MECHANISM OF MUSCULAR WASTING IN LEPROSY

S. N. Chatterjee, M.B., D.T.M.

Lately of the Leprosy Research Department
School of Tropical Medicine
Calcutta, India

INTRODUCTION

In my article on muscular wasting in leprosy and its peculiarities ([4]) I showed that muscular wasting in leprosy could not always be explained as due to degeneration of the motor nerve. There were reasons to suspect that some diminution of the blood circulation took place in the affected muscles which later caused their malnutrition and wasting. As direct observation was not possible to prove this and as the temperature of a part depends on its blood circulation, I undertook with the help of a galvanometer and a thermoneedle to investigate the muscle temperature in leprosy cases. The results are reported in this paper.

MATERIALS AND METHODS

A light-trace galvanometer made by Hartmann and Braun, as previously described ([3]), and a thermoneedle were used for detection of muscle temperature.

Some of the cases reported in my previous paper ([4]) were available for this study (marked with asterisks in the tabulations), but as their number was insufficient other similar cases were also selected. There were 44 cases with muscular wasting, and they are included in Group I. Another 20 cases without muscular wasting in the hand but with thickening of the ulnar nerve of one side, were selected for the temperature study of the fourth dorsal interosseus muscle. They are included in Groups II and III.

The experiment was done during the winter months when the difference between body and room temperatures was most marked. The testing of the affected and the unaffected muscles of a patient was done at the same time and at the same room temperature. The parts to be tested were free from reacting skin lesions, or from local inflammation due to any other cause. The skin and the needle were cleaned with alcohol, which was allowed to evaporate completely before the skin was pierced; any trace of alcohol on the skin or needle adversely affected the temperature record because of its cooling effect. The most wasted part was tested. The same length of the needle was thrust into the muscle on either side. Usually one-fourth to one-half inch of the needle was pushed in perpendicularly, according to the thickness of the muscle. In case of the orbicularis oculi, which is thin, about one-fourth inch of the needle was pushed in subcutaneously in the lower lid at an acute angle. Fig. 1 shows the galvanometer and the thermoneedle in position while testing the thenar muscles of the right hand which were wasted.

GROUP I. TEMPERATURE OF WASTED MUSCLES

The muscles tested, wasted on one side and normal on the opposite side (control) are divided into six subgroups: (*a*) face, (*b*) shoulder, (*c*) upper extremity, elbow and the upper part of the forearm (*d*) do, hand, (*e*) lower extremity, thigh, and (*f*) do, lower leg. The results are presented in the following tabulation.

Case number	Part affected by wasting	Parts tested (wasted and normal)	Temperature records	Difference
A. *Face and scalp*				
In one case it was possible to test the temperature of the frontal belly of the occipitofrontalis muscle, which was paralyzed. In 8 cases I tested the orbicularis oculi, which was paralyzed causing lagophthalmos. In case of lagophthalmos the lower eyelid was found to be more affected than the upper one; so the temperature of the palpebral part of the muscle in the lower eyelid was tested in all cases.				
1. (4347)	Right lagophthalmos	Right eyelid Left eyelid	32.0°C 32.5°C	0.5°C
2. (4374)	Right frontal belly, occipitofrontalis, paralysis	Right belly Left belly	32.0°C 33.5°C	1.5°C
3. (4170)*	Right lagophthalmos	Right eyelid Left eyelid	33.5°C 34.0°C	0.5°C
4. (4346)*	Right lagophthalmos	Right eyelid Left eyelid	32.7°C 33.7°C	1.0°C
5. (4376)*	Left lagophthalmos, slight	Left eyelid Right eyelid	33.0°C 33.0°C	None
6. (4348)*	Right lagophthalmos	Right eyelid Left eyelid	33.0°C 33.5°C	0.5°C
7. (2047)*	Left lagophthalmos	Left eyelid Right eyelid	31.0°C 32.0°C	1.0°C
8. (4315)*	Right lagophthalmos, very slight	Right eyelid Left eyelid	33.5°C 33.5°C	None
B. *Shoulder*				
1. (2828)*	Right scapular	Right scapular Left scapular	34.5°C 35.5°C	1.0°C
2. (3919)°	Left scapular	Left scapular Right scapular	32.7°C 33.2°C	0.5°C
3. (9391)*	Left scapular	Left scapular Right scapular	32.0°C 32.5°C	0.5°C
4. (3698)*	Right scapular	Right scapular Left scapular	33.3°C 34.0°C	0.7°C
C. *Upper extremity, elbow and forearm*				
1. (2365)*	Right elbow and below, ulnar side	Ulnar side, right, 2″ below the olecranon Do, left	32.5°C 33.0°C	0.5°C
2. (4288)°	Left elbow, forearm and hand	Radial side, 2″ below olecranon, left Do, right	30.2°C 30.7°C	0.5°C
3. (3935)°	Left forearm	Radial side, 2.5″ below olecranon, left Do, right	31.0°C 31.5°C	0.5°C

