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Alterations in Sweat Response in Skin Lesions of Leprosy

A Dermometric Study*

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Sweat response and tactile sensibility has been studied by the use of dermometry and strength of tactile stimulus in the skin lesions of 45 leprosy patients. Tuberculoid lesions showed severe impairment to sweating, while the majority of lepromatous macules showed a fairly normal sweat response. The sweat response in borderline leprosy appeared variable. There was a close correlation between alteration in sweat response and impairment of tactile sensibility in these lesions.

Anhidrosis as a clinical manifestation of leprosy has been well recognized and adequately documented (Duhring, 1877; Jeanselme and Giraudeau, 1931; Degotte, 1942). It is characterized clinically by the absence of visible sweating on the affected skin in the presence of an appropriate stimulus and environment. A study of anhidrosis therefore immediately leads to the problems of (1) stimulating the sweat glands, and (2) estimating the degree of sweat response. The sweat glands may be stimulated by cholenergic drugs. They may also be stimulated to produce thermoregulatory sweating by elevation of body temperature. The sweat response may be judged by direct visualization (Kahn and Rothman, 1942), by various colorimetric procedures (Minor, 1927; Gutmann, 1940; Oden and Holstein, 1954; Karat *et al.*, 1969) and by measurement of electrical skin resistance (Richter and Katz, 1943).

Muir (1938) applied the pilocarpine test and Arnold (1944) the mecholyl test to study the sweat response in leprosy. Wade (1954) in an editorial entitled "Neglected Electrical Testing" records some observations made by Suskind who, using a neurodermometer on 9 patients, found a neat correlation between loss of sweat response and a clear-cut measurable rise in electrical skin resistance. These observations of changes in sweat response have often been used to record sensory changes in denervated skin (Moberg, 1958).

The purpose of this paper is to present the technique of dermometry as used to study the sweat response in skin lesions in various types of leprosy. It also attempts to find a correlation, if any, between sweat response and tactile sensibility in these areas of skin.

NEUROHISTOLOGY OF SKIN LESIONS OF LEPROSY

The neurohistology of skin lesions of leprosy has been studied and reported by Decoud (1948), Gass and Balasubrahmanyan (1954) and more recently by Dastur (1955) and Weddell *et al.*, (1964). Dastur considers the density of innervation greatest in "normal" skin, rather less dense in skin from "lepromatous" lesions and least dense in skin from "neural" lesions. He states that "in anaesthetic patches, no nerves were seen and it was significant that in such specimens the cells of sweat and sebaceous glands as well as smooth muscle fibres could no longer be identified".

Weddell maintains that in established untreated tuberculoid lesions the tissues of the dermis are grossly disorganized but that the only structures which appear to be completely destroyed are the neural elements. He remarks that in established untreated lepromatous lesions, there is, as far as can be judged, a full

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complement of axons. He describes the neurohistological picture in these lesions as that of ischaemia with a minimal amount of nerve destruction. In borderline leprosy, he suggests, the pictures seen form a series between those in tuberculoid lesions and those in lepromatous lesions; those in the borderline to tuberculoid spectrum show a greater tendency towards destruction of neural elements, while those in the borderline to lepromatous spectrum show less damage to axis-cylinders and more of the changes which are seen following ischaemia.

MATERIAL

In this study, in which 45 leprosy patients took part, the series included 10 cases of tuberculoid leprosy, 10 of lepromatous leprosy, 20 of borderline leprosy (of which 10 were borderline to tuberculoid (BT) and 10 were borderline to leproma (BL)), and 5 cases of indeterminate leprosy. Since interpretation of the results requires experience with normal individuals under similar experimental conditions, 15 normal subjects were also included to study "area differences", individual variations in sweat response, and tactile sensibility.

METHODS

Heating

Thermoregulatory sweating was produced by local application of heat, using radiant heat lamps, or by placing the patient in direct sunlight to raise the skin temperature to about 38°C. Visible sweating occurred within 7 to 10 min. under these conditions; the patient was then removed to a closed room to minimize evaporation of sweat and the skin lesions were tested.

