

ISCHAEMIC SENSORY LOSS IN PATIENTS WITH PERIPHERAL NERVE LESIONS

BY

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During an investigation of patients with painful paraesthesiae due to median nerve compression at the wrist it was found that arresting the circulation to the affected arm by a pneumatic cuff sometimes resulted in intense median paraesthesiae, with pain resembling that of the patients' spontaneous attacks. It was also found in these patients that the inflation of a pneumatic tourniquet round the arm might result in the rapid onset of numbness where none had been present before, and that it would make a mild sensory deficit more clear cut. This abnormally rapid failure of sensation in an area of initial sensory impairment did not prove to be restricted to patients with median nerve lesions at the wrist, but was found also in patients with other peripheral nerve lesions.

The object of the present paper is to describe the effects of sudden circulatory arrest on the sensory loss of patients with peripheral nerve and root lesions affecting the hand, and to compare them with the effects of artificially induced ischaemia in normal subjects. The ischaemic paraesthesiae of patients with median nerve lesions have already been described (Gilliatt and Wilson, 1953), and will not be discussed further.

A number of writers have described the effects of arterial arrest on sensation in the normal arm, and there appears to be agreement on the main sequence of changes. The inflation of a pneumatic cuff above the elbow is followed within a minute or so by ischaemic paraesthesiae, the "compression tingling" of Weddell and Sinclair (1947), and this is thought to be due to transient hyper-excitability of the nerve trunks lying under the cuff (Kugelberg, 1944). In normal subjects these ischaemic paraesthesiae usually take the form of a soft, diffuse tingle lasting three or four minutes, felt most strongly on the ulnar side of the hand (Weddell and Sinclair, 1947).

Some 10 to 15 minutes after the beginning of ischaemia the earliest indication of sensory loss

appears in the form of a change in the quality of sensation in the finger-tips. When the skin is rubbed it may feel numb and yet rough or prickly, and this change precedes tactile anaesthesia when fine hairs are used for testing (Lewis, Pickering, and Rothschild, 1931; Sinclair, 1948). The time of onset of these changes and the order in which the finger-tips become involved is obviously of great importance in relation to the findings in peripheral nerve lesions, and for this reason a fresh series of observations on control subjects has been included in the present work.

Material and Methods

Thirty patients were investigated who were considered to be suffering from median nerve compression at the wrist. Twelve of them, who were studied both before and after operation on the median nerve, formed part of the series of 40 patients with acroparaesthesiae, treated by surgical decompression of the median nerve in the carpal tunnel, reported by Kremer, Gilliatt, Golding, and Wilson (1953). The remainder were patients seen and treated after September, 1952, and therefore excluded from the original report. All of the 30 patients had typical nocturnal attacks of pain and tingling, and in some of them pain was present in the arm as high as the shoulder. In this respect no distinction could be made between the idiopathic group of cases, mostly middle-aged women, and the smaller group of cases in which the syndrome followed definite local trauma to the wrist. Nor were the acroparaesthesiae in those with abnormal neurological signs in the hand different from those without, and every gradation of sensory disturbance was seen, from the merest suggestion of a subjective change to gross anaesthesia throughout the median territory. The average age of the 30 patients with median nerve compression was 47, with a preponderance of females over males of four to one.

The remaining patients who were investigated included four with ulnar nerve lesions, one with a radial nerve lesion, two patients with cervical rib syndromes, and two with cervical disc degeneration causing numbness in the hand.

Fifty patients without any neurological disorder affecting the hand were used as control subjects. The

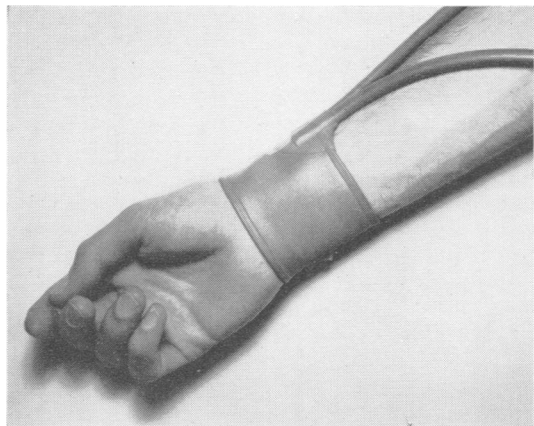


FIG. 1A

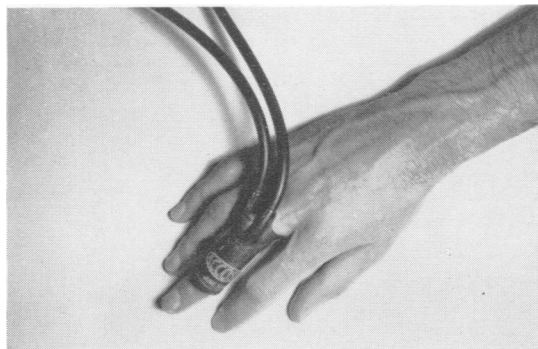


FIG. 1B

majority of them were suffering from general medical diseases, and their average age was 48, with a sex ratio of five females to two males.

Nerve ischaemia was induced by a pneumatic cuff inflated to above arterial pressure round the limb, in most cases on the arm just above the elbow. In all occlusions at this level a rubber bag 6 in. by 15 in. was used in a wide sleeve so that the arm was uniformly compressed. Cuff pressures of 200 to 220 mm. Hg were adequate save in hypertensive patients in whom 250 to 260 mm. Hg was required. Even this pressure was well tolerated for short periods.

To induce ischaemia of nerves in the forearm the same pneumatic cuff was suitable, but as the arteries and nerves in this region are partly protected from compression by their relation to bone, pressures of 220 to 260 mm. Hg were required to prevent venous congestion in the hand. A cuff round the forearm gave rise to some discomfort, owing to local compression of muscle, and in a few experiments a narrow cuff round the wrist was used (Fig. 1A). In this position there was little muscle directly under the cuff, but higher pressures were needed for complete arterial occlusion.

