Acute Renal Failure

**FIGURE 9-23 (see Color Plate)**
Urine sediment from a patient with acute tubular injury showing tubular cells and cell casts (Papanicolaou stain, original magnification × 250). Many of these cells are morphologically intact, even by electron microscopy. Studies have shown that a significant percentage of the cells shed into the urine may exclude vital dyes, and may even grow when placed in culture, indicating that they remain viable. Such cells clearly detached from tubular basement membrane as a manifestation of sub-lethal injury [7].

**FIGURE 9-24 (see Color Plate)**
Myoglobin casts in the tubules of a patient who abused cocaine. **A**, Hematoxylin and eosin stained casts have a dark red, coarsely granular appearance (original magnification × 250). **B**, Immunoperoxidase stain for myoglobin confirms positive staining in the casts (original magnification × 250). These casts may obstruct the nephron, especially with dehydration and low tubular fluid flow rates. Rhabdomyolysis with formation of intrarenal myoglobin casts may also occur with severe trauma, crush injury, or extreme exercise.
FIGURE 9-25 (see Color Plate)
Apoptosis of tubular cells following tubular cell injury. Note the shrunken cells with condensed nuclei and cytoplasm in the central tubule. The patient had presumed ischemic injury (hematoxylin and eosin, original magnification × 400). The role of apoptosis in injury to the renal tubule remains to be defined. The process may be difficult to quantitate, since apoptotic cells may rapidly disintegrate. In experimental models, the degree of apoptosis versus coagulative necrosis occurring following injury is related to the severity and duration of injury, with milder injury showing more apoptosis [9].

FIGURE 9-26
Apoptosis-schematic of histologic changes in tubular epithelium. The process begins with condensation of the cytoplasm and of the nucleus, a process which involves endonucleases, which digest the DNA into ladder-like fragments characteristic of this process. The cell disintegrates into discrete membrane-bound fragments, so-called “apoptotic bodies.” These fragments may be rapidly extruded into the tubular lumen or phagocytosed by neighboring epithelial cells or inflammatory cells. (Modified from Arends, et al. [10]; with permission.)
Acute Renal Failure

FIGURE 9-27
A schematic showing the relationship between morphologic and functional changes with injury to the renal tubule due to ischemia or nephrotoxins. Morphologic changes are shown in italics.

Histology reflects the altered hemodynamics, epithelial derangements, and obstruction which contribute to loss of renal function. (Modified from Racusen [11]; with permission.)

References