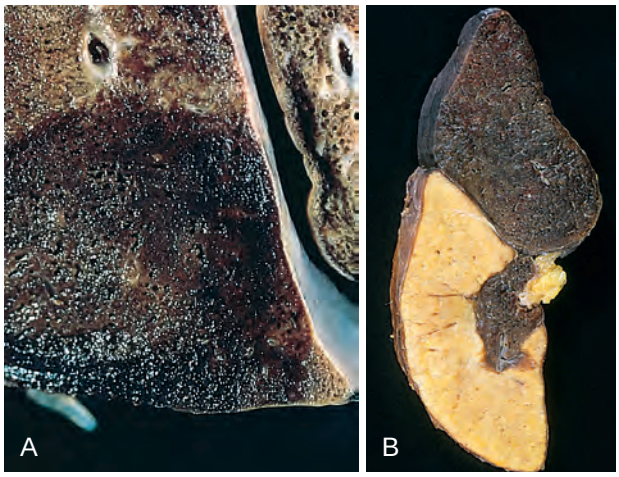


**Figure 4-18** Red and white infarcts. **A,** Hemorrhagic, roughly wedge-shaped pulmonary *red infarct*. **B,** Sharply demarcated *white infarct* in the spleen.

congested by sluggish venous outflow, and (5) when flow is reestablished to a site of previous arterial occlusion and necrosis (e.g., following angioplasty of an arterial obstruction).

- **White infarcts** (Fig. 4-18*B*) occur with arterial occlusions in solid organs with end-arterial circulation (e.g., heart, spleen, and kidney), and where tissue density limits the seepage of blood from adjoining capillary beds into the necrotic area.

Infarcts tend to be wedge-shaped, with the occluded vessel at the apex and the periphery of the organ forming the base (Fig. 4-18); when the base is a serosal surface there may be an overlying fibrinous exudate resulting from an acute inflammatory response to mediators release from injured and necrotic cells. Fresh infarcts are poorly defined and slightly hemorrhagic, but over a few days the margins tend to become better defined by a narrow rim of congestion attributable to inflammation. With further passage of time, infarcts resulting from arterial occlusions in organs without a dual blood supply typically become progressively paler and more sharply defined (Fig. 4-18*B*). In comparison, in the lung hemorrhagic infarcts are the rule (Fig. 4-18*A*). Extravasated red cells in hemorrhagic infarcts are phagocytosed by macrophages, which convert heme iron into hemosiderin; small amounts do not grossly impart any appreciable color to the tissue, but extensive hemorrhage can leave a firm, brown hemosiderin-rich residuum.

The dominant histologic characteristic of infarction is **ischemic coagulative necrosis** (Chapter 2). Importantly, if the vascular occlusion has occurred shortly (minutes to hours) before the death of the person, histologic changes may be absent; it takes 4 to 12 hours for the dead tissue to show microscopic evidence of frank necrosis. Acute inflammation is present along the margins of infarcts within a few hours and is usually well defined within 1 to 2 days. Eventually a reparative response begins in the preserved margins (Chapter 3). In stable or labile tissues, parenchymal regeneration can occur at the periphery where underlying stromal architecture is preserved. However, most infarcts are ultimately replaced by *scar* (Fig. 4-19). The brain is an exception to these generalizations, in that central nervous system infarction results in **liquefactive necrosis** (Chapter 2).

**Septic infarctions** occur when infected cardiac valve vegetations embolize or when microbes seed necrotic tissue. In these cases the infarct is converted into an *abscess*, with a correspondingly greater inflammatory response (Chapter 3). The eventual sequence of organization, however, follows the pattern already described.

***Factors That Influence Development of an Infarct.*** A vascular occlusion can cause effects ranging from virtually nothing to tissue dysfunction and necrosis sufficient to result in death. The variables that influence the outcome of vascular occlusion are the following:

- *Anatomy of the vascular supply.* The availability of an alternative blood supply is the most important determinant of whether vessel occlusion will cause tissue damage. As mentioned, the lungs have a dual pulmonary and bronchial artery blood supply that protects against thromboembolism-induced infarction. Similarly, the liver, with its dual hepatic artery and portal vein circulation, and the hand and forearm, with their dual radial and ulnar arterial supply, are all relatively resistant to infarction. In contrast, renal and splenic circulations are end-arterial, and vascular obstruction generally causes tissue death.
- *Rate of occlusion.* Slowly developing occlusions are less likely to cause infarction, because they provide time for development of collateral pathways of perfusion. For example, small interarteriolar anastomoses—normally with minimal functional flow—interconnect the three major coronary arteries in the heart. If one of the coronaries is occluded slowly (i.e., by an encroaching atherosclerotic plaque), flow within this *collateral circulation* may increase sufficiently to prevent infarction, even though the larger coronary artery is eventually occluded.
- *Tissue vulnerability to hypoxia.* Neurons undergo irreversible damage when deprived of their blood supply for only 3 to 4 minutes. Myocardial cells, although hardier than neurons, are also quite sensitive and die after only 20 to 30 minutes of ischemia (although, as mentioned, changes in the appearance of the dead cells take 4-12 hours to develop). In contrast, fibroblasts within myocardium remain viable even after many hours of ischemia (Chapter 12).

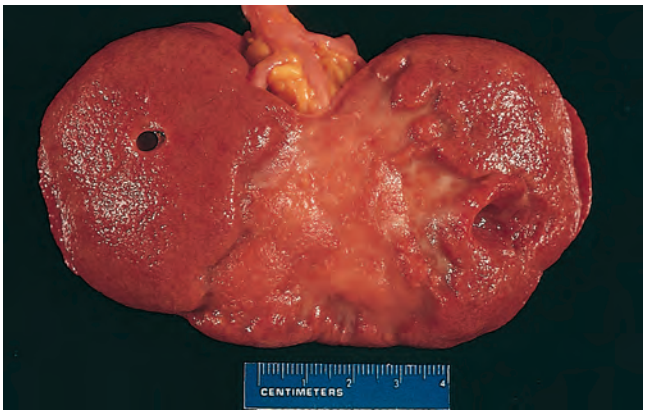


**Figure 4-19** Remote kidney infarct replaced by a large fibrotic scar.