Functional Anatomy and Assessment of the Hand

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INTRODUCTION

This chapter reviews the anatomy and (patho)kinesiology of the hand as it relates to weakness and paralysis of the intrinsic and/or extrinsic muscles. First, the muscles of the hand and their innervation will be discussed. Second, normal hand function will be reviewed followed by a discussion of the primary and secondary impairments that may be present, or that may develop when there is weakness of the muscles of the hand.

The chapter concludes with a discussion of specific examination techniques for the hand. This will be discussed in relation to tendon transfer procedures aimed at improving the function of the hand.

ANATOMY REVIEW

The muscles of the hand can be divided into extrinsic and intrinsic muscles. The extrinsic muscles are the long muscles for movements of the wrist and fingers originating proximal to the hand. The intrinsic muscles are the small muscles within the hand itself.

The extrinsic muscles can be divided into five groups of three muscles each: 1) wrist flexors; 2) wrist extensors; 3) finger flexors; 4) finger extensors and 5) a fifth group comprising the thumb extensors (Table 5-1). An additional group of five muscles make up the pronators and supinators of the forearm (Table 5-2).
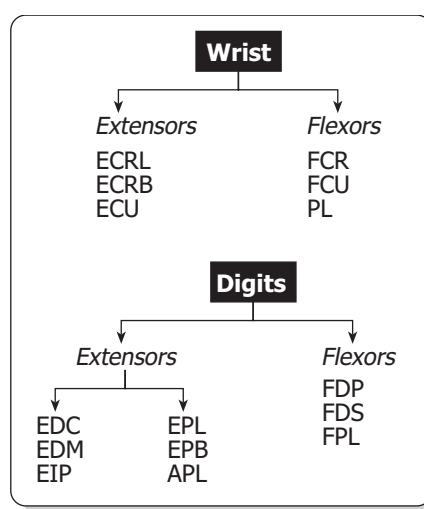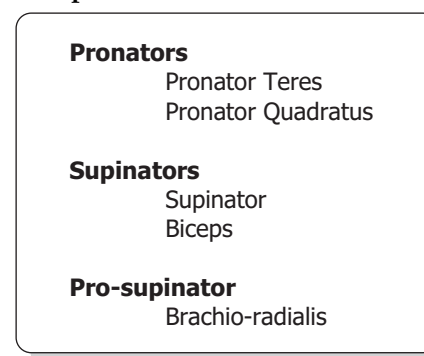
Table 5.1: Extrinsic Muscles of the Hand (5x3)

Table 5.2: Primary Pro- and Supinators of the Hand/Wrist

Pronators
Pronator Teres
Pronator Quadratus

Supinators
Supinator
Biceps

Pro-supinator
Brachio-radialis

The intrinsic muscles can be divided into five groups of four muscles each: 1) hypothenar: 2) thenar; 3) dorsal interossei 4) palmar interossei and 5) lumbricals (Table 5-3).

Table 5.3: Intrinsic Muscles of the Hand (5x4)

Hypothenar	Thenar	
Abd. dig. min.	Abd. poll. brevis	
Flex. dig. min.	Flex. poll. brevis	
Opp. dig. min.	Opp. poll.	
Palm. brevis	Add. poll.	
Lumbricals	**Dorsal Interossei**	**Palmar Interossei**
Index	Index	Index
Middle	Middle (2)	
Ring		Ring
Small	Small	Small
		(Thumb)

Innervation

1 All *extrinsic extensors* are innervated by the *radial nerve*.
2 All *extrinsic flexors* are innervated by the *median nerve* except: the ulnar half of the flexor digitorum profundus (FDP) and the Flexor Carpi Ulnaris (FCU) which are innervated by the ulnar nerve.
3 All *intrinsic muscles* are innervated by the *ulnar nerve* except: the muscles that will bring the thumb into opposition, and the lumbricals for the index and middle finger which are innervated by the median nerve.

When dealing with a peripheral neuropathy or nerve injury it may be helpful to recall the innervation of the individual muscles. This will help to establish the extent of the paralysis, monitor changes in muscle strength and to determine which muscles might be available for transfer.

