Dermatologic problems in children are frequent, and of great concern to the parent. It is estimated that approximately 20 to 30 percent of children seen in the practitioner's office have a primary or secondary skin eruption. Occasionally, an unusual dermatitis seen during a routine examination may alert the physician or nurse to a systemic disorder.

Most dermatologic problems in children can be diagnosed easily and, ideally, should be treated by the primary-care physician or health provider, who is the best person to manage the patient's entire illness. Compliance is greatly increased in the primary-office setting where the parent has confidence in the physician's diagnosis and treatment.

In children, some of the most common skin problems are due to hypersensitivity reactions. In most instances, a careful history and examination, with a bright light and a magnification lens, will lead to a morphologic diagnosis based on the type of primary and secondary skin lesions and on their distribution and configuration. Consultation with a dermatologist is suggested if conventional therapy does not result in resolution of the lesion.

In this article, I will discuss the primary clinical features, differential diagnosis, and practical management of some of the more common allergic dermatoses seen in children (Table 1). Less-common dermatoses that are not entirely due to hypersensitivity are omitted because of space limitations, but interested physicians will find them treated in some standard pediatric texts.\textsuperscript{1,2}

**ATOPIC DERMATITIS (ECZEMA)**

In many cases the cause of atopic dermatitis is not known and might better be called hypersensitivity dermatitis rather than allergic

**TABLE 1**

<table>
<thead>
<tr>
<th>ALLERGIC DERMATOSES IN CHILDREN</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atopic dermatitis (eczema)</td>
</tr>
<tr>
<td>Allergic contact dermatitis</td>
</tr>
<tr>
<td>Infectious eczema</td>
</tr>
<tr>
<td>Erythema multiforme</td>
</tr>
<tr>
<td>Erythema nodosum</td>
</tr>
<tr>
<td>Drug eruptions</td>
</tr>
<tr>
<td>Photosensitivity dermatitis</td>
</tr>
<tr>
<td>Serum sickness</td>
</tr>
<tr>
<td>Urticaria</td>
</tr>
</tbody>
</table>
or atopic dermatitis. The word *atopy*, used to describe hypersensitivity characteristics of eczema, means "out of place," and indicates a familial tendency for persons to acquire certain allergic conditions, such as hay fever or asthma. Eczema may appear in up to 40 per cent of patients with atopic dermatitis, but in only 10 per cent of those with nonatopic dermatitis.

Atopic dermatitis is one of the most common dermatologic problems of childhood and is difficult to treat. The characteristic picture is that of a young infant (over age 2 months) whose skin is easily irritated and has marked pruritus. Typically, atopic dermatitis has remissions and exacerbations, with a tendency toward spontaneous clearing by the time the child is between three and five years of age. In uncomplicated cases the skin will not be scarred after final recovery; in most cases, therapy will minimize the skin problem.

Jacobs outlined various factors that apparently act on genetically abnormal skin to exacerbate the rash and increase pruritis — e.g., sweating, heat and humidity, contact allergens, dry skin, allergic reactions, psychologic stress, infection, repeated trauma due to harsh soaps and other external irritants, and, above all, scratching. The majority of eczema patients have elevated IgE levels, which may fluctuate with the severity of the eczema. The incidence, pathogenesis, and natural course of eczema have been reviewed. Table 2 lists the exogenous causes producing and aggravating eczema, including those disorders in which another cutaneous lesion is predominant and those secondary to a systemic disease.

**Clinical features.** The child with atopic dermatitis exhibits poorly defined eczematosid patches that are frequently inflamed, with many secondary excoriations located on the cheeks, extensor surfaces of the forearms and legs, or — less commonly — on the trunk and diaper area. The eruption is usually symmetric, with variable involvement. There is frequent itching, with serosanguineous weeping, and crusting is common. Cellulitis is rarely seen, although the presence of superficial secondary infection is not uncommon.

