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

BY

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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

increased, and two days later the swelling had involved all her fingers, the palm, and the anterior surface of her wrist.

On examination her temperature was 100.6° F. (38.1° C.). The thumb and all the fingers were inflamed and swollen. The swelling extended up over the anterior aspect of the wrist. Severe pain prevented both active and passive movements of the fingers. Local tenderness was pronounced over the palm and front of the wrist. There was no evidence of any local cutaneous lesion on any part of the hand. She complained of numbness of her fingers.

Acute streptococcal infection of the hand was suspected. Treatment with systemic penicillin was started and the hand was splinted.

The next day careful examination revealed complete anaesthesia over the whole of the median-nerve area of the lateral three and a half digits and the distal part of the palm. Motor function of the thenar musculature could not be assessed because severe pain and swelling prevented movement of the thumb. Sensation over the distribution of the ulnar nerve in the hand was quite normal. Urgent decompression of the median nerve seemed to be indicated.

Operation.—This was performed under general anaesthesia, in a bloodless field. An incision was made in the crease round the base of the thenar eminence as far as the distal flexor crease of the wrist. It was extended along this in its medial two-thirds, and then a further extension at right angles was made for 2 in. (5 cm.) up the forearm. The subcutaneous tissues were very oedematous. The transverse carpal ligament was completely divided. The nerve was grossly flattened throughout its course deep to the ligament, the upper margin of which had made a well-marked indentation in the nerve. The tendon sheaths were widely opened, releasing a quantity of very thin pus. A swab was taken for culture. The wound was closed, several strands of nylon being used as a drain. The hand was again splinted and treatment with systemic penicillin was continued.

The patient was pyrexial for three days. Culture of the pus revealed the presence of β -haemolytic streptococci fully sensitive to penicillin.

By the fourth post-operative day her temperature was normal, almost all inflammation and swelling had subsided, and movement of the median-supplied thenar muscles could be demonstrated, though weakly. Sensory examination showed patchy return of sensation near the bases of the fingers, but not at the tips.

Physiotherapy was started on the fifth day. Penicillin was continued for two weeks, until all traces of inflammation had disappeared and the wound was completely healed.

Two months after operation function was almost normal. She had a strong and painless grip, and the only abnormality still detectable was slight diminution of light touch sensation over the pulps of the index and middle fingers. Two years later there was minimal blunting of light touch sensation on the pulp of the index finger, and she had no disability even when doing fine needlework.

Case 2

A male Pakistani aged 52 was referred to the General Hospital, Birmingham, on April 3, 1958. Two days previously he had punctured the flexor aspect of his right wrist and the base of his little finger with pieces of wire, which he had promptly withdrawn. Twenty-four hours later he developed pain in the wrist and then the hand.

On examination small puncture wounds at the above sites were noted. There was considerable tenderness in the little finger, together with tenderness and swelling of both palm and dorsum of the hand. An injection of benzylpenicillin was given, and also 1,500 I.U. of A.T.S. The hand was splinted.

The next day the swelling of the wrist had increased. Local exploration at the site of the puncture wound revealed no superficial pus.

On April 5 there was still no evidence of real localization. The hand remained very swollen and tender. Sensory

examination revealed slight but definite blunting of light touch and pinprick perception over the median-nerve area in the hand. Motor function could not be adequately assessed because of pain. No surgical action was taken at this stage, however. Tetracycline was given.

On April 7 an acute progressive alteration of median-nerve function was established. There was complete absence of sensation over the whole median distribution. Ulnar- and radial-nerve areas of sensation were normal. It was decided to explore the nerve in the carpal tunnel.

Operation.—This was performed on April 7, under general anaesthesia in a bloodless field. The same incision was used as in Case 1. The transverse carpal ligament was completely divided. The nerve appeared to be only moderately compressed in the tunnel, and there were none of the signs of severe local compression which were noted in Case 1. The tendon sheaths were opened, revealing a lot of thin purulent exudate. A swab was taken for culture. The wound was closed, several strands of nylon being used as a drain. The hand was splinted. Culture of the pus revealed a growth of β -haemolytic streptococci sensitive to penicillin, and also a coagulase-positive *Staphylococcus aureus* and *Streptococcus faecalis* which were insensitive to penicillin and streptomycin but sensitive to tetracycline. He was having both penicillin and tetracycline, in fact, and these were continued.

Local symptoms of pain and tenderness were slow to subside, though wound-healing was rapid. Splintage was continued for 10 days before active movements were encouraged. On April 18 a patchy recovery of perception of pinprick was noted, mainly at the bases of the fingers. Subsequent recovery was very slow. By June 3 he still had not got a full range of active movement, and he complained bitterly of pain in the scar. There was still impaired sensation in the middle finger, but recovery in the other digits was almost complete. At this stage he was lost from follow-up for some time. However, re-examination 12 months later revealed a good late result, with almost full recovery of tendon and nerve function. There was no wasting of the thenar eminence. Abduction and opposition of the thumb were powerful. Flexion of the fingers was slightly limited, but this barely affected his grip, which was only slightly less powerful than that of the uninjured hand. Sensory examination revealed only very slight patchy blunting of light touch and pinprick perception over the median-nerve distribution.

Case 3

A housewife aged 50 was admitted to the Queen Elizabeth Hospital, Birmingham, on December 31, 1954. She complained of severe throbbing pain in the right thumb and index finger, which had become very swollen and inflamed over the course of a few hours. She had had several attacks of shivering. She remembered cutting her thumb and index finger on some "tinsel" six days previously, but had taken little further notice of these apparently trivial injuries. Her doctor had given her an injection of penicillin before referring her to hospital.