Case number	Part affected by wasting	Parts tested (wasted and normal)	Temperature records	Difference
	D. *Upper extremity, hand*			
	In two cases (Cases 1 and 2) there was wasting of thenar muscles and their temperatures were tested. In all other cases (Cases 3 to 20) there was wasting of the ulnar group of muscles, and I tested the fourth dorsal interosseus muscle, which was usually the worst sufferer. About one-half inch of the needle was thrust into the dorsum of the hand between the fourth and the fifth metacarpal bones.			
1. (3698)	Left thenar	Left thenar	28.4°C	2.6°C
		Right thenar	31.0°C	
	(Left median nerve enlarged, 1+)			
2. (1523) Fig. 2	Right thenar	Right thenar	29.5°C	1.5°C
		Left thenar	31.0°C	
	(Right median nerve enlarged, 1+)			
3. (4288)°	Left hand, with deformity	Left hand	30.0°C	2.5°C
		Right hand	32.5°C	
	(Left ulnar nerve enlarged, 2+)			
4. (3935)°	Right hand	Right hand	29.0°C	3.0°C
		Left hand	32.0°C	
	(Right ulnar nerve enlarged, 1+)			
5. (4252)°	Left hand, with deformity	Left hand	28.0°C	4.0°C
		Right hand	32.0°C	
	(Left ulnar nerve enlarged, 3+)			
6. (1758)	Left hand, with deformity	Left hand	33.7°C	0.7°C
		Right hand	33.0°C	
	(Left ulnar nerve enlarged, 1+)			
7. (4328) Fig. 3	Left hand, with deformity	Left hand	33.5°C	1.0°C
		Right hand	34.5°C	
	(Left ulnar nerve enlarged, 2+)			
8. (4189) Fig. 4	Right hand, with deformity	Right hand	27.5°C	2.0°C
		Left hand	29.5°C	
	(Both ulnar nerves enlarged, 1+)			
9. (4170)	Left hand, slight wasting	Left hand	33.5°C	0.5°C
		Right hand	34.0°C	
	(Left ulnar nerve enlarged, 3+)			
10. (3610)	Right hand	Right hand	29.5°C	1.0°C
		Left hand	30.5°C	
	(Right ulnar nerve enlarged, 1+)			
11. (1432)	Left hand, with deformity	Left hand	31.5°C	2.9°C
		Right hand	34.4°C	
	(Left ulnar nerve enlarged, 2+)			
12. (2716)	Right hand	Right hand	29.5°C	2.5°C
		Left hand	32.0°C	
	(Right ulnar nerve enlarged, 2+)			
13. (3227)	Left hand, with deformity	Left hand	28.0°C	0.5°C
		Right hand	28.5°C	
	(Left ulnar nerve enlarged, 2+)			

Case number	Part affected by wasting	Parts tested (wasted and normal)	Temperature records	Difference
14. (2692)	Right hand	Right hand	29.0°C	4.0°C
		Left hand	33.0°C	
	(Right ulnar nerve enlarged, 1+)			
15. (3531)	Left hand	Left hand	30.5°C	1.3°C
		Right hand	29.2°C	
	(Left ulnar nerve enlarged, 2+)			
16. (3042) Fig. 5	Left hand, generalized	Left hand	29.8°C	0.7°C
		Right hand	30.5°C	
	(Left ulnar nerve enlarged, 1+)			
17. (3484)	Right hand, with deformity	Right hand	29.2°C	1.3°C
		Left hand	30.5°C	
	(Right ulnar nerve enlarged, 2+)			
18. (4031)	Right hand, with deformity	Right hand	30.5°C	1.5°C
		Left hand	32.0°C	
	(Right ulnar nerve enlarged, 3+)			
19. (1142)	Left hand, with deformity	Left hand	32.5°C	0.5°C
		Right hand	33.0°C	
	(Left ulnar nerve negative; slightly thickened)			
20. (4372)	Left hand, with deformity	Left hand	22.0°C	1.0°C
		Right hand	23.0°C	
	(Left ulnar nerve enlarged, 4+)			
21. (4340)	Left hand, generalized	Left hand	29.5°C	2.0°C
		Right hand	31.5°C	
	(Left ulnar nerve enlarged, 1+)			
E. *Muscles of lower extremity, thigh*				
1. (4231)*	Left thigh, outer side, 5″ above patella	Left thigh	33.5°C	0.5°C
		Right thigh	34.0°C	
2. (3109)*	Right thigh, inner side, near knee	Right thigh	31.5°C	0.9°C
		Left thigh	32.4°C	
F. *Muscles of lower extremity, leg*				
1. (3793)*	Right leg, knee and thigh; right foot-drop	Dorsiflexors, 5″ below patella, right leg	29.0°C	0.5°C
		Do, left leg	29.5°C	
2. (1428)	Left leg, with foot-drop	Dorsiflexors, 5″ below patella, left leg	34.0°C	1.0°C
		Do, right leg	35.0°C	
3. (2697)*	Right dorsiflexors, with foot-drop	Dorsiflexors, 5″ below patella, right leg	30.5°C	0.7°C
		Do, left leg	31.2°C	

