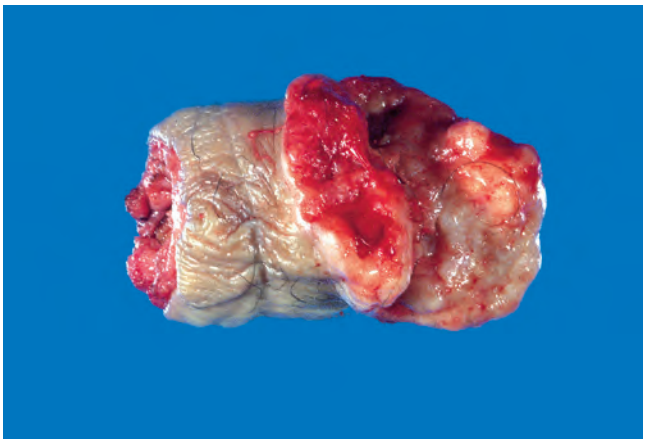

Figure 21-19 Carcinoma of the penis. The glans penis is deformed by a firm, ulcerated, infiltrative mass.

MORPHOLOGY

Squamous cell carcinoma of the penis usually begins on the glans or inner surface of the prepuce near the coronal sulcus. Two macroscopic patterns are seen—papillary and flat. The papillary lesions simulate condylomata acuminata and may produce a cauliflower-like fungating mass. Flat lesions appear as areas of epithelial thickening accompanied by graying and fissuring of the mucosal surface. With progression, an ulcerated papule develops (Fig. 21-19). Histologically, both the papillary and the flat lesions are squamous cell carcinomas with varying degrees of differentiation. **Verrucous carcinoma** is an exophytic well-differentiated variant of squamous cell carcinoma that are locally invasive, but rarely metastasize. Other, less common, subtypes of penile squamous carcinoma include basaloid, warty, and papillary variants.

Clinical Features. Invasive squamous cell carcinoma of the penis is a slowly growing, locally invasive lesion that often has been present for a year or more before it is brought to medical attention. The lesions are nonpainful until they undergo secondary ulceration and infection. Metastases to inguinal lymph nodes may occur early in its course, but widespread dissemination is extremely uncommon until the lesion is far advanced. Clinical assessment of regional lymph node involvement is notoriously inaccurate; only 50% of enlarged inguinal nodes detected in men with penile squamous cell carcinoma contain cancer, with the remainder showing only reactive lymphoid hyperplasia when examined histologically. The prognosis is related to the stage of the tumor. Without spread to lymph nodes, the 5-year survival rate is 66%, whereas metastasis to the lymph nodes carries a grim 27% 5-year survival.

KEY CONCEPTS

Lesions of the Penis

- Squamous cell carcinoma and its precursor lesions are the most important penile lesions. Many are associated with HPV infection.
- Squamous cell carcinoma occurs on the glans or shaft of the penis as an ulcerated infiltrative lesion that may spread to inguinal nodes and infrequently to distant sites. Most cases occur in uncircumcised males.
- Other important penile disorders include congenital abnormalities involving the position of the urethra (epispadias, hypospadias) and inflammatory disorders (balanitis, phimosis).

Testis and Epididymis

Distinct pathologic conditions affect the testis and epididymis. In the epididymis, the most important and frequent conditions are inflammatory diseases, whereas in the testis the major lesions are tumors.

Congenital Anomalies

With the exception of undescended testes (cryptorchidism), congenital anomalies are extremely rare and include absence of one or both testes and fusion of the testes (so-called *synorchism*).

Cryptorchidism

Cryptorchidism is a complete or partial failure of the intra-abdominal testes to descend into the scrotal sac and is associated with testicular dysfunction and an increased risk of testicular cancer. It is found in approximately 1% of 1-year-old boys. It usually occurs as an isolated anomaly but may be accompanied by other malformations of the genitourinary tract, such as hypospadias.

Testicular descent occurs in two morphologically and hormonally distinct phases. During the first transabdominal, phase, the testis comes to lie within the lower abdomen or brim of the pelvis. This phase is believed to be controlled by a hormone called *müllerian-inhibiting substance*. In the second inguinoscrotal, phase, the testes descend through the inguinal canal into the scrotal sac. This phase is androgen-dependent and is possibly mediated by androgen-induced release of calcitonin gene-related peptide from the genitofemoral nerve. Although testes may arrest anywhere along their pathway of descent, the most common site is in the inguinal canal; arrest within the abdomen is uncommon, accounting for approximately 5% to 10% of cases. Even though testicular descent is controlled by hormonal factors, cryptorchidism is only rarely associated with a well-defined hormonal disorder.

MORPHOLOGY

Cryptorchidism is unilateral in most cases, being bilateral in 25% of patients. Histologic changes in the malpositioned testis begin as early as 2 years of age. They are characterized by **arrested germ cell development** associated with **marked hyalinization and thickening of the basement membrane** of the spermatic tubules (Fig. 21-20). Eventually the tubules appear as dense cords of hyaline connective tissue outlined by prominent basement membranes. There is concomitant increase in interstitial stroma. Because Leydig cells are spared, they appear to be prominent. As might be expected with