For local compression of the digital arteries a cuff 2½ in. by 4 in. was suitable (Fig. 1B), and by a simple modification of the technique it was possible to produce

local ischaemia in all the fingers simultaneously (Fig. 2). In these experiments cuff pressures of 260 to 280 mm. Hg were used, and in fact caused no discomfort. The relative scarcity of muscle in the finger seems to be the factor which permits pressures of this order to be maintained without pain, as is also the case at the wrist.

In most experiments the arm was raised for 15 seconds before the inflation of a cuff, and then lowered and rested comfortably with the palm uppermost, care being taken to avoid local pressure on the ulnar nerve. Except in special cases the simplest possible method of sensory testing was adopted, the examiner merely stroking the tips of the patients' fingers with his own, and this massive stimulus was usually felt to be altered in some way before formal testing revealed any abnormality. However, to confirm the results obtained by this simple test, a graded set of tactile hairs was used, made up from suitably mounted nylon threads to give a range of stimuli from 0.3 g. to 9.0 g. Although nylon imbibes water unless kept in a desiccator, little fluctuation in the bending strain of the hairs was noted from one experiment to another.

In a few experiments the arm was immersed in a water bath at 37°-38° C. to above the elbow, after lightly greasing the finger-tips, as described by Lewis and others (1931). When this was done the fingers could be lifted out and tested when necessary without appreciably cooling the skin. An area of skin on the finger-tip was ringed and tested by a run of five or 10 stimuli in

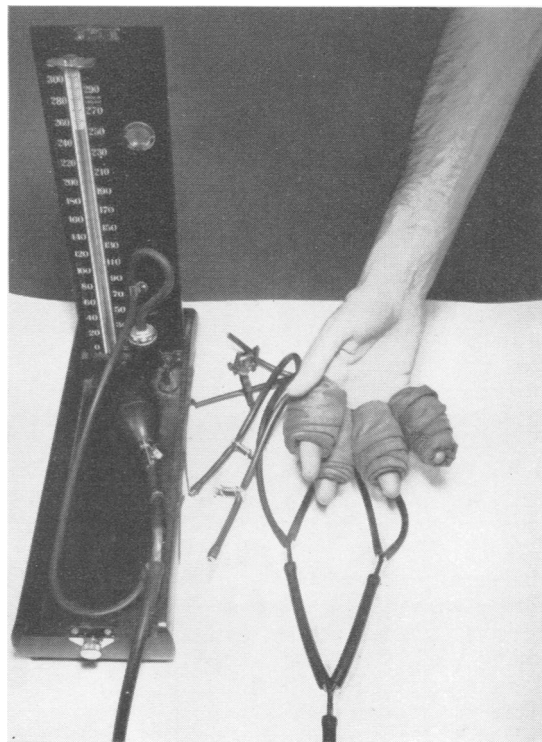


FIG. 2

succession; the subject was told not to guess, and by irregular spacing of the stimuli random answers were easily detected. As sensation failed during ischaemia, it was possible to lift the greased fingers out of water each minute, and to perform 10 tests within this period. Thus, if two separate areas were being tested, runs of five stimuli were used for each, whereas if only one area was concerned runs of 10 were possible every minute.

Some fatigue in the subject was usually noted in a run of 10 tests, and when the stimuli were near threshold the last few were often missed. This fatigue effect always disappeared by the next minute, and no progressive deterioration from this cause was noted in experiments lasting up to 20 minutes. This was shown by the fact that the tactile threshold would usually return rapidly to normal after release of the circulation, although the subject might by then have been tested 150 or 200 times.

Results

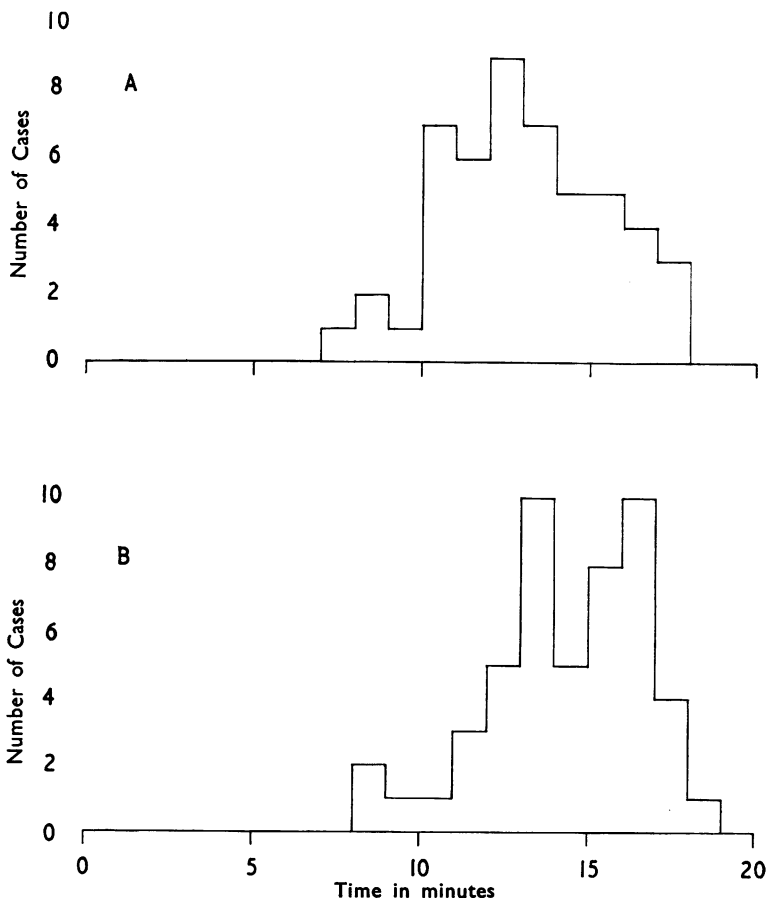
Ischaemic Sensory Loss in Normal Subjects.—Within a few minutes of inflating a cuff round the arm, ischaemic paraesthesiae would be reported by most subjects, but as these subsided sensation appeared to return to normal, and no further change would occur for several minutes. Between 10 and 15 minutes after the onset of ischaemia, however, patients reported a noticeable alteration in the quality of the sensation produced by rubbing the finger-tips, and the time at which this occurred could be elicited quite easily by careful questioning. The hands were occasionally described as “cold” or “numb” much earlier in the experiment, but in these cases enquiry revealed that the skin of the finger-tips was still as sensitive as on the other side, and the later change in the sensation produced by rubbing or stroking the pads of the fingers was immediately recognized by patients when it occurred. The change was described in a variety of ways by different patients; some referred to the feeling as “glassy” or “smoother than normal”, while others described it as “rough”, “numb but sensitive”, and later definitely

