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THE CAUSE OF CHANGES IN SWEATING RATE AFTER ULTRA-VIOLET RADIATION

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In a previous paper (Thomson, 1951) it has been shown that ultra-violet (U.V.) radiation causes a reduction in the total sweat loss of subjects generally irradiated and in the sweating rate of small areas of skin locally irradiated. During the course of the above investigation certain inferences were drawn as to the cause of the reduction in sweating; e.g. it was noted that a vesicular eruption (sudamina) occurred after both local and general radiation, indicating that blockage of the mouths of sweat gland ducts was at least partly responsible for this reduction.

This paper reports further work carried out to elucidate the pathogenesis of the changes in sweating after radiation.

METHODS

In most of the experimental work reported in this paper, a method previously described (Thomson, 1951) was used to determine quantitatively the sweating rate of small areas of skin. The ratio of sweating rates of two areas on the same person was found, and subsequently one of these areas was used as a control, while the other was submitted to various experimental procedures. For convenience the ratio of sweating rates of test to control areas was called the 'sweating ratio'. These quantitative results were verified in most experiments by the use of Minor's (1927) method. Iontophoresis was carried out by the technique of Montgomery, Holling & Friedland (1938).

RESULTS

Sudamina or crystallina caused by radiation. Sudamina is described as an eruption in the stratum corneum of small, non-inflammatory vesicles containing sweat; the vesicles communicate directly with the sweat glands. This condition occurs in the course of many acute infectious diseases (e.g. pneumonia, or acute rheumatic fever, or in the moribund, in which cases it is of little importance and requires no treatment). The rash appears suddenly on any part of the body or face, but is most commonly found on the chest or neck.

In the experiments with exposure to U.V. radiation, the condition was first noticed on a small area of skin which had been irradiated 5 days previously when general thermal sweating was induced in the hot room. Thin-walled vesicles rapidly formed under the desquamating layer of corneum and tended to become confluent as sweating proceeded. Other persons showed widespread vesicles on all exposed surfaces at a similar interval after general U.V. radiation. Pl. 1, figs. 1 and 2, shows a similar generalized eruption of sudamina after exposure to natural sunlight. The condition is, in fact, reproducible in most subjects in a hot room by administering a dose of U.V. radiation from sun or lamp sufficiently strong to cause subsequent desquamation.

In most cases the vesicles were thin walled and were readily broken by contact with any solid object, and indeed the continuing pressure of sweat would normally be sufficient to burst the vesicles. In some cases, however, notably the area of the skin shown on Pl. 1, fig. 2, the necrosed layer of corneum was thicker, and the vesicles lasted for 2 or 3 days despite brisk towelling and the pressure and friction of clothing.

It is necessary to differentiate between sudamina and thermal blister burns. The sweat-filled vesicle in sudamina is more superficial; it appears at a longer interval after injury (2–4 days, as compared with 2–4 hr.), and only in conjunction with visible sweating elsewhere. In the vesicle fluid from sudamina protein is absent (negative heat coagulation and salicyl sulphonic acid tests), no cells are present and the chloride content is that of sweat (0·2–0·3% as NaCl). Thermal blister fluid approximates more to plasma in composition. It gives a heavy coagulum of protein with heat or salicyl sulphonic acid and may on occasion clot. Its chloride content is equivalent to 0·6% NaCl and polymorphonuclear cells (8–12 per high power field) are present, together with an occasional erythrocyte.

Histological examination of radiated skin. A biopsy specimen was obtained from a subject who had been locally irradiated 3 days previously (subject Cu., Thomson, 1951), and included skin from the erythematous and adjoining unradiated areas.

The unradiated part was apparently normal (Pl. 1, fig. 3). In the erythematous part the lymphatics of the dermis were infiltrated with lymphocytes, and a great many polymorphonuclear cells lay in the capillaries and small vessels. The superficial layers of the epidermis were necrotic with pyknotic nuclei and homogeneous cytoplasm, and in some places the underlying healthy zone was very thin, consisting of only one or two layers of cells. No mitoses were seen in the epidermis.

