Current Practice

DISEASES OF THE SKIN

Management and Treatment of Contact Eczema

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The diagnosis of contact eczema is complete with the identification of the cause and an understanding of the process by which the dermatitis has been produced. Treatment begins with the elimination of the causative process. Though a discussion of diagnosis is outside the scope of this article, it is perhaps desirable to emphasize the multifactorial origin of eczema. Even when an exogenous contactant has been firmly incriminated the possibility of other less conspicuous contributory causes should be kept in mind. H. G. Adamson drew attention to the influence of sunburn in inducing specific allergic sensitivity to plants. Friction, maceration, and excoriation may contribute in the same way. A seborrhoeic background may be important and it is not unusual for contact eczema to be superimposed, either by therapy or otherwise, on a comparatively mild seborrhoeic dermatitis or intertrigo. Light sensitivity and aggravation by cleansers and other primary irritants frequently play a part in causation.

The influence of temperament and emotional factors is notoriously difficult to assess. Extreme views may result from personal bias; but to assume any case of contact eczema to be entirely free from a psychiatric factor, or at the other extreme to accept any eczema as entirely psychogenic—these attitudes are alike dangerously apt to be oversimplifications.

When contact eczema fails to recover after removal of "the" cause it is often because these secondary factors have not been taken into account—not that the primary agent has been wrongly incriminated but that contributory causes have been overlooked and are still active.

Elimination of Contacts

The elimination of specific allergic contacts is straightforward, though sometimes difficult in practice; the amateur gardener can easily give up primulas or chrysanthemums. Specific desensitization is possible with some epidermal allergens but is seldom a practical proposition, and in many cases avoidance of the allergen has to be very strict indeed. Substitutes can be found for most types of cosmetic and suitable alternatives can be chosen with the help of patch tests. Women very soon learn to avoid contact with nickel buckles and clasps, but continuing contact in less obvious ways—coinage, nickel traces in detergents—may be extremely difficult to detect and to eliminate. Rubber sensitivity elicited by rubber gloves is not difficult to deal with, but the elastic in women's clothing and the concealed rubber in shoes—even rubber adhesive—may present more of a problem.

In industrial cases a work process may be altered by the use of a substitute, but this may not be possible, and a change of occupation may be necessary. Because of the economic factor patients may have difficulty in accepting this, but it is seldom satisfactory, when allergic sensitivity is involved, to continue at the same work while attempting to avoid contact as much as possible, or with the doubtful protection of barrier cream or rubber gloves.

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Primary Irritants

Many cases of contact eczema depend not on specific allergic sensitivity but on excessive exposure to primary irritants, particularly cleansers, alkalis, and degreasers. Some people are naturally more susceptible to these sources of trauma; some appear to become so, perhaps as a result of long-continued exposure, perhaps with advancing years. Non-specific irritation of this sort contributes also to many cases of allergic contact eczema. Because this kind of trauma is non-specific, the substitution of alternative cleansers and so on is usually not very helpful; the detergent effect on the skin is shared by most or all detergents—all degreasers degrease. Fortunately the elimination of these contacts does not usually have to be so complete as the avoidance of allergens. A moderate reduction of exposure with careful removal of the contact from the skin after exposure is likely to be sufficient. When the hands are affected rubber gloves may be helpful, but the heat and moisture inside the gloves may counteract their protective effect if they are worn for periods of more than five or ten minutes at a time.

Bacterial infection is not often a source of difficulty. Once the eczematous process is being brought under control the skin can usually look after itself in this regard. The production of pus, either in vesicles or bullae or in furuncles, is not a part of the eczematous process and indicates bacterial infection. The same is true of subcutaneous cellulitis, lymphangitis, and painful lymphadenopathy.

Local Treatment

Once the causes of contact eczema have been eliminated there is a strong natural tendency towards recovery. To hasten recovery the local application of corticosteroids provides a treatment which is so effective that all the older methods pale into insignificance. The fluoridated corticosteroids—triamcinolone, betamethasone, and fluocinolone—are the ones in most common use. Hydrocortisone is relatively so much less effective that it no longer deserves a place. There is little if anything to choose between an ointment and a cream base, but a lotion may be easier to apply to a weeping surface or to the scalp. Most of these ointments can be diluted with resulting economy without significant loss of efficacy. This has been a measure of economy in many hospitals for the past few years, the standard strengths being diluted 1:10 or 1:4 with a suitable ointment or cream base. For greatest effect the corticosteroids should be applied four times daily but in many cases twice

daily is enough. A thin dressing of Tubegauze may be convenient.

When an adequate response is not obtained occlusion under polyethylene after applying the steroid very greatly enhances absorption.

Prolonged local treatment with corticosteroids has its drawbacks, and may lead to atrophy of the treated area of skin and subcutaneous tissues. However, if contacts are satisfactorily eliminated prolonged local treatment should not be required, and the suppressive effect of steroids should not be a substitute for accurate identification of causes and their removal.

