# Vascular Effects of a Local Anesthetic Mixture in Atopic Dermatitis

LENNART JUHLIN and OLA ROLLMAN

*Department of Dermatology, University Hospital, Uppsala, Sweden*

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An anesthetic cream with an eutectic mixture of 5% lidocaine and 5% prilocaine has been tested on the skin of healthy subjects and patients with atopic dermatitis and generalized eczema. In healthy subjects a blanching was seen when the analgesia was complete after 30–60 min. In the dry skin and eczematous lesions of atopic dermatitis an application time of 5–15 min caused a blanching and a good analgesia. When applied for 30–60 min the eczematous skin became increasingly red and in one patient purpura appeared. The white dermographism turned red in treated areas. The abnormal vascular reactions to the cream in diseased skin can be explained by a rapid and increased percutaneous absorption of the anesthetics. A shortened application time is here recommended. *Key words: Anesthetic cream; Atopic dermatitis; White dermographism.* (Received May 8, 1984.)

L. Juhlin, Department of Dermatology, University Hospital, S-751 85 Uppsala, Sweden.

An eutectic mixture of 5% lidocaine and 5% prilocaine in a water emulsion cream base has previously been shown to produce an effective superficial dermal analgesia when applied under an occlusive dressing for 60 min (1). The mixture was especially useful in children for superficial surgical procedures. A slight blanching of the skin was noted as a useful marker for complete anesthesia which persists for 2 hours. In children an application time of 30 min was usually enough and on mucous membranes analgesia was evident within a few minutes.

Since abnormal vascular reactions to pharmacological stimuli are well known in atopic dermatitis we have now studied the local reactions to the epicutaneously applied anesthetic mixture in patients with this skin disorder.

## METHODS

The anesthetic cream contained an eutectic mixture of 5% lidocaine and 5% prilocaine with Arlatone® as emulsifier and Carbopol® as thickener. The base buffered to pH 8.4 was used as a control. The creams were gently applied to the skin of the shoulder region and covered with a soft paper and an occlusive bandage over 2×2 cm large areas. They were left for 1, 5, 10, 15, 30 and 60 min and removed with a soft paper. The colour of the treated skin was observed and the onset of analgesia was tested with needle pin pricks (1).

### *Patients*

Ten patients (age 18–25 years) with atopic dermatitis since childhood were studied. They all had lichenified plaques and dry, rough skin on the trunk and arms. Three patients with chronic generalized eczema of unknown cause (age 51–70 years) were also studied. Ten healthy subjects (aged 20–40 years) were used as controls.

## RESULTS

In healthy subjects the anesthetic cream applied under occlusion for 30–60 min produced a slight blanching of the skin and analgesia which lasted for 1–2 hours. When applied for 2–4 hours the blanching was more pronounced and replaced in ½–2 hours by a slight to moderate erythema, which persisted for 1–3 hours. No reaction was seen where the cream-base had been tested.

When the anesthetic cream was applied for one minute on the dry itching skin and lichenified patches of patients with atopic dermatitis a blanching was seen in 4 patients 1–5

min after removal of the dressing. The pale areas did not attain analgesia and the blanching disappeared in 15 min. Application for 5–10 min was followed within 5–15 min by a blanching and analgesia in all patients with atopic dermatitis and chronic eczema. After prolonged exposures (30–60 min) to the anesthetic cream the white area turned pale-red or red and 60 min after removal of the cream the test area was markedly red centrally. Sometimes there was a 2–3 mm white border. In the test area of one patient purpura appeared after 60 min application. The purpura slowly faded within a week.

The white dermographism observed in all atopics was inhibited or immediately turned red in areas treated with the cream for 15–60 min. The anesthesia and reddening of the treated area persisted for about 2 hours. In 2 of the atopic patients the reddening disappeared before the anesthesia and here the white dermographism reappeared despite anaesthesia. No change in vascular reactions to mechanical stroking was seen in placebo treated areas.

## DISCUSSION

We do not know why the anesthetic cream causes a blanching in the skin of healthy subjects. Attempts to block it have been negative. Thus pretreatment with intradermal injections of 0.2 ml phentolamine (5 mg/ml), reserpine (0.025 mg/ml), quanetidine (0.25 mg/ml) or marcaine (2.5 mg/ml) did not influence the degree or duration of blanching (unpublished data).

In both the dry, rough skin and lichenified plaques of patients with atopic dermatitis and in non-atopic eczematous areas there was a quicker onset of both blanching and anesthesia indicating a more rapid percutaneous absorption than in normal skin. Actually, preliminary experiments in various dermatoses have shown abnormally high blood levels of lidocaine and prilocaine after epicutaneous application of the anesthetic cream. The blanching effect observed in the patients often appeared before the analgesia indicating that the vascular responses of the anesthetic cream may not depend on blocking effects on nervous tissue. The blanching was not seen with the placebo cream and was thus not due to a delayed blanching caused by gently applying the cream to the skin.

The later appearing erythema caused by the anesthetic cream in atopics may be due to an increased penetration of the anesthetics causing a reactive hyperemia. It is less likely that it is due to the high pH since there was no effect with the placebo cream having the same pH. Therefore, the active components probably cause vasodilatation in atopic patients as has been found in healthy subjects by another anesthetic, ketocaine (2).

In skin treated with the anesthetic cream for 15–30 min the white dermographism in atopic patients sometimes turned red. The explanation could be that the cream had started its vasodilating activity which was not seen before provocation by stroking. Intradermal injection of procaine does not abolish the white dermographism (3, 4) and in two cases we found white dermographism when the skin was still anesthetic. Therefore the inhibition of the white dermographism in atopic dermatitis seems to be due to the late onset vasodilatation, and not due to the nervous effects, caused by the anesthetic cream.

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