So far as is known, the following findings have not been described previously in relation to U.V. radiation. While in some cases the eccrine sweat gland ducts were followed through in serial sections, no evidence could be found of a patent channel through the superficial damaged layer of the corneum. Pl. 1, fig. 4,

shows a section of one of these ducts, and it will be seen that the keratin ring surrounding the coil of the duct nearest to the surface is occluded. Structurally this resembles the early stage of sweat duct closure in prickly heat as described by O'Brien (1947).

Pl. 1, fig. 5, shows a neighbouring sweat gland duct with the damaged layer of the epidermis raised slightly in the region of the duct orifice. Although vesicle formation had not been noted visually on this area, it is apparent that a vesicle of the same nature as sudamina had been in process of formation. The sweat gland duct was occluded near its orifice, and sweat from the still functioning gland had forced apart the damaged layer from the remainder of the epidermis. In its epidermal portion the lumen of the duct is dilated and the lining cells are flattened as though from sweat under pressure. The dilatation, which extends right up to the superficial necrosed layer, is as great as that found by O'Brien (1947) in thermal anhidrosis after complete blockage of sweat ducts. The related sweat gland acinus appears normal.

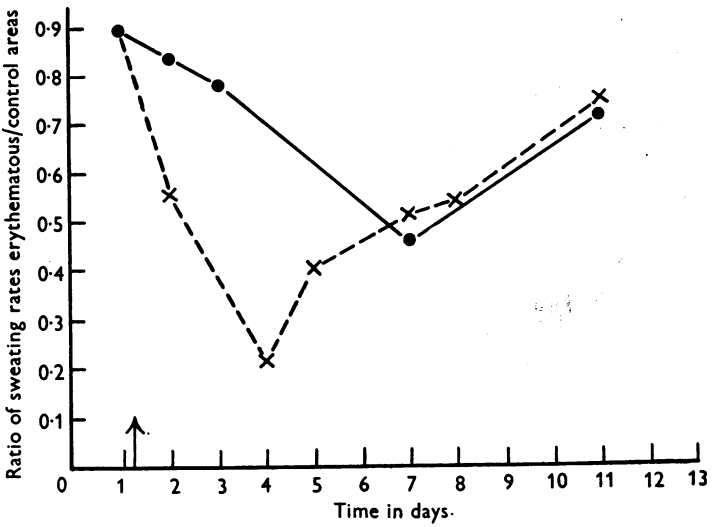
A similar picture of superficial occlusion was found in a biopsy specimen of another subject taken from an area (flexor surface, forearm) which had been radiated 5 days previously, where defective sweating had already been demonstrated by Minor's test.

A third specimen was also obtained from the scapular area of one of the subjects who had shown no reduction of sweating in a hot dry environment (Thomson, 1951; second series of trials). Signs of radiation damage were minimal and no abnormality was found in the sweat ducts.

Effect of other skin traumata on the sweating rate (*mustard plaster*). Lewis (1927) has shown that there is a similar sequence of events (the triple response) in many forms of skin injury including that caused by U.V. radiation. He believes that these changes are caused by the liberation in the skin of a histamine-like substance in response to the injury. It was therefore decided to compare the effect on the sweating rate of another variety of trauma with that of U.V. radiation.

Text-fig. 1 shows the effect of applying a mustard plaster (allyl isothiocyanate) to a skin area, whose sweating rate relative to that of a control area had previously been ascertained. In this case, erythema developed rapidly (within an hour), oedema was noted on the second day, desquamation began about the fifth day, and was complete by the seventh day. The sweating rate of the experimental area, as compared with that of the control area (sweating ratio) fell on the third day to a quarter of the value obtained before application of the mustard. On each day thereafter the rate of sweating rose, approaching the control value on the tenth day after application of the injurious agent. The observations on which the interrupted line-curve in Text-fig. 1 is based were made after sweating had been in progress for at least an hour. On some days observations were made within the first half hour of sweating, and these results

form the continuous line-curve in Text-fig. 1. This curve descended more slowly than the other, and joined it between the third and sixth days, after which the two rose together.



