INTERNATIONAL JOURNAL OF LEPROSY

Volume 42, Number 1
Printed in the U.S.A.

A Study of Surface and Deep Temperatures Along the Course of the Ulnar Nerve in the Pisohamate Tunnel[1, 2]

C. D. Enna, H. T. Berghtholdt and F. Stockwell[3]

The degree of infiltration of human tissues by *Mycobacterium leprae* is considered by many to be temperature related, in which instances dermal lesions occur in relatively cool areas and specific segments of peripheral nerve trunks become involved at levels where the deep temperature is lower than core body temperature ([6, 11, 13, 14, 16]).

Reported temperature studies along the course of the ulnar nerve revealed a difference greater than 2°C between three selected sites: (a) proximal to the medial epicondyle of the humerus, (b) the proximal end of the cubital groove, and (c) proximal to the flexor crease of the wrist and medial to the flexor carpi ulnaris tendon, from a deeper site in the proximal third of the forearm ([14]). The position and relationship of the ulnar nerve in the distal arm are anatomically comparable with the course of the nerve at the carpal level. In both areas the nerve lies subcutaneously before entering the cubital groove at the elbow and the pisohamate tunnel at the wrist. This temperature study was done to lend clarification to certain issues. A lower temperature of the ulnar nerve in the distal arm and elbow is incriminated as one of several features that appear to promote the development of leprous neuritis at that site which is causative for the claw deformity of the hand. An equivalent low temperature affects the ulnar nerve in its subcutaneous course in the distal forearm proximal to its passing into the pisohamate tunnel. It is recognized that this is a second site of predilection for leprous infiltration of the ulnar nerve, yet very little attention has been directed to the latter process as possibly representing a similar situation to that at the elbow. Two different patterns of ulnar paralysis limited to the intrinsic hand muscles are described by Minato ([12]). These could result from leprous ulnar neuritis at the wrist, as is recognized for the median nerve which is affected at the same depth and level of the wrist with an equivalent lower nerve temperature ([14]). Moreover, a number of pathologic conditions not related to leprosy are reported involving the ulnar nerve in the pisohamate tunnel ([3, 4, 7, 8, 9, 10, 15, 18, 19, 20, 21]).

In leprosy, involvement of the ulnar nerve alone causes 52% of claw hand deformities, and its involvement in combination with the median nerve provides an additional 46% of complete claw deformity. Whereas the areas at the elbow and wrist are sites of predilection for leprous infection of the ulnar nerve, it has been empirical to treat the nerve surgically at the elbow but not the wrist.

Whether or not ulnar nerve involvement at the wrist is incriminating as a primary cause of the leprous claw hand has been discussed by Antia ([1]). He felt that the pain and tenderness elicited along the course of the ulnar nerve in the palm could be due to leprous neuritis. Studies were reported later which demonstrated a prolongation of wrist to palm latency accompanied by slowing of nerve conduction velocity. These functional changes were supported by operative findings consisting of pathologic foci of thickening, adherence, and compression of the ulnar nerve as it entered the tunnel and "gumming up" of digital nerves in the palms of 15 of 21 patients operated upon ([2, 5]).

These findings implicate leprous involvement of the ulnar nerve at the wrist as a possibly primary site for the development of claw deformity. As tissue temperature is one of multiple features which predisposes neural segments to infiltration by *M. leprae*, this study was undertaken to ascertain if any relationship exists between temperatures related to the ulnar nerve at the carpal level and the development of deformity due to paralysis of the intrinsic muscles of the hand.

[1]Revised edition received for publication 22 January 1973; original received 13 October 1972.

[2]This work was supported by Social and Rehabilitation Grant No. RC 75 MPO.

[3]C. D. Enna, M.D., F.A.C.S., Chief, Clinical Branch and Surgical Department; H. T. Berghtholdt, Research Therapist; F. Stockwell, B.S., Statistician, USPHS Hospital, Carville, Louisiana 70721.

