ACROPARAESTHESIAE AND ACROMEGALY

BY

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The syndrome of acroparaesthesiae due to compression of the median nerve in the carpal tunnel, with the relief obtained by section of the transverse carpal ligament, has recently attracted much attention. It might be expected that diseases causing abnormalities of the carpal tunnel should frequently give rise to acroparaesthesiae, but examples have not often been recorded. Bony lesions, such as a dislocated lunate, and haemorrhage have been reported by Watson-Jones (1949). A neuroma was mentioned by Cannon and Love (1946), though it is not clear from their paper that this was a tunfour in the strict sense. The syndrome has been reported in myxoedema (Schiller and Kolb, 1954; Murray and Simpson, 1958) and in acromegaly in nine cases (List, 1932; Woltman, 1941; Cannon and Love, 1946; Kellgren *et al.*, 1952; Schiller and Kolb, 1954). The present paper adds five more.

Case 1

A police officer aged 39 was admitted to hospital under Dr. Michael Ashby in June, 1958. He had always been tall, but acromegalic features had been apparent in photographs for only about 10 years. There had been no increase in the size of his hands during the previous five years. For nine months he had complained of painful tingling in the thumb and middle three fingers of the right hand, principally at night. For two months the left hand had been affected similarly though less severely. His sleep had been disturbed by the attacks.

Examination showed the typical features of acromegaly. There was wasting of both thenar eminences, particularly of abductor pollicis brevis, more marked on the right. There was no sensory loss.

On June 11 Mr. P. T. Savage carried out bilateral carpal-tunnel section. On the right the median nerve was red and swollen, with definite constriction. Similar but lesser changes were seen on the left.

In November, 1958, he reported that he had been completely relieved by the operations, though on examination there was still some thenar wasting. At this time ischaemia produced by inflation of a sphygmomanometer cuff above the systolic blood-pressure (Gilliat and Wilson, 1953) did produce impairment of sensation over the median area in both arms. This was repeated on several occasions, but the sensory deficit developed only after 15 to 20 minutes. On one occasion, however, a severe attack of pain, identical with his preoperative symptoms, was produced in five minutes in the right arm. The cuff was over the middle of the forearm, and it is interesting that the pain radiated to the elbow above the cuff. It proved impossible to precipitate further attacks by various manoeuvres, including inducing venous engorgement or holding the arm dependent.

Case 2

A plumber aged 49 first attended Dr. Ashby's out-patient clinic in September, 1955, complaining of pain and numbness in the hands, occurring mainly at night over the previous 18 months. Acromegaly had been present for 10 years. A diagnosis of mild carpal-tunnel syndrome was made, and as his symptoms subsided with rest no further action was

taken. He again attended in September, 1958, because of a recurrence of the acroparaesthesiae five weeks previously. The middle three fingers were mainly affected, more so on the right. The attacks occurred almost entirely at night except that hammering at work would sometimes precipitate one.

Examination showed typical acromegaly. In the hands there was doubtful flattening of the right thenar eminence but definite impairment of sensation of median-nerve distribution on the right and to a lesser extent on the left.

On October 8 Mr. Savage performed bilateral carpal-tunnel section and found that on the right there was definite compression of the median nerve.

Ischaemic tests before operation enhanced the sensory deficit, but only after 17 minutes. When the sphygmomanometer cuff was released the patient could then distinguish between the ulnar-nerve and the median-nerve distribution, as the post-ischaemic tinglings disappeared first in the ulnar area. These findings could be reproduced three months after operation when he was symptom-free in the right hand, but he had had occasional tingling in the left since returning to work.

Case 3

A box-maker aged 47 was admitted under Dr. Ashby on May 19, 1959. She first noticed persistent swelling of her face 26 years ago after an attack of erysipelas. Increase in the size of her hands was noted during the war and of both hands and feet about three years ago. She has required treatment for diabetes since May, 1958 (Dr. A. Bloom), and for dermatitis for nine years.

Pain and tingling in her hands at night first began during the war while working in an aircraft factory. The right hand was affected much more than the left. She had had occasional attacks since then, but none had persisted until the present series, which began in December, 1958. She then had, in addition, permanent roughness of the radial aspect of the ring finger and the whole of the middle finger of the right hand.

Examination showed typical acromegalic features with patches of dermatitis. The only abnormalities in the central nervous system were wasting of the thenar eminences with weakness of abductor pollicis brevis and impairment of sensation of median-nerve distribution. All these abnormalities were greater on the right.

Ischaemic compression of the arm sometimes enhanced the sensory impairment within seven minutes, but occasionally there was no change after a quarter of an hour.

X-ray examination of the skull showed an enlarged pituitary fossa.

The patient was seen by Mr. Savage, who performed carpal-tunnel decompression on July 2, 1959, with relief of symptoms.