Text-fig. 1. The effect of mustard oil on rate of sweating. The graphs are ratios (A/B) of the quantities of sweat produced simultaneously in 9 min. from two equal areas of skin A and B: ●——●, in the first ½ hr. after the onset of sweating each day; ×- - -×, after 1 hr. of continuous sweating. Mustard oil was applied for 45 min. to area A at the arrow.

TABLE 1. Effect of iontophoresis with histamine on the sweating rate

		Weight of sweat (mg.)		Average ratio $\frac{\text{Right}}{\text{Left}}$ arm
		Right arm	Left arm (control)	
Before histamine		50	36	
		43	31	
		—	—	
	Total	93	67	1·39
After histamine		17	22	
		26	32	
		34	40	
	Total	77	94	0·82

The weights of sweat were obtained simultaneously over the same interval (total 9 min.) from two equal areas on the right and left arms. Histamine was applied to the right arm only.

The effect of iontophoresis with histamine. In view of the similarity of the reactions caused by histamine and other forms of skin trauma, it was decided to find whether histamine alone would cause reduced sweating. A solution of histamine acid phosphate (0·1% w/v) was applied by iontophoresis (10 min. at 6 mA. and 51 V.) to an area of skin after its sweating rate relative to that of a control area had been ascertained. There was very obvious oedema (which

lasted for 2 or 3 hr.) on the treated area, with the exception of a 5-day-old patch of U.V. erythema which had been inadvertently covered by the electrode. This area was refractory to the histamine, and showed a complete and striking absence of oedema (Lewis, 1927). The sweating ratio fell in the first 20 min. after iontophoresis from 1·39 to 0·82 (Table 1).

The effect of iontophoresis with acetylcholine and acetyl-β-methylcholine on areas of ultra-violet erythema which showed marked diminution of sweating. The 3-day-old irradiated area on the chest of the white subject (Text-fig. 2), which showed a markedly reduced sweating ratio from a normal of 1·10 to 0·22, was treated by iontophoresis with 2% (w/v) solution of acetylcholine hydrochloride under 51 V. for 5 min. at 14 mA. Before the iontophoresis was terminated, sweating and hyperaemia began to appear for an inch or so beyond the edge of the electrode, while still in the comparative coolness of the antechamber (75° F.). On entering the hot room there was markedly enhanced sweating on a roughly circular area of 5 in. diameter, extending up to, but excluding, the radiated area which showed no sweat drops and appeared to be more or less dry. The sweating ratio on the radiated area was now found to be 0·19, or the same substantially as before the acetylcholine was administered.

This experiment was repeated on a 4-day-old erythematous area on another subject using acetyl-β-methylcholine chloride (0·3% w/v) for 6 min. at 20 mA. under 51 V. The same very profuse sweating was observed over the zone of application of the drug, but not on the erythematous area. The sweating ratio which had been 0·93 prior to radiation, was 0·55 before and 0·84 after iontophoresis (Table 2).

TABLE 2. Effect of iontophoresis with acetyl-β-methylcholine on skin showing reduced sweating as the result of previous U.V. radiation

			Weight of sweat (mg.)		
				After radiation	
Area	No.		Before radiation (day 1)	Before acetyl-β-methyl-choline (day 4)	After acetyl-β-methyl-choline (day 4)
Left chest	1		26	38	54
	2		62	25	40
	3		32	23	28
		Total	120	86	122
Right chest	1		34	53	67
	2		55	48	40
	3		41	54	38
		Total	130	155	145
Av. ratio $\frac{\text{Left}}{\text{Right}}$			0·93	0·55	0·84

Three simultaneous pairs of sweat collections were made on two equal areas of skin over the same time (total 9 min.). Radiation and acetyl-β-methylcholine were applied to the area on the left side of the chest only.

