

Review Article

Hand kinematics: Application in clinical practice

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ABSTRACT

Pathological conditions of the hand consequent to injuries, paralysis, disease, arthritis and congenital difference results in loss or limitation of function, deformities, stiffness, inadequate power and poor position for pinch. The pathogenesis of deformities is influenced by bio-mechanical principles of joints and muscle function. The crippling impact of secondary changes due to edema, soft tissue contractures, muscle shortening and functional adaptations also have a mechanical basis. For clinicians and hand therapists, it is necessary to understand these fundamental principles of biomechanics to plan treatment modalities. Interpretation of mechanics of hand deformities in rheumatoid arthritis and paralysis will enable the treating team to identify the appropriate interventions of splinting, therapy and surgical procedures. Basic knowledge of the principles of hand clinical bio-mechanics will help the beginner to sail through the multitude of tendon transfers described in the text books of hand surgery and find the best solution for a particular clinical presentation. Similarly, knowledge of bio-mechanics will provide solutions to an experienced surgeon to plan treatment protocols for complex situations. The article presents a concise summary of the basic principles of hand bio-mechanics for common hand conditions seen in clinical practice. Understanding and applying these principles will help clinicians in planning and devising treatment options for common and complex hand conditions.

KEY WORDS

Hand Biomechanics; deformity mechanics; paralytic hand reconstruction

INTRODUCTION

Mobility, stability and strength are the three prerequisites for prehensile hand function. Stability during loading without compromising its mobility is essential for the hand to achieve the various digital positions for activities. In other words, during

a specific function, the mobile segments of the hand should be able to achieve a certain desirable position in space (stability) and retain the said position against resistance (strength). The human hand has evolved to achieve stability and strength without compromising mobility of the organ. Function difficulties arise when any of the three components of the working hand, that is, mobility, stability and strength, are affected in diseases, injury or congenital difference. The kinematic basis of these clinical situations and treatment principles are discussed below.

Few basic concepts of kinematics and terminology used to analyze joint function needs to be outlined. These are as follows:

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- *Degrees of freedom:* Joints normally have one, two or three axes of rotation.^[1] Each axis of rotation represents a degree of freedom for that joint to move around and for tendons and external forces to act on. Each degree of freedom requires a minimum of two motors for control: one to move it one way and one to move it the other way. Joints such as the interphalangeal (IP) joint with one axis of rotation have only an extensor and a flexor. Two-axis joints such as the finger metacarpophalangeal (MCP) joint have two sets of muscles: a flexor/extensor set and an abductor/adductor set.
- In a chain of joints such as the finger, the rule of at least two motors for each degree of freedom applies. Therefore, more than one flexor is required to balance the finger joints distal to the MCP joint.
- The proximal interphalangeal (PIP) joint and the distal interphalangeal (DIP) joint motion is linked; that is, the motion at the DIP joint is dependent on and follows motion at the PIP joint.^[2] The extensor apparatus and its interconnections with retinacular ligaments and intertendinous bands produce a unique situation in which there is simultaneous or linked flexion and extension of both IP joints.
- The extensor digitorum longus along with the extensor apparatus is able to extend the three-digit joints by itself if the proximal phalanx (PPX) is prevented from hyperextending at the MCP joint.
- The intrinsic tendons extend the IP joints in all positions of the PPX on the metacarpal head but are stronger if the PPX is simultaneously extended by the long extensor tendon.
- Axes of rotation of the shoulder, elbow, forearm and wrist: The hand is at the end of the chain of bones and joints that makes the upper extremity. The shoulder is a ball and socket joint with three degrees of freedom. The shoulder places the hand in space, and a limitation to do so in disease and stiffness will limit the reach of the hand and severely incapacitate its function. The elbow (ulna humeral) joint is a hinge joint with one degree of freedom; that is, flexion and extension as stability is its virtue. The forearm rotations increase the sphere that the hand can perform its activity, and restriction of rotation significantly compromises essential activities of daily living. The wrist has two degrees of freedom, that is, flexion and extension and ulnar and radial deviation. The shoulder, elbow and forearm rotation places the hand in a desired position in space for functioning. The wrist makes finer adjustments for the final hand placement. The wrist is

a complex multi-articulated joint that provides many finer arcs of motion while maintaining its stability and strength. Restriction of motion or pain at the wrist impairs the grip and pinch strength of the hand.