“prickly”. It was commonly recognized that the finger had “gone to sleep” and was “dead” or “numb”, as if it had “been in the snow”.

Weddell and Sinclair (1947) referred to this change as “velvety numbness” and considered that it was similar to the sensation of rubbing velvet between the fingers, but when this analogy has been suggested to patients they have usually rejected it and preferred some less picturesque description, such as one of the terms mentioned above.

In all the patients, the appearance of subjective numbness of the skin when it was rubbed or stroked proved a most reliable indication of early sensory failure. If there were any doubt as to its presence, it was often useful to ask the patient to confirm the change by feeling the affected finger-tips with his other hand, and when sensation was examined in

FIG. 3.—Time to onset of ischaemic numbness, pneumatic tourniquet placed above the elbow. A: time to onset of numbness in one finger in 50 control subjects. B: time to onset of numbness in all fingers in control subjects.



this way the results were as consistent as those obtained by careful tests with graded nylon threads. In the majority of patients, stroking of the finger pads was the only method of testing employed, and the term "subjective numbness" has been adopted to indicate sensory loss detected in this way.

Subjective numbness often occurred first in the thumb and spread gradually from finger to finger across the hand, affecting the little finger a minute or two later. Although this spread from radial to ulnar border was not invariable, it was common and occurred in 23 of the 50 control subjects. In seven of them subjective numbness was present in the tips of the fingers supplied by the median nerve for a minute or more before the other fingers were affected.

It was uncommon for numbness to appear first in the fifth finger, and in only one of the 50 subjects did ulnar sensory loss involving one-and-a-half fingers clearly precede any change in the rest of the hand. The patient concerned had been bedridden for many weeks, and although no other signs of ulnar nerve damage were present the possibility of a decubitus ulnar nerve lesion was considered.

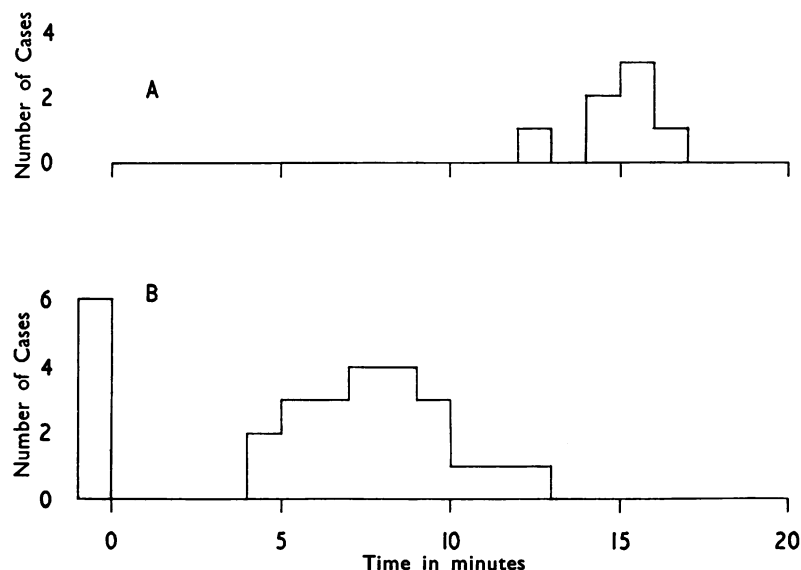
The reason for the unequal involvement of different fingers in the normal hand is obscure. Sinclair (1948) considered that individual peripheral nerves were normally affected in a definite sequence during

ischaemia, and that a difference between median and ulnar territories could usually be demonstrated in the normal hand at some stage of sensory failure. In our own cases clear-cut differences between the median and ulnar fingers were uncommon, and this appeared to be due to the fact that ischaemia was not usually continued beyond a very early stage of sensory failure. If the experiment were continued beyond this point, it often happened that sensory loss, which had appeared almost simultaneously in all the fingers, would gradually become more marked in the median than the ulnar territory, the difference becoming increasingly obvious as the anaesthesia increased.

The earliest time at which subjective numbness appeared in any digit was recorded in each case, and when, after a further interval, numbness was clearly present in all the finger-tips, the time was again noted. The results in 50 control subjects are shown graphically in Figs. 3A and 3B. From these it can be seen that the "time to numbness" for the earliest digit to be affected varied from 7 minutes to 17 minutes in different subjects with a mean of 12.5 minutes (S.D. ± 2.4). The time required for the numbness to involve the tips of all digits was slightly longer, ranging from 8 minutes to 18 minutes (mean 14.0 minutes, S.D. ± 2.3). In all the cases recorded in this way, the site of arterial occlusion was above the elbow.

In the seven control subjects in whom the median territory appeared to be involved one minute or more before any change occurred in the ulnar fingers, the "time to numbness" for the median-supplied digits is shown in Fig. 4A. From this figure it can be seen that in no case was the median numbness present within 10 minutes of the onset of ischaemia.