MATERIALS AND METHODS

The surface and deep temperatures were taken at the hypothenar eminence and the pisohamate tunnel respectively in four volunteer groups. The groups consisted of (a) a nonpatient control group, (b) a patient control group, (c) a group possessing ulnar clawing, and (d) a group with complete claw deformity due to combined ulnar and median nerve paralysis. The nature and/or degree of deformity was not necessarily the same bilaterally in an individual; therefore, we felt it would be more appropriate to discuss the findings as related to the hands rather than in terms of the number of individuals studied.

All subjects were tested between 10:00 and 11:00 a.m. Smoking was restricted for one hour before examination. The room temperature was controlled by air conditioning maintained at 23.8°C ± 1° for two hours preceding the examination, and each subject was acclimated to the room temperature for 15 minutes during which time the palms were held exposed. The temperature readings were taken by the same examiner (H.T.B.), employing a thermister attached to a telethermometer (YSI Model 46 TU).[4] A YSI Flat Thermister was used to take the surface temperature, and a YSI 514 probe was used to obtain the deep temperature. All probes were checked for accuracy in a controlled temperature water bath before and after testing with calibration curves established for the YSI 514 probes for correction of the direct deep temperature read-offs. The site to apply the thermister was established by identifying the pisiform and marking a joint just medial and beyond but in line with the base of the middle finger. The surface temperature was taken prior to the deep temperature and overlying the site of insertion of the probe into the pisohamate tunnel.

RESULTS

Eighteen hands of the nonpatient control group and eleven hands of the patient control group were examined. In the paralytic groups examined, there were six hands with ulnar clawing and twenty hands with combined ulnar and median clawing.

There were no significant differences in the mean surface, deep, and deep minus surface temperatures of the nonpatient and patient control groups (Table 1); therefore, these data were pooled into one group for this study. The data on the surface and deep temperatures of the control and ulnar groups

TABLE 1. *Means and standard deviation by group and location.*

Group	Mean (°C)			Standard Deviation		
	Surface	Deep	Deep minus surface	Surface	Deep	Deep minus surface
Normal nonpatient	30.4	34.2	3.79	1.8	1.5	0.57
Normal patient	30.6	34.4	3.78	1.4	1.4	1.17
Ulnar	30.2	33.2	3.00	1.6	2.0	0.76
Median-ulnar	28.3	31.4	3.06	1.8	2.6	1.19

TABLE 2. *Simple effects of surface and deep temperatures.*

Group	Mean (°C)		Group	Mean (°C)		Mean Difference	
	Surface	Deep		Surface	Deep	Surface	Deep
Normal control	30.48	34.26	Median-ulnar	28.29	31.37	2.19	2.89
Normal control	30.48	34.26	Ulnar	30.20	33.19	0.28	1.07
Ulnar	30.20	33.19	Median-ulnar	28.29	31.37	1.91	1.82

[4]Yellow Springs Instrument Co., Yellow Springs, Ohio 45387. Company names are mentioned in this paper for the purpose of identification, and this does not imply endorsement by the U.S. Public Health Service.

TABLE 3. *Simple effects of deep minus surface temperature differences.*

Group	Mean (°C)	Group	Mean (°C)	Mean difference
Normal control	3.79	Median-ulnar	3.06	0.73
Normal control	3.79	Ulnar	3.00	0.79
Ulnar	3.00	Median-ulnar	3.06	0.06

do not differ significantly; however, they differ from the group with median and ulnar deformity (Table 2). On the other hand, the deep minus surface temperature difference for the combined control groups differs significantly from both the ulnar and median-ulnar paralytic groups (Table 3).

DISCUSSION

The findings in the clinically normal patients and the nonpatient groups were similar for surface, deep, and deep minus surface temperatures. These patients had quiescent disease and were without dermal lesions or neural deficits which might produce temperature variations.

Whereas the surface and deep temperatures noted in the ulnar paralytic group were not significantly different from the control group, the deep temperature reflects a slight change which could be encountered in early ulnar palsy, considering the varying degree of atrophy of the intrinsic musculature of the hand innervated by the ulnar nerve.