ALTERATION OF JOINT MOVEMENT

In a hand the normal transmission of force requires that the joint axis stays in its relationship to both bones at the joint and the joint glides in a normal pattern. In injury, arthritis or paralysis, there is an alteration of pattern of movement around an axis. The axis may change considerably when the joint no longer glides or when the joint subluxates or collapses.

FAILURE OF GLIDING

When the joint surface becomes rough or irregular as in arthritis or intra-articular fractures or when there is a mechanical obstruction as with osteophytes, the normal sequence of motion is compromised. The joint surface in these situations instead of gliding becomes a tilting joint [Figure 1]. The functional axes will then immediately

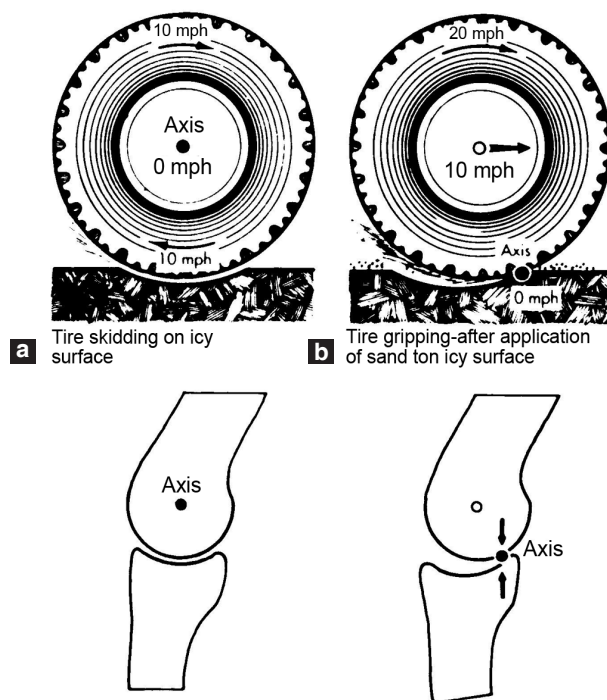


Figure 1: (a) A wheel spinning on its axle on a slippery road is like the convex surface of the head of femur moving in the concave acetabulum, (b) If, with gravel on the road, the gliding-sliding is prevented and the tire grips the road, the wheel moves forward and now the point of contact with the road becomes the axis around which the wheel turns. In the joint, if gliding is blocked and the joint tilts without sliding, the effective axis moves to the joint surface where the tilting occurs

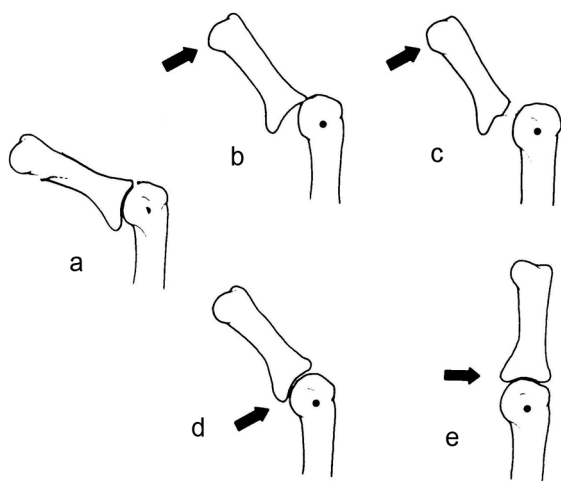


Figure 2: (a) In rheumatoid arthritis, if a phalanx begins to subluxate in flexion, (b) it is tempting to use passive external force to reduce the joint. If the force is applied distally, (c) it results in tilting, and in high stress on the dorsal lip, which over time becomes absorbed. (d) If the external force is applied at the base of the phalanx, (e) it tends to restore joint congruence and correct the subluxations

move and come to lie at the joint surface. The pressure at these new points becomes manyfolds, and the pattern of the joint movement becomes pathological. There is a tremendous increase in the compressive forces at the cartilage where the joint comes in contact for the tilting motion and these results in progressive joint damage and deformity.

