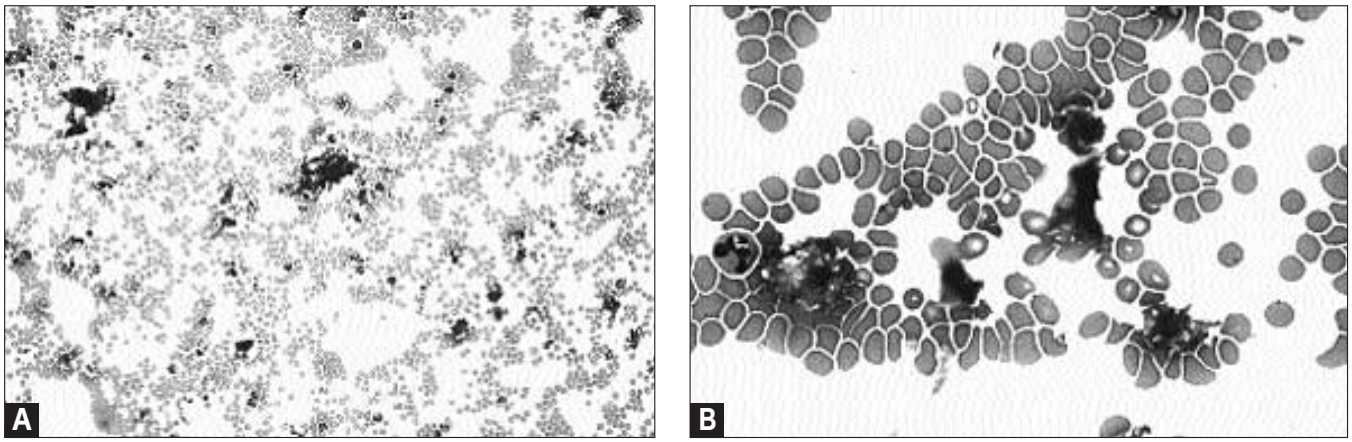

FIGURE 9-6 (see Color Plate)

A fine-needle aspirate in renal infarction. **A**, Low magnification shows many degenerating cells with a "dirty background" containing cellular debris and scattered neutrophils. Compare to acute tubular necrosis, which has only scattered degenerated or necrotic cells without the extensive necrosis and cell debris. Neutrophils may be numerous if the edge of an infarct is aspirated (May-Grunwald Giemsa, original magnification × 40). **B**, Diffusely degenerated and necrotic cells with condensed and disrupted cytoplasm and pyknotic nuclei, and an adjacent neutrophil. No significant numbers of viable tubule epithelial cells remain (May-Grunwald Giemsa, original magnification × 160).

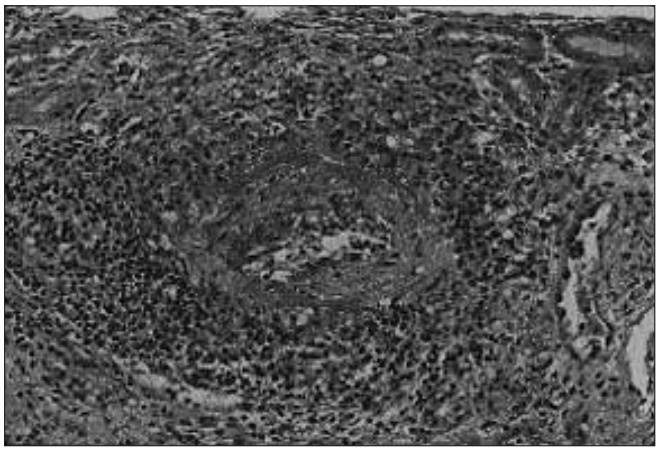

FIGURE 9-7 (see Color Plate)

A small artery with severe inflammation in a patient with a small vessel vasculitis. The wall of the vessel is infiltrated by lymphocytes, plasma cells, and eosinophils (hematoxylin and eosin, original magnification × 250). The patient was p-ANCA positive. ANCA may play a pathogenic role in the vasculitis process [4]. Vasculitis in the kidney is often part of a systemic syndrome, but may occur as an apparently renal-limited process.