Occasionally, an eruption will be seen in the scalp, postauricular areas, posterior neck, and upper buttocks. Rarely, if the entire body becomes affected, the infant becomes very irritable and the mother more anxious. Day-to-day variation in pruritus and severity of the rash is characteristic. In the older child and in those with persistent itching, the dermatitis becomes more papular, with plaques and areas of lichenification. Occasionally, parents remark that itching is infrequent or does not occur; however, careful examination will reveal secondary excoriations (Figure 1), produced by nocturnal scratching. Often the child’s skin will be markedly inflamed and blood-stained when he awakens (Figure 2). As the stratum corneum is desquamated, hypo-

continued
### Table 2

**CAUSES OF ECZEMATOID ERUPTIONS IN CHILDREN**

<table>
<thead>
<tr>
<th>Exogenous</th>
<th>Endogenous</th>
<th>Cutaneous lesion secondary to a systemic disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Contact dermatitis:</td>
<td>Predominant cutaneous lesion</td>
<td>Wiscott-Aldrich syndrome</td>
</tr>
<tr>
<td>Primary irritant</td>
<td>Infantile eczema, atop ic seborrhea, cradle cap</td>
<td>Leiner’s disease with C₅ dysfunc tion</td>
</tr>
<tr>
<td></td>
<td>Desquamative erythrodema</td>
<td>Chronic granulomatous disease</td>
</tr>
<tr>
<td>Infection:</td>
<td>Dyshidrosis (pompholyx)</td>
<td>Phenylketonuria</td>
</tr>
<tr>
<td>Bacteria</td>
<td>Ritter’s disease (toxic epidermal necrolysis)</td>
<td>Mucopolysaccharidoses</td>
</tr>
<tr>
<td>Fungi</td>
<td></td>
<td>Hartnup disease</td>
</tr>
<tr>
<td>Physical agents:</td>
<td>Congenital ichthyosiform erythrodema</td>
<td>Acrodermatitis enteropathica</td>
</tr>
<tr>
<td>Light</td>
<td>Diaper (napkin) dermatitis</td>
<td>Gluten-sensitive enteropathy</td>
</tr>
<tr>
<td>Chemical</td>
<td>Ichthyosis simplex</td>
<td>Anhidrotic ectodermal dysplasia</td>
</tr>
<tr>
<td>Cold</td>
<td></td>
<td>Histio cytosis X</td>
</tr>
<tr>
<td>Heat</td>
<td>Lichen simplex chronicus</td>
<td>Others.*</td>
</tr>
</tbody>
</table>

*List is incomplete, but includes the more common diseases.
Source: Modified from Solomon, L. M., and Esterly, H.B.*

---

**Figure 2.** Two-year-old with chronic atopic dermatitis and secondary infection in the classic distribution.

Pigmentation may be noted that will gradually repigment with healing.

Generally the skin is very dry (xerosis) and rough, with a “goose-bump” appearance, and with follicular hyperkeratosis of the upper arms and thighs — i.e., keratosis pilaris. The eyelids may be affected, and, if so, will appear wrinkled. Children with allergic rhinitis often have a distinct facial pallor, with allergic “shiners” and a transverse nasal crease due to their persistent allergic “saluting.” There will be a family history of atopic dermatitis, hay fever, or asthma in 30 to 40 per cent of such patients.

**Differential diagnosis.** In making the differential diagnosis of atopic dermatitis, nummular eczema, seborrheic dermatitis, candidal infection, pityriasis alba (occasionally), and lichen simplex (rarely), miliaria (rarely), and ichthyosis (rarely) should be considered. Usually, a nonpruritic eruption should not be considered in making the differential diagnosis of eczema.

During infancy there are a number of rare systemic diseases with rashes resembling atopic dermatosis (Table 2). In the winter-
time, when the skin becomes much drier, ichthyosis may be confused with so-called winter eczema. The large, flat scales that have a generalized distribution easily identify the child with ichthyosis simplex. Congenital bullous ichthyosiform erythroderma (epidermolytic hyperkeratosis), characterized by blistering, erythroderma, and hyperkeratosis at birth or very soon thereafter, is easily distinguished from atopic dermatitis. Lichen simplex chronicus may resemble atopic dermatitis but is usually sharply localized to one or two areas and tends to persist for many months to years without remissions and exacerbations. Miliaria is characteristically localized in the face and neck and is thus easily differentiated from atopic dermatitis (Figure 3).

The greatest confusion in making the differential diagnosis occurs with seborrheic dermatitis, which usually is found in early infancy.\(^6\)\(^7\) It may appear alone or in association with atopic dermatitis (Table 3). It is characterized by greasy, thick, yellow scaling of the scalp and postauricular areas and often is in the intertriginous areas. "Potato-chip" scales on an erythematous base may develop, particularly in the very young infant (Figure 4). Pruritus is mild or absent, and lichenification does not occur. Frequently, the entire diaper area is affected, and in about one-third of the infants, secondary candidal infections will develop in the diaper area. Without treatment, seborrheic dermatitis usually persists and may become generalized.

