce reversible injury, whereas large doses of the same toxin or more prolonged ischemia might