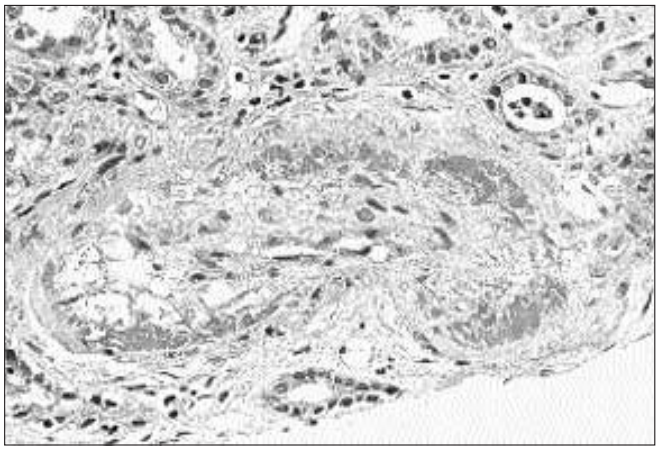

FIGURE 9-8 (see Color Plate)

Microangiopathic changes in a small artery, with endothelial activation, evidenced by the large endothelial cells with hyperchromatic nuclei and vacuolization. There is intimal edema with some cell proliferation, and a prominent band of fibrinoid necrosis is seen; the latter appears dark red-pink on this hematoxylin-eosin stain, and represents insudation of fibrin and plasma proteins into the wall of the injured vessel (original magnification × 250). The differential diagnosis includes hemolytic uremic syndrome, thrombotic thrombocytopenic purpura, malignant hypertension, scleroderma, and drug toxicity, the latter due most commonly to mitomycin C or cyclosporine/FK506 [5].

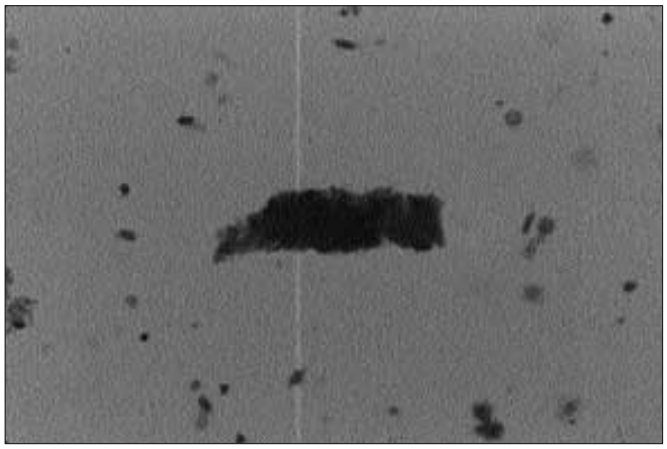

FIGURE 9-9 (see Color Plate)
A cast of necrotic tubular cells in urine sediment (Papanicolaou stain, original magnification × 400). The most likely causes of damage to the renal tubules with such findings in the urinary sediment are severe ischemia/infarction, or tubular necrosis due to exposure to toxins which injure the renal tubules. The latter include antibiotics, including aminoglycosides and cephalosporins, and chemotherapeutic agents.

