PARALYSIS OF THE ULNAR NERVE AND MANAGEMENT OF ITS DEFORMITY
WITH SUGGESTION FOR TEMPORARY PARTIAL BLOCKAGE
DURING RECOVERY

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Paralysis of the ulnar nerve has been more frequent and unfortunately the results of suture of this nerve have given less gratifying results than in any other peripheral nerve. Reasons for this are: (1) the ulnar is a mixed motor and sensory nerve carrying different types of nerve fibers, (2) distal innervation is to small highly specialized muscles for the finer movements of the hand, (3) another reason may be the poor blood supply of the nerve which may be further impaired by anterior transplantation of the nerve.

The functional return of a useful hand should be the objective when confronted with an ulnar nerve paralysis. This may be obtained by neurorrhaphy or by a combination of primary nerve repair followed by tendon transfers for deficient regeneration. In either case we must preserve the parts of the hand that are essential for function. An innervated, but stiff hand is useless. Steps must be taken to preserve muscles, joints and soft tissues in a state of mobility.

It seems to the writer that mechanical splintage of paralyzed joints must follow more closely the anatomical and physiological characteristics of a part. The prevention of muscle stretching is important, but the joint structures on which the muscle acts must also be considered; namely, the ligaments, joint capsules and soft tissues. For example, tight skin about a joint can vitiate completely normal nerve-muscle-joint mechanism.

Paralysis of the ulnar nerve sparing the nerves to the flexor digitorum profundus muscle presents a deformity of the hand that is not seen when the lesion is above the elbow. Phylogenetically, the muscles innervated by the ulnar nerve are oldest and more highly co-ordinated than those supplied by other peripheral nerves. This nerve is peculiar by supplying muscles at different levels in the extremity, but which co-ordinate for fine hand movement involving identical joints. An active flexor digitorum profundus muscle sets in motion the platform of origin of the lumbrical muscles. The latter, however, have their insertion upon the dorsal expansion of the metacarpophalangeal joint.

Paralysis of the ulnar nerve above the elbow, producing a flail state of all the muscles supplied by this nerve, causes a minimal deformity of the hand. However, a lesion below the elbow sparing the innervation of the
flexor digitorum profundus produces an unequal pull on the "a" joint of the little and ring fingers. The deep flexors to these digits flex the interphalangeal joints. The lumbrical muscles initiate the arc of flexion in the "a" joints. When the latter are paralyzed, this joint is tipped into extension by the extensor tendon. This position is maintained by the continuous pull of the active flexor digitorum profundus muscle. Apparently, an active

lumbrical muscle is the key to a neutral balance between extension and flexion of the metacarpophalangeal joint. Though less noticeable, the same pattern of deformity is present in median nerve paralysis if the lesion is distal to the innervation of the deep flexor muscles to digits 2 and 3. If the median nerve alone is paralyzed, the intact interossei help maintain the balance. However, a combined ulnar and median nerve paralysis gives rise to the claw hand. This deformity is produced by the same mechanism.

An obvious and interesting fact of ulnar nerve paralysis is that the deformity associated with lesions below the elbow is greater than that with
The lateral view of the deformity shows the dorsal extensor apparatus of the "a" joint pulled upwards by the active extensor muscle. The paralyzed lumbrical muscle has allowed the proximal phalanx to tip into extension. The flexor digitorum profundus muscle pulls the distal phalanges into flexion.

The dorsal view of the extensor apparatus reveals the lumbrical and volar interosseous muscles inserted to the dorsal hood. The lumbrical muscle originates on the flexor tendon but is inserted on the dorsal expansion. In the little finger the latter muscles are inserted on the radial side. The abductor digit quinti substitutes for the interossei on the ulnar side. (Redrawn from Grant)
lesions above the elbow region. This results from the muscle imbalance exerted on the hand when the innervation to the ulnar portion of the profundus flexor is intact. This observation has been recorded by Bunnell and by Davis. When this muscle is activated, a flexion contracture is superimposed upon the little and ring fingers. This accentuates the hyperextension deformity of the “a” joint because the paralysis of the interossei and lumbral muscles, which extend joints “b” and “c” and flex the “a” joint, allows the unopposed action of the extensor tendons to throw the proximal phalanx into extension. This attitude of the digit allows the middle and distal phalanges to fall into flexion; only however, if the flexor tendons are active.

The physiological effect of this uneven muscle pull manifests itself early by deformity of digits 4 and 5, especially so in the latter, described as the typewriter position of the fingers, and later by limitation of motion in “a” joints. The limitation of motion in the “a” joint is caused by thickening and shortening of the collateral ligaments of the joint and peri-articular fibrosis at the interphalangeal joints. This effect apparently is not from disuse, but from the uneven pull exerted about the joint by the tendons involved. Clinically, this is obvious in brachial plexus lesions when the small joints of the hand are flail from a complete paralysis in the extremity. No motion is possible in the joints except passively, but they remain supple even months after injury.