FIG. 4.—Time to onset of ischaemic numbness, pneumatic tourniquet placed above the elbow. A: time to onset of median numbness in control subjects. B: time to onset of median numbness in carpal tunnel syndrome. (Median numbness was already present initially in six cases.)



In the experiments in which careful sensory tests with nylon threads were carried out, the findings were of great interest. An early deterioration in performance occurred associated with ischaemic tingling, but, as the latter subsided, awareness of the stimuli improved and did not fall off again until a well marked subjective change was present (Fig. 5). This was the case even when the stimulus was so close to the tactile threshold that a

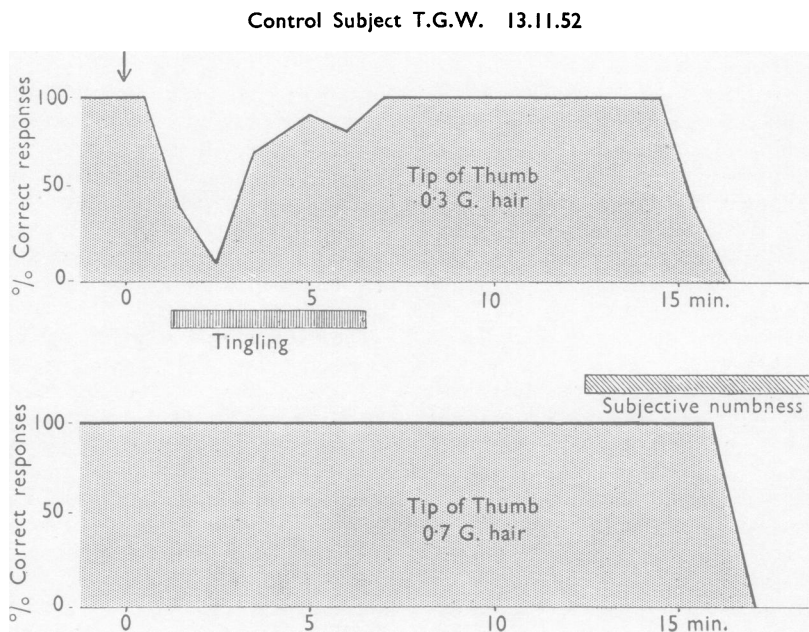


FIG. 5.—Control subject, left arm in water bath at 37°C. Ten tests per minute, tip of thumb; ischaemia at arrow, from pneumatic tourniquet above elbow.

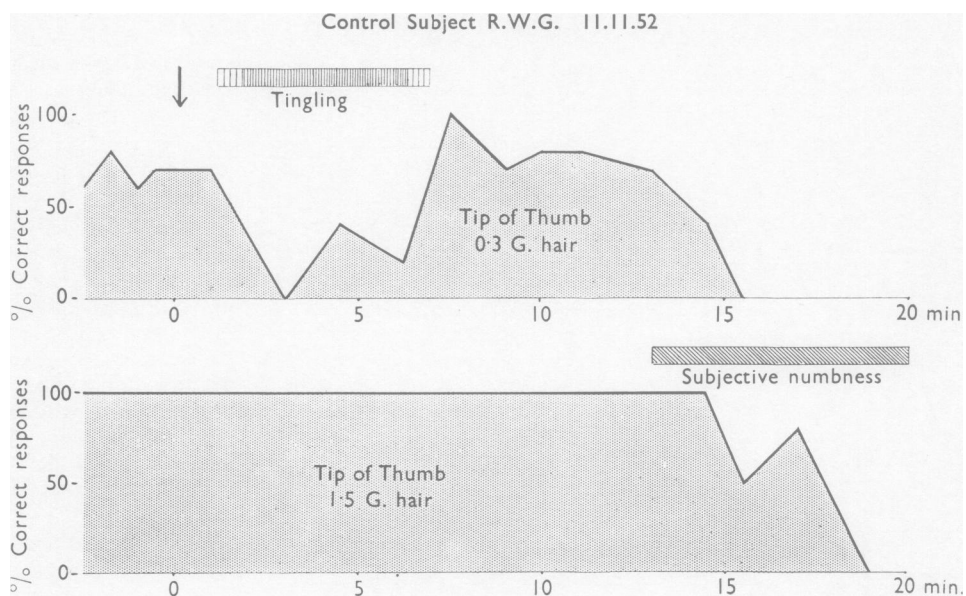


FIG. 6.—Control subject, left arm in water bath at 37°C. Ten tests per minute, tip of thumb; ischaemia at arrow, from pneumatic tourniquet above elbow.

proportion of touches was being missed before the ischaemia had begun (Fig. 6). After release of the circulation, sensation usually returned to normal within 30 seconds, but a second rise in tactile threshold always occurred as soon as post-ischaemic pricking and tingling began.

Ischaemic Sensory Loss in the Carpal Tunnel Syndrome.—Thirty patients with median nerve compression at the wrist were subjected to a period of ischaemia long enough to produce numbness of the fingers. In 20 patients some subjective numbness was already present in the hand, but in only six did it involve the whole median distribution. In all cases existing sensory loss became very much more obvious during ischaemia and in the 14 patients with partial median nerve involvement, numbness spread within a few minutes to the remaining fingers supplied by the nerve, the ulnar territory remaining normal.