This situation classically occurs in rheumatoid patients when the MCP joint fails to glide and begins to subluxate into the palm. Attempts to log lever the finger to extend the MCP joint results in the axis becoming localised to the dorsal edge of the PPX, where it impinges on the head of the metacarpal. This leads to erosion of the dorsal lip of the base of PPX, pain and progressive deformity [Figure 2]. In this situation the therapist should not use the full length of the PPX as a lever to restore angular movement at the MCP joint as this will aggravate the situation. Instead, the PPX should be held near its base, and congruence and gliding at the MCP joint should be restored first. After restoring the gliding pattern of joint and the axis of the joint motion, angular movements can then be achieved without damaging the joint further.

JOINT COLLAPSE

In severe derangement of the joint surface as in intra-articular fracture or in dislocated or collapsed joint, the axis of the joint surface is lost altogether. An example is excisional arthroplasty where the joint no longer moves

about the axes of rotation. The type and the range of motion are entirely different from those of a normal joint. This pattern of motion is a poor compensation and is only useful in limited situations where the adjacent joints can compensate. An example is the excisional arthroplasty for osteoarthritis (OA) of the base of thumb, where the aim of surgery is to relieve pain and retains stability at the basal joint, motion being provided by the distal MCP joint and IP joints. However, introducing a fibrous joint on a basal finger or a thumb joint, one changes the way the joint moves, the joint mechanics and the moment arm of all the muscles at that joint. This invariably leads to an imbalance in the distal joints as seen with thumb MCP joint hyperextension with silastic or excisional arthroplasty of the CMC joint. There is nothing wrong with the MCP joint itself, but the altered mechanics of the basal joints leads to a change in forces of the muscles on the distal joints, deformity, pain and eventually OA (Brand).

STIFFNESS

The collateral and accessory collateral ligaments provide the stability of the lateral finger joints while allowing a full range of motion. The length of these ligaments is critical to allow normal joint motion. The collateral ligaments are fully stretched with the MCP joint in flexion and the IP joints in extension, a position commonly named as the 'intrinsic plus position' (after the action produced by the intrinsic muscles). Immobilization of the hand for any conditions requires the joints to be placed in a position that maintains the full length of the collateral ligament, that is, the 'intrinsic plus position'. The improper immobilization of the hand causes shortening of the collateral ligament, which limits the passive range of motion and is the common cause of joint stiffness. The normal cascade of the hand motion is that with wrist flexion the MCP joints extend and with wrist extension the MCP joint moves into flexion. Keeping the wrist joint at an angle of 30° of extension facilitates maintaining the 'intrinsic plus position' by relaxing the long extensor tendons and the dorsal skin of the hand.

The normal dorsal skin laxity is essential for full flexion of the MCP joint. Injury or disease that produces oedema of the hand inadvertently reduces dorsal skin laxity, encourages the MCP joint to assume a position of extension and reduces flexion at the joint. In such a situation, the long flexors produce only IP joint flexion. In

painful conditions of the hand, there is a natural tendency of the wrist to be flexed and the stretched Extensor digitorum communis (EDC) causes the MCP joint to stay in extension, which leads to further increase in IP joint flexion. The position of wrist flexion, MCP joint extension and IP joint flexion is the classic attitude of the hand in painful conditions and when kept in these positions for a prolonged period it leads to contracture of the collateral ligaments and capsules as noticed with neglected hand stiffness. This position is the complete antithesis to the position of the hand function. Stabilizing the wrist in extension is necessary for all painful condition of the hand and fingers to avoid the sequence of events that causes stiffness of the uninvolved joints. This is the rationale for supporting the whole hand, including the wrist in a finger-tip injury.

The viscoelasticity of soft tissue of the hand allows it to be deformed under stress and provides the forces to restore their resting shapes. Hand oedema affects the viscoelasticity of the soft tissues and increases stiffening. The resistance to gliding tissue and stress on tendon repair site increases with oedema, and this may lead to tendon rupture. Oedema limits the longitudinal movement of fibres by a reorientation in a transverse direction. This is similar to the situation of an anchored ship that has a high mobility at low tide and much less mobility at high tide [Figure 3].

