areas should be lubricated with a moisturizer. Approximately 30 g of a topical agent is required to cover the entire body surface of an average adult.

Low- to mid-potency topical glucocorticoids are employed in most treatment regimens for AD. Skin atrophy and the potential for systemic absorption are constant concerns, especially with more potent agents. Low-potency topical glucocorticoids or nonglucocorticoid anti-inflammatory agents should be selected for use on the face and in intertriginous areas to minimize the risk of skin atrophy. Two nonglucocorticoid anti-inflammatory agents are available: tacrolimus ointment and pimecrolimus cream. These agents are macrolide immunosuppressants that are approved by the U.S. Food and Drug Administration (FDA) for topical use in AD. Reports of broader effectiveness appear in the literature. These agents do not cause skin atrophy, nor do they suppress the hypothalamic-pituitary-adrenal axis. However, concerns have emerged regarding the potential for lymphomas in patients treated with these agents. Thus, caution should be exercised when these agents are considered. Currently, they are also more costly than topical glucocorticoids. Barrier-repair products that attempt to restore the impaired epidermal barrier are also nonglucocorticoid agents and are gaining popularity in the treatment of AD.

Secondary infection of eczematous skin may lead to exacerbation of AD. Crusted and weeping skin lesions may be infected with *S. aureus*. When secondary infection is suspected, eczematous lesions should be cultured and patients treated with systemic antibiotics active against *S. aureus*. The initial use of penicillinase-resistant penicillins or cephalosporins is preferable. Dicloxacillin or cephalexin (250 mg qid for 7–10 days) is generally adequate for adults; however, antibiotic selection must be directed by culture results and clinical response. More than 50% of *S. aureus* isolates are now methicillin resistant in some communities. Current recommendations for the treatment of infection with these community-acquired methicillin-resistant *S. aureus* (CA-MRSA) strains in adults include trimethoprim-sulfamethoxazole (1 double-strength tablet bid), minocycline (100 mg bid), doxycycline (100 mg bid), or clindamycin (300–450 mg qid). Duration of therapy should be 7–10 days. Inducible resistance may limit clindamycin's usefulness. Such resistance can be detected by the double-disk diffusion test, which should be ordered if the isolate is erythromycin resistant and clindamycin sensitive. As an adjunct, antibacterial washes or dilute sodium hypochlorite baths (0.005% bleach) and intermittent nasal mupirocin may be useful.

Control of pruritus is essential for treatment, because AD often represents "an itch that rashes." Antihistamines are most often used to control pruritus. Diphenhydramine (25 mg every 4–6 h), hydroxyzine (10–25mg every 6 h), or doxepin (10–25 mg at bedtime) are useful primarily due to their sedating action. Higher doses of these agents may be required, but sedation can become bothersome. Patients need to be counseled about driving or operating heavy equipment after taking these medications. When used at bedtime, sedating antihistamines may improve the patient's sleep. Although they are effective in urticaria, non-sedating antihistamines and selective H_2 blockers are of little use in controlling the pruritus of AD.

Treatment with systemic glucocorticoids should be limited to severe exacerbations unresponsive to topical therapy. In the patient with chronic AD, therapy with systemic glucocorticoids will generally clear the skin only briefly, and cessation of the systemic therapy will invariably be accompanied by a return, if not a worsening, of the dermatitis. Patients who do not respond to conventional therapies should be considered for patch testing to rule out allergic contact dermatitis (ACD). The role of dietary allergens in AD is controversial, and there is little evidence that they play any role outside of infancy, during which a small percentage of patients with AD may be affected by food allergens.

LICHEN SIMPLEX CHRONICUS

Lichen simplex chronicus may represent the end stage of a variety of pruritic and eczematous disorders, including AD. It consists of a circumscribed plaque or plaques of lichenified skin due to chronic scratching or rubbing. Common areas involved include the posterior nuchal region, dorsum of the feet, and ankles. Treatment of lichen simplex chronicus centers on breaking the cycle of chronic itching and scratching. High-potency topical glucocorticoids are helpful in most cases, but, in recalcitrant cases, application of topical glucocorticoids under occlusion, or intralesional injection of glucocorticoids may be required.

CONTACT DERMATITIS

Contact dermatitis is an inflammatory skin process caused by an exogenous agent or agents that directly or indirectly injure the skin. In *irritant* contact dermatitis (ICD), this injury is caused by an inherent characteristic of a compound—for example, a concentrated acid or base. Agents that cause ACD induce an antigen-specific immune response (e.g., poison ivy dermatitis). The clinical lesions of contact dermatitis may be acute (wet and edematous) or chronic (dry, thickened, and scaly), depending on the persistence of the insult (see Fig. 70-10).

Irritant Contact Dermatitis ICD is generally well demarcated and often localized to areas of thin skin (eyelids, intertriginous areas) or to areas where the irritant was occluded. Lesions may range from minimal skin erythema to areas of marked edema, vesicles, and ulcers. Prior exposure to the offending agent is not necessary, and the reaction develops in minutes to a few hours. Chronic low-grade irritant dermatitis is the most common type of ICD, and the most common area of involvement is the hands (see below). The most common irritants encountered are chronic wet work, soaps, and detergents. Treatment should be directed toward the avoidance of irritants and the use of protective gloves or clothing.

Allergic Contact Dermatitis ACD is a manifestation of delayed-type hypersensitivity mediated by memory T lymphocytes in the skin. Prior exposure to the offending agent is necessary to develop the hypersensitivity reaction, which may take as little as 12 h or as much as 72 h to develop. The most common cause of ACD is exposure to plants, especially to members of the family Anacardiaceae, including the genus *Toxicodendron*. Poison ivy, poison oak, and poison sumac are members of this genus and cause an allergic reaction marked by erythema, vesiculation, and severe pruritus. The eruption is often linear or angular, corresponding to areas where plants have touched the skin. The sensitizing antigen common to these plants is urushiol, an oleoresin containing the active ingredient pentadecylcatechol. The oleoresin may adhere to skin, clothing, tools, and pets, and contaminated articles may cause dermatitis even after prolonged storage. Blister fluid does not contain urushiol and is not capable of inducing skin eruption in exposed subjects.

TREATMENT CONTACT DERMATITIS

If contact dermatitis is suspected and an offending agent is identified and removed, the eruption will resolve. Usually, treatment with high-potency topical glucocorticoids is enough to relieve symptoms while the dermatitis runs its course. For those patients who require systemic therapy, daily oral prednisone—beginning at 1 mg/kg, but usually ≤60 mg/d—is sufficient. The dose should be tapered over 2–3 weeks, and each daily dose should be taken in the morning with food.

Identification of a contact allergen can be a difficult and time-consuming task. Allergic contact dermatitis should be suspected in patients with dermatitis unresponsive to conventional therapy or with an unusual and patterned distribution. Patients should be questioned carefully regarding occupational exposures and topical medications. Common sensitizers include preservatives in topical preparations, nickel sulfate, potassium dichromate, thimerosal, neomycin sulfate, fragrances, formaldehyde, and rubber-curing agents. Patch testing is helpful in identifying these agents but should not be attempted when patients have widespread active dermatitis or are taking systemic glucocorticoids.