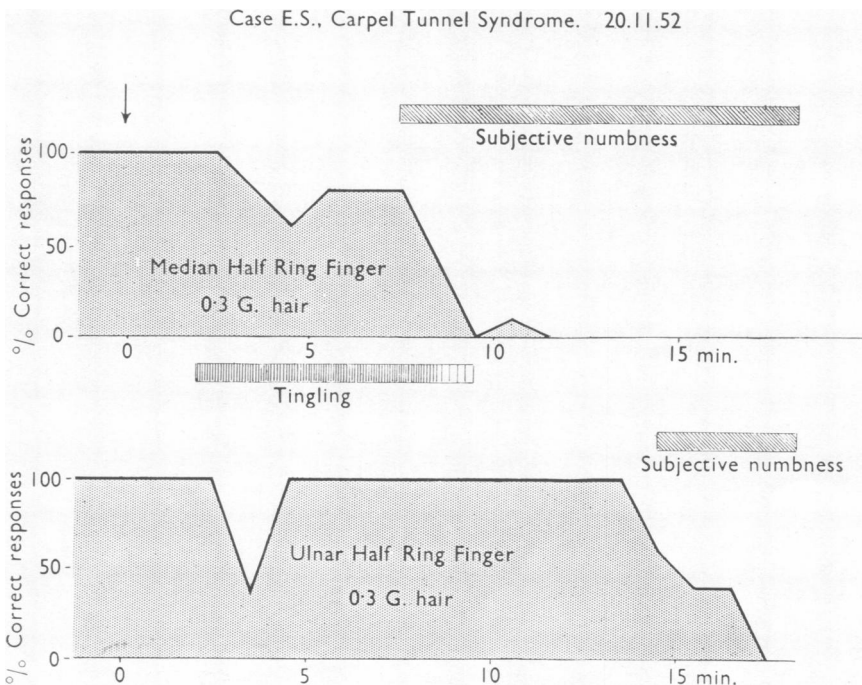
In the 10 patients without subjective numbness of the hands, ischaemia caused it to appear in a median distribution in seven, with a well-marked difference between the two sides of the ring finger. In only three of the 30 patients did a median

distribution of numbness fail to occur at all during ischaemia. Two of the three failures were in mild early cases for whom operation had not been advised, and in the third the test was negative at first, but subsequently became positive while the patient was awaiting admission to hospital.

The earliest time by which subjective numbness was present in all the median-supplied digits was noted in each case and is shown graphically in Fig. 4B. From this it can be seen that in the 28 cases in which it occurred, the "time to numbness" for the median fingers ranged from zero to 12 minutes, and that in 26 cases the numbness was present within 10 minutes.

The early onset of median sensory loss during ischaemia was easily confirmed by quantitative tests with graded hairs, and one experiment is illustrated in Fig. 7. In this patient slight subjective sensory loss was present initially in the thumb, index, and middle fingers, but the ring finger was described as normal. During ischaemia subjective numbness developed on the median side of the ring finger after eight minutes, with anaesthesia to the 0.3 g. hair by 10 minutes, the corresponding changes on the opposite side of the finger occurring after a further seven or eight minutes of ischaemia.

FIG. 7.—Carpal tunnel syndrome, left arm in water bath at 37° C. Five tests per minute on each side of ring finger; ischaemia at arrow as in Fig. 5.



In that it often provided confirmatory evidence of median nerve damage, the inflation of a pneumatic tourniquet round the arm was of considerable practical value in obscure cases of acroparaesthesiae, as is shown by the following cases.

Mrs. E. W.—This patient aged 55 had attacks of painful tingling in both hands at night, the right more than the left, with numbness of the fingers on waking, for many years. Examination of the right hand revealed subjective numbness of the middle finger, with a slight change in the index and median half of the ring finger, but sensation in the thumb was described as normal. The right arm was placed in water at 37°–39° C. and a pneumatic cuff inflated

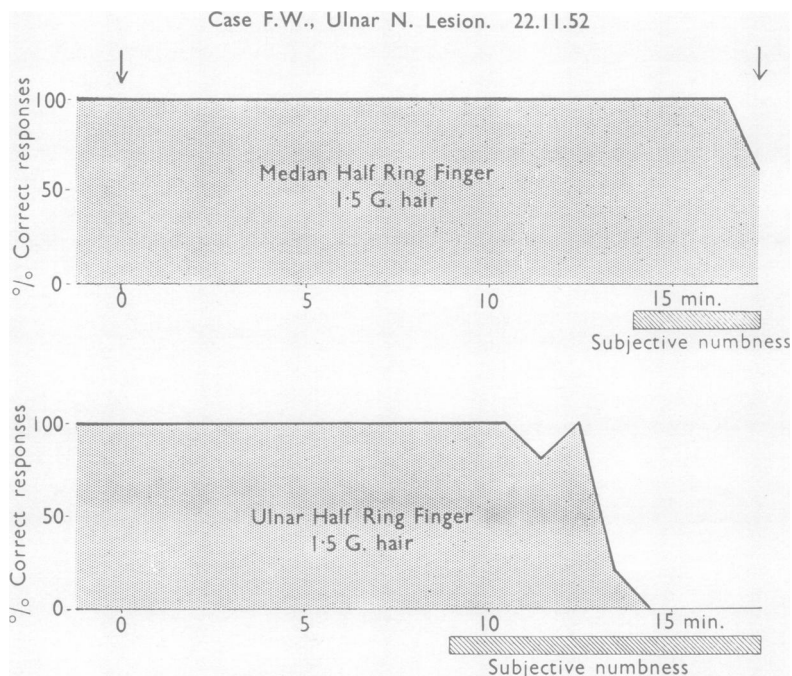
to 220 mm. Hg above the elbow. Ischaemic tingling was not abnormal, being mild in intensity and most marked in the ulnar fingers, but when, after five minutes, the hand was withdrawn from the water-bath and dried, the median fingers and thumb were definitely numb, particularly at the tips, and the patient described them as "much more dead" than before the experiment. The hand was replaced in water and when it was taken out and retested after 10 minutes, the thumb, index, and middle fingers, and half the ring finger were described as "very dead", the ulnar fingers being normal. The hand was replaced in water and the tourniquet released, resulting in generalized post-ischaemic paraesthesiae, and a rapid return of sensation to its initial condition.