Significant change was noted in the "deep minus surface" temperature difference of both paralytic groups. In the ulnar paralytic group, clawing involved the ring and little fingers; however, atrophy of the hypothenar eminence varied in its extent. In the combined median-ulnar paralytic group in which all digits were clawed, atrophy of the ulnar innervated intrinsic muscles was always complete with partial to complete involvement of the median innervated intrinsic muscles. The hypothenar eminence tended to be flattened; however, the circulation of the ulnar aspect of the hand was impaired in all cases of ulnar paralysis, as reflected in thermal patterns of thermographic studies of these hands. It has been proposed that the fibrofatty pad and the palmaris brevis muscle protect the ulnar nerve from extrinsic trauma as it courses through the pisohamate tunnel ([17]). Assuming that the degree of infiltration of peripheral nerves by *M. leprae* is temperature related, it is proposed that protection is also provided against infection by this bacillus when a significantly high deep temperature is maintained by an intact hypothenar eminence. It is noted that a decrease in the deep temperature ranges from 1.2°C to 3.0°C less in the ulnar and the median-ulnar group respectively than in the control group.

The deep temperature accompanying the ulnar nerve in the pisohamate tunnel of the nonparalytic hand does not present a situation analagous to that encountered with the ulnar nerve at the proximal end of the cubital groove. The deep temperatures of the ulnar nerve proximal to and at the elbow are reported to be essentially the same, registering 32.8°C and 32.6°C at a 6 mm depth, the sites where the ulnar nerve is often involved in leprosy and the involvement is believed to cause claw hand deformity. Whereas the temperature at the wrist (proximal to the carpus) is registered as 33.7°C at 10 mm ([14]), the mean average temperature of the nerve in the pisohamate tunnel of the combined normal groups at approximately 10 mm depth was 34.3°C. Thus in the nonparalytic hand, the deep temperatures at the distal forearm and wrist are greater than that at corresponding levels in the distal arm and elbow.

On the other hand, in the paralytic groups a decrease in the deep temperatures within the pisohamate tunnel existed concomitantly or developed after the onset of claw deformity. Whereas the infiltration of human tissues by the *M. leprae* is probably multifactoral in origin, in considering that it is temperature related, it appears that the primary site for development of ulnar neuritis would more apt to be at the elbow as is

recognized clinically. Similarly, it appears that involvement of the ulnar nerve at the wrist is secondary since it is observed concomitantly with paralysis of the intrinsic muscles of the hand due to involvement of the ulnar nerve at the elbow.

SUMMARY

The surface and deep temperatures were taken at the hypothenar eminence and the pisohamate tunnel respectively in four groups of subjects, a nonpatient and a patient control group, a group possessing ulnar clawing, and a group with ulnar-median clawing.

The results were compared with findings reported from similar studies on the ulnar nerve at the elbow in persons without leprosy.

The results of this study suggest that the development of ulnar neuritis at the wrist is secondary to involvement at the primary site at the elbow.

RESUMEN

Se tomó la temperatura de la superficie y la temperatura profunda en la eminencia hipotenar y el tunel pisoamato respectivamente en cuatro grupos de sujetos: un grupo control formado por pacientes y un grupo control de sanos, un grupo que tenía garra cubital y un grupo con garra cubital-media.

Se compararon los resultados con hallazgos reportados en estudios similares del nervio cubital a la altura del codo en individuos sin lepra.

Los resultados de este estudio sugieren que el desarrollo de neuritis cubital en la muñeca es secundario al compromiso del sitio primario, en el codo.

RÉSUMÉ

Dans quatre groupes de sujets, un groupe de sujets sains, un groupe de malades témoins, un groupe présentant une griffe cubitale et un groupe avec une griffe cubito-médiane, on a procédé à la mesure des températures superficielles et profondes au niveau de l'éminence hypothénare et du tunnel pisiforme.

Les résultats ont été comparés avec ceux rapportés à la suite des études similaires sur le nerf cubital au niveau du coude, chez des personnes non atteintes de lèpre.

