

FRICITION BLISTERS AS A MANIFESTATION OF PATHOMIMIA

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Abstract. Self-inflicted skin lesions (pathomimia, artefacts) can be produced in a variety of ways, by the help of nails, razor blades, cigarettes, chemicals, etc. The present study reports three cases of pathomimia, in which the lesions consisted of vesicles or bullae produced by friction. Friction blisters can easily be produced in sites where the epidermis is both thick and firmly attached to the underlying tissues. The blisters have a characteristic histological appearance, which makes it possible to distinguish them from the blisters of different forms of vesicular and bullous diseases and from burn blisters which may also occur in pathomimia.

Key words: Artefact; Blister; Pathomimia; Skin manifestation

The terms pathomimia or artefacts are used here to denote self-inflicted skin damage. In many cases the phenomenon expresses some deeper psychological disturbance. The patient can be unaware that he himself has inflicted the injury, or he can deny it. In the latter case there can be an underlying desire for some form of social benefit such as pension or sick pay.

These artefacts can be caused in various ways, for instance by finger nails, razor blades, cigarette burns, or chemical irritants. This means that the clinical picture can vary enormously from case to case and the diagnosis can be difficult. The self-inflicted lesions sometimes simulate known dermatoses. In other cases the lesions can have such unusual appearance, localisation, distribution and powers of healing that artefact is strongly suspected. It is, however, often very difficult to confirm these suspicions because of lack of cooperation from the patient.

Vesicles and blisters produced by friction are forms of pathomimia of which is little known. This prompted us to report the following three cases.

CASE REPORTS

Case 1. A 60-year-old male psychiatric nurse was admitted to the dermatology department because of chronic urticaria

and recurrent blisters on his hands. His symptoms had persisted for many years. The man stated that he periodically developed itchy blisters on his hands. Scratching the blisters reduced the itching. On admission he had several erosions, both new and old, on the dorsal and palmar aspects of his hands and fingers (Fig. 1). There were no blisters. We suspected that the lesions were self-inflicted. His medical history was, however, somewhat complicated since he had had, a year earlier, a lymph node removed from his mediastinum and which contained islands of epithelioid cells of the kind seen in sarcoidosis. A complete investigation was done in an attempt to disclose sarcoidosis, malignant tumour, blood disease or liver disease, including porphyria cutanea tarda. None of these possible diagnoses could be substantiated, but he was found to have considerable fatty degeneration of the liver. After 16 days of observation, a new blister was noted on the palm of his hand. This was excised for histological examination.

Case 2. A 53-year-old man, alcoholic and unemployed, had recently had his application for invalid pension on grounds of backache refused. During a period of 2 months the patient had consulted his doctor three times for bullae on his palms and soles; the first time his right sole and left palm, second time left palm and third time right sole. After the third consultation he was referred to the dermatology department for examination. He had then had the blister for 2 days. At the examination he stated that he had been lounging on the sofa all day and that he had suddenly experienced a throbbing in his foot and discovered the blister. On his right sole there was a tense bulla with clear contents, the size of a child's hand. The bulla had a dry, scaly surface and two small, irregular excoriations. The surrounding skin was normal (Fig. 2). In the left palm he had a well healed scar as from an erosion. His skin and mucosae were otherwise normal. His general condition was completely unaffected. A biopsy was taken from the edge of the blister and Tzanck's test was performed at the same time.

Case 3. A man aged 32, labourer, came to the dermatological outpatient clinic complaining of recurrent blisters on his wrists, forearms, backs of hands, lower legs, front of thorax and face. The disease had started at the age of 18 years. The blisters, which were small and not itchy, broke out every one or two months and developed during 1-2 days. They burst rapidly and healed leaving pigmented scars or small yellowish brown papules. At his first visit only scars could be

¹ Case 1 kindly referred by Prof. Hans Rorsman, Department of Dermatology, Lund.