**Management.** In comparison to atopic dermatitis, management of seborrheic dermatitis is simple. For the patient with the latter condition, Sebizon lotion (10%) sodium sulfacetamide cream in a hydrophilic base) massaged into the scalp nightly for one week is very effective. Each morning a mild shampoo is used to wash the hair. The lotion is applied every other night during the second week, and every third night during the third week, with a shampoo the following morning. Improvement should be noted during the first week, with clearing during the third week. Recurrence is rare; if it occurs, the above treatment will again be effective. Steroid creams may be useful but are expensive and occasionally ineffective. Overnight application of 3% salicylic acid ointment is also effective.\(^8\)

---

**Table 3**

<table>
<thead>
<tr>
<th>Clinical feature</th>
<th>Seborrheic dermatitis</th>
<th>Atopic dermatitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>FAMILY HISTORY-ALLERGY</td>
<td>+</td>
<td>+++</td>
</tr>
<tr>
<td>ONSET UNDER 2 MONTHS</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>SCALP AND GROIN</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>LICHENIFICATION</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>PRURITUS</td>
<td>+</td>
<td>+++</td>
</tr>
<tr>
<td>RECURRENT</td>
<td>-</td>
<td>+++</td>
</tr>
<tr>
<td>SECONDARY INFECTION</td>
<td>-</td>
<td>+</td>
</tr>
</tbody>
</table>

- RARE, +, UNCOMMON; ++, COMMON; +++ USUAL
Source: Adopted from Kahn, G.\(^7\)

---

*Figure 3.* Twenty-one-day-old infant with miliaria rubra, crystallina, and pustulosa aggravated by ointments.
Management of atopic dermatitis is complex and can be difficult for the busy practitioner. Five components must be monitored, with repeated education of the parents:

1. Itching must be stopped.
2. Skin irritants must be avoided.
3. Skin hydration and local care must be given.
4. Environmental control, dietary advice, and other appropriate general measures must be instituted.
5. The parents must be reassured that eczema will clear or be reduced in most children after they reach the age of two or three (Figure 5).

In talking to parents of young infants, the physician should de-emphasize the allergic connotations and give reassurance that, although there is no cure, daily therapy can control eczema until it runs its course. Since infantile eczema usually is self-limited, one should avoid using systemic steroids. In specific patients, desensitization measures may be effective, especially if allergic respiratory disease is present at the same time.

It is essential that written instructions be given to the parent at each visit. We give printed material to the parent (Figure 5); sections not appropriate for that particular stage of skin care are crossed out. The parent must be reassured in order that tensions in the home can be reduced since the psychosocial sequelae of eczema are most important. An interested pediatrician can provide his patient with the emotional support needed while the child “outgrows” his disease.

To stop itching, sufficient diphenhydramine (Benadryl), 4 mg./kg. daily, or ciproheptadine HCL (Periactin), 1-4 mg. daily for infants, 4-12 mg. daily for two-to-six-year-olds, is recommended. The Benadryl dose may be doubled at bedtime; 6 to 8 mg./kg. daily may be necessary at times to stop itching. Occasionally the addition of aspirin, 100 mg./kg. daily, or hydroxyzine HCl (Atarax), 10-25 mg. t.i.d., to the Benadryl will break the itch-scratch cycle. It is absolutely necessary to avoid wool, silk, harsh soaps, and occlusive medications, and one should strive to avoid overwarming, chilling, or drying of the skin. Saline or tap-water compresses are effective for the acute weeping stage. An effective alternative is the use of dressings wet with Burow’s solution, applied continuously for a day or two, and then three or four times a day for a period ranging from 30 minutes to an hour. Usually the skin improves within 48 hours, and one can progress to the subacute stage of treatment indicated in Figure 5.

For secondary infection, (staphylococcus or group A hemolytic Streptococcus) systemic penicillin or erythromycin, orally, 40 mg./kg. daily for 10 or more days, is effective. For small areas of local infection we use Betadine skin cleanser, four to five times daily, followed by thorough rinsing. A light film of bacitracin ointment, 500 units/gm., or gentamicin cream, 0.1 per cent, may be applied.
four or five times daily. Burow’s solution dressings may also clear superficial infection. It is essential to clear the secondary infection before improvement can occur and before steroid creams are used.

