Exzema craquelé can be induced by repeated open application of a topical glucocorticoid, viz. 0.05% clobetasol 17-propionate cream. This might not be invariably due to the active component. Comparison of the skin surface roughness as assessed by profilometry and as expressed by $R_{Z}$ showed a decrease after repeated open application of 0.1% betamethasone 17-valerate cream and 0.25% prednicarbure cream, but an increase following the vehicle of the latter preparation. Thus commercial oil-in-water emulsion preparations seem to be potentially injurious to human skin, though this may be masked when a glucocorticoid is added.

(Accepted October 16, 1990.)


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As early as in 1979 Kligman & Frosch (1) described a clinical picture encountered after discontinuing prolonged application of topical glucocorticoids, characterized by reddening, tenderness, itch, scratching and scaling. While Kligman & Frosch traced these findings to what they called steroid addiction it has subsequently been described how regular application of topical glucocorticoid preparations can even in a very limited period of time lead to a diseased state presenting as eczema craquelé (erythema craquelé) (2). According to Björnberg (2) this is especially linked to repeated application of potent glucocorticoids in a cream base, such as 0.1% betamethasone 17-valerate or 0.1% hydrocortisone 17-butyrate, when applied under occlusion. Yet in rare cases it may also be due to the open application of a potent steroid cream (over a 6-week period) or to the repeated application of pH 5 ethanol base under occlusion. Eczema craquelé is associated with skin dryness. Skin dryness defined as a clinical condition meaning a rough, finally scaling non-inflamed skin surface can adequately be assessed by using surface profilometry (3). When repeatedly confronted with eczema craquelé as a side effect of the repeated open application of clobetasol 17-propionate cream (0.05%) during a trial performed for the comparative assessment of skin atrophy due to various topical glucocorticoids, we looked for skin surface roughness before and after the application period in a subgroup of 6 healthy volunteers.

MATERIAL AND METHODS

During a double-blind randomized trial, four different formulations were applied to the proximal flexor part of the forearms of 24 healthy volunteers. Application followed a randomized blocks design to ensure that either preparation A or B was applied to the one forearm and preparation C or D to the other. Preparation A representing 0.25% prednicarbure cream, B the corresponding vehicle; C, 0.1% betamethasone 17-valerate cream; C, 0.05% clobetasol
RESULTS

In 8 out of 12 volunteers applying 0.05% clobetasol 17-propionate cream, clear-cut eczema craquelé was found, making immediate discontinuation of treatment inevitable for safety reasons. Once only treatment had to be stopped after 14 days; 7 times after 21 days. Fig. 1 shows the typical clinical picture of eczema craquelé caused by 0.05% clobetasol 17-propionate cream. The ages of the volunteers showing this side effect ranged from 24 to 39 years, the ages of the others from 24 to 32 years. Five volunteers showing manifest eczema were female, while 2 were female in the other subgroup.

Of the 6 volunteers in whom skin roughness was assessed, 3 received 0.25% prednicarbate cream, 3 the corresponding vehicle, 4 0.1% betamethasone 17-valerate cream, and 2 0.05% clobetasol 17-propionate. Both volunteers applying the latter preparation had to discontinue treatment earlier than planned due to eczema craquelé. Hence data representing the treatment effect after 6 weeks are not available for this subgroup. While $R_{ZDN}$ values tended to decrease with preparation A as well as C, they tended to increase with preparation B (Fig. 2).

DISCUSSION

Eczema (or erythema) craquelé following the repeated open (or closed) application of glucocorticoid creams to normal human skin has been interpreted as an unwanted effect of the active ingredient (2). In one case, however, identical findings have been linked to the (closed) application of ethanol pH 5 (21). Moreover it has been stated that even widely accepted commercial moisturizers might act as irritants (7). The present findings first substantiate the idea that eczema craquelé can be a frequent sequela of the repeated open application of a potent glucocorticoid cream. This can be deduced from the frequency of Etat craquelé during the use of 0.05% clobetasol 17-propionate cream. This effect, however, need not be linked to the glucocorticoid itself. This can be concluded from the findings with 0.25% prednicarbate cream and the corresponding vehicle. It was in fact the vehicle which was irritant in this case, while the corresponding glucocorticoid preparation was not. Hence, prednicarbate seems to be able to over-compensate the unwanted effect of the cream base chosen by its manufacturer. This in a way seems to contradict a recent finding by Van der
Valk & Maibach (8) that several topical glucocorticoids are not able to suppress irritant skin reaction elicited by sodium laurylsulfate solution. As commercial glucocorticoid cream bases can be assumed to be free of components with well-known irritancy potential, one might be tempted to speculate that it is simply the comparatively high water content of the oil-in-water emulsion which reduces the water-binding capacity and thus gives way to desiccation. This question must be subjected to further analysis. The area of application of the drug chosen here might in fact represent the optimum area for pertinent analysis, as the potential irritation has been shown to increase from the wrist to the cubital fossa (9).

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Immediate Contact Reactions to Cow's Milk and Egg in Atopic Children

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Forty children (0-5 years old), presented with immediate contact urticaria, rash and often atopic dermatitis (n = 34). Redness or urticaria around the mouth appearing after consuming cow's milk or egg, were the major complaints in all. These symptoms suggested a food-induced immediate contact reaction, which can be immune-mediated or irritative. To re-induce this reaction, a skin provocation test, called SAFT, was performed. SAFT stands for Skin Application Food Test. This test is based on direct skin contact, during a maximum of 30 min with food in its 'ordinary consumptive state'. The SAFT can be regarded as a 'physiological' provocation patch test. If positive, contact urticaria develops most often within a few minutes. The results of SAFT and IgE RAST correlated significantly well. Total IgE values were not informative. The rapid onset of the SAFT reaction, induced by proteins, supported by RAST results, strongly indicates an immune-mediated mechanism. In 52% of the 34 patients with atopic dermatitis, dermatitis was exacerbated following food-to-skin contact. Immune-mediated contact reactions to foods play an important role in (dermal) food allergy. Key words: Contact urticaria; Dermal food allergy; Atopic dermatitis.

(Accepted October 31, 1991.)
Acta Derm Venereol (Stockh) 1991; 71: 263–266.
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The diagnosis of food allergy is a controversial issue. A reliable conclusion is often hampered by misleading histories from the parent(s) and psychological