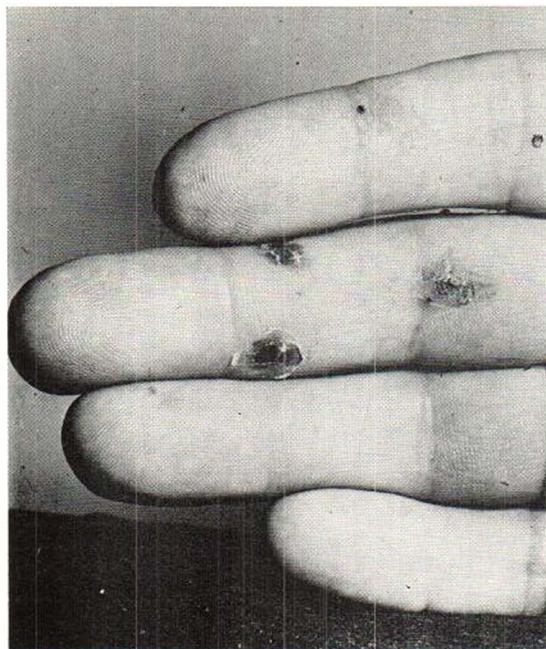


Fig. 1. Case 1. Fresh and older erosions on the fingers.

found. The patient returned a month later and demonstrated some fresh lesions on his fingers consisting of almost confetti-sized reddened maculopapules with either a suspected vesicle, a pustule or a scratch mark in the middle. A punch biopsy was taken from a fresh and probably vesicular lesion on the back of a finger (Fig. 3).

METHODS

The skin biopsies were fixed in 10% formalin, embedded in paraffin wax, sectioned and stained with hematoxylin-eosin, according to van Gieson and the periodic acid Schiff method. The material from cases 1 and 3 was sectioned in series. For the cytological examination, a modified Tzanck's test was used. The smears were fixed immediately in 95% alcohol for a minimum of 30 minutes. The staining was done with Mayer's hematoxylin and erythrosin.

Microscopical observations

The excised blister from case 1 and the punch biopsy from the edge of the bulla in case 2 showed, in principle, the same histological picture. In both cases the blisters were formed intra-epidermally due to a wide cleft within or under the stratum granulosum. Thus the top of the blister consisted of stratum corneum and a few rows of well preserved cells from the stratum granulosum and in some places also of one or more rows of degenerated cells (Fig. 4). The floor was composed of remaining parts of epidermis, outermost of strongly degenerated or necrotic epithelial cells, while the intermediate and deep parts of the floor consisted of totally undamaged cells. In the papillae and upper part of the corium a sparse perivascular infiltrate of lymphocytes and histio-

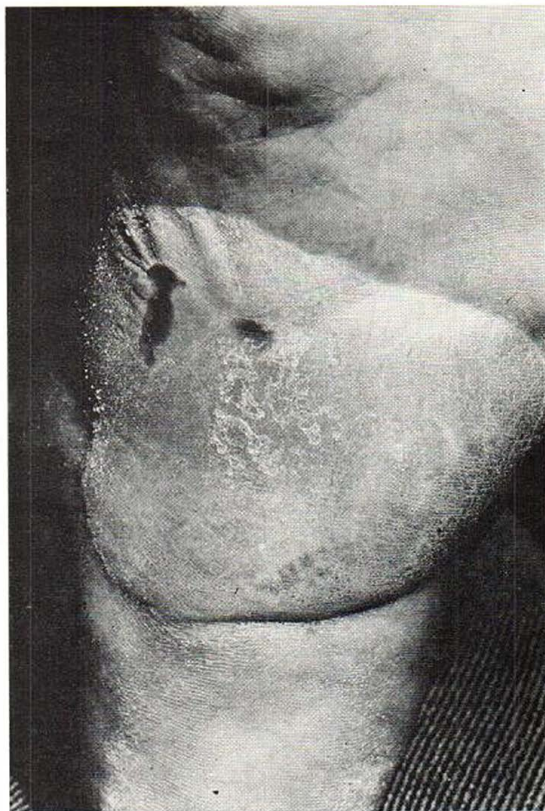


Fig. 2. Case 2. Bulla on right foot. The bulla has a dry scaling surface and 2 small irregular shaped excoriations. The surrounding skin is normal.

cytes was found. The blisters contained red blood cells and small cellular debris (Figs. 5, 7).