For the subacute stage, hydrocortisone cream, 0.5 or 1 per cent, applied every four hours, is very effective. If eczema is severe, Synemol cream (fluocinolone acetonide), 0.025 per cent, or Aristocort A cream (triamcinolone acetonide), 0.1 per cent, is effective, but prolonged use should be avoided. Occlusive wrapping with Saran Wrap every 12 hours may be helpful but usually is unnecessary. A tap-water bath at least once daily can be very soothing. Ordinary soaps should be

---

**ATOPIIC ECZEMA MANAGEMENT: INSTRUCTIONS FOR PARENTS**

1. **Give your child reassurance:** reduce tension, program therapy, explain course.

2. **itching must be stopped:**
   A. Benadryl or Periactin
   B. Aspirin may be given with Benadryl or Periactin
   C. Restraints and/or hydroxyzine HCL (Atarax) (10 to 25 mg t.i.d.) for sedation
   D. Avoid wool, silk, regular soaps, and occlusive medications
   E. Keep cool and comfortable, avoid heat and humidity
   F. Satin or percale sheets (bandaging if necessary)
   G. Cut fingernails short

3. **Skin care:**
   A. **Acute weeping stage** (Expect improvement in 1 to 3 days) (Use #1 or 2)
      1) Baths: Saline or tap water 2 or 3 times/day. For cleansing use Aveeno oatmeal or diluted Aveeno in bath or as compresses.
      2) Compresses (three layers of dressing): Burow’s solution (1 Domeboro packet/tablet to 1 quart water) continuous first day (new dressing every 3-4 hours), then 4 x daily ½ to 1 hours at a time.
      3) Infection, secondary: Culture, Rx-Betadine skin cleanser (1:1 with water) then apply local gentamicin, 0.1%, cream. If ineffective, use oral penicillin or erythromycin if necessary for 10-14 days.
   B. **Subacute stage** (Expect improvement in 48 hours)
      1) Hydrocortisone cream (½ to 1% every 4 hours) (Hytone) or Tredinon cream, 0.05%.
      2) If severe Synemol cream (0.025%) or Aristocort A Cream, 0.1% (Saran Wrap every 12 hours) for 1 to 2 days.
      3) Bath once daily (no soap); cleanse with Cetaphil lotion.
   C. **Chronic stage** (dry) (Use only one of the 3 following treatments or combinations)
      1) Tar: Coal tar ointment, 1 to 5%, or Tarbonic cream or
         Liquor carbonis detergens (5 to 10%) in vanishing cream or in zinc oxide ointment or combined
         with a steroid cream.
      2) Quinolones: 3% Violom in ½% hydrocortisone cream
      3) Steroids: Synalar 0.01% cream or solution of Valiseone 0.1% ointment or lotion.

4. **General Care:** (No soaps — use Lowila cake or Neutrogena)
   A. Oil baths: Alpha-Keri or Nivea oil 1 tsp./quart, 2 to 4 X daily for 5-15 minute baths or;
   B. Tar baths: Balnetar or Zetar, one capful as directed in bath water.
   C. For cleansing use: Oilated Aveeno Bath 1 X daily or Cetaphil lotion; wipe lightly with soft cotton cloth
      to leave a protective film.
   D. Emollients: Applied t.i.d. while skin is still wet especially to involved areas. Use Nivea cream, Eucerin,
      Lubriderm, Aquaphor, Aquacare HP lotion or cream or Carmol cream. For very dry/scaly skin
      use urea-containing emollients.

5. **Diets:** Elimination diet trial for 3 to 4 weeks in infants. (Cow’s milk, chocolate, nuts, wheat, eggs, legumes,
      fuzzy and citrus fruits, raw tomatoes).

6. **Repeated visits** and phone calls to clarify management program.

---

Figure 5. Instructions for parents used by the author.
avoided; cleansing should be done with Cetaphil lotion or Lowila or Neutrogena soap when inflammation is present.

For the chronic, noninflamed, dry stage one of three medications should be used. We prefer a quinolone, 3 per cent Vioform in 0.5 per cent hydrocortisone cream, which is effective, inexpensive, and nontoxic. With temporary flareups, 1 to 2½ per cent hydrocortisone cream (Hytone without paraben) is effective. In severe cases we use fluorinated steroids but for short periods only — since they can cause cutaneous atrophy and hypopigmentation, and they are expensive. Since many of our patients are dark-skinned, we use hydrocortisone creams routinely. Occasionally an ointment may be more soothing for black skin; however, it may be occlusive and, hence, irritating. We have not used systemic steroids in eczema. Severe complications from chronic steroid use in other diseases has been observed, and we do not recommend it.