When dealing with peripheral neuropathies or nerve injuries, the surgeon and therapist need to be aware of the presence of a Martin Gruber 'anastomosis'.[4,11,15] In the Martin-Gruber anastomosis nerve fibers from the median nerve in the forearm cross over into the ulnar nerve (Fig. 5-1). Reported prevalence of this occurrence is between 10-25 percent. In such cases a variable number of intrinsic muscles on the radial side of the hand, but especially the flexor pollicis brevis and first dorsal interosseus, are median innervated through the distal ulnar nerve. In 'confirmed' high ulnar lesions the clinical signs and symptoms may therefore be fewer or less obvious. In the presence of a Martin-Gruber anastomosis, a high ulnar palsy and a low median palsy in the same extremity may confuse the surgeon and therapist that are unaware of this 'anomaly'. A patient with a high ulnar lesion and a low median lesion may still be able to abduct the thumb and may have a very active first dorsal interosseus muscle.
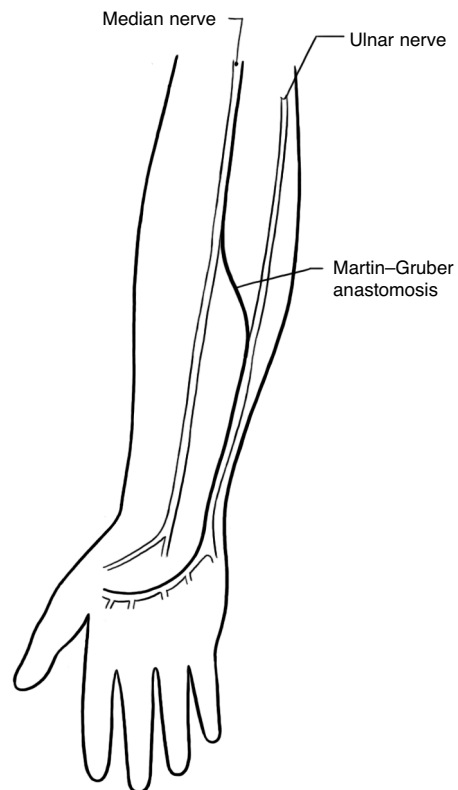

FIGURE 5-1 Martin-Gruber 'anastomosis'.

BASIC FUNCTIONS OF THE HAND: GRASPING AND PINCHING

Many hand functions require an extended and stable wrist. Weakness in wrist extension may cause the wrist to go into flexion when a strong grip is required. Wrist extension (radial innervated muscles) may be corrected 'statically' by providing a splint that will support the wrist in extension. It may also be corrected 'dynamically' by a tendon transfer.

Full finger extension is both a function of the radial innervated wrist muscles and the intrinsic finger muscles, primarily the interossei. Full finger extension can also be enhanced by either a splint or a tendon transfer. Restoring full finger extension, however, in a radial palsy is functionally less important than restoring wrist extension.

In a radial nerve palsy thumb interphalangeal (IP) extension is usually still possible. Surgeons and therapists should not mistake this to be the result of 'anomalous' innervation or think that the radial nerve is only partially damaged. The intrinsic muscles of the thumb have an expansion into the thumb extensor mechanism similar to the extension of the interossei and lumbricals into the extensor mechanism of the fingers.

Loss of strength in the median innervated thenar muscles will 'dis-able' the thumb. The thumb will often not be able to 'oppose' the fingers in various activities of daily living for which a pinch is required. Again, a splint could be provided which will enable the thumb to meet the other fingers in opposition. A single tendon transfer, an 'internal-dynamic' splint, will achieve the same. Some cases with an isolated median nerve palsy in which the FPB is entirely ulnar innervated may show full opposition of the thumb.

If there is also weakness of the adductor-flexor muscles of the thumb then grip strength may also be impaired. Generally speaking, the *median* innervated muscles *position* the thumb for pinching, the *ulnar* innervated muscles, adductor-flexor group, provide the *power* that is needed in pinch activities. In addition, the first dorsal interosseus is an important muscle providing pinch strength.

Weakness or paralysis of the ulnar innervated thenar muscles may result in a flexion deformity of the thumb IP joint (Froment's sign) or hyperextension of the metacarpophalangeal (MCP) joint (Jeanne's sign). Whether these impairments occur depends on such factors as: use of the hand, hand dominance, degree of weakness, innervation pattern of the Flexor Pollicis Brevis (FPB), MCP joint articulating surfaces, and joint laxity.