Interstitial Disease

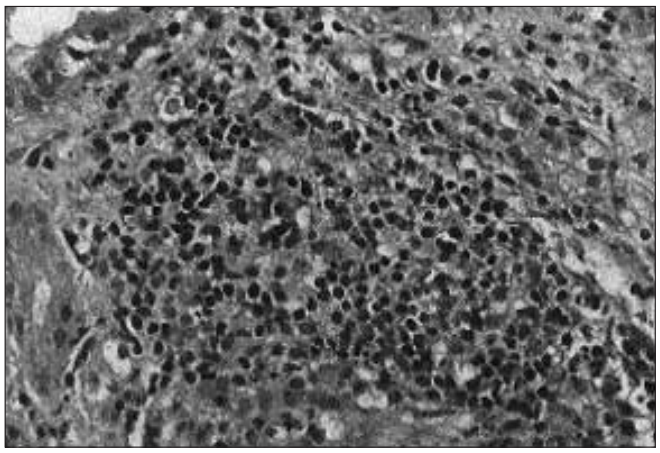

FIGURE 9-10 (see Color Plate)
Interstitial nephritis with edema and a mononuclear inflammatory infiltrate. Eosinophils in the infiltrate suggest a possible hypersensitivity reaction (hematoxylin and eosin, original magnification ×400). Drugs are the most common cause of such a reaction, which often presents with acute renal failure [6]. Inflammatory cells and cell casts may be seen in the urine sediment in these cases, as inflammatory cells infiltrate the tubular epithelium.

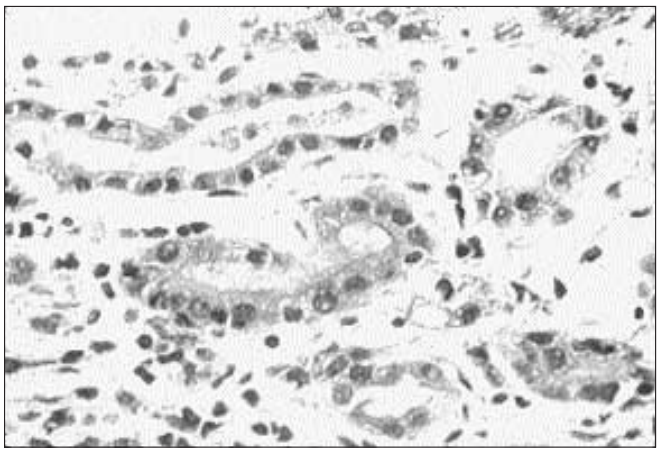

FIGURE 9-11 (see Color Plate)
Tubulitis, with infiltration of mononuclear cells into the tubular epithelium (hematoxylin and eosin, original magnification × 400). There is a mononuclear infiltrate and edema in the surrounding interstitium. Tubule cells may show evidence of lethal or sublethal injury as the inflammatory cells release damaging enzymes. Tubulitis is often seen in interstitial nephritis especially if the targets of the inflammatory reaction are tubular cell antigens or antigens deposited around the tubules. Immunofluorescence may reveal granular or linear deposits of immunoglobulin and complement around the tubules.

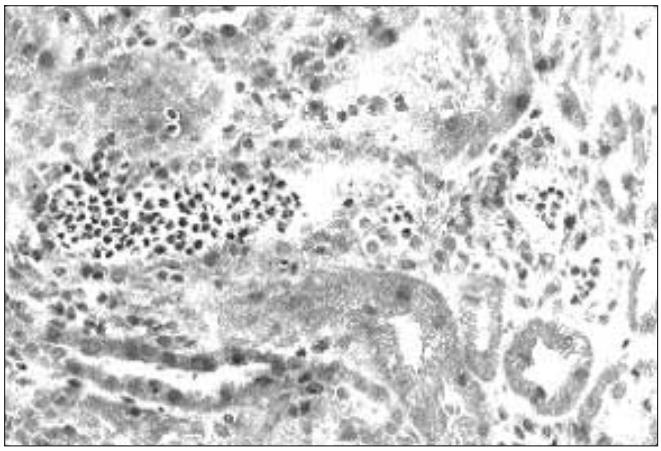

FIGURE 9-12 (see Color Plate)

Polymorphonuclear leukocytes forming a cast in a cortical tubule (hematoxylin and eosin, original magnification × 400). Note edema and inflammation in adjacent interstitium. These intratubular cells are highly suggestive of acute infection, and may be seen in distal as well as proximal nephron as part of an ascending infection. Intratubular PML may also be seen in vasculitis and other necrotizing glomerular processes, in which these cells escape across damaged areas of the inflamed glomerular tuft.