On examination her temperature was 101° F. (38.3° C.). She was obviously acutely ill. The whole of the right hand was swollen, the swelling extending to just above the wrist. The thumb and index and middle fingers were principally involved and were very tender. Small, apparently clean, shallow cuts were present on the pulps of the thumb and index finger. There were tender enlarged glands in the axilla.

She was thought to have a rapidly spreading cellulitis of the right hand and forearm, probably of streptococcal type. The arm was splinted and elevated. She was given 1,500 units of antitetanus serum, and put on systemic penicillin and oral tetracycline. Her general condition slowly improved, and her temperature fell to 99° F. (37.2° C.) in three days. During this time her hand became less painful, but the swelling did not subside. On January 4, 1955, she complained of tingling, then numbness of median-nerve distribution, suggesting that the nerve was compressed in the

carpal tunnel. Further swelling slowly developed, together with acute tenderness both in the palm and just above the transverse carpal ligament on the front of the wrist. There was almost complete loss of median-nerve sensation in the thumb and index and middle fingers. Motor function of the short thenar muscles could not be tested because of the extreme swelling and tenderness. On January 7 it was decided to explore the front of the wrist.

Operation.—This was performed by Mr. A. Gourevitch under general anaesthesia, in a bloodless field. An incision was made along the ulnar border of the wrist, and Parona's space was opened. The tissues were all oedematous and a small quantity of blood-stained exudate was evacuated. A second incision was made, curving round the base of the thenar eminence and up to the distal wrist-crease. The palmar fascia and transverse carpal ligament were exposed. The latter was divided completely, exposing the median nerve. No macroscopic evidence of compression was noted, but some further blood-stained fluid was evacuated from here and from the mid-palmar space, which was explored by an incision through the palmar fascia. No frank pus was found. A small rubber drain was placed in each wound and the incisions were closed with silk sutures.

Organisms were not cultured from the swab taken at operation. Nevertheless, antibiotic treatment was continued in view of the persistent inflammatory changes in the hand. These slowly subsided over the next 48 hours, and there was patchy return of sensation except at the tips of the affected digits. Movements were encouraged from this stage as there was gross stiffness of the hand. Improvement was steady but slow. She was allowed home three weeks after operation, at which time sensory recovery was full except for the terminal pulp areas. Physiotherapy was continued for a further two months, during which period hand and finger movements returned almost to normal.

She was seen again recently, five years after her illness. Power of grip was very slightly weaker than on the left. Movements were normal. There was persistent minimal blunting of light touch sensation on the tips of the thumb and index finger, but this caused her hardly any disability apart from, she stated, making her slightly clumsy when doing very fine needlework.

Discussion

In their two cases of the same condition, Bailey and Bolton Carter (1955) suggested that interference with median-nerve function resulted from compression by inflammatory exudate in the radial and ulnar bursae deep to the transverse carpal ligament. They preferred this hypothesis of compression ischaemia to the alternative explanation that the changes were produced by the direct spread of infection to the nerve.

The present series would appear to support their view. Particularly in Case 1 did the appearance at operation suggest compression. In this case and also in Case 2 there was tight distension of the flexor tendon sheaths in the carpal tunnel. The somewhat later onset of nervous signs in Case 3, after the infection was coming under control, also suggests a pressure effect rather than an infective neuritis.

On the basis of the experience of these three patients, and in view of the two similar cases previously described, it is urged that a close watch be kept for signs of interference with median-nerve function in all severe acute hand infections, and particularly in those in which the tendon sheath is involved. Such cases should be treated by division of the transverse carpal ligament as soon as any signs of damage to the median nerve are noted. This division must be complete, and at the same time the ulnar and radial bursae should be opened to drain any infective exudate therein.

Summary

Three cases of acute infection of the hand complicated by median-nerve paralysis are described.

In two patients the tendon sheaths deep to the transverse carpal ligament were shown to be involved in an acute streptococcal infection.

The nerve lesion was probably due to pressure in the carpal tunnel as a result of the acute inflammatory swelling. All three cases were treated by division of the transverse carpal ligament.

I thank Mr. A. B. Watson for permitting me to treat Cases 1 and 2, and Mr. A. Gourevitch for allowing me to publish the details of Case 3; also for their help and encouragement in the preparation of this paper.

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AUTOMATIC CLOTTING-TIMER FOR CONTROL OF ANTICOAGULANT THERAPY

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In the control of anticoagulant therapy there are two basic requisites: first, the laboratory control; and, secondly, the equally important experience needed to prescribe anticoagulant drugs so as to allow for the inevitable changes in the maintenance dose which may occur owing to changes in health, diet, administration of other drugs, etc. Much can be done to maintain adequate anticoagulant control by recognizing these "periods of danger" (Toohey, 1959) and making any necessary changes in the dose as early as possible, and by taking more frequent blood tests during these "periods of danger." Here the great advantage of the physician who actually prescribes the anticoagulant drug seeing the patient at the time the blood test is taken, as opposed to a "postal service," is clearly evident. With regard to the actual anticoagulant drug, there are now available several good oral anticoagulants for the control of both short- and long-term anticoagulant therapy (Toohey, 1959).

Unfortunately, even to-day, after many years of experience with anticoagulant therapy, there is no universal agreement on the best laboratory method for the control of this treatment. Controversy still exists about the relative merits of the two most widely used laboratory techniques—Quick's one-stage technique and Owren's "P and P" method. Quick's one-stage technique has the great advantage of speed and simplicity. Owren's method is more sensitive and also allows more accurate comparison of results between different laboratories. Owren's method, however, has great disadvantages for widespread use: first, the

*Dr. Toohey died on March 21, 1960, after this paper had been prepared.