Smears made from the floor of the bulla on the sole (case 2) contained red blood cells and small clusters of strongly degenerated epithelial cells, each with a very pale nucleus containing a nucleolus and dense eosinophilic cytoplasm (Fig. 6).

The punch biopsy (serially sectioned) from the back of the finger in case 3 showed no real blister, but the epithelial cells in the outer and intermediate parts of the stratum spinosum were strongly degenerative. The zone with degenerative changes was stained weakly, in contrast to the well preserved parts of the stratum granulosum and spinosum. In the upper part of the corium very sparse perivascular infiltrates of lymphocytes and histiocytes were found (Fig. 8).

DISCUSSION

The histological appearances described in cases 1 and 2 are not characteristic of any kind of vesicular or bullous disease, but they correspond to the kind of blister which can be produced by friction as, for example, in the palms after unaccustomed manual

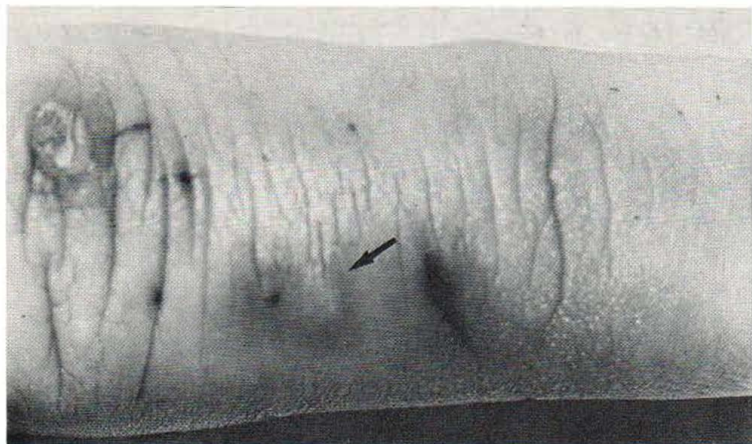


Fig. 3. Case 3. Back of a finger with scratch marks and an efflorescence with a suspected vesicle (arrow).

labour, or from ill-fitting shoes. Friction blisters in these places are a well known phenomenon and are seldom removed for biopsy. In the armed forces, however, this type of injury is so common and so important that it stimulated Sulzberger and co-

workers (4) to carry out a systematic study including histological examination of the lesions.

When the first patient was asked directly if he himself had produced the blisters, he at once admitted that he produced them by scratching with his fingernails. On request he scratched up a blister on his palm (Fig. 9). This was excised and showed exactly the same histological pattern as the first one examined.