Oedema also has direct effects on the joint movement when changing the moment arms of skin. The skin in an oedematous hand moves away from the axis of the joint and increases its moment arm and thus its resistance to motion in the opposite direction [Figure 4].

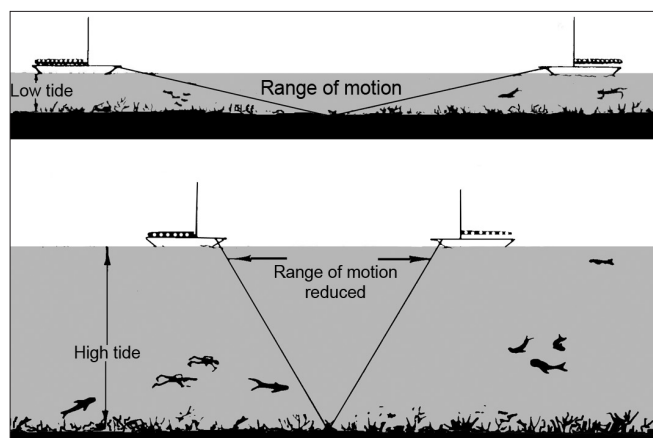


Figure 3: A ship at anchor has wide range of mobility at low tide and much less mobility at high tide. This is because the water (oedema) has lifted the boat. The anchor chain (connective tissue) is now oriented vertically, limiting the boat's range horizontally

FRACTURES

Watson Jones, the father of modern orthopaedics, described a fracture as a soft tissue injury that happens to involve the bone. This is overtly applicable to hand fractures. The impact of the injury to muscle, tendon, nerves, vessels and skin determines the final outcome of the fracture treatment. The major determinant is the amount of energy the soft tissue absorbs during the injury and the site and the pattern of fracture.

The stability of the fracture is influenced by the muscle/tendon forces acting on the bone and the distal articulations. For example, a transverse fracture of the PPX introduces an additional site of motion in the multi-articulated system. Stability of the biarticulated finger is derailed by the introduction of another mobile segment. The proximal fragment is flexed by the strong pull of the interosseous muscles leading to volar angulations. Neutralising the force of the interosseous muscle by full flexion of the MCP joint is crucial to achieve and retain reduction in non-operative management of these fractures. The deformity pattern in the fracture of the middle phalanx (MPX) is determined by the site and the forces. A fracture of the proximal one-fourth angulates dorsally due to unbalanced extensor insertion into the base of MPX. Middle two-fourth fractures could angulate in either direction, and distal fourth fractures have volar angulations as the superficialis tendon pulls on the proximal fragment. Many of these fractures can be managed by applying nail traction and maintaining the finger in 'intrinsic plus position', that is, flexion of MCP joint with IP joints in extension. This position neutralises

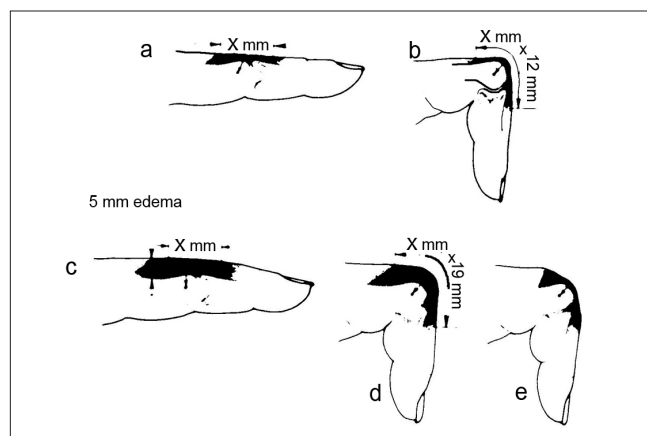


Figure 4: (a,b) Dorsal skin of the digit requires 12 mm of lengthening for 90 degree of flexion, (c,d) With 5-mm thickness of edema, skin requires 19 mm of lengthening for 90 degree of flexion, (e) with continuing application of force for flexion, the oedema fluid moves around the joint, permitting the skin to cross closer to the joint axis and requires less stretch

the intrinsic muscle action, and the longitudinal traction maintains the phalange in reduced position. Periarticular and intra-articular fractures, however, may or may not be amenable to these principles due to ligament attachments.

