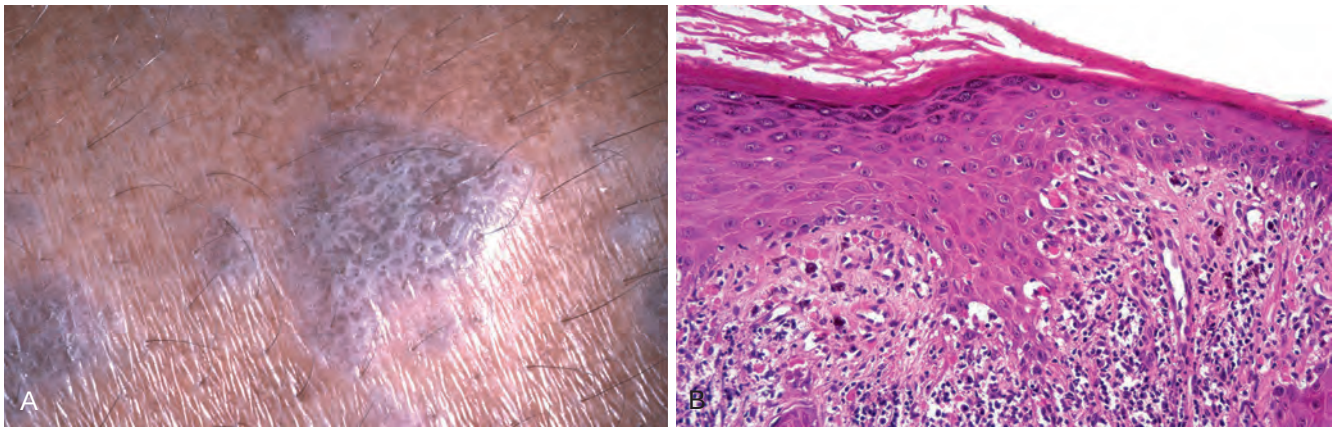

Figure 25-26 Lichen planus. **A,** This flat-topped pink-purple, polygonal papule has a white lacelike pattern of lines that are referred to as Wickham stria. **B,** There is a bandlike infiltrate of lymphocytes at the dermoepidermal junction, hyperkeratosis, and pointed rete ridges (sawtoothing), the latter as a result of chronic basal cell layer injury.

MORPHOLOGY

Cutaneous lesions consist of itchy, violaceous, flat-topped papules that may coalesce focally to form plaques (Fig. 25-26*A*). These papules are often highlighted by white dots or lines called **Wickham striae**, which are created by areas of hypergranulosis. In darkly pigmented individuals, lesions may acquire a dark-brown color due to release of melanin into the dermis as the basal cell layer is destroyed. Lesions are usually multiple and symmetrically distributed, particularly on the extremities and often about the wrists and elbows. The glans penis is another common site of involvement. In 70% of cases, oral lesions are present as white, reticulated, or netlike areas involving the mucosa.

Lichen planus is characterized histologically by a dense, continuous infiltrate of lymphocytes along the dermoepidermal junction, a prototypic example of **interface dermatitis** (Fig. 25-26*B*). The lymphocytes are intimately associated with basal keratinocytes, which show degeneration, necrosis, and a resemblance in size and contour to more mature cells of the stratum spinosum (squamatization). As a consequence of this destructive lymphocytic infiltrate, the dermoepidermal interface takes on an angulated zigzag contour (sawtoothing). Anucleate, necrotic basal cells may become incorporated into the inflamed papillary dermis, where they are referred to as **colloid** or **Civatte bodies.** Though characteristic of lichen planus, these bodies may be detected in any chronic dermatitis in which basal keratinocytes are destroyed. Although the lesions bear some similarities to those in erythema multiforme, lichen planus shows changes of chronicity, namely, epidermal hyperplasia (or rarely atrophy) and thickening of the granular cell layer and stratum corneum (hypergranulosis and hyperkeratosis, respectively).

KEY CONCEPTS

Inflammatory Dermatoses

- Many specific inflammatory dermatoses exist, which can be mediated by IgE antibodies (urticaria) or antigen-specific T cells (eczema, erythema multiforme, and psoriasis)
- These disorders are diagnosed based on the distribution and gross appearance of skin lesions and the microscopic patterns of inflammation (e.g., interface dermatitis in lichen planus and erythema multiforme)

Blistering (Bullous) Diseases

Although vesicles and bullae (blisters) occur in several unrelated conditions such as herpesvirus infection, spongiotic dermatitis, erythema multiforme, and thermal burns, there exists a group of disorders in which blisters are the primary and most distinctive features. These *bullous diseases*, as they are called, produce dramatic lesions and in some instances are fatal if untreated. Blisters in the various disorders occur at different levels within the skin (Fig. 25-27); histologic assessment is essential for accurate diagnosis and provides insight into the pathogenic mechanisms. Knowledge of the structure of desmosomes and hemidesmosomes (described in Chapter 1), which you will recall provide the skin with mechanical stability, is helpful in understanding these diseases, as they are often caused by acquired or inherited defects in proteins that make up or bind to these structures (Fig. 25-28).

Inflammatory Blistering Disorders

Pemphigus

Pemphigus is a blistering disorder caused by autoantibodies that result in the dissolution of intercellular attachments within the epidermis and mucosal epithelium. The pathobiology of blistering disorders provides important insights into the molecular underpinnings of keratinocyte adhesion. The majority of individuals who develop pemphigus are in the fourth to sixth decades of life, and men and women are affected equally. There are multiple variants: (1) pemphigus vulgaris, (2) pemphigus vegetans, (3) pemphigus foliaceus, (4) pemphigus erythematosus, and (5) paraneoplastic pemphigus. These disorders are usually benign, but in extreme cases can be fatal without treatment.