General care consists of oil or tar baths daily if the child enjoys them. Following a bath, while the skin is still wet, an emollient (Eucerin) should be applied (Figure 5). Urea-containing emollients are quite effective for the severe case. If the steroid cream is applied within the next hour or so while the skin is hydrated, it will be more effective. Hospitalization is rarely necessary when this regimen is followed.

Although food intolerance is uncommon, the dramatic effect of an elimination diet after a three-to-four-week trial in a young infant is remarkable. Chocolate, nuts, wheat, eggs, legumes and raw vegetables should be avoided and a trial of a soybean or other milk substitute is advised. The key to effective therapy is the physician’s reassurance and supportive care.

For persistent eczema in older children, consultation with an allergist is advised. Inhalant allergens, especially dust, may aggravate eczema and may require specific immunotherapy, especially in older children and adolescents.

In about one out of every 20 infants the above measures will be ineffective, and a modified Scholtz-Jacobs regimen is recommended. Basically this consists of avoiding soap and water completely. For candida infections, 1 or 2 per cent aqueous gentian violet applied t.i.d. with a Q-tip for no more than three days will clear more than 90 per cent. When the purple discoloration has cleared, any residual infection should be treated with another three-day course of gentian violet.

Nummular eczema (“coinlike”) is usually seen in older children (Figure 6). It can be managed effectively with the local application of a fluorinated steroid cream or with a 1 or 2.5 per cent hydrocortisone cream three or four times daily. Benadryl is effective for itching, and emollients applied t.i.d. alternately with the steroid cream will hydrate the skin. For the rare patient with extensive involvement, the eczema regimen (Figure 5) will be effective.

---

**ALLERGIC CONTACT DERMATITIS**

Allergic contact dermatitis is a cell-mediated, delayed hypersensitivity reaction to an antigenic substance applied to the skin. True allergic contact dermatitis should be distinguished from primary irritant dermatitis caused by simple chemical or physical irritation of the skin.

**Clinical features.** The characteristic clinical feature of allergic contact dermatitis is an irregular and asymmetric distribution of linear lesions not conforming to dermatomal cleav-
age lines and nerve distributions. Angular, irregular, and bizarrely shaped patches with asymmetric appearances on one arm or one leg or a portion of the trunk will distinguish contact dermatitis from other allergic dermatoses. The most common causes in children are irritants — i.e., nickel, plastic, poison ivy, and other members of the Rhus family, shoe or sock contact, clothing, cosmetics, and diaper dermatitis. The classic distribution of lesions due to contact dermatitis can be seen in a child with poison ivy (Figure 7).

Finding the cause. Good clues to the cause of contact dermatitis usually can be obtained by taking a careful history, particularly of prior contact with similar clothing, plants, or cosmetics and by noting the distribution and configuration of the lesions. Rhus dermatitis (poison ivy, poison oak, poison sumac) accounts for the more common eruptions in children with lesions usually found on the exposed areas of the face, arms, and legs (Figures 7 and 8). The antigen may also be transmitted to the genitalia or eyes by the fingers. In those previously sensitized, the rash begins to appear within one to three days after

Figure 6. Nummular eczema, chronic, recurrent over nine-month period.

Figure 7. Contact dermatitis due to poison ivy. Note classic linear vesicular and microvesicular eruption and eyelid edema.

Figure 8. Contact dermatitis due to poison sumac with uninfected vesicles and minimally secondarily infected bullae due to S. aureus.
contact. The intensity of the eruption usually increases over the next seven days, and then it remits spontaneously. The rash occurs in vesicular, and occasionally bullous, erythematous streaks and patches, and the intense itching is characteristic. (Figure 8).

Management. Management is best achieved by educating the child to avoid future contacts with the substances causing the problem. When contact has occurred, immediate washing of the area or wiping with alcohol (70 per cent strength) is effective. Once the eruption has occurred, Benadryl or Atarax is very helpful, and tap water, saline, or 1:20 Burow’s solution soaks can be soothing. Applications of hydrocortisone cream (0.5 to 1 per cent) will be useful; in milder cases, plain calamine lotion can be effective.