Since neither acetylcholine nor acetyl-β-methylcholine was applied to the control areas in the above experiments, two comparisons were possible: (1) a quantitative comparison between the sweating rate of normal untreated skin (control areas) and that of the radiated areas before, and after, application of the drugs; and (2) the observed comparison between the sweating rate of normal and radiated skin, both under the influence of the drugs. The quantitative comparison (probably invalid in the case of acetylcholine because of its short-lived action) showed that acetyl-β-methylcholine produced a rise in sweating rate on the radiated area. The observed contrast between the profuse sweating caused by acetylcholine and acetyl-β-methylcholine on normal skin, and the almost dry condition of radiated areas was, however, so marked as to establish beyond question that sweat glands in the erythematous areas were relatively insensitive to both drugs, or alternatively that any increase in secretory pressure caused by the drugs was insufficient to force sweat to the surface of the skin.

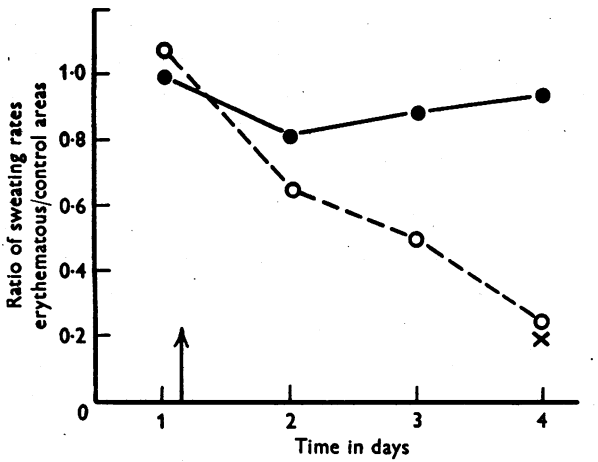
It is known that drugs most readily pass the skin barrier via the appendages (Calvery, Draize & Lang, 1946). It might, therefore, be argued that the acetylcholine and acetyl-β-methylcholine did not penetrate to the sweat-secreting epithelium of the radiated skin in the above two cases because of occlusion of the sweat ducts. It has been pointed out, however, that profuse sweating extended about 2–3 cm. beyond the edge of the electrode, and the influence of the drugs would similarly be expected to spread along the skin towards the centre of the erythematous area, which was about 4 cm. in diameter. Since the non-sweating zone was sharply limited to the erythematous area, it was concluded that either the sweat glands were relatively refractory to the drugs, or that many of the ducts were blocked.

Comparison between a black (negro) and a white skin as regards the effect of ultra-violet radiation on the sweating rate. Text-fig. 2 shows the sweating ratios of skin areas on a West African and a white subject before and after exposure to identical doses of U.V. radiation from a mercury-vapour lamp. The white subject had been exposed to radiation in the form of sun-bathing for many hours in preceding weeks, and showed definite tanning of his skin.

The white skin developed the usual sequence of changes from erythema, with slight oedema and hyperaesthesia, to pigmentation and desquamation. The black skin showed at least as much oedema as the white skin on the day following radiation, and perhaps because of this, was paler than the normal surround. Desquamation began in both cases on the fourth day; sudamina was not observed in either subject during the course of the experiment. The sweating ratio of the white skin fell from 1·10 to 0·52 on the second day after radiation, and to 0·22 on the third day. The sweating ratio of the black skin showed an insignificant reduction from 1·02 to 0·82 on the first day, was 0·89 on the second day, and 0·95 on the third day. Although the black skin reacted visibly to

radiation in much the same way as the white skin, it appeared to be more protected as regards fall in sweating rate.

Sweating rate of an irradiated area still reduced after complete desquamation. A radiated area (½ min. Kromayer lamp) which had shown reduced sweating on the fourth day had almost completely desquamated by the sixth day. It was demonstrated by Minor's (1927) method that the desquamated area still sweated less than the adjacent normal skin.



Text-fig. 2. Sweating rates of negro and white skins after the same exposure to radiation. White skin, O- - -O; negro skin, ●——●; after iontophoresis of acetylcholine (day 4), ×. Radiation was applied at the arrow.

