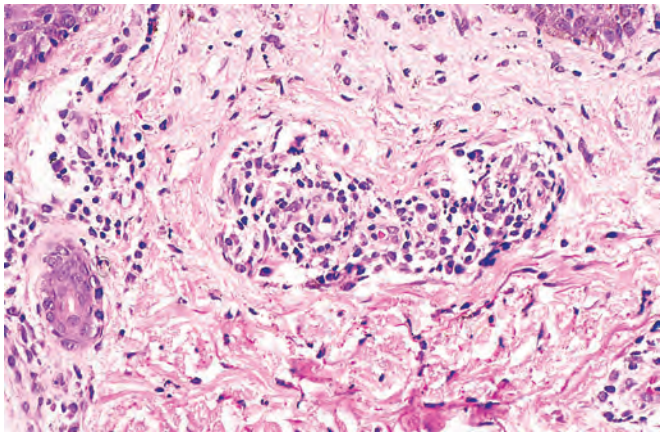

Figure 8-5 Secondary syphilis in the dermis with perivascular lymphoplasmacytic infiltrate and endothelial proliferation.

mononuclear inflammation usually evoked by infectious agents that resist eradication and are capable of stimulating strong T cell–mediated immunity (e.g., *M. tuberculosis, Histoplasma capsulatum*, schistosome eggs). Granulomatous inflammation is characterized by accumulation and aggregation of activated macrophages called "epithelioid" cells, some of which may fuse to form giant cells. Granulomas may contain a central area of caseous necrosis (see Chapter 3 and "Tuberculosis" in this chapter).

Cytopathic-Cytoproliferative Reaction

These reactions are usually produced by viruses. The lesions are characterized by cell necrosis or cellular proliferation, usually with sparse inflammatory cells. Some viruses replicate within cells and make viral aggregates that are visible as inclusion bodies (e.g., herpesviruses or adenovirus) or induce cells to fuse and form multinucleated cells called polykaryons (e.g., measles virus or herpesviruses). Focal cell damage in the skin may cause epithelial cells to become detached, forming blisters (Fig. 8-6). Some viruses can cause epithelial cells to proliferate (e.g., venereal warts caused by HPV or the umbilicated papules of molluscum contagiosum caused by poxviruses). Finally, viruses can contribute to the development of malignant neoplasms (Chapter 7).

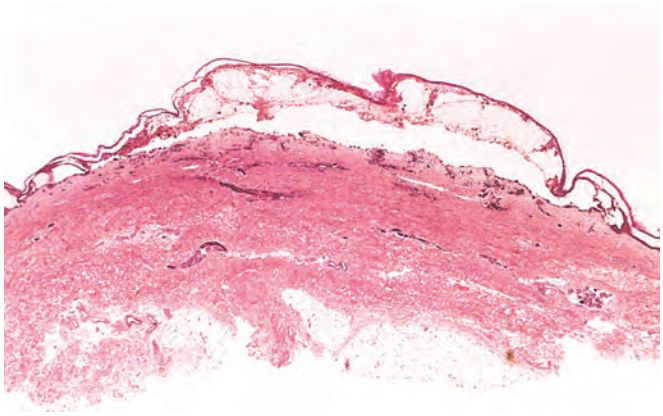

Figure 8-6 Herpesvirus blister in mucosa. See Figure 8-9 for viral inclusions.

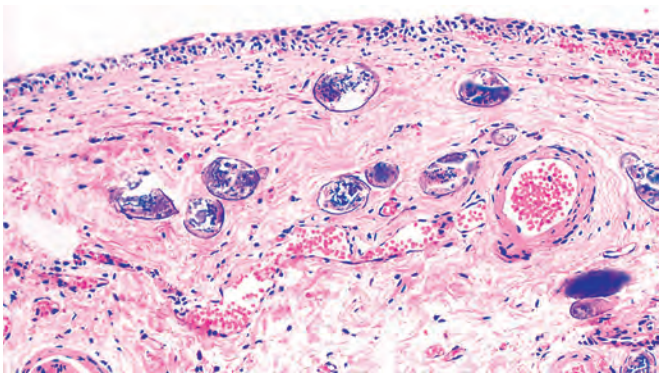

Figure 8-7 *Schistosoma haematobium* infection of the bladder with numerous calcified eggs and extensive scarring.

Tissue Necrosis

Clostridium perfringens and other organisms such as *C. diphtheriae* that secrete powerful toxins cause such rapid and severe necrosis (gangrenous necrosis) that tissue damage is the dominant feature. The parasite *E. histolytica* causes colonic ulcers and liver abscesses characterized by extensive tissue destruction with liquefactive necrosis and little inflammatory infiltrate. Some viruses can cause widespread and severe necrosis of host cells associated with inflammation, as exemplified by total destruction of the temporal lobes of the brain by herpesvirus or the liver by HBV.

Chronic Inflammation and Scarring

Many infections elicit chronic inflammation, which can lead either to complete healing or to extensive scarring. For example, chronic HBV infection may cause cirrhosis of the liver, in which dense fibrous septae surround nodules of regenerating hepatocytes with complete loss of normal liver architecture and consequent changes in blood flow.. Sometimes the exuberant scarring response is the major cause of dysfunction (e.g., the "pipestem" fibrosis of the liver or fibrosis of the bladder wall caused by schistosomal eggs [Fig. 8-7] or the constrictive fibrous pericarditis in tuberculosis).

These patterns of tissue reaction are useful guidelines for analyzing microscopic features of infectious processes, but they rarely appear in pure form because different types of host reactions often occur at the same time. For example, the lung of an AIDS patient may be infected with CMV, which causes cytolytic changes, and at the same time by *Pneumocystis*, which causes interstitial inflammation. Similar patterns of inflammation also can be seen in tissue responses to physical or chemical agents and in inflammatory diseases of unknown cause (Chapter 3).

Special Techniques for Diagnosing Infectious Agents

The gold standards for diagnosis of infections are culture, biochemical or serologic identification, and, in some cases, molecular diagnosis, depending on the organism in question. Some infectious agents or their products can be directly observed in hematoxylin and eosin–stained