Metacarpal fractures

The fracture through the neck of fifth and fourth metacarpals, commonly referred to as a Boxer's fracture, angulates dorsally and there is usually a volar comminution of the metacarpal neck. The deformity is aggravated and maintained by the intrinsic muscles that cross the MCP joint volar to the axis of rotation. The dorsal angulation at the fracture in turn leads to a compensatory hyperextension at the MCP joint and flexion at the PIP joint, that is, a 'pseudoclawing'. Reduction of the metacarpal neck fracture is clinically indicated when there is pseudoclawing, and this is achieved by flexion of the MCP joint and PIP joint to 90° and exerting upward pressure through the flexed PPX and simultaneous downward pressure on the metacarpal shaft. The hand is immobilized with the MCP joint in full flexion and the IP joints in extension to neutralize the pull of the intrinsic muscles. Attempting to maintain position in a ball bandage (as was used earlier) will displace the reduction and produce PIP stiffness.

Intra-articular fractures

Fracture–dislocation of the small finger CMC joint: Intra-articular fractures of the hamate-metacarpal joint are usually associated with proximal and dorsal migration of the metacarpal. The displacement is accentuated by the pull of extensor carpi ulnaris and flexor carpi ulnaris through the pisometacarpal ligament. Pull of the abductor digiti minimi also contributes to the instability. These fractures when displaced are inherently unstable due to the forces of these muscles. Closed reduction and cast immobilisation can be risky as these tend to re-dislocate. Reduction and percutaneous pinning of the fifth metacarpal in an extra-articular fashion to the fourth metacarpal following reduction is the optimal treatment.

Avulsion fractures involving the joint at the insertion of tendons are unstable; that is, a Bennets' fracture at the base of thumb metacarpal. The injury may be associated with a dislocation of the injured joint. Closed reduction of these fragments is feasible but redisplacement with external immobilisation is commonly found. The stability of these joints is best restored by operative reinsertion

of these avulsion fragments. If these avulsion fragments are displaced by more than 2 mm, accurate reduction and internal fixation are necessary to prevent joint deformity. Examples are avulsion fracture of the dorsal base of the middle phalanx with the attachment of the central slip. Inadequate reduction leads to extensor lag and boutonnière deformity. Dorsal base distal phalanx fractures lead to Mallet deformity. Similarly, displaced avulsion fracture with ulnar collateral ligament injury at the thumb MCP joint requires operative treatment to restore stability and pinch power.

Fractures of the thumb

Transverse fractures of the thumb metacarpal are angulated with its apex dorsally so that the distal fragment is flexed and adducted. These are due to the pull of the thenar intrinsic on the distal fragment and dorsal pull of abductor pollicis on the proximal fragment. Rolando's fracture at the base of the first metacarpal is the commonest example. Closed reduction is usually stable and 30° of angulations is accepted due to the compensatory movement at the adjoining CMC and MCP joints. An angulation of more than 30° is unacceptable, as this leads to hyperextension deformity at the MCP joint and extensor lag at the IP joint. Open reduction and fixation are usually indicated.

The transverse fracture of the PPX angulates the apex volarly secondary to the pull of the thenar intrinsic on the proximal fragment and the extensor pollicis longus on the distal fragment. Closed reduction is usually stable, but angulations more than 30° will lead to an extensor lag at the distal joint.

Distal phalangeal transverse shaft fractures are unstable. The fracture angulates with apex volar due to the pull of the flexor pollicis longus on the proximal fragment. If reduction cannot be held in a splint, then a longitudinal K wire will maintain stability.

Bennett's fracture

Fracture base of the first metacarpal with subluxations of the CMC joint: The anterior oblique ligament holds the ulnar fragment to the trapezium and the remaining metacarpal base subluxates radially, dorsally and proximally by the pull of the abductor pollicis longus. These fractures if displaced by more than 2 mm should be reduced under fluoroscopic guidance and extra articular K wire pinning of the first to the second metacarpal.

It is not necessary to pin the metacarpal to its ulnar fragment or pin through the CMC joint. Failure to reduce the fracture will result in OA of CMC and compensatory hyperextension of the MCP joint and flexion of the IP joint. The kinematics of joint collapse in these situations and in paralysis is discussed below.