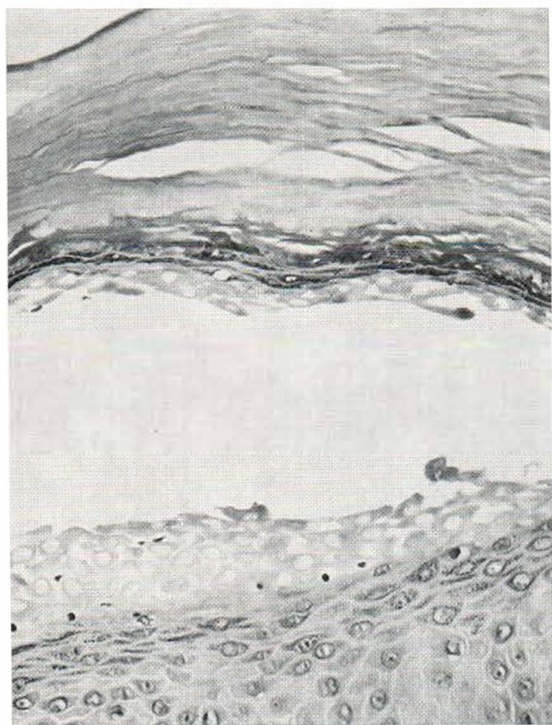


Fig. 4. Case 2. Bulla in the upper part of the epidermis. The roof of the bulla consists of stratum granulosum and a couple of rows of strongly degenerative cells. The floor of the blister is composed of the remaining part of epidermis. Outmost is a layer of strongly degenerative cells; underneath, a few rows of well preserved stratum granulosum. Hematoxylin-eosin, $\times 260$.

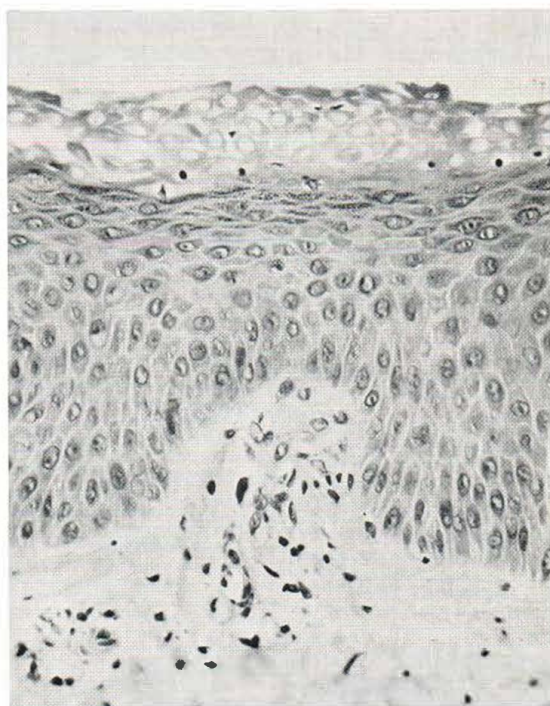


Fig. 5. Case 2. Floor of bulla. Outmost, strongly degenerative cells are seen; underneath, normal epithelium. In the papillae, sparse perivascular infiltrates of lymphocytes and histiocytes. Hematoxylin-eosin, $\times 260$.

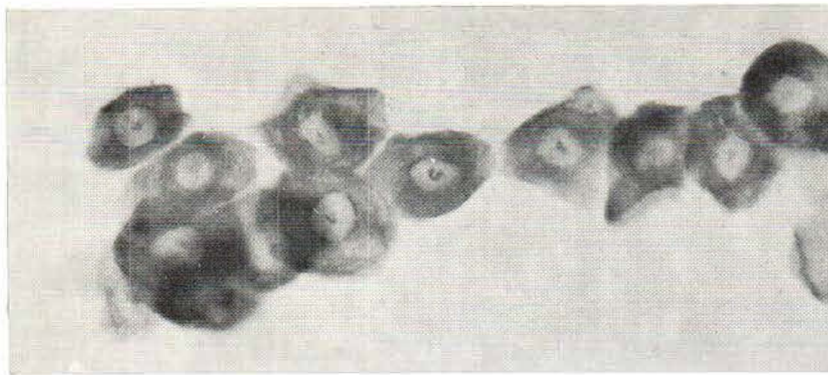


Fig. 6. Case 2. Smear from bulla in arch of foot. Small clusters of strongly degenerative epithelial cells, each with a very pale nucleus and with an obvious nucleolus and dense, eosinophilic cytoplasm. Mayer's hematoxylin-erythrosin, $\times 750$.