Mrs. N. G.—This patient aged 53 had had nocturnal attacks of pain in both arms for many years, with tingling in the fingers and aching in the arms as high as the shoulder. Examination revealed subjective numbness of the index and middle fingers with an unpleasant prickly component when they were touched, and slight numbness of the thumb, but the median half of the ring finger was described as "more sensitive" than the ulnar half. Testing with a pin gave inconclusive results owing to horny skin, but suggested if anything an ulnar distribution of hypalgesia. Careful testing with compass points showed some diminution in sensation on the median fingers. A pneumatic tourniquet was inflated round the arm and resulted in tingling that was normal in intensity and distribution, but by five minutes the thumb, index and middle fingers were "dead" and the median half of the ring finger prickled when touched. By nine minutes these fingers were "very dead" and it was possible to demonstrate a change described as "numbness and a buzz" all over the median territory in the palm, the ulnar fingers and hypothenar region and the back of the hand and proximal phalanges remaining normal until 12 minutes when the cuff was released.

Ischaemic Sensory Loss in Other Nerve Lesions Affecting the Hand.—Four patients were seen with mild ulnar nerve lesions due to pressure at the elbow. In all four patients there was slight sensory impairment initially in the fifth finger, but in only two of them was there a difference between the two sides of the ring finger. Early ischaemic sensory loss

occurred in all four cases, one of them being illustrated in Fig. 8. Within 10 minutes of the onset of ischaemia well marked numbness of the fifth finger and of half the ring finger was present in each case,

FIG. 8.—Ulnar nerve lesion at the elbow, left arm in water bath at 37° C. Five tests per minute on each side of ring finger; ischaemia at arrow as in Fig. 5. Heavier hair was used for testing than in previous figures.



and was obvious to the patient when he touched the area with his other hand. During the next 10 minutes, however, two of the patients showed a gradual reversal of the pattern of sensory loss, the normal progression of sensory failure over the median-supplied digits "overtaking" that of the ulnar fingers, so that at the end of 20 minutes anaesthesia was most dense over the thumb and radial border of the hand. This phenomenon is further discussed in a subsequent section.

Until this time it had been assumed that the effect of ischaemia was a direct one on the damaged nerve fibres at the level of the lesion, but in two of the patients with ulnar nerve lesions, cuffs were inflated round the forearm and the results were found to be similar to the effects of arterial occlusion above the elbow. Subsequently one patient was seen with a mild radial nerve lesion, whose history left no doubt that the site of the lesion was in the musculospiral groove. Slight sensory impairment was

present over the dorsal surface of the first interdigital cleft and proximal phalanx of the thumb, and in this patient also rapidly worsening sensation over the affected area resulted from circulatory arrest in the forearm.

Two patients were then studied with slight subjective sensory loss of a C7 root distribution due to cervical disc degeneration. In both of them the sensory loss was of long standing and had shown little change in the six months before testing. The effects of ischaemia followed the pattern already described, a change in the area of initial sensory loss preceding the onset of numbness elsewhere in the hand.

In these patients, whose nerve lesions were proximal to the pneumatic cuff, it seemed likely that the effect of ischaemia depended upon the additive effect of two quite separate regions of depressed function along the nerve, one at the site of the initial lesion, and the other in the peripheral segment of nerve artificially deprived of its blood supply. Some confirmation for this theory was provided by the following experiment.

Dr. P. B.—This patient, aged 29, in July, 1950, sustained a deep laceration of the palm of the right hand with complete severance of the palmar branch of the median nerve supplying the contiguous surfaces of the index and middle fingers. Primary suture was carried out within an hour of injury, and partial recovery of sensation occurred within six months, but no further change had been noticed since. At the time of examination in November, 1952, there was slight subjective numbness of the contiguous surfaces of the index and middle fingers, with a raised tactile threshold to hairs. Ischaemia was induced by the inflation of four small pneumatic cuffs round the fingers, as in Fig. 2. No change in sensation was detected after 10 minutes, but by 20 minutes marked deterioration had occurred in the hypaesthetic areas, no demonstrable alteration in the responses to graded hairs resulting elsewhere in the hand until 30 to 40 minutes. The difference between the normal and abnormal areas was particularly well brought out by the very gradual loss of sensation which is characteristic of local ischaemia of the fingers.

Further Experimental Observations in Normal Subjects.—In the patients described in the preceding section, ischaemic sensory loss had appeared earlier in a region where sensation was already impaired than elsewhere, irrespective of the site of the lesion relative to the cuff, and it seemed likely that the phenomenon depended upon simple summation of the effects of two separate nerve blocks at different levels. An alternative hypothesis remained that the nerve fibres distal to an injured segment might have undergone some mild degenerative change rendering

them more susceptible to ischaemia. In order to decide between these two possibilities the following experiments were carried out.

In a normal subject local ischaemia of the ring finger was produced by a cuff round its base. Simultaneously an ulnar nerve block was produced above the elbow by suspending the arm from a broad strap passing round the limb just above the medial condyle of the humerus, as described by Zotterman (1933). When this was done there was a brief period in which slight subjective numbness was present over the fifth finger due to the strap, and over the median side of the ring finger due to the cuff, with more marked numbness of the ulnar side of the ring finger due to summation of the two, and it was possible to select a hair which was felt on the former two areas and missed altogether on the latter. Technically this was not a very easy experiment to carry out, as the two nerve blocks developed at different rates, but it was repeated successfully on more than one occasion, and the effect reversed by releasing either the strap or the cuff at the critical moment.

In a second similar experiment, a cuff was inflated round the ring finger and shortly afterwards a partial ulnar nerve block was produced by injecting procaine into the ulnar nerve at the elbow. The resulting anaesthesia was mild over the fifth finger, as was the sensory loss over the median-supplied half of the ring finger due to the cuff, but over the ulnar half of the ring finger there was marked sensory loss, with complete anaesthesia to a 0.7 g. hair, which could still be felt without difficulty over the rest of the hand. Release of the finger cuff then resulted in prompt improvement, sensation on the ulnar side of the ring finger improving until it approximated to that over the remainder of the ulnar territory.

From these results it may be concluded that two separate regions of depressed function along a nerve have an additive effect in causing sensory loss in excess of that produced by either block alone.

