

The Ulnar Nerve and Ulnar Claw Hand: An Explanation of the Deformity

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Abstract

An understanding of how this deformity develops is very instructive and satisfying. An explanation is suggested.

Case Study

A 56-year-old electrician sustained a laceration of his left mid-forearm with transection of his ulnar nerve. A delayed repair with nerve grafting was carried out but with no return of function. He was left with significant ulnar intrinsic wasting and weakness, with sensory loss in the medial hand, his little finger, and the ulnar half of his ring finger. He also showed the classical features of an ulnar claw hand.

Figure 1 shows the typical deformity in an ulnar claw hand with extension at the level of the Metacarpophalangeal (MP) joints and flexion at both the Proximal Interphalangeal joints (PIP) and Distal Interphalangeal joints (DIP) of the ring and little fingers. Note the wasting of the first dorsal interosseous.



Figure 1: Ulnar claw hand.

Discussion

The explanation of this deformity is very instructive and satisfying. However, two different issues need to be understood in this regard. The first is the difference between balanced and unbalanced paralysis and the second, is an understanding of the function of the intrinsic muscles of the hand.

The difference between balanced and unbalanced paralysis

- Say for example, a person has a complete lesion of the radial nerve in the upper arm which does not recover.
- This leads to loss of extensor function of the wrist with resultant wrist drop and eventual deformity due to the unopposed action of the wrist flexors.
- This is an unbalanced paralysis, which leads to deformity.
- Now assume a person has a brachial plexus lesion with loss of both flexor and extensor function.
- This is a balanced paralysis which leads to flailness.
- An unbalanced paralysis therefore leads to deformity; a balanced paralysis leads to flailness.

It is worth noting that the treatment for each of these conditions is very different. (Please note that these are general surgical/orthopaedic principles which would apply to any similar situation).

- If one has an unbalanced paralysis of the wrist due to paralysis of the extensor muscles with a still mobile deformity, the treatment is to transfer one of the flexor tendons of the wrist to the extensor tendons, to give some extensor function and therefore, restore balance.
- On the other hand, if one has a balanced paralysis due to paralysis of the flexor and extensor muscles which leads to flailness, the treatment is stabilisation, either externally by means of a splint or internally by fusion of the wrist.

➤ Condition	Outcome	Treatment
Unbalanced paralysis →	Deformity →	Tendon transfer
Balanced paralysis →	Flailness →	Stabilisation – (external/internal)

Intrinsic muscle function

- The second issue or principle to understand is the function of the intrinsic muscles of the hand.
- **Figure 2** shows that contraction of the intrinsic muscles causes flexion at the MP joints and extension at the IP joints.



Figure 2: Intrinsic function: MPs flexed and IPs extended.

- It is important to realise that the long flexors do not have any direct effect on the MP joints and that flexion of these joints follows flexion of the DIP and PIP joints in a 'roll-up' action.
- Therefore, when the fingers are extended, there can be no 'roll-up' action, and the only way one can flex the MP joints is via the intrinsics.

Explanation of the ulnar claw hand deformity

- Note again the deformity in an ulnar claw hand with extension or hyper-extension at the level of the MP joints and flexion at the IP joints of the ring and little fingers.
- Concentrating first on the MP joints and recall that the intrinsics cause flexion of these joints. In an ulnar nerve lesion with loss of function of the intrinsics, there is loss of flexion of the MP joints while there is still active (unbalanced) extension.
- So, one has an unbalanced paralysis which leads to deformity in favour of extension.
- At the level of the DIP joints, the long flexors (flexor digitorum profundus - FDP) are still functioning whereas the extensors (that is the intrinsics) are not functioning, leading to a deformity in favour of flexion.

Summary

In an ulnar claw hand, there is unbalanced paralysis at the level of the MP joint leading to deformity in favour of extension and unbalanced paralysis at the level of the IP joints leading to deformity in favour of flexion.

(Recall that flexor digitorum superficialis is supplied by the median nerve as are FDP to the index and middle fingers. FDP to the ring and little fingers are supplied by the ulnar nerve).

The ulnar paradox

We need to consider the difference between a high and a low ulnar nerve lesion.

- In the low lesion, the function of the long flexor tendons (FDP) is preserved leading to unbalanced paralysis at the level of the DIP joints which in turn leads to deformity in favour of flexion as noted above.
- However, in a high ulnar nerve lesion, the long flexors (FDP) of the little and ring fingers lose their nerve supply and therefore, instead of an unbalanced paralysis at the DIP joint in favour of flexion, there is a balanced paralysis which leads to flailness rather than deformity.
- The deformity is therefore less noticeable.
- Hence the use of the term 'paradox'. The high ulnar nerve lesion results in less deformity than a low lesion whereas one would normally expect the opposite.

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