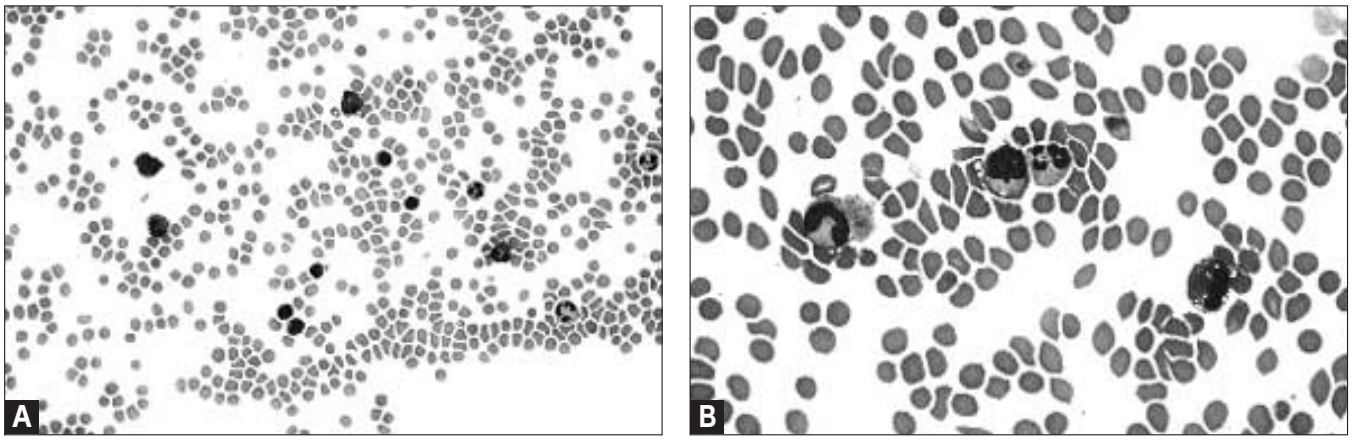

FIGURE 9-13 (see Color Plate)

Fine-needle aspirate of acute infectious interstitial nephritis (acute pyelonephritis). A 25-gauge needle attached to a 10-cc syringe was utilized to withdraw the aspirate into 4 cc of RPMI-based medium. The specimen was then cytocentrifuged and stained with May-Grunwald Giemsa. **A**, The renal aspirate contains large numbers of intrarenal neutrophils, which are focally undergoing degenerative changes with cytoplasmic vacuolization and nuclear breakdown. In bacterial infection there are many infiltrating neutrophils and there may be associated necrosis of tubule epithelial cells (original magnification × 80). **B**, A neutrophil contains phagocytosed bacteria within the cytoplasm; bacteria stain with Giemsa, so are readily detectable in this setting. Adjacent tubule epithelial cells have cytoplasmic granules but do not phagocytize bacteria (original magnification × 160).

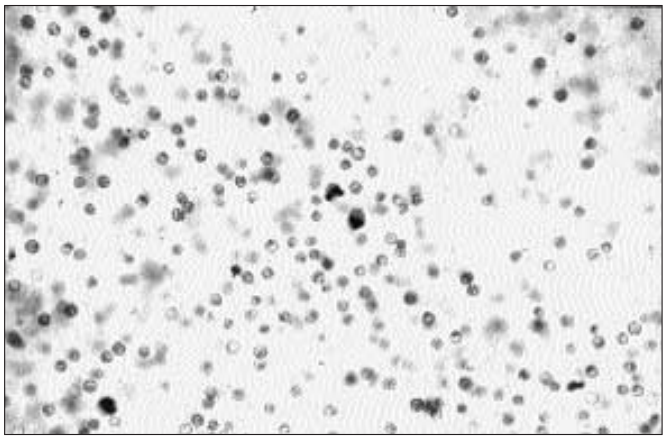

FIGURE 9-14 (see Color Plate)

Numerous polymorphonuclear leukocytes (PML) in the urine sediment of a patient with acute pyelonephritis (hematoxylin and eosin, original magnification × 400). Some red blood cells and tubular cells are seen in the background of this cytospin preparation. PML may be found in the urine with acute infection of the lower urinary tract as well, or as a contaminant from vaginal secretions in females. PML casts, on the other hand, are evidence that the cells are from the kidney.