“Time to Numbness” as a Test of Sensory Recovery.—Two patients with cervical rib syndromes were studied. Both showed mild weakness of the intrinsic muscles of the hand, with a slightly raised tactile threshold to hairs, particularly along the ulnar border of the hand and forearm. In neither case was there any evidence of arterial insufficiency or vasomotor disturbance in the arm itself, and the radial pulse was not easily obliterated by the usual manoeuvres.

Both these patients were tested repeatedly with ischaemia before and after operation, and the results

in one of them are shown in Fig. 9. In this patient spontaneous tingling was present continuously in the fingers before operation, and the 0.3 and 0.7 g. hairs were not felt. When a pneumatic cuff was inflated above the elbow, tingling in the fingers increased for a few minutes and then declined, and within four minutes the patient complained that the fingers were "numb" and "dead".

Ten days after resection of the cervical rib the hand was apparently normal, all tingling having disappeared, and the 0.3 g. hair was easily felt on all fingers. At this time no errors occurred in discrimination of the two points of a compass 0.5 cm. apart. However, a pneumatic tourniquet round the

arm produced subjective numbness within seven minutes, and it was on the fifth finger that responses first failed to the touch of a hair.

A further test on the fifteenth post-operative day gave a much more normal result, sensory loss appearing first on the radial side of the hand and involving the fifth finger after 12 minutes.

This result shows that for a single patient repeated measurement of the "time to numbness" can give a reliable quantitative estimate of sensory recovery. It also indicates that the "time to numbness" may still be abnormal at a stage of recovery when formal tests of superficial sensation reveal no abnormality.

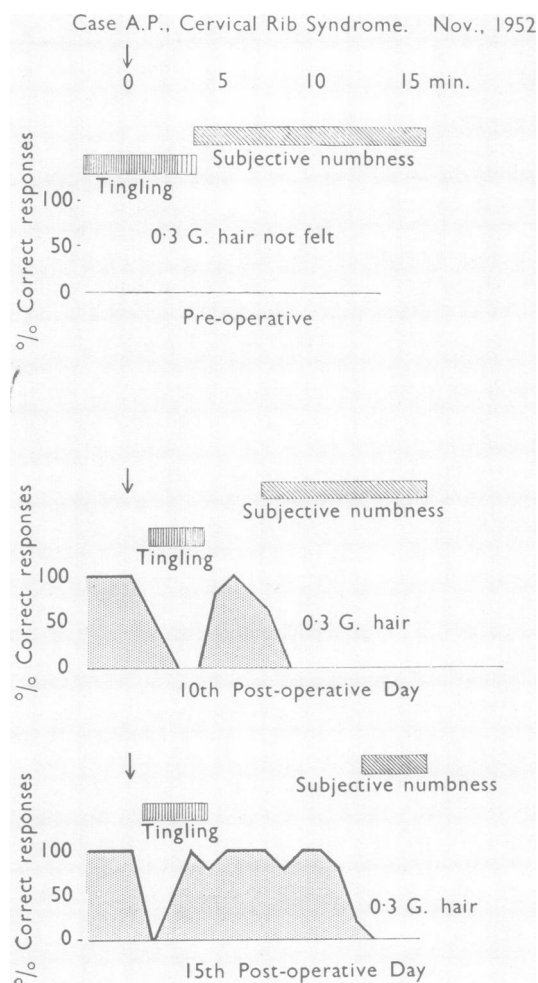
Discussion

In patients with severe compression of the median nerve at the wrist, arrest of the circulation to the arm has been shown to result in intense paraesthesiae and pain, followed by rapid sensory failure throughout the median territory (Gilliatt and Wilson, 1953). At first it seemed likely that these changes were due to a direct effect of ischaemia on the damaged segment of the nerve in the carpal tunnel, but later studies of other peripheral nerve lesions somewhat modified this view, as it appeared that any area of sensory impairment might be adversely affected by ischaemia, regardless of the site of the lesion relative to the occluding cuff.

In their original investigation of the effects of circulatory arrest in normal subjects, Lewis and others (1931) showed that the earliest ischaemic sensory loss was due to depression of function in the segment of nerve immediately underneath a cuff. In one of their experiments slight sensory loss was produced by a cuff round the arm; a second cuff was then inflated round the forearm and the upper cuff released, as a result of which numbness disappeared from the fingers, although the circulation had not been restored to the distal part of the limb. With longer periods of circulatory arrest, ischaemia of nerves distal to the cuff did contribute to the sensory loss, and local return of the circulation above the elbow did not restore normal sensation to the hand.

At the suggestion of Dr. Michael Kremer the experiment of Lewis and others (1931) was repeated in three patients with severe acroparaesthesiae due to median nerve compression at the wrist. When, after five or six minutes, intensely painful paraesthesiae with numbness of the fingers had been induced by a cuff above the elbow, a second cuff was inflated just above the wrist and the upper one released. In all three patients painful paraesthesiae and numbness persisted and appeared to be unchanged

Fig. 9.—Cervical rib syndrome, left arm at room temperature. Five tests per minute, tip of fifth finger: ischaemia at arrow as in Fig. 5.



until the lower cuff was released. Detailed sensory testing was not possible in these patients owing to severe pain in the hand, but the conclusion seemed inevitable that the segment of nerve in the carpal tunnel was in fact more sensitive to deprivation of its blood supply than the proximal part of the nerve above the elbow. The result is thus in contrast to that of Lewis and others (1931), in whose normal subjects the peripheral portions of the nerves contributed little to the early sensory disturbance resulting from a cuff above the elbow.

From this it may be concluded that in patients with peripheral nerve lesions distal to an occluding cuff round the arm, the resulting sensory loss in the hand may depend in part upon a direct effect on damaged nerve, whereas in patients with lesions proximal to an occluding cuff the result must be due to the simple additive effect of two separate regions of depressed function.