Case number	Part affected by wasting	Parts tested (wasted and normal)	Temperature records	Difference
4. (1560)*	Left leg, with foot-drop	Dorsiflexors, 6″ below patella, left leg	31.8°C	0.9°C
		Do, right leg	32.7°C	
5. (4107)*	Right leg, with foot-drop	Dorsiflexors, 7″ below patella, right leg	29.5°C	1.0°C
		Do, left leg	30.5°C	
6. (2263)°	Right leg, with foot-drop	Dorsiflexors, 4″ below patella, right leg	32.0°C	0.5°C
		Do, left leg	32.5°C	

The data here recorded reveal clearly that the temperature of the wasted muscles is usually lower than that of the corresponding normal muscles. In 42 out of 44 cases (i.e., 95.4%) the wasted muscles recorded lower temperatures varying from 0.5° C to 4.0° C. In only 2 cases (i.e., 4.6%) was no difference detected. Both of these were cases of early lagophthalmos.

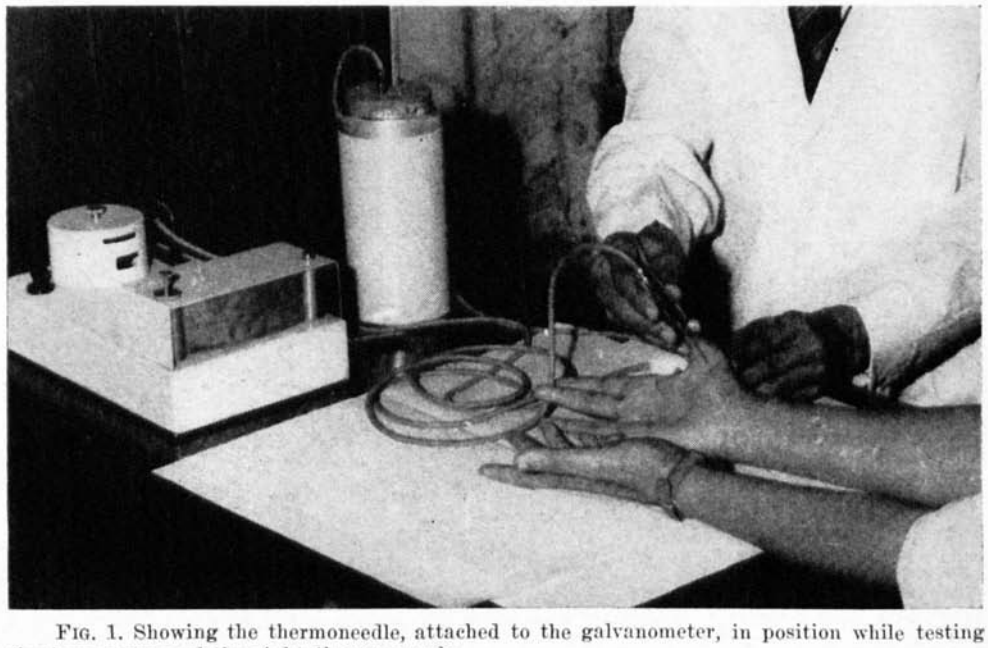

FIG. 1. Showing the thermoneedle, attached to the galvanometer, in position while testing the temperature of the right thenar muscle.

The question arises whether the lower temperature recorded in the 42 cases was the after-effect of atrophy of the muscles, or whether the atrophy was due to the lowering of temperature, i.e., due to diminution of the blood supply. To find that out, 20 cases with thickening of the ulnar nerve of one side but without wasting of the muscles supplied by it were selected for investigation . In all these cases the temperature of the fourth dorsal interosseous muscle was tested. These cases are divided into two groups, those with anesthesia and those without sensory changes.