Patient no. 2 maintained decidedly that his blisters appeared spontaneously; however he was referred to the dermatology department again 5 weeks later for two infected necrotic patches 7×10 cm, one on the medial side of his right lower leg, the other on the lateral side of his left lower leg. The patient stated that the injuries had appeared spontaneously and had begun as bullae. Clinically, however, it was obvious that the lesions were artefacts. Biopsy showed a central necrosis extending deep into the corium with non-specific inflammation at the margin of the viable tissue. The histological appearance was non-specific and gave no clues as to the aetiology. Another 8 months went by before the patient reappeared with a superficial 7×7 cm ulceration with well defined edges below his left knee. This

lesion too was judged, by its clinical appearance, to be an artefact. The blisters on his palms and soles healed spontaneously. The necrotic patches on his legs received local treatment and, because they were infected, penicillin was also administered. As the damaged areas showed little tendency to spontaneous healing, a skin transplantation was done with a satisfactory result.

In the case of patient no. 3 the clinical picture was bewildering. There was no similarity to any form of dermatosis. However, dermatitis herpetiformis, porphyria cutanea tarda and some other vesicular or bullous diseases were considered and suitable tests for each condition were performed, all of which proved to be normal. After a second visit nearly 3 years ago on which occasion the patient

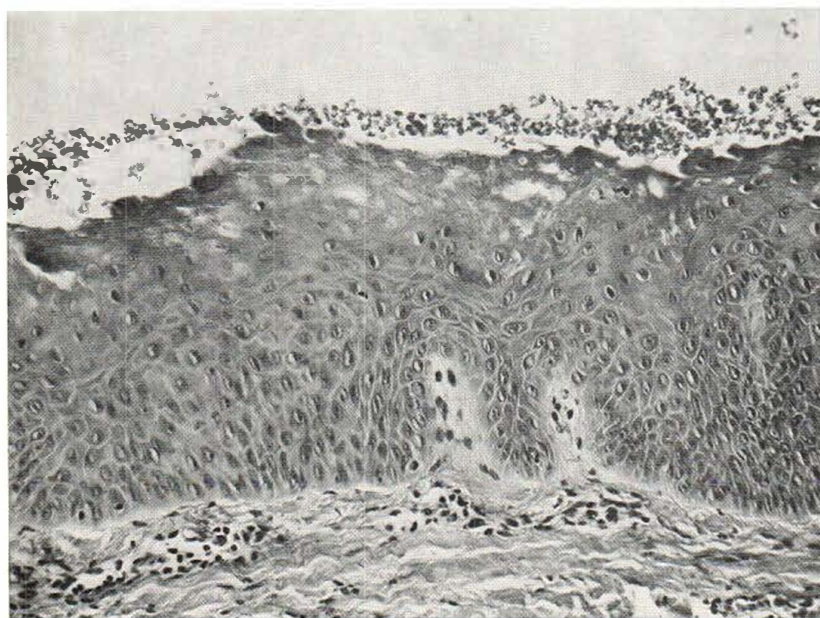


Fig. 7. Case 1. Floor of blister. The uppermost part consists of necrotic epithelial cells, the undermost part of well preserved epithelium. In the papillae and upper part of the corium, sparse perivascular lymphocyte infiltrate. Above the epithelium, red blood cells. Hematoxylin-eosin, $\times 188$.