No evidence of a direct effect on the sweat-gland acini. A layer of loose skin on the forearm was elevated and held above the surface by two strong paper clips. A Kromayer lamp was then applied to one side of the elevation. When the clips were removed the result was a roughly circular area of skin, one half of which had received an erythemal dose of radiation, and the other half only such radiation as would pass through the whole thickness of skin plus an indeterminate layer of subcutaneous tissue. Erythema with reduced sweating (Minor's method) developed only on the segment which had received direct radiation. Apparently the U.V. radiation did not penetrate to a sufficient depth to affect noticeably the acini of the sweat glands of the other segment.

DISCUSSION

The diminution in sweating rate after radiation may have been caused by any or all of the following; (*a*) blockage of the sweat-gland ducts, (*b*) failure of secretion of the sweat glands, (*c*) reduced thermal stimulus to sweating.

Blockage of the sweat-gland ducts. The occurrence of sudamina in many of the cases described in this report constituted definite evidence of occlusion of the

mouths of the sweat ducts in the necrosed layer of desquamating epithelium. This was confirmed by histological examination of radiated skin. Superficial occlusion was indicated by dilatation of the epidermal portion of the sweat-gland ducts with flattening of the lining cells.

It has been suggested that the oedema which follows U.V. radiation might have caused mechanical obstruction of the sweat gland ducts at a deeper level. Diminished sweating, however, persisted after all visible oedema had subsided. The oedema in the negro skin was not accompanied by significant depression of sweating, and the change in sweating rate on an area of massive histamine oedema was small compared with that after U.V. radiation or mustard-oil injury. Moreover, this explanation was rendered unlikely by the histological findings that dilatation of the sweat-gland ducts extended right up to the superficial layers of the epidermis.

Failure of secretion of the sweat glands. Positive evidence of diminution in the actual secretion of sweat in these cases is lacking, but the following observations support this theory. The secretory pressure of the sweat glands is said to be high; Best & Taylor (1945) give a value of 240 mm. mercury. In thermal anhidrosis (O'Brien, 1947), and in atopic dermatitis, ichthyosis and seborrheic dermatitis (Sulzberger, Herrmann & Zak, 1947) where the sweat glands are active and their ducts are occluded, vesicles or papules accompanied by pruritus usually appear when sweating commences. Such elevations were not seen at the time of minimum sweating after radiation or mustard-oil injury, nor did they occur on application of acetylcholine. Rupture of the sweat ducts, which has been shown by O'Brien (1947) to occur in thermal anhidrosis, was not noted in any of these cases. Only one subject complained of pruritus. In view of the lack of response of radiated skin to locally applied acetylcholine, it is interesting that in idiopathic heat stroke (Hearne, 1932) and also in thermogenic anhidrosis (Wolkin, Goodman & Kelley, 1944) the parasympathomimetic drug pilocarpine did not cause visible increase in sweating. A less regular return to normal of the curve of reduced sweating (Thomson, 1951) might be expected if superficial blockage were the only cause, since the obstructing layer of damaged epidermis usually comes off, or is removed, in large pieces. It has been shown above that the sweating rate does not return to normal after complete desquamation of one layer. There is, therefore, indirect evidence that the secretory pressure of sweat is diminished by U.V. radiation; some possible causes for this are now discussed.

X-rays suppress the function of sweat and sebaceous glands by direct action on the secreting cells or by interfering with their blood supply (Jadassohn, 1929; Borak, 1936). The action of X-rays is more drastic than U.V. rays, and readily leads to permanent anhidrosis, with complete disappearance of gland tissue after a strong dose. In the case of U.V. radiation any damage sustained by the secreting epithelium is unlikely to be caused by direct action, since,

apart from the inconclusive experiment described above, there is overwhelming evidence against penetration of such radiation to the depth at which the sweat-gland acini are situated (Blum, 1945; Lacassagne, 1945).

The similarity between the effects of mustard oil and U.V. radiation suggests that diminished sweating should perhaps be listed along with Lewis's (1927) other signs of skin injury. The behaviour of skin subjected to very large doses of histamine did not, however, support the theory that an H-like substance was responsible for this particular effect. The diminution in sweating rate produced was much less than had been anticipated, and may have been caused by mechanical obstruction due to the massive histamine oedema.