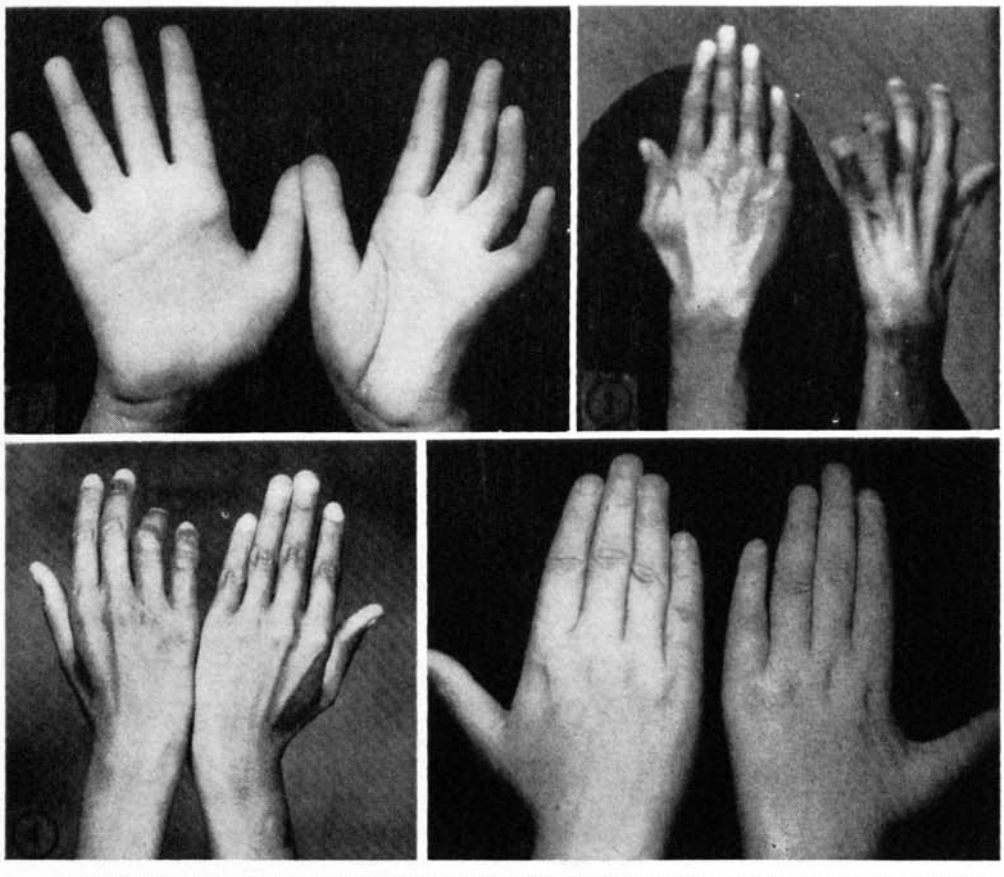

FIG. 2. Wasting of the muscles of the right thenar eminence. Difference of temperature (thenar eminences), right (atrophied) less than left by 1.5°C. (Case 1532.)

FIG. 3. Wasting of the muscles of the left hand. Difference of temperatures (fourth dorsal interossei), left (atrophied) less than right by 1.0°C. (Case 4328.)

FIG. 4. Wasting of the muscles of the right hand. Difference of temperatures (fourth dorsal interossei), right (atrophied) less than left by 2.0°C. (Case 4189.)

FIG. 5. Generalized wasting of the muscles of the left hand. Difference of temperatures (fourth dorsal interossei), left (atrophied) less than right by 0.7°C. (Case 3042.)

GROUP II. HANDS WITH ANESTHESIA

Temperature of the fourth dorsal interosseous muscle in cases hav-

ing thickening of the ulnar nerve of one side, with anesthesia of the hand supplied by it but without wasting.

Case number	Part affected	Part tested	Temperature records	Difference
1. (2898)	Left hand, with anesthesia (Left ulnar nerve enlarged, 1+)	Left hand Right hand	30.7°C 33.0°C	2.3°C
2. (3699)	Right hand, with anesthetic lesion (Right ulnar nerve enlarged, 1+)	Right hand Left hand	31.7°C 33.5°C	1.8°C
3. (3489)	Right hand, with anesthetic lesion (Right ulnar nerve enlarged, 2+)	Right hand Left hand	33.5°C 35.2°C	1.7°C
4. (2314)	Left hand, with anesthesia, ulnar side (Left ulnar nerve enlarged, 2+)	Left hand Right hand	28.2°C 29.7°C	1.5°C
5. (3602)	Right hand, with anesthetic lesion (Right ulnar nerve enlarged, 1+)	Right hand Left hand	28.0°C 29.0°C	1.0°C
6. (4226)	Right hand, with anesthetic lesion (Right ulnar nerve enlarged, 3+)	Right hand Left hand	33.0°C 34.0°C	1.0°C
7. (3450)	Right hand, with anesthetic lesion (Right ulnar nerve enlarged, 1+)	Right hand Left hand	30.0°C 30.8°C	0.8°C
8. (4223) Fig. 6	Left hand, with anesthetic lesion (Left ulnar nerve enlarged, 3+; nerve abscess)	Left hand Right hand	33.8°C 34.5°C	0.7°C
9. (3917)	Right hand, with anesthetic lesion (Right ulnar nerve enlarged, 1+)	Right hand Left hand	28.8°C 29.0°C	0.2°C
10. (405)	Right hand, with anesthetic lesion (Right ulnar nerve enlarged, 1+)	Right hand Left hand	30.5°C 30.7°C	0.2°C
11. (3882)	Left hand, with anesthetic lesion (Left ulnar nerve enlarged, 2+)	Left hand Right hand	32.7°C 32.7°C	None
12. (3934)	Left hand, with anesthetic lesion (Left ulnar nerve enlarged, 2+)	Left hand Right hand	33.5°C 33.5°C	None
13. (3433)	Right hand, with anesthetic lesion (Right ulnar nerve enlarged, 2+)	Right hand Left hand	30.0°C 30.0°C	None