When the ulnar nerve lesion is above the elbow, the patient retains the ability to clench a fist though there is little power in the little finger. If the flexor digitorum profundus is paralyzed the ugly deformity of the hand is absent, and with the passage of time incident to nerve regeneration following
suture the “a” joint maintains its mobility and function until the deep flexor is re-innervated. As the flexor muscle gains power, the deformity becomes more evident.

Anatomically the metacarpophalangeal joint is supported by two pairs of ligaments: the volar and the dorsal; and the two collateral ligaments. The collateral ligaments have fibers running parallel to the joint surface. When the “a” joint is in extension the collateral ligaments loosen up and allow abduction and adduction of the joint. When the joint is in complete flexion, the collateral ligaments shorten and prevent abduction and adduction. Therefore, the latter motions are impossible when the finger is in flexion, whether this is caused by muscle imbalance or because the finger is kept in flexion by external splintage. It seems that the usual type of splint for this deformity directs its force to further tighten the collateral ligaments, by increasing the flexion of the joint and eliminating abduction and adduction movements. The volar ligaments do not seem to hypertrophy as much as the collateral, probably because the line of directional stress upon the ligament is different.

It has been noted on various occasions following anterior transplantation of the ulnar nerve necessary for nerve suture that for several days following surgery the 4th and 5th digit deformity in the hand disappears and the patient is relieved from the constant tension on these digits. He feels better! This is probably secondary to a temporary paralysis of the fibers to the deep flexors from handling the nerve fibers in dissecting them from the main nerve trunk and from edema in the surgical wound. The power to make a fist does not seem to be impaired. The decreased deformity has also been noted in high lesions of the ulnar nerve; this becoming worse as the nerve fibers regenerate to activate the deep flexor muscles. On several occasions, the neuroma-in-continuity involved sacrificing the motor branches to the flexor profundus muscle before normal-appearing fascicles could be obtained for nerve suture. In these cases also there was a disappearance of the deformity and each patient presented a better hand. These observations suggest the benefit that may accrue from a partial temporary blockage of the nerve in treatment.

The muscle imbalance in low ulnar nerve lesions could be temporarily
eliminated by dissecting free the innervation to the flexor digitorum profundus from the main nerve trunk, a sufficient distance proximal so that the fibers could be crushed much in the fashion that a phrenic nerve is, to produce a temporary paralysis of this muscle. If this were done from 10 to 12 cm. above the myoneural junction the tension effect of this muscle upon the metacarpophalangeal joint producing thickening and deformity of the small joints in digits 4 and 5 would be postponed sufficiently long to allow innervation of the distal muscles, namely, the interossei and the lumbricals. We have not as yet performed this procedure but there is no reason to believe that a permanent paralysis of the deep flexor would result from crushing its nerve supply; however, even if it did the ability to make a fist would not be impaired.

We have performed several amputations of the little finger with its metacarpal head because of deformity secondary to irreparable lesions of the ulnar nerve below the elbow. This was necessary because the metacarpophalangeal joint was stiff from fibrosis of its capsule. These patients had
routine physiotherapy consisting of passive exercises of the finger joints, hot bakes, massage, hot paraffin treatment and electrical stimulation. In one of these irreparable lesions the sensory component arose from the nerve trunk at a higher level than the penetrating wound. This man had sensation over the ulnar distribution in the hand. If the small joints could be saved in a case like the above, the patient would still have a good functioning hand or one amenable to tendon transplants; assuming, of course, that the nerve suture had failed completely. This could have been accomplished by defunctionalizing the ulnar half of the flexor digitorum profundus muscle. This procedure seems like destructive surgery, but in the long run would have proven to be conservative therapy eliminating amputation of the 5th digit and its corresponding distal third of the metacarpal bone and would have resulted in a useful hand without deformity.

SUMMARY

1. Paralysis of the ulnar nerve below the elbow gives more deformity of the hand than paralysis above the innervation of the flexor digitorum profundus muscle.

2. Constant flexion of the 5th metacarpophalangeal joint produces fibrosis of its capsule with ankylosis of this joint.

3. Defunctionalizing the flexor digitorum muscle would diminish this fibrosis and deformity of the 5th metacarpophalangeal joint. This could be done by crushing the motor fibers to this muscle.

4. Splinting the 4th and 5th digits in flexion for ulnar nerve paralysis may be unphysiological.

5. Temporary denervation of the deep flexors to digits 2 and 3 from median nerve paralysis alone or accompanied by ulnar paresis would minimize main en griffe or claw hand.

6. Prevention of stiff hand joints by eliminating the factor of tension gives the constructive surgeon a better chance to effect tendon transfers. Hands requiring tendon transfers must be supple and pliable.

7. An innervated but stiff hand is useless.

REFERENCES