Les résultats de cette étude suggèrent que le développement d'une névrite cubitale au poignet est secondaire à l'atteinte du site primaire au niveau du coude.

REFERENCES

1. ANTIA, N. H. Personal communication.
2. ANTIA, N. H. and DASTUR, D. K. Nerve lesions in leprosy. Ann. Report of V.R.A. Project for year ending 31 January 1965. Grant No. OVE 2 NDI-22-62.
3. BROOKS, D. M. Nerve compression by simple ganglia. J. Bone Joint Surg. **34B** (1952) 391-400.
4. *Ibid.* Nerve compression syndromes. J. Bone Joint Surg. **45B** (1963) 445-446.
5. DASTUR, D. K. The peripheral neuropathology of leprosy. From the Univ. of Bombay Symposium on Leprosy. Antia, N. H. and Dastur, D. K. eds. Fort Bombay: Bombay University Press, 1967.
6. HASTINGS, R. C., BRAND, P. W., MANSFIELD, R. E. and EBNER, J. D. Bacterial density in the skin in lepromatous leprosy as related to temperature. Leprosy Rev. **39** (1968) 71-74.
7. HOWARD, F. M. Ulnar nerve palsy in wrist fractures. J. Bone Joint Surg. **43A** (1961) 1197-1201.
8. HUNT, J. R. Occupational neuritis of the deep palmar branch of the ulnar nerve. A well-defined clinical type of professional palsy of the hand. J. Nerv. Ment. Dis. **35** (1908) 673.
9. JENKINS, S. A. Osteoarthritis of the pisiform triquetral joint. J. Bone Joint Surg. **33B** (1951) 532-534.
10. KLEINERT, H. E. and HAYES, J. E. The ulnar tunnel syndrome. Plast. Reconstr. Surg. **47** (1971) 21-24.
11. LEVAN, N. E., BYSTRYN, J. C. and HYMAN, C. Temperature and blood flow in macules of lepromatous leprosy. Internat. J. Leprosy **37** (1969) 249-252.
12. MINATO, J. Functional and surgical classification of paralysis, in particular of motor paralysis in the upper extremity in leprosy. Rep. Res. Inst. Tuberculosis and Leprosy. Tohoku Univ. Series C (Medicine) **9** (1960) 345-350.
13. SABIN, T. D. Temperature-linked sensory loss. Arch. Neurol. **20** (1969) 257-262.
14. SABIN, T. D., HACKETT, E. R. and BRAND, P. W. Temperatures along the course of certain nerves affected in lepromatous leprosy. (To be published)
15. SEDDON, M. J. Carpal ganglion as a cause of paralysis of the deep branch of the ulnar nerve. J. Bone Joint Surg. **34B** (1952) 386-390.
16. SHEPARD, C. C. Temperature optimum of *Mycobacterium leprae* in mice. J. Bacteriol. **90** (1965) 1271-1275.
17. SHREWSBURY, M. M., JOHNSON, R. K. and OUSTERHOUT, D. K. The palmaris brevis—A reconsideration of its anatomy and possible function. J. Bone Joint Surg. **54A** (1972) 344-348.

18. SWANSON, A. E., BIDDULPH, S. L., BAUGHMAN, F. A., Jr., and DEGROOT, G. Ulnar nerve compression due to an anomalous muscle in the Canal of Guyon. Clin. Orthop. **83** (1972) 64-69.
19. THOMAS, C. G. Clinical manifestations of an accessory palmaris muscle. J. Bone Joint Surg. **40A** (1958) 929-930.
20. VAINIO, K. J. Surgery of Arthritis. *In:* Hand, Chapter 8. R. A. Milch, ed. Baltimore: Williams and Wilkins, 1964, p 154.
21. VANDERPOOL, D. W., CHALMERS, J., LAMB, D. W. and WHISTON, T. B. Peripheral compression lesions of the ulnar nerve. J. Bone Joint Surg. **50B** (1968) 792-803.