GROUP III. HANDS WITHOUT ANESTHESIA

Temperature of the fourth dorsal interosseous muscle in cases having thickening of the ulnar nerve of one side, but without any anesthesia or wasting in the hand supplied by it.

Case number	Part affected	Part tested	Temperature records	Difference
1. (3960)	Right ulnar (3+)	Right hand Left hand	23.7°C 24.8°C	1.1°C
2. (4264)	Right ulnar (3+)	Right hand Left hand	27.2°C 27.8°C	0.6°C
3. (3803)	Left ulnar (1+)	Left hand Right hand	28.0°C 28.3°C	0.3°C
4. (3021)	Right ulnar (2+)	Right hand Left hand	32.4°C 32.4°C	None
5. (2329)	Left ulnar (1+)	Left hand Right hand	34.0°C 34.0°C	None
6. (4289)	Right ulnar (4+)	Right hand Left hand	28.5°C 28.5°C	None
7. (4386)	Left ulnar (3+, with abscess)	Left hand Right hand	35.5°C 35.5°C	None

ANALYSIS OF TEMPERATURE RECORDS

A summary tabulation of the degrees of lowering of the temperature records follows:

Temperature lower by	Group I (44 cases)	Group II (13 cases)	Group III (7 cases)
4.0°C	2	—	—
3.0°C	1	—	—
2.9°C	1	—	—
2.8°C	1	1	—
2.6°C	1	—	—
2.5°C	2	—	—
2.0°C	2	—	—
1.8°C	—	1	—
1.7°C	—	1	—
1.5°C	2	1	—
1.3°C	1	—	—
1.1°C	—	—	1
1.0°C	8	2	—
0.9°C	2	—	—
0.8°C	—	1	—
0.7°C	4	1	—
0.6°C	—	—	1
0.5°C	15	—	—
0.3°C	—	—	1
0.2°C	—	2	—
None	2	3	4

From this tabulation it is seen that in Group I, 42 of the 44 cases (95.4%) showed lowering of the temperature of the wasted muscles as compared with that of the corresponding normal muscles. The differences varied from 0.5°C to 4.0°C. Therefore, the wasting of muscles had some relation with their low temperature records.

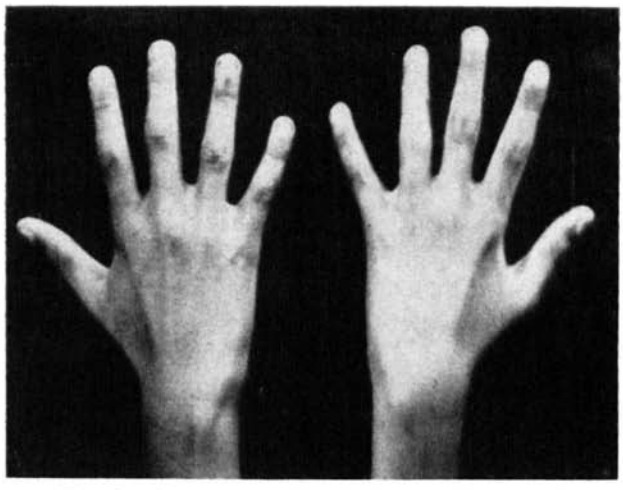

FIG. 6. Wasting of the muscles of the left hand. A small tuberculoid patch on left ring finger; left ulnar nerve (3+ involvement) with abscess. Difference of temperatures (fourth dorsal interossei), left (atrophied) less than right by 0.7°C. (Case 4223.)

In the Group II cases there was no muscular wasting in the hand, but thickening of the ulnar nerve of one side and anesthesia of the hand on the same side. In 10 out of 13 cases in this group (77.7%) the temperature of the fourth dorsal interosseous muscle in the affected hand was lower than that of the normal hand. The differences varied from 0.2°C to 2.3°C.

In the Group III cases there was no wasting or anesthesia in the hand, but the ulnar nerve of one side was thickened. In only 3 of the 7 cases in this group (32.8%) was the temperature of the fourth dorsal interosseous muscle supplied by the thickened nerve lower than that of the other side. The differences varied from 0.3°C to 1.1°C.

