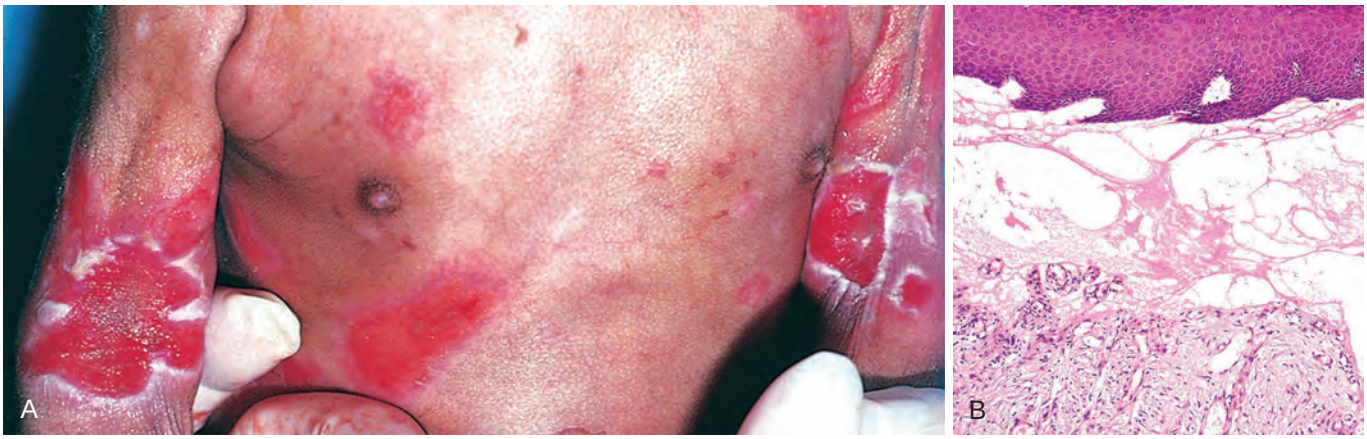

Figure 25-35 Epidermolysis bullosa. **A,** Junctional epidermolysis bullosa showing typical erosions in flexural creases. **B,** A subepidermal blister at the level of the lamina lucida. There is no associated inflammation.

sebaceous glands, such as heavy clothing, cosmetics, and tropical climates. Some families seem to be particularly prone to acne, suggesting a hereditary component.

Acne is divided into noninflammatory and inflammatory types, although both types may coexist. Noninflammatory acne may take the form of open and closed comedones.

- *Open comedones* are small follicular papules containing a central black keratin plug. This color is the result of oxidation of melanin pigment (not dirt).
- *Closed comedones* are follicular papules without a visible central plug. Because the keratin plug is trapped beneath the epidermal surface, these lesions are potential sources of follicular rupture and inflammation.

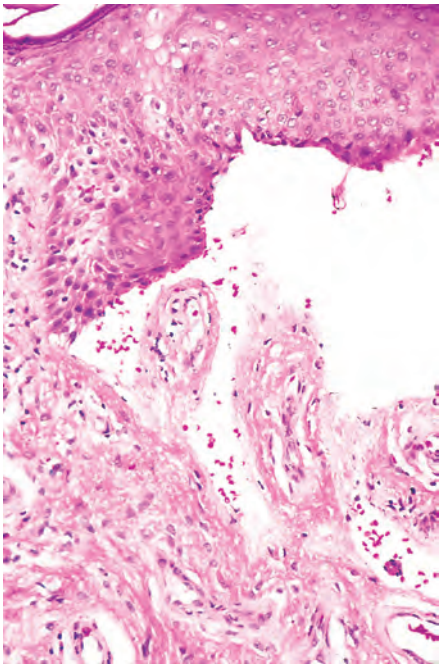

Figure 25-36 Porphyria. A noninflammatory blister at the dermoepidermal junction; note the seemingly rigid dermal papillae at the base that contain abnormal superficial vessels.

Pathogenesis. The pathogenesis of acne is incompletely understood and is likely multifactorial. At least four factors contribute to its development: (1) keratinization of the lower portion of the follicular infundibulum and development of a keratin plug that blocks outflow of sebum to the skin surface, (2) hypertrophy of sebaceous glands during puberty under the influence of androgens, (3) lipase-synthesizing bacteria *(Propionibacterium acnes)* colonizing the upper and midportion of the hair follicle, converting lipids within sebum to proinflammatory fatty acids, and (4) secondary inflammation of the involved follicle. Androgens, which increase sebum production, were first implicated in times past when it was noted that young castrated males generally did not develop the condition (a questionable tradeoff). Elimination of *P. acnes* is the rationale for administration of antibiotics to individuals with inflammatory acne. The synthetic vitamin A derivative 13-*cis*-retinoic acid (isotretinoin) brings about remarkable improvement in some cases of severe acne through its strong antisebaceous action.

MORPHOLOGY

Inflammatory acne is marked by erythematous papules, nodules, and pustules (Fig. 25-37*A*). Severe variants (e.g., **acne conglobata**) result in sinus tract formation and dermal scarring. Depending on the stage of the disease, open or closed comedones, papules, pustules, or deep inflammatory nodules may develop. **Open comedones** have large, patulous orifices, whereas those of **closed comedones** are identifiable only microscopically (Fig. 25-37*B, C*). Variable infiltrates of lymphocytes and macrophages are present in and around affected follicles, and extensive acute inflammation accompanies follicular rupture. Dermal abscesses may form in association with rupture (Fig. 25-37*B*) and lead to scarring.

Rosacea

Rosacea is a common disease of middle age and beyond, affecting up to 3% of the US population, with a predilection for females. Four stages are recognized: (1) flushing episodes (pre-rosacea), (2) persistent erythema and