Case 4

A man aged 67 was admitted under Dr. A. L. Jacobs in July, 1957. Acromegalic changes were first noted in 1918, the diagnosis being made in 1925. He complained at that time of tingling in the hands. Two courses of radiotherapy were then given to the pituitary.

In July, 1955, he was again seen at another hospital complaining of tingling in the fingers of both hands. This affected mainly the middle fingers, especially at night. There was no motor or sensory loss and no change in the reflexes. A diagnosis of carpal-tunnel syndrome was made.

His complaints on admission were varied, and he soon developed a paranoid state requiring his transfer to a mental observation ward. He has not been seen since.

Case 5

A man aged 53 was admitted under Dr. Bloom in September, 1958. The changes of acromegaly were present in 1942. He was given a course of radiotherapy to the pituitary in April, 1958. In addition he had chronic bronchitis

and emphysema, a rounded mass in the lingula, thought to be a hamartoma, as well as some weakness and wasting of the legs without reflex changes.

In 1954 and again in 1958 he had wakened at night with numbness of his hands, banging them on the bed for relief. All fingers were affected. It also occurred when he was out walking.

No neurological signs were found in the hands. Occlusion of the brachial artery was tolerated for only about seven minutes because of a feeling of severe tightness in the hands and forearms which was apparently similar to his attacks. There was no localization of the paraesthesiae or sensory impairment to any particular area. A diagnosis of carpal-tunnel syndrome was made.

Median nerves of patient with acromegaly showing constriction in the carpal tunnel.

The following case is recorded because a necropsy was performed.

A female clerk aged 53 was admitted under Dr. Ashby on May 15, 1959, as an emergency case. During the evening she had vomited several times and then lost consciousness.

On examination she was deeply comatose, with extensor plantar responses. There was

no thenar wasting. A diagnosis of cerebral haemorrhage was made. Her condition steadily deteriorated and she died four hours later.

The previous history was that increase in the size of her hands and feet was first observed in 1933 and change in her features 10 years later. There was no history of pain or tingling in her fingers.

Necropsy showed acromegalic features with an enlarged cystic pituitary gland. Section revealed an adenoma of the anterior lobe which was mainly eosinophil. The anterior carpal ligaments were thickened, with compression of both median nerves (see photograph). Histologically there was marked perineural fibrosis, with moderate epineural and endoneural fibrosis distal to the sites of the constriction. Silver-staining showed moderate reduction in conducting elements distal to the constriction on the right side but only slight reduction on the left.

Discussion

These five patients with acromegaly all developed a syndrome similar to that described by Brain *et al.* (1947). It is perhaps surprising that only nine cases of acromegaly and carpal-tunnel syndrome have been reported in the literature, as it is likely that the real incidence is much higher. Sternberg (1899), in his review of acromegaly, regarded acroparaesthesiae as an early symptom of the disease but one which rarely persisted in the fully developed condition. He quoted one suggestive history and said that thenar wasting might occur. Atkinson (1932) also reported that acroparaesthesiae were quite common.

In the present series occupation did not appear to be important, as only one of the patients is employed in a trade, though the right hand was more severely affected in three cases. In the literature the details are incomplete.

One notable difference from the usual syndrome is the sex incidence. Kremer *et al.* (1953) stated that in their series of patients with the carpal-tunnel syndrome women were affected five times as often as men. Dick and Zakik (1958) gave a 6/1 ratio. In the 14 cases so far recorded there are equal numbers of men and women. We know that the carpal tunnel is smaller in women than in men (Garland *et al.*, 1957), but this difference may well be abolished by the characteristic local overgrowth of bones and ligaments in acromegaly.

The frequent onset of acroparaesthesiae in women around the menopause suggests an endocrine abnormality. Schiller and Kolb (1954) therefore treated three cases of menopausal median neuropathy with oestrogens, with relief of symptoms. A spontaneous remission may have been responsible for this effect.

Other lines of evidence support a soft-tissue component—for example, the considerable spontaneous variations when reviewed over the course of 24 hours or over many years. In the acroparaesthesiae of acromegaly the symptoms are sometimes quickly relieved after treatment of the pituitary either surgically (Schiller and Kolb, 1954) or by adequate radiotherapy (List, 1932; Woltman, 1941). The rapidity of response supports the presence of a soft-tissue factor. However, in some patients—for example, Cases 1 and 2 of the present series and Schiller and Kolb's second case—the disease appeared to be inactive at the time of the development of the acroparaesthesiae. Nevertheless, in view of the equal sex incidence of acromegaly and of acroparaesthesiae in acromegaly, the alteration in bones and probably ligaments predisposes to the development of paraesthesiae.