DISCUSSION

In a previous article ([2]) I drew attention to the fact that in leprosy the skin temperature of a deformed hand was about 1°F below normal, indicating diminution of blood circulation in the affected hand. That the blood circulation in anesthetic parts was less than that of the corresponding normal parts was confirmed in a later article ([3]). In my clinical study of muscular wasting in leprosy I found sufficient reasons to suspect that the wasting of muscles had some relation with their blood supply. This was explained in my most recent article ([4]). As direct observation of blood circulation in the muscles is not possible, and as the temperature of muscles depends on their blood circulation, I made the investigation of temperatures here reported, by means of the galvanometer and thermoneedle. In this study I had three categories of cases, and the findings in them are discussed separately.

1. In the first category there were the 20 cases (Groups II and III) in which the ulnar nerve of one side was thickened at the elbow, quite considerably in some cases, but the muscles supplied by it were not wasted. It was not known why that should be so. A study of the temperature of the muscles showed that in 7 out of the 20 cases there was

no lowering of temperature of the fourth dorsal interosseous muscle on the affected side, indicating that there was no diminution of blood supply to the muscle. This explains why there was no wasting.

In 10 of the remaining 13 cases the temperature of the fourth dorsal interosseous muscle on the affected side was lower than that of the unaffected side. This indicated that there was diminution of blood supply to the muscle on that side. In the beginning of this stage there may not be any visible muscular wasting, but continued diminished supply of blood to the muscles ultimately leads to gross muscular wasting, due to prolonged malnutrition. From this it is evident that muscular wasting in leprosy is preceded by diminution of blood supply to the muscles, causing a lowering of muscle temperature.

In cases where the nerves supplying the muscles are thickened, possibly diminution of blood supply also takes place in the nerves when the thickening subsides. Diminution of blood supply to the nerves and in consequence to the muscles is usually very slow and gradual, due to gradual narrowing of the vasa nervorum. This is why muscular wasting in leprosy is very slow and gradual. But sometimes there is ischemia of the vasa nervorum due to sudden swelling of the nerve in the phase of reaction, causing sudden diminution of blood supply to the nerves and to the muscles. This is how paralysis and wasting develop suddenly in the reaction phase of leprosy.

Usually more attention is given to the nerve supply of muscles than to the equally important matter of blood supply of the nerves and muscles. The importance of the vascular factors in the production of peripheral nerve affections has, however, been considered by various authors with reference to both clinical and experimental aspects of the problem. Kher *et al.* ([6]) found, by dissection of 16 upper limbs of human bodies, that in the region of the arm the vasa nervorum to the median nerve were more plentiful than those of the ulnar nerve. Therefore, the ulnar nerve is liable to suffer more readily from ischemic conditions.

It was previously believed that for the function of the peripheral nerves only the anatomical and physiologic continuity of the axon with the cell body was essential. Kher *et al.* mentioned the work of Bülbring and Burn([1]), who proved that whenever there was occlusion of the blood supply to the nerves there was definite impairment in their function. Ferguson and Liversedge([5]) discussed ischemia of the lateral popliteal nerve causing foot-drop. They concluded that the lateral popliteal was especially vulnerable because it receives only one nutrient vessel, whereas the medial popliteal receives a fairly rich blood supply. They referred to the work of Lewis *et al.* ([7]) who concluded that ischemia was of prime importance in neural symptoms, and that sensory manifestations in an ischemic limb were not dependent upon direct nerve compression.

The relation of the blood supply to the nerves and their functions

can be demonstrated in another way. If a tourniquet applied to the thigh is kept on for some time, the parts distal to it gradually become anesthetic and the dorsiflexion of the foot is affected. Here the nerves remain intact but the blood circulation distal to the tourniquet is disturbed, and that affects the cutaneous sensation, producing stocking-like anesthesia as seen in leprosy. The strength of the dorsiflexor muscles of the foot is also affected, simulating early foot-drop.

2. In the second category of the cases studied the nerves were thickened and the muscles supplied by them were wasted. The affected nerves were mixed nerve trunks like the ulnar, the median, the radial, and the lateral popliteal. The prevailing concept is that when the thickening of the nerve subsides, fibrosis starts and the nerve fibers are destroyed by fibrous tissue, and that the degeneration of the nerve fibers is irreversible. I am of the opinion, however, that degeneration of nerve fibers may be found in some cases but that in the majority of cases there is no degeneration, but dysfunction due to malnutrition. When the nerve lesion subsides the vasa nervorum usually become narrower, which leads to malnutrition of the nerve fibers and the muscles supplied by them; ultimately there is wasting of the muscles. That the blood supply in the muscles becomes less is reflected in the temperature of the wasted muscles, which is lower than in the corresponding normal muscles.