Dermometry

It has been known for some time that denervated dry skin and normally moist skin show distinct differences in electrical resistance. The study of this phenomenon, called dermometry, was popularized by Richter and Katz in 1943, and the dermometer used in our experiments was similar to that used by those workers for their study. The fixed electrode consists of a zinc plate about 6 cm square which is fastened to a sweating area, usually the axilla. The movable electrode consists of a copper rod about 5 mm in diameter with an insulated handle. The electrodes are connected to a box which contains a microammeter reading 0 to 50, 2 rheostats, a switch, and a 4.5 V dry battery.

The movable electrode is pressed lightly against the skin to be tested. By means of the variable resistance in the instrument the current flowing through the completed circuit is adjusted so that only a minimal flow is allowed. The movable electrode is then moved over the area of the skin lesion and the deflections of the microammeter, if any, carefully noted. When a sudden change in the flow of current was found, the area was further explored until the line of demarcation between areas of high and low resistance was located. The point was then marked with a skin marking pencil. When a second line of demarcation was determined, a line was drawn between the 2 points. The process was repeated until finally the entire areas of high and low resistance were defined. Often the change from high to low resistance occurred within a few millimetres of each other. Over non-sweating, dry skin no reading was obtained. The movable electrode was then passed over a comparable area of apparently normal skin on the contralateral side and the needle deflections again carefully recorded. These areas were then tested for tactile sensibility.

Tactile sensibility

In all cases the areas to be tested were shaved before the application of stimuli. Threshold stimulus in skin lesions and mirror areas on the corresponding sides of the body was determined by using the bending pressure of 1-in. long nylon filaments graded from 1 to 5 according to increase in diameter of the filament. The subjects, who remained seated in a relaxed position, were asked to keep their eyes closed during the application of stimuli and to point to the spot when a touch was felt. Stimulation was commenced with the filament of smallest diameter and was progressed to the larger diameter filaments in each case.

RESULTS

Table 1 shows the results of sweat tests in microamperes in 15 normal individuals from corresponding areas of skin on either side of the body.

TABLE 1
Normal subjects

Area tested	*Right (mean) microamps*	*Left (mean) microamps*
Face (cheek)	50	50
Axilla	50	50
Chest	48	47
Back	47	46
Buttock	46	42
Thigh (anterior)	30	30
Abdomen	48	48
Forearm (anterior)	27	26
Knee	10	8
Elbow (posterior)	5	5

Table 2 shows the results of sweat tests conducted on skin lesions and normal areas of skin in 10 patients with tuberculoid leprosy. In all cases tactile sensibility was tested in the corresponding areas.

TABLE 2
Tuberculoid leprosy

Over skin lesions		*Over control areas*	
Sweat response (microamps)	*Loss of tactile sensibility 1–5 nylon*	*Sweat response (microamps)*	*Loss of tactile sensibility 1–5 nylon*
0	1–5	30	Nil
0	1–5	50	Nil
0	1–5	50	Nil
0	1–5	50	Nil
0	1–5	50	Nil
0	1–5	50	Nil
0	1–5	50	Nil
0	1–5	25	Nil
0	1–5	30	Nil
0	1–5	40	Nil

Table 3 shows the results of sweat tests in 10 cases of borderline leprosy in the tuberculoid spectrum (BT), with corresponding tests for tactile sensation in skin lesions and apparently normal areas.