PATHOKINESIOLOGY OF THE ULNAR PARALYSED HAND

In lesions of the ulnar and median nerves, the intrinsic muscles of the hand are paralysed and will fail to modulate and balance the role of the extrinsic muscles. In peripheral neurological conditions, weakness and paralysis follow the innervation pattern of the nerve. This will not be the case in 'central' neurological conditions or a motor neuron disease. The impairments commonly seen in isolated nerve lesions will then be less severe or may even be absent.

Clawing

Weakness or paralysis of the intrinsic muscles of the fingers may result in the so-called claw position. Loss of the primary flexors of the MCP joints (interossei) causes the fingers to 'claw' when the patient is asked to fully straighten the fingers, or open the hand. Often patients will get into the habit of flexing the wrist when asked to straighten the fingers. Wrist flexion enhances opening of the hand because it decreases tension in the long finger flexors thereby facilitating opening of the hand by the extrinsic finger extensors.

Clawing may be *overt,* obvious, or it may only become evident on finger loading: *latent,* or hidden clawing. A push on the volar side of the proximal phalanx by the examiner will reveal weakness and will cause the finger to buckle. The interossei are the anti-clawing muscles.

Alternatively, to reveal weakness in the interosseus muscles, the examiner could ask the patient to grasp his own forearm. The fingers with weak/paralysed interossei will show hyperflexion of the proximal inter-phalangeal (PIP) and limited flexion of the MCP joints (compare both hands when only one is weak/paralysed). The degree and extent of clawing may be dependent on such factors as: duration of the paralysis, degree of weakness of the extrinsic finger muscles, whether there is a high or low ulnar palsy, and the presence of a Martin-Gruber nerve anastomosis.

Clawing is a visible, and functional, impairment. However, it is not the only functional impairment when there is weakness or paralysis of the ulnar innervated muscles (Table 5-4).

Table 5-4: Primary Impairments

1. Loss of protective sensation and functional sensibility.
2. Clawing due to loss of primary finger (MCP) flexors (interossei).
3. Reversed finger closing pattern: distal to proximal.
4. Loss of ab- and adduction of fingers.
5. Loss of metacarpal arch / ulnar opposition (hypothenar muscles).
6. Decreased grip strength.
7. Deceased pinch strength (pulp-pulp and key-pinch).

A second functional defect is the 'reversed' finger closing pattern. In the normal hand the finger joints more or less flex sequentially: first the MCP joints, followed by the PIP joints and finally the distal inter-phalangeal (DIP) joints.

This makes it possible for the fingers to grasp larger objects. This pattern is reversed in the fingers with paralysis of the intrinsic muscles. The fingers start flexing from a hyperextended position in the MCP joints and the finger closing sequence will be from distal to proximal making it difficult for patients to grasp larger objects.

Abduction and adduction of the fingers will also be lost. A tendon transfer, by virtue of the insertion of the grafts, is aimed at bringing the fingers together in an adducted position, but active abduction and adduction will not be restored.

Paralysis of the hypothenar muscles causes the hand to become 'flat'. Ante- or propulsion of the fifth and fourth metacarpals is important in cupping of the hand. Cupping of the hand is important in cultures in which for drinking and/or eating, no other 'tool' but the hand is used. Propulsion of these metacarpals also contributes to grip strength and secures the grip.[4]

The main functional impairment, however, may be loss of functional sensibility and loss of protective sensation. Patients may unwittingly injure their hands. They must learn to substitute with their eyes (vision) for the loss of functional sensibility and protective sensation and use adaptive (safety) devices to protect their insensitive hands.

Secondary impairments (Table 5-5)

The hand with paralysis of intrinsic and extrinsic muscles may be in danger of developing secondary impairments.[5] These will develop as a result of muscle imbalance, 'clawing', and loss of opposability of the thumb. Splinting and specific exercises will prevent these secondary defects (Chapter 4). If present at the time of surgery they may influence the final outcome of tendon transfer surgery.

In the a-sensate hand, injuries (e.g. burns, wounds) are considered secondary impairments. Tool adaptations and an adapted life

style modifications should minimise the occurrence of these impairments.

Table 5.5: Secondary Impairments

1. Ulcers/Scars/Absorption
2. Contractures (PIP joints: skin/joint structures)
3. Long Finger Flexor Tightness
4. Contracture Oblique Retinacular Ligament
5. 'Hooding'/attenuation of extensor mechanism
6. Habitual wrist flexion
7. Z-thumb

Joint contractures

The joints most likely to contract are the PIP joints. As long as there is sufficient strength in extrinsic finger extensors and flexor muscles patients will continue to use their hands thereby maintaining flexion and extension range of motion of the MCP joints. The DIP joints are usually pushed into extension when objects are grasped which will maintain the mobility in these joints. To test for this, passive extension of the PIP joint is attempted with the wrist and MCP joints flexed. Extension limitation of the PIP joint will be present.