Had there been actual degeneration of the nerves in all the cases where there was muscular wasting, there could not be any improvement in such cases. The fact that some cases improve, partially or completely, after physiotherapy and other medical measures shows that there was no actual degeneration of the nerves in those cases, and that the condition was not irreversible. These measures had no effect other than vasodilation in the affected part. Similarly, improvement after decapsulation of a thickened nerve is due to relief of pressure of the vasa nervorum and improvement of blood circulation.

In the same way we can explain the phenomenon of sudden appearance of muscular wasting and paralysis in the phase of reaction, and their correction in some cases after subsidence of reaction. In these cases the affected nerves swell up suddenly during the reaction, and because the nerve sheath is thick and unyielding it causes ischemia of the vasa nervorum, and therefore the affected nerves and the muscles supplied by them suffer from diminution of the blood supply. After the subsidence of the reaction the swelling of the nerves subsides, and with that —if the blood circulation improves—the muscular wasting may be corrected. All these facts cannot be explained on the ground of degeneration and regeneration of nerves.

In this connection we should also consider whether it is possible for any regenerated nerve fiber to make its way through the dense granulomatous mass inside the thickened nerve trunk to reinnervate the wasted muscles.

Furthermore, some cases of muscular wasting are not associated with thickening of the nerves supplying them, and in those cases muscular wasting cannot be due to nerve degeneration. This point is well illustrated in our cases of the third category, where the nerves supplying the muscles were purely motor and they were not thickened.

3. In the third category the muscles were wasted but the nerves supplying them were not found thickened. These nerves being purely motor, there was no possibility of the infection spreading from the skin to these nerves via the cutaneous sensory nerves. Therefore, the theory of nerve degeneration is not applicable here.

Since there was no thickening of the nerves in these cases and their degeneration was therefore out of the question, the muscular wasting can be explained by our finding of a lower temperature in the wasted muscles, indicating less blood supply to them. That the diminished supply of blood was responsible for the wasting and paralysis is substantiated by a finding in the cases with lagophthalmos. Patients who had lagophthalamos could close their eyes better immediately after massaging of their eyelids. This return of strength in the orbicularis oculi and the ability to close the eyelids again was due only to improved blood circulation in the muscle after massaging.

The most interesting point in this group of cases is that the muscular wasting was usually limited to the area of the skin lesions, the wasting affecting only the muscles underlying them. The relationship of subsidence of a skin lesion and wasting of the muscle underlying it is very well seen in cases with lesions on the face. So long as a tuberculoid lesion on the face is active and very warm, there is no evidence of lagophthalmos. But with the subsidence and cooling of the skin lesion, lagophthalmos becomes evident. Therefore, it is reasonable to think that with the subsidence of the skin lesion there is diminution of blood supply to it. With the lowered temperature there is also diminution of the blood supply of the subcutaneous tissue, and the underlying muscle becomes wasted due to malnutrition. This condition is supported by the lower temperatures of the affected orbicularis oculi in cases of lagophthalmos in comparison to the corresponding normal muscle.

SUMMARY

Since muscular wasting in leprosy cases cannot always be explained as due to degeneration of motor nerves, a study of muscle temperature has been made with thermoneedle and galvanometer.

In 44 cases (Group I) with unilateral muscle wasting the temperatures of the affected muscles were compared with those of the normal contralateral muscles. In 42 of these cases (95.4%) the temperature of the wasted muscle was found to be the lower, the difference varying from 0.5°C to 4.0°C. The lowered temperatures of the atrophied muscles signify diminution of their blood supply, and that is believed to be

associated with or responsible for the atrophy. Some of these cases did not have thickening of the corresponding nerves, and nerve degeneration cannot explain the muscular wasting.

Another 20 cases without muscular wasting but with varying degrees of unilateral nerve thickening were also tested with respect to the temperature of the fourth dorsal interosseous muscles. In 13 of these cases (Group II) there was anesthesia of the hands; in the other 7 (Group III) there was no change associated with the thickened nerves. In 10 of the Group II cases, but only 3 of the Group III cases, the temperature on the affected side was lower than that on the other.

This variation of effects is explained on the basis of probable state of the nerve lesion. In the first, or earliest stage the blood supply of the thickened nerve is not affected. In the second stage there is some subsidence of the nerve lesion; it becomes less vascular and in consequence there is also diminution of the blood supply of the corresponding muscles and lowering of their temperature. In the third stage these conditions and effects are increased, with malnutrition and wasting of the affected muscles.

ADDENDUM.—It is suggested that the reader take note of the story in the News and Notes department, on the recent announcement by the Eastman Kodak Company of a new infrared method of detecting local changes of the body temperature.—EDITOR.

SUMARIO

Ya que la decadencia muscular en la lepra no siempre se puede explicar como debida a la degeneración de los nervios motores se he hecho un estudio de la temperatura muscular con un galvanómetro y con una aguja para tomar la temperatura.