MECHANICS OF FINGER STABILITY

The mobility of the multi-articulated finger allows it to achieve various positions required for function. The muscle forces acting on the MCP and IP joints and the response to application of loads are determined primarily at the MCP joint. The MCP joint, therefore, is the key to the chain of the three finger joints; the position of the IP joints follows that of the MCP joint in a predictable pattern. The IP joint motion is linked and they function as one unit. In mechanical terms, a finger is a biarticular system by virtue of its extensor expansion and the retinacular system of ligaments. The forces acting on these joints are two extrinsic flexors: extensor and intrinsic.

To understand the mechanical stability in this multi-articulated system, we need to apply the stability principles in biarticular systems, as described by Landsmeer.^[3] To simplify this, let us analyse the stability in a uniarticular system. The IP joint is a hinge joint and has one degree of freedom. The MCP joint has two degrees of freedom, that is, flexion and extension and ulnar/radial deviation. There is also a small amount of rotation due to the anatomical configuration of the joint. Let us for a moment consider MCP joint flexion--extension to be of one degree of freedom in the following discussion.

For each degree of freedom, two forces acting on a joint in the opposite direction can provide stability in any position (i.e. stability by a flexor and extensor force acting on the knee or elbow). In a biarticular system like that the one in the finger, two forces are inadequate and a third force is required to achieve stability. This is provided by the intrinsic muscles that produce a flexion force at the MCP joint and extension force at the PIP joint. A loss of this third force, as in intrinsic paralysis, leads to a zigzag deformity producing a claw digit. The pattern of the collapse of the biarticular system depends on the moment arm or leverage of the extrinsic forces. The moment arm for external forces tends to be the largest at the proximal joint than that at the distal joint. For example, on applying a load on the finger, the extensor

torque is highest at the MCP joint and the long flexors are unable to keep the MCP flexed in the absence of intrinsic. Therefore, the deformity pattern in intrinsic paralysis is always predictable; that is, hyperextension at the MCP joint and flexion at the IP joint are commonly referred to as a 'claw deformity'.

PARALYTIC DEFORMITIES: CLAW FINGER

Based on the above mechanical basis of a claw finger deformity, preventing hyperextension at the MCP joint by mechanical obstruction by a dorsal block splint or by a volar capsulodesis can correct the claw deformity. Alternatively, providing an MCP joint flexion force to replace that of the lost intrinsic as done for tendon transfer can restore balance in the digit and prevent clawing. This is the principle of claw correction for all tendon transfers, that is, to provide a flexion force at the MCP joint. The caveat to this is that the integrity of long extensor/extensor apparatus is intact as this extends the IP joints when the PPX is prevented from the hyperextension. The ability of the long extensors to extend IP joints while the PPX is prevented from hyperextension is the basis of the Bouvier manoeuvre for differentiating between a simple and a complicated claw finger. The function of the extensor apparatus is intact in a simple claw finger, so a splint, volar capsulodesis and a flexion force at the MCP joint will correct the claw deformity. When the long extensor is unable to extend the IP joints commonly due to damaged extensor expansion, it becomes a complicated claw finger. Procedures suitable to treat a simple claw finger such as the Zancolli's 'lasso' or a capsulodesis will fail for a complicated claw finger based on the aforementioned mechanical considerations. Complicated claw finger will require replacement of an IP joint extension force in addition to those procedures required for correction of a simple claw finger. Therefore, the surgical option of tendon transfer for treating a simple and complicated claw finger has to be determined on kinematics for a successful outcome. The complicated claw finger will require a procedure such as Bunnells/Brands technique of tendon transfer insertion to the lateral bands. The route is volar to the MCP joint in all these tendon transfer.

Camptodactyly

The cause for this may be abnormal intrinsic insertion or intrinsic insufficiency although the deformity presents as a congenital PIP flexion contracture. In these cases correcting the PIP flexion contracture by Z plasty will

be unsuccessful in the long term. An additional MCP joint flexor as for the claw finger is necessary to prevent recurrence.