Although little is known about the blood supply to the corium of erythematous skin, it is thought unlikely that the blood flow to the sweat gland acini was so reduced as to impair sweat secretion. Kuno (1934) has shown that complete occlusion of the artery to a limb must be maintained for 20 min. or so before a significant reduction in sweating rate takes place. It is reported that obliterative vascular disturbances, e.g. Raynaud's or Buerger's disease, have little influence on sweating (Burch & Sodeman, 1944). Sheard (1935) believes that the blood flow to the skin is *increased* after radiation. This increase is thought by Laurens & Foster (1937) to explain the diminished temperature gradient found by these authors in radiated skin between the surface temperature and that of deeper layers (16 mm.).

It is therefore concluded that the secretory pressure of sweat is more likely to have been affected by diffusion of toxic products from the injured epidermis than by direct action of the radiation, or by alterations in blood supply to the glands.

Reduced thermal stimulus to sweating. The skin temperature on an area rendered erythematous by U.V. radiation at ordinary environmental temperatures may be as much as 2·4° C. (4·3° F.) higher than that of normal skin close by (Koch, 1947). This would have the effect of favouring heat loss to an environment of which the temperature was lower than that of the skin, and might have resulted in a less than normal stimulus to sweating. The extra heat lost in this way would, however, be inappreciable or absent under the conditions of these trials where the gradient between the skin and surroundings was small. In any case, the slight response obtained to the strong dose of acetylcholine appears to rule out the possibility that changes in the temperature gradient play other than a minor role in causing diminished sweating after radiation.

Racial differences in susceptibility to ultra-violet radiation

Hausser & Vahle (1927) found that ten times as much radiation was required to produce the same degree of erythema in a Togo negro as in white persons. The results obtained above suggest that the negro skin is protected in some way against the effect of U.V. radiation on the sweating rate.

In view of the alleged secondary role played by melanin in conferring protection against U.V. radiation it has been difficult to demonstrate that the skin of the coloured races equips them better than that of the white races for life in the tropics. There is no significant difference in the temperature gradient of white and negro skins when submitted to infra-red rays (Laurens & Foster, 1937). Guillaume (1926) and Miescher (1930) have shown that relative insensitivity to U.V. radiation is mainly acquired by thickening of the corneum. Negro skins might therefore be expected to have thicker horny layers, but Miescher (1931) found no constant difference between the two races in this respect. The black skin is apparently at a disadvantage in that it absorbs a greater proportion of heat rays and reflects less than a white skin (Martin, 1930). On the other hand, the coloured races have a larger number of sweat glands (Glaser, 1934) and sebaceous glands (Homma, 1926) per unit area of skin than white races, and the individual glands are said to be larger (Daubler, 1900). It has also been shown by Miescher (1931) that even after long residence in a temperate climate, the melanin in an Ethiopian's skin is not confined to the melanoblasts in the basal layer as it is in the skin of unhabituated white persons. It is distributed plentifully throughout all layers up to the corneum affording protection to the prickle cells which otherwise would sustain direct damage from U.V. light. It appears probable that these differences in pigmentation account for the relative insensitivity of a negro's sweat function to the effects of U.V. radiation.

SUMMARY

1. The vesicular rash found in many subjects after ultra-violet radiation provided evidence that blockage of sweat gland ducts was at least partly responsible for the diminished sweating which followed the radiation. The rash was identified as sudamina or crystallina. Occlusion of a proportion of the sweat-gland ducts in radiated skin was confirmed histologically.

2. Blockage of sweat-gland ducts, however, appears not to be the sole factor responsible for the decreased sweating. Indirect evidence, in fact, suggests that the secretion itself is reduced. Since it is unlikely that the ultra-violet rays have penetrated to the sweat glands and impaired their function, it is suggested that the reduced sweat secretion is brought about by toxins which have diffused from the injured epidermis.