Contrary to what is sometimes supposed the local application of corticosteroids does not impair resistance of the skin to bacterial infection. It is therefore not necessary to incorporate an antibiotic or antiseptic as a routine in order to guard against intercurrent infection; this only adds a possible hazard of additional sensitivity to the antibiotic. When bacterial infection is present the addition of neomycin to the steroid is usually effective, but recently neomycin sensitivity is being increasingly recognized. The quinoline antiseptics Vioform and Chinoform are safer and are usually preferred by dermatologists. When there is gross superficial infection baths of potassium permanganate or hexachlorophane solution twice daily are useful in cleaning up the area, and systemic antibiotics are seldom needed. Systemic administration of an antibiotic may, however, be indicated in the presence of subcutaneous cellulitis and lymphangitis, though even here the treatment of the skin surface is more important.

Systemic Treatment

Systemic treatment with steroids is in fact seldom indicated, since local applications are so effective. But in a severe hair-dye dermatitis, for example, where the whole head and face are involved in an acute process, systemic treatment will produce an immediate result and will perhaps be justified by the rapid relief of the great distress and discomfort which is often present in such a case. When systemic treatment is given it should be accompanied by local treatment and can ordinarily be terminated in a week or so. Prolonged systemic treatment should not be given as a way of avoiding the inconveniences of thorough local (and much safer) treatment.

Antihistamine drugs have little or no effect on the eczematous process, a reaction in which histamine has not been shown to play a part. Promethazine is sometimes useful, chiefly when given at night for its sedative effect. All the antihistamine drugs have some antipruritic action but their practical value in eczema is doubtful. Itching and discomfort rapidly disappear as the inflammatory process is controlled with local treatment, and though the antihistamine drugs are often given it is doubtful how much they contribute. Very often, one suspects, they are quite useless in this type of case. Other sedatives may be helpful, according to the usual indications; contact eczema calls for no special consideration in this context.

Psychotherapy

Whatever may be thought about the importance of the psychiatric factor the effect of psychotherapy and psychotropic drugs on the eczematous process remains in considerable doubt. Often enough these patients are benefited by treatment of this sort, the benefit being to their mental state and to their own attitude to the skin disease, but the objective physical progress of the eczema is not so clearly influenced. Psychiatric treatment in contact eczema should therefore be given on psychiatric indications with the object of obtaining psychiatric benefit, and not physical cutaneous ones. If this view is correct the indiscriminate treatment of all cases of contact eczema with sedatives or tranquillizers is mistaken.

Under suitable treatment, and when causes have been eliminated, recovery of contact eczema is often rapid—days or very few weeks. Delayed recovery usually indicates the presence of other hitherto unrecognized causative factors, secondary infection, or superadded irritation or allergy from treatment. Allergy to neomycin, lanolin, and ointment bases is a possible source of trouble.

TODAY'S DRUGS

With the help of expert contributors we publish below notes on a selection of drugs in current use.

Ethacrynic Acid

This drug is marketed by Merck Sharp and Dohme Ltd. under the name Edecrin.

Chemistry and Pharmacology

Ethacrynic acid is 2,-3-dichloro-4-(2-methylenebutyryl)-phenoxyacetic acid. It is a diuretic agent with a clinical effectiveness as great or greater than that of mersalyl, and it is more potent than thiazide diuretics.[1-3] The only other orally active diuretic with a potency approaching that of ethacrynic acid is frusemide, which has a very similar action on the renal tubules. Renal clearance studies indicate that ethacrynic acid has a unique mode of action. It has little effect on glomerular filtration or on renal plasma flow. It induces its saluretic and diuretic effect by action predominantly on the proximal convoluted renal tubule and on the ascending loop of Henle.[2 4 6] This novel influence on the loop of Henle is not shared by thiazide compounds.

Within 30 minutes of oral administration ethacrynic acid induces a prompt diuresis, which reaches its maximum in two hours and lasts for six to eight hours. After intravenous administration a diuretic response occurs within 15 minutes. The urinary excretions of sodium and chloride are both strikingly increased. The loss of chloride ions exceeds that of sodium; hydrogen ion excretion is increased and there is a tendency for a metabolic hypochloraemic alkalosis to develop. Potassium excretion is also increased; the degree of this is similar to that after frusemide and thiazide diuretics, but it is difficult to compare the kaluretic effect of different diuretics because endogenous aldosterone secretion plays a large part in determining the amount of potassium excreted after any agent. The practical lesson, however, is clear. Potassium supplements, preferably potassium chloride in the case of ethacrynic acid and frusemide, must be given with *all* diuretics.

The maximum effective single dose of ethacrynic acid is 150–200 mg., but because individual patients may have a phenomenal diuresis, even when resistant to older-established diuretics,[1] the initial amount should not exceed 50 mg.[2 3] Depending on the response, the dosage can be increased if