TABLE 3
Borderline-Tuberculoid leprosy

Over skin lesions		*Over control areas*	
Sweat response (microamps)	*Loss of tactile sensibility 1–5 nylon*	*Sweat response (microamps)*	*Loss of tactile sensibility 1–5 nylon*
5	1–3	50	Nil
30	1	50	Nil
20	Nil	30	Nil
30	1	50	Nil
*0/10	1–5/1–3	50	Nil
30	1	50	Nil
*0/20	1–5/1–4	30	Nil
*0/20	1–5/1–3	25	Nil
*10/20	1–3/1–2	25	Nil
0	1–5	25	Nil

Correlation coefficient $(r)=0.911$; $P<0.001$.
*These skin lesions showed a variation in sweat reponse within the area tested and 2 readings were obtained in each case. A corresponding variation in tactile sensibility was also recorded as shown in the tabulation.

Table 4 records the results of sweat tests in 10 cases of borderline leprosy in the lepromatous spectrum with results of tests for tactile sensibility in skin lesions and apparently normal areas.

TABLE 4
Borderline-Lepromatous leprosy

Over skin lesions		*Over control areas*	
Sweat response (microamps)	*Loss of tactile sensibility 1–5 nylon*	*Sweat response (microamps)*	*Loss of tactile sensibility 1–5 nylon*
50	Nil	50	Nil
30	Nil	30	Nil
10	1–5	50	Nil
20	Nil	25	Nil
15	Nil	20	Nil
10	1–2	20	Nil
5	1–3	20	Nil
10	1–2	50	Nil
5	1–2	30	Nil
50	Nil	50	Nil

Correlation coefficient $(r)=0.739$; $0.01<P<0.02$.

In Table 5 the results of sweat tests and tactile sensibility tests conducted in lepromatous macules are recorded and compared with those in apparently normal areas on the contralateral side.

TABLE 5
Lepromatous leprosy

Over skin lesions		*Over control areas*	
Sweat response (microamps)	*Loss of tactile sensibility 1–5 nylon*	*Sweat response (microamps)*	*Loss of tactile sensibility 1–5 nylon*
15	1–3	30	Nil
20	Nil	20	Nil
0	1–5	20	Nil
25	Nil	30	Nil
20	Nil	25	Nil
20	Nil	25	Nil
20	Nil	20	Nil
0	1–5	15	Nil
30	Nil	30	Nil
25	Nil	30	Nil

Correlation coefficient (r)=0.946; $P<0.001$.

Table 6 records the results of sweat tests and tactile sensibility tests in skin lesions and apparently normal areas in 5 cases of indeterminate leprosy.

TABLE 6
Indeterminate leprosy

Over skin lesions		*Over control areas*	
Sweat response (microamps)	*Loss of tactile sensibility 1–5 nylon*	*Sweat response (microamps)*	*Loss of tactile sensibility 1–5 nylon*
0	1–5	20	Nil
15	Nil	15	Nil
20	Nil	20	Nil
20	Nil	20	Nil
20	Nil	20	Nil

Correlation coefficient (r)=0.968; $0.001<P<0.01$.

COMMENTS

Sweat glands are governed by the sympathetic nervous system, through post-ganglionic fibres from the thoraco-lumbar sympathetic plexus. Under ordinary circumstances generalized sweating occurs in response to a rise in the environmental temperature and this is regulated by the anterior hypothalamus. Emotional sweating is caused by pain, anxiety, and such other emotional changes and is known to occur in the palm of the hand and sole of the foot. The final stimulus is mediated by the release of acetylcholine at nerve endings whereby the sweat gland is stimulated to activity.

In the 15 normal healthy control subjects studied it was found that there was a variation in sweat response in different parts of the body. Thus, the response appeared to be high in certain areas such as the axilla, chest, back, abdomen and face. It was particularly low at the point of the elbow and in front of the knee. It was also observed that sweating varied in "mirror areas" in the same individual. These observations were similar to those previously reported (Shelley *et al.*, 1950).

In the 10 cases of tuberculoid leprosy (Table 2) there was complete absence of sweat response in the skin lesions, with a clear line of demarcation between the lesion and the surrounding skin as indicated by deflection of the microammeter needle. In all cases there was complete loss of touch to all grades of nylon filament (1 to 5). This picture of loss of touch concurrently with loss of sweat response suggests destruction of nerve elements in the affected area. The loss of sweating, however, could be in part due to destruction of sweat glands by tuberculoid granulomatous infiltration (Sato, 1938; Arnold, 1948).