Albright *et al.* (1946) showed that decrease in the volume of the hands and feet of acromegalic patients occurred on oestrogen therapy. The change was attributed to an alteration in the amount of sodium-and-water retention and could be as much as 10% of the initial volume. They further pointed out that active acromegaly resembled the post-menopausal state. In both there is a high serum inorganic phosphorus level and negative calcium and phosphorus balances, with a tendency to osteoporosis. With oestrogen therapy these changes are reversed. There may therefore be a similar mechanism underlying the production of acroparaesthesiae in both conditions—possibly imbalance between oestrogens and growth hormone—producing its effects by sodium retention and increase in the extracellular fluid. Again, there is increased sodium-and-water retention in pregnancy. It is not surprising, therefore, that an increased incidence of acroparaesthesiae in pregnancy has been reported, and usually it has been relieved immediately after delivery (Garland, 1955; Heathfield, 1957). Again, in myxoedema the relief of the acroparaesthesiae shows a clear relationship to the clinical response to treatment. Further, relapse to the myxoedematous state may be associated with a return of the acroparaesthesiae, only to disappear again when the patient becomes euthyroid (Murray and Simpson, 1958).

The importance of the anatomical background in acroparaesthesiae is suggested by the dramatic relief

obtained by section of the carpal tunnel—for example, Kellgren *et al.* (1952). This is also supported by the occasional cases where space-occupying lesions are found—for example, a displaced lunate bone (Watson-Jones, 1949). Previous bony injury is often contributory, especially malunion of a Colles fracture (Newman, 1948; Garland, *et al.*, 1957). In addition there is the occasional case where relief is not obtained until an incomplete decompression is remedied at a second operation (Kremer *et al.*, 1953).

Though it is axiomatic that there is local overgrowth of the extremities, by the very derivation of the name acromegaly, the bony changes do not appear to be responsible alone. There is a varying soft-tissue component as well, since some cases are burnt out when first developing acroparaesthesiae. That the two factors are involved is strongly supported by the necropsy findings in Case 6. The constriction of the median nerves was striking, and histologically there is evidence of nerve damage, yet the patient had no symptoms. Apparently the critical soft-tissue component was not present.

The residual space in the carpal tunnel is probably smaller in acromegals. The development of symptoms in both cases seems, however, to be more dependent on changes in the soft-tissue component. These variations, though possibly small, can be highly critical in a confined space.

Why the characteristic story of hanging the arms out of bed or moving them about should give relief is not so clear. Contraction of the muscles will increase the lymphatic flow and so remove any increase in the extracellular fluid. This would take some minutes to produce its effects, which tallies with the clinical story. Osborne (1959) suggests that venous stagnation and local tissue changes rather than arterial compression are responsible for the symptoms. It is surely ischaemia developing at capillary level which is responsible. Hanging the arm down, however, might be expected to increase the extracellular fluid. It may be that just enough fluid flows into the palm to tip the balance and relieve the pressure. Alternatively, it is well known that when an injured hand is dependent it throbs severely. It is possible that a similar increase in blood supply is sufficient to relieve the anoxia of the median nerve. Stammers (1959) suggests that it is the peripheral vasodilatation during sleep which is responsible for the classical nocturnal occurrence, by adding to the squeeze already present.

It would seem reasonable to conclude that the general thickening of the bones and soft tissues, often so striking in acromegaly, probably reduces the capacity of the carpal tunnel to a critical level, at which a small increase in the extracellular fluid induces anoxia of the median nerve.

Summary

The syndrome of acroparaesthesiae occurring in acromegaly is described in five cases, together with the necropsy findings in one patient who had no symptoms but showed evidence of median-nerve compression. Only nine similar cases have been recorded previously.

The sex incidence is equal in the acroparaesthesiae of acromegaly, compared with the marked preponderance of females in acroparaesthesiae without acromegaly.

It is suggested that the great variation in symptoms of acroparaesthesiae is due to a soft-tissue component acting in the confined space of the carpal tunnel and

that this effect is exacerbated by the local overgrowth of the structures forming the tunnel in acromegaly.

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MEDIAN-NERVE PALSY AS A COMPLICATION OF ACUTE PYOGENIC INFECTIONS OF THE HAND

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Since the "carpal-tunnel syndrome" was first described by Brain, Wright, and Wilkinson (1947), many cases of median-nerve palsy due to compression of the nerve as it passes deep to the transverse carpal ligament have been reported. Causes commonly cited include degenerative arthritis due to old fractures of the carpal bones (notably the scaphoid), chronic rheumatoid arthritis, non-specific proliferative tenosynovitis of the flexor tendons, and cases of tuberculous tenosynovitis. All these are *chronic* conditions. Not so well recognized, however, is the occurrence of acute median-nerve compression resulting from severe pyogenic infection of the hand. I have been able to find only one other reference to this condition (Bailey and Bolton Carter, 1955).

These writers described two cases of acute infection of the tendon sheath which subsequently developed median-nerve palsy. Both cases were treated successfully by decompressing the nerve.

Three further cases are described below.

Case 1

A housewife aged 68 was referred to the General Hospital, Birmingham, on January 21, 1958. Three days previously she had developed a painful swelling of her right thumb. She could not remember injuring the part. Pain and swelling