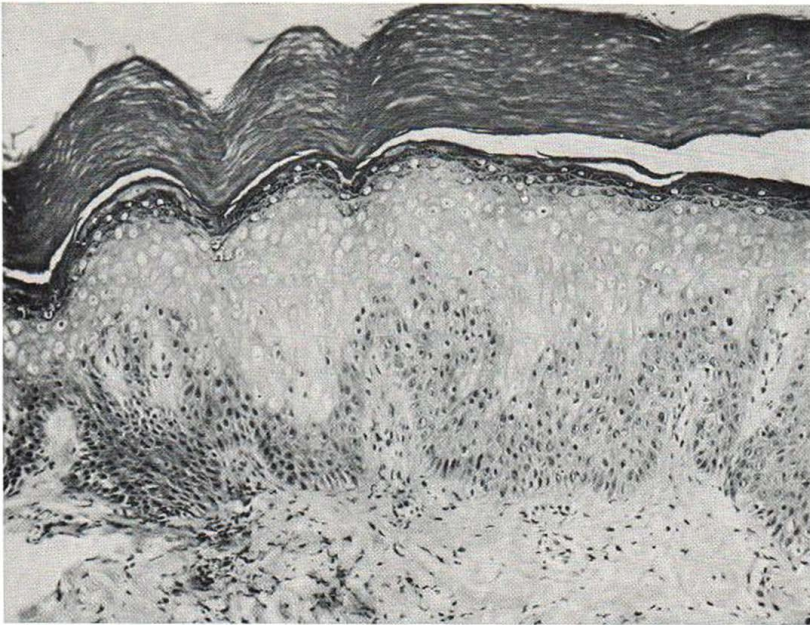


Fig. 8. Case 3. Epithelial cells of the stratum spinosum's outer and middle layers are strongly degenerative and stain weakly in contrast with the well preserved parts of the stratum granulosum and spinosum. The margin between the normal and the altered epithelium is well defined. Hematoxylin-eosin, $\times 120$.

presented fresh lesions which were photographed and biopsied, he has not shown up again. It is our opinion, however, that, in this case too, we are concerned with friction artefacts and that the distinctly demarcated devitalized zone in the outer and intermediate parts of the stratum spinosum are proof of damage by rubbing, but that the damage had not yet gone to the lengths of forming a blister.

Friction blisters have been studied experimentally by Naylor (1) and by Sulzberger and co-workers (4) using volunteers. Both these groups of authors examined the induced blisters histologically and found that they were due to clefts in the epidermis caused by necrosis of the cells in the upper and intermediate layers of the stratum spinosum. The roof of the blister thus consisted of stratum corneum and stratum granulosum together with a thin layer of amorphous cellular debris. This corresponds well with our findings. The histological pattern in our cases, however, varied somewhat between different parts of the blisters. In some regions the roof of the blister consisted of stratum corneum only and a well preserved stratum granulosum. In case 2 the cleft formation went in some places through the stratum granulosum so that it was possible to identify granulated cells even in the floor of the bulla (Fig. 4).

Naylor induced friction blisters in the skin over the middle third of the anterior surface of the tibia with a special machine which he had constructed. Sulzberger and co-workers produced blisters in two

different ways, one by linear rubbing, the other by twist rubbing. For the linear rubbing, a modified version of the instrument used by Goldblum and Piper and by Naylor was used. Twist rubbing was achieved with the help of an eraser on an ordinary pencil. The pencil was held between the palms of the operator, perpendicular to the skin surface with the flat end of the eraser pressed down firmly on the area to be rubbed. The pencil was then briskly rotated in clockwise-counter-clockwise directions. By this technique it was possible to produce blisters in some people in as little as 30 seconds and in no case did it take longer than 3 minutes.

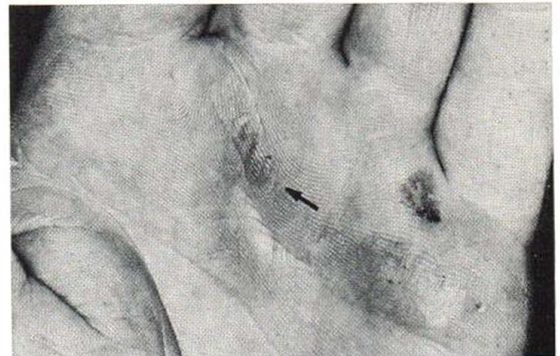


Fig. 9. Case 1. A fresh blister in the palm of the hand produced by scratching with the nails (arrow) and a healing erosion.