THUMB

The thumb consists of three joints with the saddle-shaped CMC joint providing two axis of rotation, one in the trapezium and the other in the metacarpal. The MCP joint is also a two-axis joint: that is, flexion/extension and abduction/adduction. The IP joint is uniaxial. Muscles that cross the MCP joint also cross the CMC joint. The muscles that abduct the CMC also abduct the MCP joint, and the muscle that adducts the CMC also adducts the other joint. This arrangement of the muscles allows the two joints to act together as a unit in the normal thumb to produce circumduction and opposition.

The intrinsic muscles of the thumb – abductor pollicis brevis (APB), opponence pollicis (OP), flexor pollicis brevis (FPB) and adductor pollicis (AP) – merge with each other to form a continuous sheet of muscle around the CMC joint. Although each muscle is an anatomical entity with independent nerve supply, during thumb function they merge with one another to produce a ripple or wave of activity. At one end of this quadrant fan of muscle, the action is flexion abduction (by APB), and at the other end it is flexion adduction (by AP). One end is supplied by the median nerve and the other by the ulnar nerve. The division between the two is variable. In median palsy, there is rarely enough abductor function remaining to avoid the need for a tendon transfer; that is, an opposition transfer for APB replacement is mandatory. The pattern of innervation of FPB determines the need for flexor–adductor replacement in ulnar nerve palsy. If the FPB is supplied by the median nerve, there is enough flexion power at the MCP joint to position the thumb for pinch. If during power pinch, the MCP joint is stable and does not buckle into hyperextension or if the patient does not complain of functional difficulty, then tendon transfer for adductor replacement in ulnar nerve palsy is not required.

There is much disagreement in the choice of procedures in combined median–ulnar palsy. Much of this is due to the lack of understanding of the mechanics for balance of the thumb (Brand). The flexion of the tip of the thumb (Froment's sign) [Figure 5] has been thought to be a failure of the extensor. Therefore, transfers of the abductor–opponence function have been attached



Figure 5: Froment's sign

to the extensor tendon in the hope that this extra pull would result in extension of the IP joint during pinch. The extensor in majority of these cases is normal. The cause of this deformity is a weakness of flexion at the MCP joint, not lack of extension at the IP joint. As soon as a flexion power is added to the MCP joint, the IP joint extension is restored. Therefore, in combined median–ulnar paralysis we need two tendon transfers: one for abduction and the other for MCP flexion/thumb adduction to restore the balance of the thumb. A single tendon transfer for abduction in such a situation will lead to MCP joint hyperextension or a Froment's sign during pinch following a successful opposition transfer. Brand recommends that the stronger muscle should be near the adductor end of the transfer.

There are five mechanical considerations in the choice of tendon transfer operations to restore muscle balance to the hand. These are

- Choice of donor,
- Number of degree of freedom the transfer will control,
- Relationship of the motor to the axes of rotation of the joint that the transfer crosses,
- Pathways in the hand and
- Method of insertion.

The potential excursion of the muscle depends on the muscle fiber length, and the cross-sectional area of the muscle fiber determines the maximum tension the muscle can produce. The ideal donor muscle should have excursion and strength similar to those of the paralyzed muscle for achieving a suitable result. For example, transferring the wrist extensor to flexor digitorum profundus provides enough power but the excursion of

Extensor carpi radialis longus (ECRL) is far below that of Flexor digitorum profundus (FDP). Full fist closure is achieved only with hyperextending the wrist. Similarly, Flexor carpi ulnaris (FCU) or Flexor carpi radialis (FCR) transfer for digit extension requires wrist flexion to achieve full digital extension.

Two muscle forces are required to control each degree of freedom and the force vector of each muscle depends on its relationship to the axes of the joint. This information helps in deciding the number of motors to be transferred and the direction of routing the transfer. Finally the site of insertion of the tendon transfer as near as to the paralyzed muscle will restore the lost function.

Understanding these basic concepts of hand mechanics will help the clinician in planning operations and therapy strategies for hand injuries, paralysis, and hand and

wrist deformities from rheumatoid arthritis. Most of these concepts enumerated above are based on Paul Brand's classic monograph *Clinical Mechanics of Hand*.^[1] This reference manual provides enormous amounts of laboratory and clinical information that can be applied in day-to-day practice.

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