In the 10 cases of borderline leprosy in the tuberculoid spectrum (Table 3), the sweat response in the skin lesions was less than that in the apparently normal areas in all cases. It was also noted that there was some degree of loss of tactile sensibility in every case. It was interesting to record in 4 patients with this type of skin lesion a variation of sweat response in the same lesion with a corresponding loss of tactile sensibility to various grades of nylon filaments. The pattern of sensory changes generally followed the pattern of sweat response in these lesions. The decreased sweat response associated with impairment of tactile sensibility suggests that there is partial destruction of nerve elements to a variable extent within the lesion. In this group of patients sweat response and tactile sensibility were found to be closely correlated (r=0.911).

Among the 10 borderline cases in the lepromatous spectrum (Table 4), a diminished sweat response was found in 5 skin lesions. Loss of

tactile sensibility to various grades of nylon filaments was also noted within these lesions. The other 5 skin lesions showed a normal sweat response with no loss of tactile sensibility. In this group there is probably less direct involvement of nerve endings in the skin lesion until late in the course of the disease, with the result that the lesions still retain a fair amount of tactile sensibility and the ability to sweat. These skin lesions showed a fair degree of correlation between sweat response and tactile sensibility ($r=0.739$).

In macular leproma, the sweat response and tactile sensibility were similar in the lesion and in apparently normal skin in 7 out of a total of 10 cases (Table 5). Since in lepromatous leprosy the disease is widely disseminated and large areas of skin are involved, the sweat response was further compared with that of healthy controls and found to be similar. Three skin lesions showed a marked reduction in sweat response and tactile sensibility. These lesions could have deteriorated from the borderline or indeterminate zone to the lepromatous zone. Loss of tactile sensibility and sweat response were found to be highly correlated in this group of lesions ($r=0.946$).

In 5 cases of indeterminate leprosy, loss of both sweat response and tactile sensibility was recorded in only one instance. The remaining 4 patients showed a normal sweat response and tactile sensibility within the skin lesions. This suggests that local destruction of nerve endings in skin lesions of the indeterminate type is relatively uncommon and probably a late manifestation. In this group also, sweat response and tactile sensibility were found to be highly correlated ($r=0.968$).

This study suggests a fairly good correlation between loss of tactile sensibility in a skin lesion in leprosy and loss of sweating in the same lesion when tested by dermometry. The study also suggests the existence of a gradation in loss of tactile sensibility which corresponds to a gradation in the degree of sweat loss. One may therefore infer that the major cause of interference with sweat response in specific skin lesions of leprosy is probably the loss of autonomic nerve supply rather than specific involvement of sweat glands in the granulomatous inflammatory process.

SUMMARY

Sweat response and tactile sensibility have been studied by the use of dermometry and strength of tactile stimulus as represented by 5 grades of nylon filaments in the skin lesions of 45 patients with tuberculoid, borderline, lepromatous and indeterminate leprosy.

Practically all tuberculoid lesions showed severe impairment of sweating whereas the majority of lepromatous macules showed a fairly normal sweat response. A normal sweat response was also noted in the majority of indeterminate lesions.

The sweat response in borderline leprosy appeared to be variable. Diminished sweat response was more frequently recorded in borderline-tuberculoid lesions than in borderline-leproma lesions.

There was a close correlation between alteration in sweat response and impairment of tactile sensibility in these lesions, suggesting that the alteration in sweat response in skin lesions of leprosy is probably mostly due to loss of autonomic supply rather than to destruction of sweat glands.

The simplicity and accuracy of the technique makes it a useful objective method of testing sweat response and tactile sensibility in the skin lesions of leprosy.

It is suggested that dermometry be employed to determine tactile sensibility in those patients, such as children and the aged, whose co-operation is generally difficult to obtain.

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