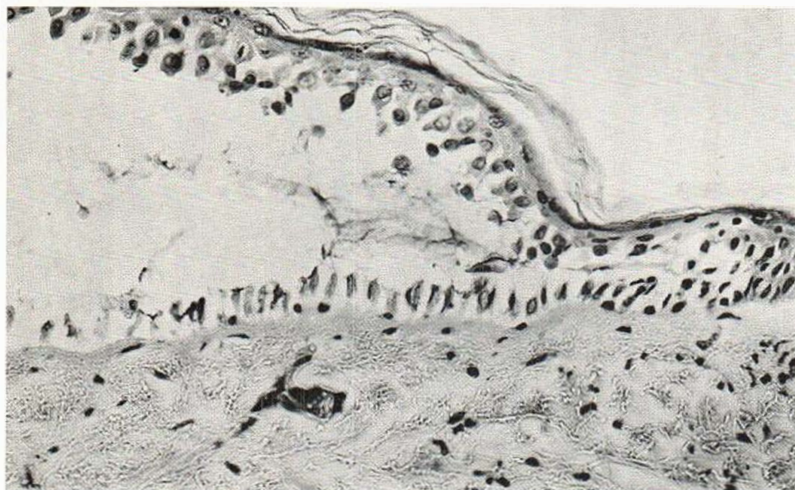


Fig. 10. The edge of a burn blister in which the epithelium was necrotic in the centre. At the edges of the necrotic area, intra-cellular oedema is seen and the cells are degenerative. A number of cells are vacuolised, others have dense, eosinophilic cytoplasm. The nuclei too vary in their stainability. The floor of the vesicle is covered by a row of strongly degenerated basal cells which are elongated in a characteristic way. Hematoxylin-eosin, $\times 250$.

Attempts to produce blisters, with the linear rubbing machine and the twisting rubber eraser technique, were made on apparently normal skin on the back, gluteal regions, shins, under- and overarms, thighs, palms and soles. Sulzberger and co-workers found that it was very difficult to produce complete fluid-filled blisters in all these places, with the exception of the palms and soles. In the other sites the blisters cracked rapidly, leaving an erosion. More lasting fluid-filled blisters could thus only be produced in such sites as where the skin is taut, i.e. where the lower epidermis is firmly attached to the underlying tissues, and, at the same time, the stratum corneum and stratum granulosum are thick. This is the case in the palms, soles, heels and backs of the fingers.

Naylor, in his trials, was able to show that the blisters did not depend on oedema in the epidermis, and were probably not a consequence of enzyme activity or of a local heating effect produced by the friction. Both Naylor and Sulzberger were of the opinion that the necrosis in the stratum spinosum was a mechanical injury caused by repeated distortion of the prickle cells. Their studies also show that friction blisters have a characteristic histological appearance and can therefore be distinguished from the lesions seen in the various kinds of vesicular and bullous diseases.

A relatively common form of artefact is the burn blister. This too has a characteristic histological appearance. The degree of damage to the epithelium can vary but, even in the more serious first-degree

burns, it is possible to find vacuolisation in the cytoplasm of the basal cells (3). The epidermal changes vary considerably within the same lesion and the histological picture is therefore much more complicated than in the case of the friction blister (Fig. 10). Coagulation or liquefaction necrosis is very often seen in the corium. Within the damaged area the vessels are often dilated and filled with tightly packed red cells, while perivascular inflammatory cellular infiltrate is seen only on the periphery of the lesion. Our experience of blisters caused by chemical irritants is limited. From the literature it is difficult to obtain a good idea of the histological appearance of such changes, except in the case of blisters produced by cantharidin in which acantholysis is a predominant phenomenon (2). It would seem, however, that chemical irritants, in the same way as burning, have a more deep-going effect than has friction.

Our attention was focused on this form of artefact by patient no. 1, who not only revealed but also demonstrated willingly his manner of producing blisters. The localisation and the histological appearance of the lesions were in good agreement with the findings reported by Naylor and Sulzberger. Self-inflicted mechanical damage can, however, be suspected if not actually confirmed also in lesions in other places than those where the skin is thick and taut and no whole blisters are to be found, viz. when the outermost parts of the epithelium are necrotic even though negligible or no changes are seen in the deeper layers of the epidermis and corium.

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