Flexor tightness

Longstanding claw position of the fingers, together with the often present habitual wrist flexion position, may result in adaptive shortening of the extrinsic finger flexors.

The clinical test for tightness is as follows: The examiner keeps the wrist and MCP joints in flexion. Complete PIP extension, in the absence of joint contractures, should then be possible without feeling restraint from the extrinsic finger flexors. The test is now repeated with both the wrist and MCP joints fully extended. Complete DIP-PIP joint extension may now no longer be possible when adaptive shortening of the multi-articular finger flexors

is present.

Skin Contracture

Longstanding clawing of the fngers will result in contracture of the skin on the volar surface of the finger. To test for this, passive extension of the finger is attempted with the MCP joints extended. When a skin contracture is present the extension limitation decreases as the MCP joint flexes, but there is no change with wrist flexion. Tightness of the skin can also be felt with forced extension of the finger.

Contracture of oblique retinacular ligament

Some patients with long-standing claw position of the fingers may develop a contracture of the oblique retinacular ligament. The function of this ligament has been explained as contributing to the synchronisation of finger flexion and extension.

To test for contracture of this ligament the examiner keeps the PIP joint extended and then assesses range of motion and resistance towards DIP flexion. In the second part of this test DIP flexion is assessed with the PIP joint in flexion. A significant difference between range of flexion in the two tests denotes a contracture of this ligament. In severe contractures hyperextension of the DIP joint may be seen when the PIP joint is extended.

"Hooding" or attenuation of extensor mechanism

A chronic flexion posture of the fingers may result in adaptive changes (growth-lengthening) of the extensor hood. In severe cases this may result in anterior displacement of the lateral bands which then often come to lie on the anterior side of the PIP joint axis. The result is functionally rather like the Boutonnière deformity. A lag in active-assisted extension may develop.

Asking the patient for assisted- active extension reveals 'stretching' of the extensor mecha-

nism. The examiner stabilises the MCP joints and asks for active extension. The lag (difference) between active-assisted and passive extension angles is an indication of the severity of 'hooding' of the extensor mechanism. This, of course, is only a valid test if the extrinsic finger extensors are not weak/paralysed and there are no severe contractures of the PIP joints.

Habitual wrist flexion posture

In the presence of paralysis of the intrinsic muscles of the hand the patients may develop the habit of flexing the wrist to enhance opening of the fingers. With wrist flexion the tension in the extrinsic finger flexors decreases which will facilitate a better opening of the hand (less clawing).

Z-thumb

Depending on the innervation pattern of the FPB, the MCP joint articulating surfaces (mobility) and use of the hand, a patient may develop IP joint flexion deformity and MCP joint hyperextension deformity.

SECONDARY DEFECTS IN THE PRESENCE OF PARALYSIS OR WEAKNESS OF THE MEDIAN AND/OR RADIAL INNERVATED MUSCLES

Patients who have lost the ability to extend the wrist have a poorly functioning hand. Many activities will require an extended wrist. Wrist extension can be achieved with a splint. Loss of wrist and finger extension may contribute to 'tightening' of the wrist- and finger flexors.

With paralysis of the intrinsic muscles of the thumb, the thumb looses its ability to oppose the other fingers in pinch activities. The thumb will come to lie in an adducted and supinated position next to the 2nd metacarpal. A thumb

web contracture is the main complication that may occur. Splinting and exercises will prevent or may overcome a thumb web contracture.

ASSESSMENT

In the preceeding paragraphs the relevant basic anatomical features of the hand and the assessment of specific primary and secondary impairments that may occur in the hand with paralysis of the intrinsic and extrinsic muscles were discussed. Now we will briefly discuss the four main general assessment areas: 1) angle measurement; 2) muscle testing; 3) sensory testing (impairment level) and 4) functional assessment (activity level). It should be remembered that functionality of the hand and the result of interventions (splints/tendon transfers) are determined by the presence and extent of primary and secondary impairments as discussed in the first section of this chapter. (See also Chapter 21 on pre- and postoperative rehabilitation, Chapter 4 on prevention *of* impairments and the assessment forms in the appendices).