In severe recurrent cases when the eruption is acute, exudative, and vesicular or bullous, oral corticosteroids may be necessary. Prednisone, 1 mg./kg. for four days followed by gradual tapering of the dose over a period of four or more days for a total of eight days’ treatment is suggested. In severe cases a loading dose, 2 mg./kg., not to exceed 60 mg. for the first 24 hours, is suggested. Premature discontinuation of oral steroids may result in a generalized eczematous flare.

Cosmetic dermatitis is usually caused by hair dyes, perfumes, sun-screen preparations, lipsticks, and antiperspirants. Contact dermatitis of the eyelids may be due to nail-polish allergy even though the fingernails show no reaction.

Clothing dermatitis is due to formaldehyde and formaldehyde resins impregnated in cloth to prevent wrinkling. In children with clothing dermatitis, lesions usually will be found over the trunk and buttocks, where the clothing binds closely to the body. The axillary folds may be severely affected, due to the leaching of the formaldehyde onto the skin because of perspiration.

Substitution of polyester, nylon, or acetate clothing may avoid further irritation. Occasionally, contact dermatitis may have been caused by elastic or rubber in undergarments or from the application of plastic Band-Aids (Figure 9). Detergents or bleaches that contain optical whiteners may be the cause of clothing dermatitis. In hot weather, “sweaty sock dermatitis” may be due to the reinforced nylon or Dacron heels and toes in socks (Figure 10).
Management consists of avoiding the material causing the problem.\cite{8} Washing of new clothes will be helpful.

Diaper dermatitis, one of the most common nonallergic dermatoses in infants, is usually due to the wetness and maceration, which may be aggravated by such irritants as ammonia that are produced by urea-splitting B. ammoniagenes. The sites of the dermatitis are usually on the convex prominences of the pubis, thighs, and buttocks and usually spare the intertriginous folds. In patients with true allergic dermatitis the entire groin area becomes erythematous, with edema and maceration. Occasionally, creams prescribed for ordinary diaper dermatitis contain neomycin, parabens and ethylendiamine, which may be primary sensitizers. Management consists of keeping the area as dry and cool as possible, preferably leaving the diaper off for a 48 hour period. Application of a bland cream or ointment, or in severe cases, a topical hydrocortisone cream not containing any of the above irritating substances is quite effective. Secondary candidal infection should be treated with 1 per cent aqueous gentian violet t.i.d. for three days or nystatin powder or cream t.i.d. for five to seven days.\cite{8}

**INFECTIONOUS ECZEMA**

Infectious eczematoid dermatitis may occur in children who have very dry skin (xerosis) with or without atopic dermatitis. Such children are prone to superficial infection with *Staphylococcus aureus* or group-A β-hemolytic streptococci.\cite{8} Eczematoid dermatitis may appear in the axillary or intertriginous moist areas, with itching, followed shortly by secondary infection. More often the patient has a contact dermatitis that becomes infected. We routinely culture the affected skin and prescribe tap water or saline compresses for 15 to 30 minutes every four hours, followed by gentle washing with Betadine skin cleanser. After air-drying, a thin film of bacitracin ointment (500 u./gm.) or gentamicin cream, 0.1 per cent, is applied every four hours. If

Figure 11. Eczema herpeticum (Kaposi’s varicelliform eruption) due to herpesvirus hominis, of two to three days’ duration.

group-A βHS is recovered, and particularly if response to local treatment is minimal during 48 hours, the patient is given Bicillin IM, 600,000 U., if weighing under 60 pounds, or 1.2 million units, if over 60 pounds. If *S. aureus* is isolated, an antistaphylococcal drug may be given orally for seven to 10 days if local therapy has been ineffective.\cite{11}

The most serious complication of atopic dermatitis is infection with herpes simplex or vaccinia viruses (Kaposi’s varicelliform eruption). Because children with atopic dermatitis are especially susceptible to this complication, they should be isolated from persons recently vaccinated or infected with herpes simplex virus. In eczema herpeticum, the skin — particularly the face — is usually covered with vesiculopustular umbilicated lesions of 10-to-15-mm. diameter (Figure 11). Marked permanent scarring may develop, and about 5 per cent of the children with this condition die. Coxsackie A-16 virus can cause a similar problem.