En 44 casos (grupo I) con desgasto muscular unilateral la temperatura de los musculos afectados se comparó con la temperatura de los musculos contralaterales normales. En 42 de estos casos (99.4%) la temperatura de los músculos gastados se encontró ser menor, la diferencia siendo entre 0.5 grados centígrados a 4.0 grados centígrados. La temperatura menor de los musculos atrofiados significa una diminución de la provisión sanguínea y se crée, que está asociada ó es responsable por el estádo de atrófia. Algúnos de éstos cásos no tenían engrosamiento de los nervios correspondientes y la degeneración nerviosa no puede explicar el desgasto muscular.

Otros 20 casos sín desgasto muscular, pero cón varios grados de engrosamiento nervioso únilateral también fueron examinados por lo que se trata a la temperatura de los músculos correspondientes al cuarto interoseo dorsal. En trece de éstos casos (grupo II) hubo anestésia de las manos; en los otros 7 (grupo III) no se encontró cambio asociado con engrosamiento nervioso. En 10 de los casos del grupo II, y en 3 de los casos del grupo III la temperatura del lado afectado se encontró menor que en el lado opuesto. Esta variación de efectos se explica a base del estado probable de la lesion nerviosa. En el estado primario, ó séa el primero, la provisión sanguínea del nervio engrosado no está afectada. En el segúndo estado hay algo de diminución de la lesión nerviosa, se vuelve menos vascular y por consecuencia también existe una diminución de la provision sanguinea a los musculos correspondientes, y una diminución de su temperatura. En el tercer estado éstos afectos y condiciones aumentan, con malnutrición y con desgasto de los músculos afectados.

RESUMÉ

Puisque la dégénérescence musculaire chez les malades atteints de lèpre ne peut toujours être rapportée à une dégénérescence des nerfs moteurs, nous avons entrepris une étude de la température musculaire au moyen du thermocouple avec aiguille et du galvanomètre.

Dans 44 cas (Group I) atteints de dégénérescence musculaire unilatérale, les températures des muscles affectés ont été comparées avec celles des muscles normaux homologues de l'autre côté. Chez 42 de ces malades (95.4% des cas), les températures des muscles dégénérés ont été trouvées plus basses, la différence variant de 0.5°C à 4.0°C L'abaissement de la température dans les muscles atrophiés signifie que leur circulation est diminuée, et ceci est considéré comme étant soit associé avec l'atrophie, soit la cause de celle-ci. Certains de cas ne présentaient pas d'épaississement des nerfs correspondants, et la dégénérescence nerveuse ne peut expliquer la dégénérescence musculaire.

La température des quatrièmes interosseux dorsaux fut étudiée dans un autre groupe de 20 cas sans dégénérescence musculaire, mais présentant des degrés variables d'épaississement nerveux unilatéral. Chez 13 de ces malades (group II) il y avait anesthésie des mains; chez les 7 autres (groupe III) aucun changement n'était associé à l'epaississement des nerfs. Chez 10 malades du groupe II, mais chez 3 seulement du groupe III, la température du côté atteint était plus basse que celle notée de l'autre côté.

Cette divergence d'effets est expliquée sur la base de l'état probable de la lésion nerveuse. Dans le premier cas, ou lors du stade le plus précoce, l'irrigation vasculaire du nerf épaissi n'est pas troublée. Dans le second cas, il se produit une certaine consolidation de la lésion nerveuse, le nerf devient moins vascularisé, et en conséquence l'approvisionnement en sang des muscles correspondants diminue, entraînant un abaissement de leur température. Dans le troisième stade, ces conditions et leurs effets sont plus accusés, et sont marqués par la malnutrition et la dégénérescence des muscles affectés.

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REFERENCES

1. Bülbring, E. and Burn, J. H. Vascular changes affecting transmission of nervous impulses. J. Physiol, **97** (1939) 250-264.
2. Chatterjee, S. N. The mechanism of the neural signs and symptoms of leprosy. Internat. J. Leprosy **23** (1955) 1-18.
3. Chatterjee, S. N. Mechanism of blister formation in leprosy patients. Internat J. Leprosy **27** (1959) 305-320.
4. Chatterjee, S. N. Muscular wasting in leprosy and its peculiarities. Internat J. Leprosy **31** (1963) 280-302.
5. Ferguson, F. R. and Liversedge, L. A. Ischaemic lateral popliteal nerve palsy. British Med. J. **2** (1954) 333-335.
6. Kher, G. A., Ramu, G. Hussain, F. and Bose, S. Blood supply of the nerves of the upper limb. J. Indian Med. Assoc. **36** (1961) 395-396.
7. Lewis, T., Pickering, G. W. and Rothschild, P. Observations upon muscular pain in intermittent claudication. Heart **15** (1931) 359-383.