Reflux nephropathy, or renal parenchymal scarring associated with vesicoureteral reflux (VUR), is an important cause of renal failure. Some studies have shown that in up to 10% of adults and 30% of children requiring renal replacement therapy for end-stage renal disease, reflux nephropathy is the cause of the renal failure. Reflux nephropathy is thought to result from the combination of VUR of infected urine into the kidney by way of an incompetent ureterovesical junction valve mechanism and intrarenal reflux. Acute inflammatory responses to the infection result in renal parenchymal damage and subsequent renal scarring. Loss of functioning renal mass prompt compensatory changes in renal hemodynamics that, over time, are maladaptive and result in glomerular injury and sclerosis.

Clinically, reflux nephropathy may cause hypertension, proteinuria, and decreased renal function when the scarring is extensive. The identification of VUR raises the theoretic possibility of preventing reflux nephropathy. The inheritance pattern of VUR clearly is suggestive of a strong genetic influence. Familial studies of VUR are consistent with autosomal dominant transmission, and linkage to the major histocompatibility genes has been reported. Identification of infants with reflux detected on the basis of abnormalities seen on prenatal ultrasound examinations before urinary tract infection occurs may provide an opportunity for prevention of reflux nephropathy. In persons with VUR detected at the time of diagnosis of a urinary tract infection, avoidance of further infections may prevent renal injury. Nevertheless, the situation is far from clear. Most children with reflux nephropathy already have renal scars demonstrable at the time of the urinary tract infection that prompts the diagnosis of VUR. Most children found to have VUR do not develop further renal scarring after diagnosis, even after subsequent urinary tract infections. Other children may develop renal scars in the absence of further urinary tract infections. The best treatment of
Intraluminal
Calculus, clot, renal papilla, fungus ball

Intrinsic
Congenital:
Calyceal infundibular obstruction
Ureteropelvic junction obstruction
Ureteral stricture or valves
Posterior urethral valves
Anterior urethral valves
Urethral stricture
Meatal stenosis
Prune-belly syndrome
Neoplastic:
Carcinoma of the renal pelvis, ureter, or bladder
Polyps

Extrinsic
Congenital (aberrant vessels):
Congenital hydrocalycosis
Ureteropelvic junction obstruction
Retrocecal ureter
Neoplastic tumors:
Benign tumors:
Benign prostate hypertrophy
Pelvic lipomatosis
Cysts
Primary retroperitoneal tumors:
Mesodermal origin (e.g., sarcoma)
Neurogenic origin (e.g., neurofibroma)
Embryonic remnant (e.g., teratoma)
Retroperitoneal extension of pelvic or abdominal tumors:
Uterus, cervix
Bladder, prostate
Rectum, sigmoid colon
Metastatic tumor:
Lymphoma
Inflammatory:
Retroperitoneal fibrosis
Inflammatory bowel disease
Diverticulitis
Infection or abscess
Gynecologic:
Pregnancy
Uterine prolapse
Surgical disruption or ligation

Functional
Neurogenic bladder
Drugs (anticholinergics, antidepressants, calcium channel blockers)

FIGURE 8-1
Obstructive nephropathy is responsible for end-stage renal failure in approximately 4% of persons. Obstruction to the flow of urine can occur anywhere in the urinary tract. Obstruction can be caused by luminal bodies; mural defects; extrinsic compression by vascular, neoplastic, inflammatory, or other processes; or dysfunction of the autonomic nervous system or smooth muscle of the urinary tract. The functional and clinical consequences of urinary tract obstruction depend on the developmental stage of the kidney at the time the obstruction occurs, severity of the obstruction, and whether the obstruction affects one or both kidneys.
Anatomy of Vesicoureteric Reflux

**Figure 8-2**
Anatomy of the ureterovesical junction. The ureterovesical junction permits free antegrade urine flow from the upper urinary tract into the bladder and prevents retrograde urinary reflux from the bladder into the ureter and kidney. Passive compression of the distal submucosal portion of the ureter against the detrusor muscle as a result of bladder filling impedes vesicoureteral reflux (VUR). An active mechanism preventing reflux also has been proposed in which contraction of longitudinally arranged distal ureteral muscle fibers occludes the ureteral lumen, impeding retrograde urine flow [1–3]. (From Politano [4]; with permission.)

**Figure 8-3**
Tissue sagittal sections (upper panels) and cystoscopic appearances (lower panels) of the ureterovesical junction illustrating varying submucosal tunnel lengths. The length of the submucosal segment of the distal ureter is an important factor in determining the effectiveness of the ureteral valvular mechanism in preventing vesicoureteral reflux (VUR). In children without VUR, the ratio of tunnel length to ureteral diameter is significantly greater than in children with VUR [5,6]. (From Kramer [7]; with permission.)

**Figure 8-4**
Simple and compound papillae are illustrated [8,9]. Two types of renal papillae have been identified. Simple papillae are the most common type. They have slitlike papillary duct openings on their convex surface. These papillae are compressed by increases in pelvic pressure, preventing urine from entering the papillary ducts (intrarenal reflux). Compound papillae are formed by the fusion of two or more simple papillae. In compound papillae, some ducts open onto a flat or concave surface at less oblique angles. Increased intrapelvic pressure may permit intrarenal reflux. Compound papillae usually are found in the renal poles.