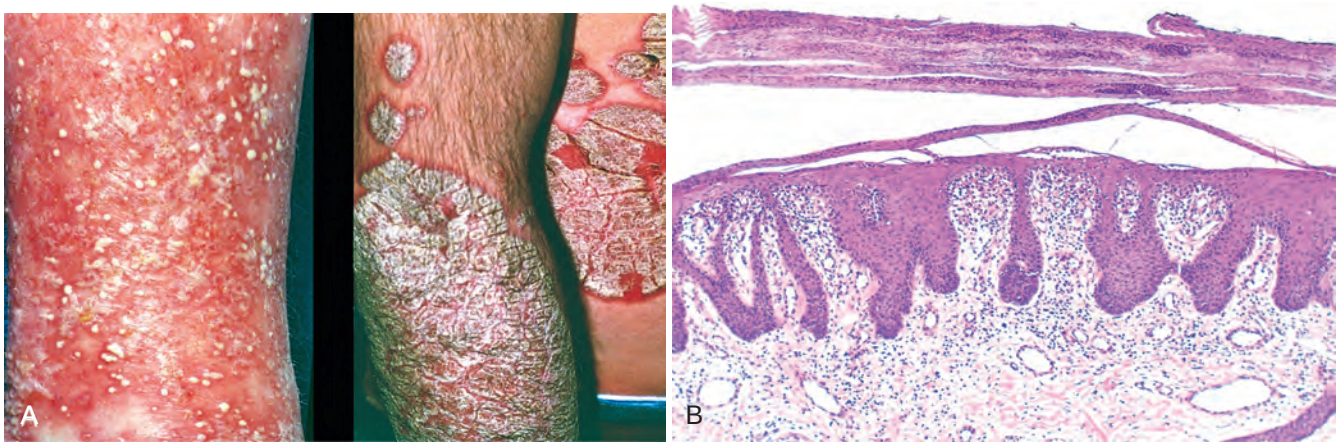

Figure 25-25 Psoriasis. **A,** Early lesions may be dominated by inflammation, marked by the presence of small pustules and erythema *(left).* Established chronic lesions are erythematous and covered by a characteristic silver-white scale *(right).* **B,** Microscopically there is epidermal hyperplasia, parakeratotic scale, and accumulation of neutrophils within the superficial epidermis.

the characteristic clinical phenomenon of multiple, minute, bleeding points when the scale is lifted from the plaque **(Auspitz sign).** Neutrophils form small aggregates within slightly spongiotic foci of the superficial epidermis **(spongiform pustules)** and within the parakeratotic stratum corneum **(Munro microabscesses).** In pustular psoriasis, larger abscess-like accumulations of neutrophils are present directly beneath the stratum corneum.

Seborrheic Dermatitis

Seborrheic dermatitis is a chronic inflammatory dermatosis that is even more common than psoriasis, affecting up to 5 % of the general population. It classically involves regions with a high density of sebaceous glands, such as the scalp, forehead (especially the glabella), external auditory canal, retroauricular area, nasolabial folds, and the presternal area. Despite this association and its name, however, seborrheic dermatitis is associated with inflammation of the epidermis and is not a disease of the sebaceous glands per se.

Pathogenesis. The precise etiology of seborrheic dermatitis is unknown. Increased sebum production, often in response to androgens, is one possible contributory factor. Involvement of sebum is supported by clinical observations of patients with Parkinson disease, who typically show increased sebum production secondary to dopamine deficiency and have a markedly increased incidence of seborrheic dermatitis. Once treated with levodopa, the oiliness of the skin decreases and the seborrheic dermatitis improves. However, other conditions associated with increased sebum production such as acne (discussed later) are not associated with seborrheic dermatitis, and sebum production is probably best viewed as being necessary but not sufficient to cause the disorder. Other work has suggested a relationship with colonization of the skin by certain fungal species of the genus *Malassezia*, but there is no definitive evidence of a cause and effect relationship. A severe form of seborrheic dermatitis that is difficult to treat is seen in many HIV-infected individuals with low CD4 counts; as with other forms of the disorder, its etiology is also unknown.

MORPHOLOGY

The individual lesions are macules and papules on an erythematous-yellow, often greasy base, typically in association with extensive scaling and crusting. Fissures may also be present, particularly behind the ears. Dandruff is the common clinical expression of seborrheic dermatitis of the scalp. Microscopically, seborrheic dermatitis shares features with both spongiotic dermatitis and psoriasis, with earlier lesions being more spongiotic and later ones more acanthotic. Typically, mounds of parakeratosis containing neutrophils and serum are present at the ostia of hair follicles (so-called **follicular lipping**). A superficial perivascular inflammatory infiltrate generally consists of an admixture of lymphocytes and neutrophils. With human immunodeficiency virus infection, apoptotic keratinocytes and plasma cells may also be present.

Lichen Planus

"Pruritic, purple, polygonal, planar, papules, and plaques" are the tongue-twisting "six Ps" of lichen planus, a disorder of skin and mucosa. Lichen planus is usually self-limited, most commonly resolving spontaneously 1 to 2 years after onset. Resolution often leaves a residuum of postinflammatory hyperpigmentation. Oral lesions, however, may persist for years. Squamous cell carcinoma has been noted to occur in chronic mucosal and paramucosal lesions of lichen planus, and could be an example of carcinogenesis in the setting of a chronic inflammatory process. As in psoriasis, the Koebner phenomenon may be seen in lichen planus.

Pathogenesis. The pathogenesis of lichen planus is not known. It is plausible that expression of altered antigens in basal epidermal cells or the dermoepidermal junction elicit a cell-mediated cytotoxic (CD8+) T cell response. In support of this notion, T-lymphocyte infiltrates and hyperplasia of Langerhans cells are characteristic features of this disorder.