Angle measurement

The key joints in the hand with paralysis of the intrinsic muscles to evaluate are the PIP joints of the fingers and the IP and CMC joint of the thumb. The results of various PIP joint extension measurements indicate the presence and severity of primary and secondary impairments.

a) Active extension

The examiner asks the patient to extend the IP joints while the patient tries to maintain the MCP joints in active flexion. The lag in active PIP extension is recorded. Lag in extension is an indication of the in-effectiveness of the extensor mechanism (intrinsics) to extend the PIP joint in the weak/paralysed hand.

Alternatively, in the hand following tendon transfer for intrinsic paralysis the extension angle is an indication of the efficacy of the tendon transfer to contribute to active extension of the IP joints.

b) Assisted or assisted-active extension

The examiner stabilises the MCP joints in some flexion. The patient is asked to actively extend the fingers at the IP joints. The examiner prevents MCP extension so that all extensor force is directed at the IP joints. A possible lag in assisted-active extension, in the absence of a contracture, is indicative of 'stretching' (hooding) of the extensor mechanism. This presupposes that the radial innervated extensors are not weak/paralysed.

c) Passive extension (contracture angle)

This angle measures the possible restraint in PIP extension when the examiner passively extends the PIP joint. Different tissues could contribute towards a lag in passive extension: skin, ligament and joint capsule. In other words, causes of a lag in passive extension could be in intra-articular and/or extra articular tissues.

d) Thumb web

In an ulnar-median palsy the first metacarpal may come to rest against the 2nd metacarpal. Active opposition is not possible and the thumb web and CMC joint may become contracted. There are different ways of measuring the thumbweb angle. The examiner can measure the angle between the first and 2nd metacarpal or the distance between the centre of the first and second metacarpal heads.[13]

Muscle testing

Manual muscle strength testing is useful in establishing the extent and degree of muscle weakness. In nerve injuries and neuropathies muscles strength is a 'proxy' for evaluating the motor conduction of the nerve. The muscles are graded using the Medical Research Council 0-5 (MRC) scale. It should be realised that it is difficult and often impossible to grade individual muscles.[6] Some studies have reported on the reliability of manual muscle strength testing[8] (Chapter 2).

Grip- and pinch strength is also useful to evaluate and monitor strength. Both intrinsic and extrinsic muscles contribute to grip- and pinch strength. It has been shown that paralysis of the intrinsic muscles alone will greatly effect grip- and pinch strength. A sphygmomanometer (blood pressure cuff) can be converted into a grip- or pinch strength dynamometer.[9,10,14]

Sensory testing

Nylon filaments (Semmes-Weinstein monofilaments) are now the instrument of choice to evaluate and monitor sensory status of the skin. With a set of graded nylons the sensory status can be semi-quantitated. The level of protective sensation of the hand and foot has been established in various studies.

The main loss to the hand, however, when sensation is impaired, might be the loss of 'tactile gnosis' or functional sensibility. Some studies have determined a relation between functional sensibility and the 2-point discrimination test. For more information and testing techniques the reader is referred to Chapter 2.

Functional assessment

Different tests have been developed to assess 'functionality' of the hand. What is the hand able to do? With some degree of difficulty? Taking more time? With or without adaptation or substitution? Commonly used tests to assess hand function are grip strength and key pinch strength. The Moberg test and nine hole pegboard test assess hand dexterity, and grip

contact assesses the pattern of hand closure. The D.A.S.H. score (Disability of Arm, Shoulder and Hand) is a useful and valid assessment of hand disability, although its use has not been reported in leprosy.

To date, most studies reporting on the effects of tendon transfer surgery have reported on improved angles (appearance) and some on grip strength. It is often assumed that when the fingers have a proper opening and closing sequence, and when the thumb is an 'opposable' thumb again, that this will benefit the functionality of the hand. Studies are needed to show that this is the case. One scale is being developed, part of which will also be able to assess and monitor activities related to the hand in patients with neurologically impaired function of the hand.[1]

Postoperative assessment and review

Surgeons and therapists alike should be interested in long-term outcome of the surgery. The result at time of discharge form the hospital may not be the same after a few months or years of active use of the hand. Forms that are helpful to review the surgery are given in the appendices. A critical appraisal of corrective surgery and comparing techniques may help the surgeon and therapist to make better decisions about management of the paralysed limb.[2,7,12]

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