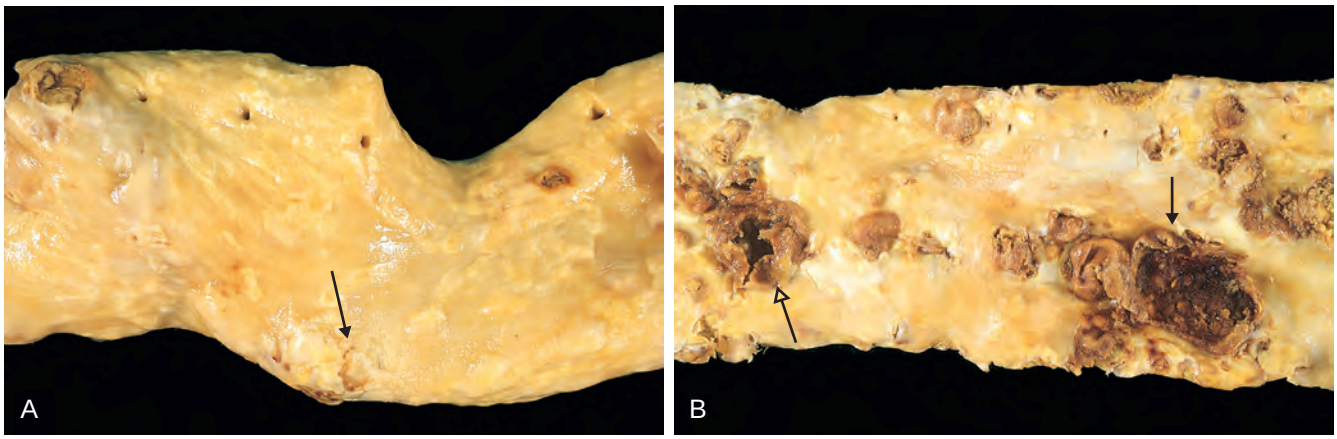

Figure 11-13 Gross views of atherosclerosis in the aorta. **A,** Mild atherosclerosis composed of fibrous plaques, one of which is denoted by the arrow. **B,** Severe disease with diffuse and complicated lesions including an ulcerated plaque *(open arrow),* and a lesion with overlying thrombus *(closed arrow).*

smoke toxins, elevated LDL, and hyperglycemia—is attributable to the vagaries of vascular hemodynamics. Local flow disturbances, such as turbulence at branch points, make certain portions of a vessel wall more susceptible to plaque formation. Although focal and sparsely distributed at first, with time atherosclerotic lesions can become larger, more numerous, and more broadly distributed. Moreover, in any given vessel, lesions at various stages often coexist.

In descending order, **the most extensively involved vessels are the lower abdominal aorta, the coronary arteries, the popliteal arteries, the internal carotid arteries, and the vessels of the circle of Willis.** In humans, the abdominal aorta is typically involved to a much greater degree than the thoracic aorta. Vessels of the upper extremities are usually spared, as are the mesenteric and renal arteries, except at their ostia. Although most individuals tend to have a consistent degree of atherosclerotic burden in the affected vasculature, severity of disease in one arterial distribution does not always predict its severity in another.

Atherosclerotic plaques have three principal components: (1) smooth muscle cells, macrophages, and T cells; (2) extracellular matrix, including collagen, elastic fibers, and proteoglycans; and (3) intracellular and extracellular lipid (Fig. 11-14). These components occur in varying proportions and configurations in different lesions. Typically, there is a superficial fibrous cap composed of smooth muscle cells and relatively dense collagen. Beneath and to the side of the cap (the "shoulder") is a more cellular area containing macrophages, T cells, and smooth muscle cells. Deep to the fibrous cap is a necrotic core, containing lipid (primarily cholesterol and cholesterol esters), debris from dead cells, foam cells (lipid-laden macrophages and smooth muscle cells), fibrin, variably organized thrombus, and other plasma proteins; the cholesterol content is frequently present as crystalline aggregates that are washed out during routine tissue processing and leave behind only empty "clefts." The periphery of the lesions demonstrate **neovascularization** (proliferating small blood vessels; Fig. 11-14*C*). Most atheromas contain abundant lipid, but some

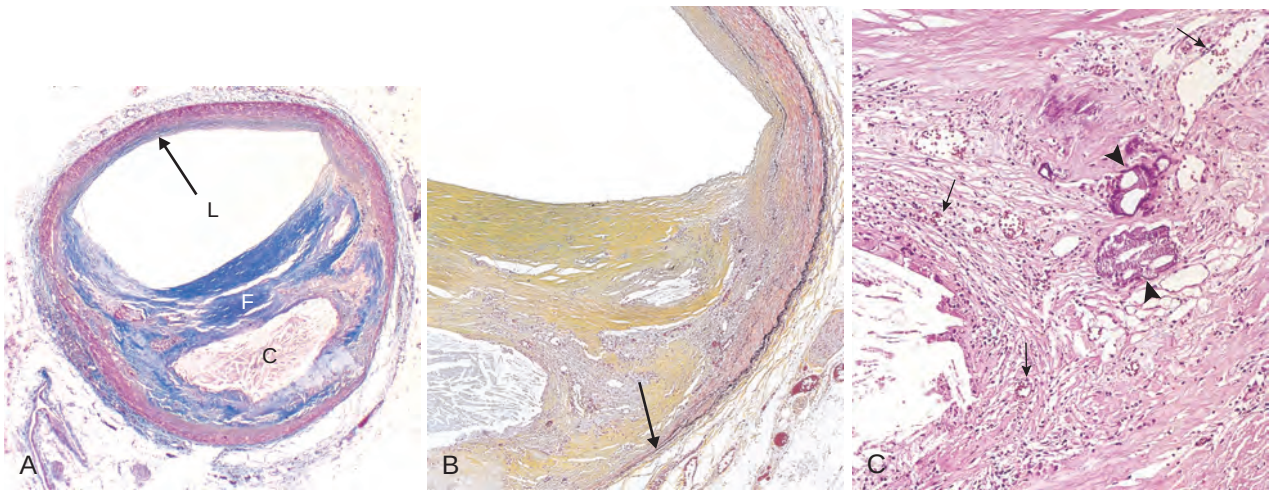

Figure 11-14 Histologic features of atheromatous plaque in the coronary artery. **A,** Overall architecture demonstrating fibrous cap (F) and a central necrotic core (C) containing cholesterol and other lipids. The lumen (L) has been moderately compromised. Note that a segment of the wall is plaque free *(arrow)*; the lesion is therefore "eccentric." In this section, collagen has been stained blue (Masson trichrome stain). **B,** Higher-power photograph of a section of the plaque shown in **A,** stained for elastin (black), demonstrating that the internal and external elastic laminae are attenuated and the media of the artery is thinned under the most advanced plaque *(arrow).* **C**, Higher magnification photomicrograph at the junction of the fibrous cap and core, showing scattered inflammatory cells, calcification *(arrowhead),* and neovascularization *(small arrows).*