3. The skin injury produced by mustard oil also caused a fall in sweating rate similar in extent and duration to that produced by U.V. radiation. The slight reduction of sweating rate caused by locally applied massive doses of histamine suggested that the above effect of U.V. radiation and mustard oil was not mediated through the release of an H-like substance.

4. Acetylcholine and acetyl-β-methylcholine locally applied did not cause a return to normal of the sweating rate after radiation.

5. The skin of a negro showed no significant alteration in sweating rate after a dose of radiation which caused marked reduction of sweating in a white skin.

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EXPLANATION OF PLATE I

Fig. 1. Sudamina on the chest (near the nipple) after exposure to the sun in England.

Fig. 2. Sudamina on the skin after exposure to the sun in England. In the area indicated the vesicles are discrete; elsewhere they are coalescing with separation of the injured layer of epidermis.

Fig. 3. A normal sweat-gland duct from the non-irradiated half of the same biopsy specimen as D and E. (× 320.)

Fig. 4. Skin of chest showing extensive injury to the epidermis 3 days after radiation. The superficial coil of the duct (*a*) appears to be blocked. (× 320.)

Fig. 5. From the same section as fig. 4. The damaged layer of the epidermis has been raised forming a vesicular space (sudamina). The lumen of the duct is enlarged and its lining cells are flattened. (× 320.)

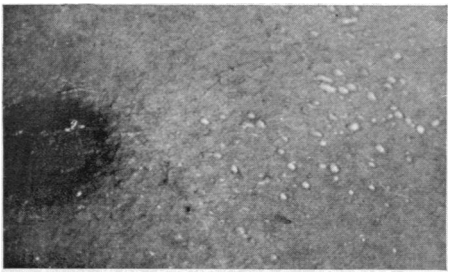

Fig. 1.

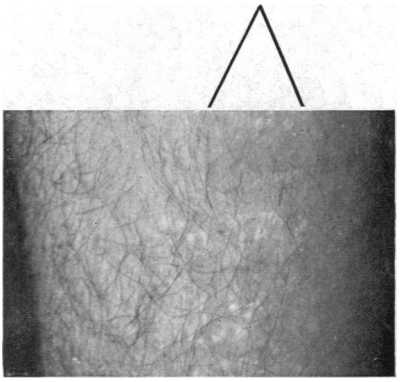

Fig. 2.

Fig. 3.

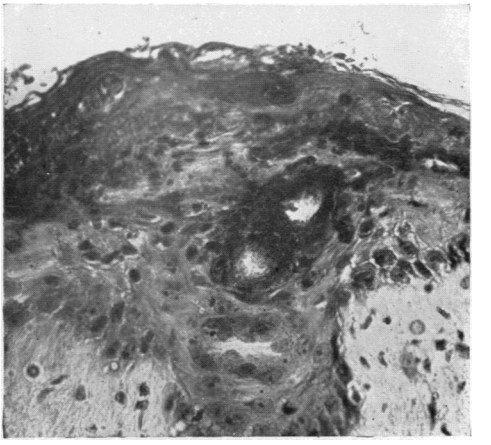

Fig. 4.

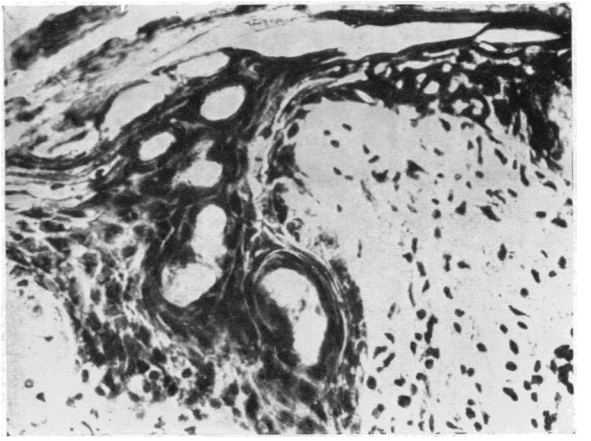

Fig. 5.

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