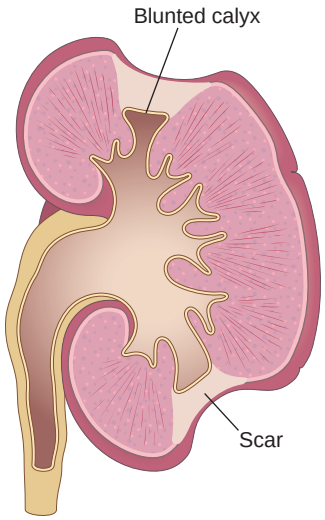

Figure 20-31 Typical coarse scars of chronic pyelonephritis associated with vesicoureteral reflux. The scars are usually polar and are associated with underlying blunted calyces.

Chronic pyelonephritis can be divided into two forms:

- *Reflux nephropathy.* This is by far the more common form of chronic pyelonephritic scarring. Reflux nephropathy occurs early in childhood as a result of superimposition of a urinary infection on congenital vesicoureteral reflux and intrarenal reflux. Reflux may be unilateral or bilateral; thus, the continuous renal damage may cause scarring and atrophy of one kidney or involve both, leading to renal insufficiency. Vesicoureteral reflux occasionally causes renal damage in the absence of infection (sterile reflux), but only when obstruction is severe.
- *Chronic obstructive pyelonephritis.* We have seen that obstruction predisposes the kidney to infection. Recurrent infections superimposed on diffuse or localized obstructive lesions lead to repeated bouts of renal inflammation and scarring, resulting in chronic pyelonephritis. In this condition, the effects of obstruction contribute to the parenchymal atrophy; indeed, it is sometimes difficult to differentiate the effects of bacterial infection from those of obstruction alone. The disease can be bilateral, as with posterior urethral valves, resulting in renal insufficiency unless the anomaly is corrected, or unilateral, as occurs with calculi and unilateral obstructive anomalies of the ureter.

MORPHOLOGY

The characteristic changes of chronic pyelonephritis are seen on gross examination (Figs. 20-31 and 20-32*A*). The kidneys usually are irregularly scarred; if bilateral, the involvement is asymmetric. In contrast, both kidneys in chronic glomerulonephritis are diffusely and symmetrically scarred. The hallmarks of chronic pyelonephritis are **coarse, discrete, corticomedullary scars overlying dilated, blunted, or deformed calyces, and flattening of the papillae** (Fig. 20-33*B*). The scars vary from one to several and most are in the upper and lower poles, consistent with the frequency of reflux in these sites.

The microscopic changes involve predominantly tubules and interstitium. The tubules show atrophy in some areas and hypertrophy or dilation in others. Dilated tubules with flattened epithelium may be filled with casts resembling thyroid colloid (thyroidization). There are varying degrees of chronic interstitial inflammation and fibrosis in the cortex and medulla. Arcuate and interlobular vessels demonstrate obliterative intimal sclerosis in the scarred areas; and in the presence of hypertension, hyaline arteriolosclerosis is seen in the entire kidney. There is often fibrosis around the calyceal epithelium as well as a marked chronic inflammatory infiltrate. Glomeruli may appear normal except for a variety of ischemic changes, including periglomerular fibrosis, fibrous obliteration and secondary changes related to hypertension. Individuals with chronic pyelonephritis and reflux nephropathy who develop proteinuria in advanced stages show secondary focal segmental glomerulosclerosis, as described later.

Xanthogranulomatous pyelonephritis is a relatively rare form of chronic pyelonephritis characterized by accumulation of foamy macrophages intermingled with plasma cells, lymphocytes, polymorphonuclear leukocytes, and occasional giant cells. Often associated with *Proteus* infections and obstruction, the lesions sometimes produce large, yellowish orange nodules that may be grossly confused with renal cell carcinoma.

Clinical Features. Chronic obstructive pyelonephritis may have a silent onset or present with manifestations of

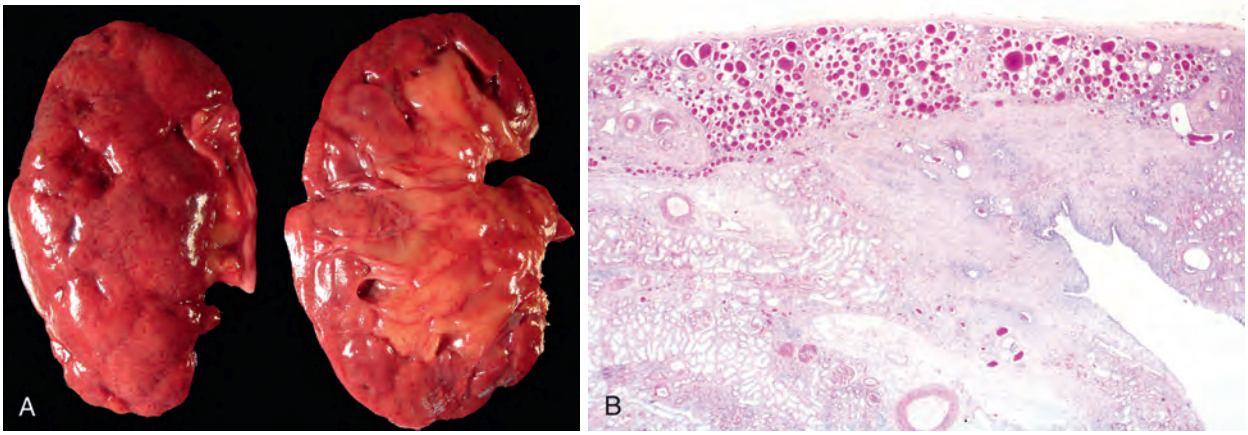

Figure 20-32 **A,** Chronic pyelonephritis. The surface *(left)* is irregularly scarred. The cut section *(right)* reveals blunting and loss of several papillae. **B,** Low-power view showing a corticomedullary renal scar with an underlying dilated deformed calyx. Note the thyroidization of tubules in the cortex.