In order to relate the severity of nerve damage to the sensory experience, it is obviously essential to know what proportion of afferent fibres must cease to conduct before detectable sensory loss results. Magladery, McDougal, and Stoll (1950) inflated a cuff round the forearm in a normal subject and stimulated the ulnar nerve at the wrist, recording the nerve action potential at the elbow, as described by Dawson and Scott (1949). Their paper provides little information about the sensory loss produced, but their records show a decrease in size of the afferent volley to less than two-thirds of normal within nine minutes of the onset of ischaemia. At this time no sensory change in the fingers would be expected from a cuff round the forearm.

If in fact 30–40% of nerve fibres cease to conduct before detectable sensory loss appears, the effect of ischaemia in our patients with ulnar nerve lesions becomes more intelligible. As a tentative theory it might be suggested that if 25% of the tactile fibres in the ulnar nerve were blocked by a local lesion at the elbow, this deficit might not be detectable clinically. If 25% were also blocked by local ischaemia in the forearm, the total deficit would be 44% (25 + 25% of 75), a proportion which might give rise to easily detectable numbness of the skin. Loss of function in 50% of fibres under the cuff would cause a total deficit of 62.5%, and blockage of 75% under the cuff a total of 81%. By this stage a normal nerve trunk would have lost 75% of its fibres, and as complete anaesthesia was approached the difference between normal and abnormal areas would become less marked. This would explain the observation that in mild ulnar nerve lesions sensory deterioration occurred first in the ulnar fingers during ischaemia, but was later "overtaken"

by the normal progress of anaesthesia starting on the radial side of the hand.

In the diagnosis of peripheral nerve lesions, the use of a pneumatic tourniquet to confirm the presence of minimal sensory loss has much to recommend it. In suspected ulnar nerve lesions, the appearance of ischaemic sensory loss restricted to the ulnar fingers is probably valid evidence of a pre-existing lesion of the nerve, as early sensory loss of this distribution does not appear to occur in the normal hand as the result of a cuff above the elbow. In suspected median nerve lesions, however, the results are more difficult to interpret as the median nerve appears to fail first in a proportion of normal subjects, and the rapidity with which sensory loss occurs is therefore of critical importance. Whereas numbness restricted to the median-supplied digits and present within five or six minutes of the onset of ischaemia is clearly abnormal and indicative of a lesion of the nerve, a similar change occurring after a longer period carries less certainty. However, when the results in the patients with median nerve lesions (Fig. 4B) are compared with those in the control group (Fig. 4A), the differences are such that it seems reasonable to accept any well marked median sensory change within the first 10 minutes of ischaemia as suggestive of median nerve damage, particularly if a further interval of several minutes elapses before involvement of the remainder of the hand.

When using ischaemia in the examination of peripheral nerve lesions, it has been possible to test sensation very simply by a massive stimulus such as the examiner's finger, and little advantage has been obtained from using a wisp of cotton wool or graded hairs. This is in accord with the observations of Lewis and others (1931), and of Weddell and Sinclair (1947), who used 0.6 and 0.5 g. hairs respectively, and found that a subjective change in the sensation aroused by a massive stimulus preceded the onset of anaesthesia to the hairs. Zotterman (1933) used a wide range of V. Frey hairs to test sensation during ischaemia, and refers to a "slow creep upwards of the threshold for the hairs from about eight minutes". Few experimental details are described, and the number of stimuli, skin temperature, and time of onset of subjective numbness are not stated. From the only figures displayed it appears that in one of his subjects a 0.2 g. hair was still felt after 10 minutes, a result incompatible with any considerable rise in threshold. In theory it is possible that an earlier change in tactile threshold does occur during ischaemia, but the technique used in the present work has not been sufficiently sensitive to demonstrate it. From the practical point of view

there is every advantage to be gained from recording merely the subjective sensation in response to a massive stimulus, as this is easily determined in unintelligent patients, and is much less affected by horny skin or cold hands than more elaborate methods of sensory testing.

In control subjects and in patients with peripheral nerve lesions it was found that ischaemic and post-ischaemic tingling were accompanied by a marked deterioration in the perception of fine hairs, and paraesthesiae that were scarcely perceptible to the subject seemed to cause a disproportionate change in the tactile threshold. It is surprising that an increase in the excitability of afferent nerve fibres should result in impairment of sensation, particularly when it is remembered that heavier stimuli actually reinforce post-ischaemic pricking and tingling and may result in marked reflex changes (Gilliatt, 1952). A raised tactile threshold associated with over-reaction to a stimulus that is perceived is analogous to the common clinical combination of hypalgesia and hyperpathia, and suggests that the latter might result from a similar increase in the excitability of pain fibres.

Summary

In 40 patients with peripheral nerve and dorsal root lesions affecting the hand, and in 50 control subjects, the circulation to the arm has been arrested by a pneumatic tourniquet above the elbow. During this period of artificially induced ischaemia, tactile sensation in the fingers has been tested repeatedly in each case in order to ascertain the time at which sensory failure occurs.

In 50 control subjects the average duration of ischaemia required to produce sensory loss in the

skin of the fingers has been 14 minutes (S.D. \pm 2.3).

In the patients with peripheral nerve lesions, ischaemic sensory loss has occurred with abnormal rapidity within the distribution of the affected nerve, and pre-existing sensory loss has become much more marked, a change usually being apparent within five or 10 minutes of the onset of ischaemia.

In the case of median nerve lesions at the wrist, the early sensory failure caused by circulatory arrest appears to be due in part to an increased susceptibility to ischaemia of damaged nerve fibres at the site of the lesion. However, in patients with peripheral nerve and root lesions proximal to the site of a pneumatic tourniquet, abnormally rapid failure of sensation also occurs during ischaemia, and in these cases the result appears to be due to the additive effect of two separate regions of depressed function along the course of the nerve, one at the site of the lesion and the other under an occluding cuff.

We wish to thank Dr. E. A. Carmichael and Dr. M. Kremer for allowing us to investigate their patients, and for much helpful criticism and advice.

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