Management. The best treatment is to avoid contact. Hospitalization is necessary for severe eczema vaccinatum. Vaccinia immune globulin 0.1 to 1 ml./kg. intramuscularly is effective, if given once and then repeated in
hypersensitivity reaction to many different antigens. It presents with a spectrum of skin lesions — e.g., urticarial, erythematopapular, and vesiculobullous, and rarely with exudative mucous-membrane involvement, conjunctivitis, and systemic manifestations (Stevens-Johnson syndrome).\textsuperscript{12}

The most common type is erythema multiforme minor. This presents with urticaria followed by erythematopapular and characteristic target (iris-type) lesions covering the entire body, including the palms and soles (Figure 12). A few patients will develop vesicles; occasionally bullae are seen. Rarely, children will develop classic Stevens-Johnson syndrome, with erosive lesions of the lips and exudative mucous membrane lesions of the mouth, periurethral, vaginal, and anal areas (Figure 13).\textsuperscript{12} Individual lesions, 1 to 2 cm in diameter with an erythematous periphery and

\textbf{ERYTHEMA MULTIFORME}

Erythema multiforme is a self-limiting eight to 10 days, especially if the initial dose is given early in the course of the infection. If this is not effective, methisazone (Marboran) therapy should be instituted at once.\textsuperscript{2} When corneal involvement is present, vaccinia immune globulin should not be used.

Primary eczema herpeticum heals spontaneously in 2 to 6 weeks. Once a patient has had a primary herpes infection, recurrent attacks are milder. Since no specific antiviral therapy is available, treatment consists of normal saline or aluminum acetate (Burow’s solution: Domeboro) compresses to remove crusts. Betadine skin cleanser should be used for general cleansing three or four times daily to decrease secondary bacterial infections.\textsuperscript{9}
a darker, violaceous center, will appear all at once, or — occasionally — in crops over a period of three to seven days. They resolve spontaneously within 10 to 14 days.

Finding the cause. Erythema multiforme in children is usually drug-related or follows an infectious disease. Drugs most commonly incriminated are the antibiotics, hydantoins, barbiturates, and sulfonamides. Common infectious agents are Mycoplasma pneumoniae, herpes simplex, Epstein-Barr virus, echovirus, Coxsackie viruses, and group A streptococci. Occasionally other causes have been implicated, including rubella virus (Figure 13), aspirin, cat-scratch fever, collagen vascular disease, diphtheria-pertussis-tetanus vaccine, horse serum, and smallpox vaccination.1,9

Management. The usual case of erythema multiforme is mild and picturesque, and since the condition is self-limiting therapy is unnecessary except for pruritus. Benadryl or Atarax is effective, especially at night. Children with vesiculobullae but without mucous membrane involvement should be treated symptomatically. Reverse isolation is necessary to prevent secondary infection. In patients with acute onset, fever, and mild toxicity associated with vesiculobullae, one must always consider toxic epidermal necrolysis. Vesiculobullae, and exudative skin lesions should be cultured to isolate S. aureus. The acute onset of red, tender skin followed shortly by bullae and a positive Nikolsky's sign suggests toxic epidermal necrolysis phage-group 1 or group 2 exfoliation-producing S. aureus.1,9,11 In our experience with 65 children with toxic epidermal necrolysis, mucous membrane invasion was limited to the lips and conjunctiva, thus excluding a diagnosis of Stevens-Johnson syndrome. The staphylococcal infection, if present, should be treated with an antistaphylococcal antibiotic; mild cases require no antibiotic therapy. Complete recovery occurs in nine to 11 days. Corticosteroids are contraindicated in patients with toxic epidermal necrolysis.11

Management of children with Stevens-Johnson syndrome includes hospitalization, reverse isolation, sedation, antipyretics, antibiotics if indicated, replacement of fluid and electrolytes, antihistamines, and mouth and local skin and eye care. Ocular treatment consists of instillation of a topical antibiotic (10 per cent sodium sulfacetamide) to prevent secondary bacterial infection. Ointments are preferred because of longer contact and their lubricating effect. For severe conjunctivitis, a topical steroid administered under an ophthalmologist's supervision is indicated. Systemic corticosteroids will not shorten the duration of this self-limited disease but will reduce fever and toxicity.

Prognosis for recovery in SJS in children is excellent. No deaths occurred during the acute or recurrent episodes in 30 patients we observed over a 15-year period. Blindness occurred in one patient and partial blindness in two others. In about 10 per cent of patients a mild recurrence of symptoms may be noted within one or two years.

BIBLIOGRAPHY