The Collateral Ligament of the Digits of the Hand: Anatomy, Physiology, Biomechanics, Injury, and Treatment

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Learning Objectives

Upon completion of this CME activity, the learner should achieve an understanding of:

- The anatomy of pathophysiology of the collateral ligaments of the digits of the hand
- The most important stabilizers to lateral deviation at the metacarpophalangeal (MCP), proximal interphalangeal (PIP), and distal interphalangeal (DIP) joints
- Diagnosis of injuries to those structures
- Management and treatment of injuries involving the collateral ligaments

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Ligament injuries are among the most common musculoskeletal injuries seen in clinical practice and ligaments are the most frequently injured structures in a joint. Ligaments play an important role in balancing joint mobility and joint stability. Disruption of joint ligaments severely impairs joint function. Over the past 10 years, a new appreciation of a neuroanatomy and neurophysiology of joint ligaments and its biofeedback loops to surrounding muscles and tendons has emerged to explain the relationship between primary and secondary restraints that allow normal joint motion yet prevent pathological motion. This review focuses on this recent information with a view to new clinical approaches to these common problems. *(J Hand Surg Am. 2017;42(11):904–915. Copyright © 2017 by the American Society for Surgery of the Hand. All rights reserved.)*

Key words Dislocation, finger, joint, injury, ligament.

Ligament injuries are among the most common musculoskeletal injuries seen in clinical practice and ligaments are the most frequently injured structure in a joint. Ligaments play an important role in balancing joint mobility and joint stability and their disruption severely impairs joint function.

In 2009, a National Electronic Injury Surveillance System study showed 3.5 million upper extremity injuries a year in the United States.[1] Finger injuries were 38% of those (1.3 million) and 16% of those (210,000) were sprains and strains. Dislocations accounted for 5% (65,000). The incidence of finger sprains is 37.3 per 100,000 a year in the United States. The proximal interphalangeal (PIP) joint is most commonly injured, followed by the thumb metacarpophalangeal (MCP) joint, and then the finger MCP joints.[1]

There are many time-honored methods of treating finger ligament injuries but, unfortunately, all too frequently this injury leads to finger stiffness, instability, chronic pain, swelling, and a substantial loss of function.

Over the past 10 years, there have been many advances in the understanding of the anatomy, physiology, and biomechanics of the ligamentous joint capsule of the MCP, PIP, and distal interphalangeal (DIP) joints. I focus on this new information with a view to new clinical approaches for these common problems.

ANATOMY

MCP joint of the thumb

The collateral ligament originates dorsally on the condyle of the metacarpal head and extends in a palmar and distal direction to insert on the tubercle of the proximal phalanx. It runs adjacent to the accessory collateral ligament (Fig. 1). The radial collateral ligament (RCL) of the thumb has been reported to be 4 to 8 mm wide and 12 to 14 mm in length.[2]

Collateral ligaments of the index MCP joint

The ulnar collateral ligament (UCL) is 4 to 8 mm wide and 12 to 14 mm long.[3] The proper UCL (pUCL) originates at the dorsoulnar MCP head (one-third of the way down from the dorsal surface) and inserts on the proximal volar aspect of the proximal phalanx (one-quarter of the distance from volar to dorsal) The proper RCL (pRCL) originates from the dorsoradial aspect (one-third of the distance from the dorsal surface) of the MCP head and inserts on the lateral tubercle of the proximal phalanx (one-quarter of the distance from volar to dorsal)[4] (Fig. 2).

The center of the origin of the pRCL is 40% volar to the dorsal cortex of the metacarpal head. The most dorsal part of the attachment on the proximal phalanx is 20% volar to the dorsal cortex.[5] The center of the insertion of the pRCL is 46% dorsal to the volar cortex of the proximal phalanx. The most volar portion of the pRCL insertion is 20% from the volar cortex of the proximal phalanx in the index and 29% in the thumb.[6] The distance between the center of the origin and the center of the insertion of the pRCL in full flexion is 15% more than in extension (Fig. 3).

PIP joint ligaments

The proper collateral ligment (pCL) arises dorsal and proximal to the fovea on the side of the proximal phalanx head with an oblong shape and inserts broadly on the base of the middle phalanx. Ligament fibers are stout and parallel to the middle phalanx in all angles of PIP flexion. The volar edge is more oblique than the dorsal edge, giving it a fan shape,

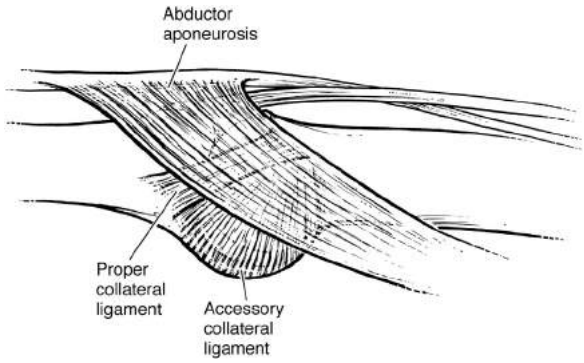

FIGURE 1: Schematic of the relationship of the RCL and the overlying abductor aponeurosis in the thumb MCP joint. Note the oblique path of the pCL from its bony origin and insertion. The aCL travels from the proximal phalanx to the volar plate (proximal and phalanx to left). (Reprinted with permission from Edelstein DM, Kardashian G, Lee S. Radial collateral ligament injuries of the thumb. *J Hand Surg Am.* 2008;33[5]:760–770.[2])

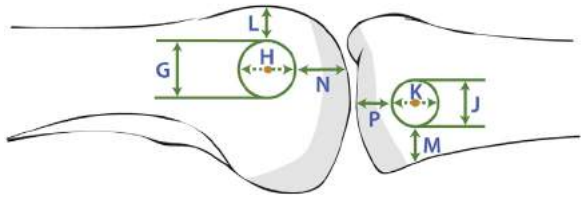

FIGURE 2: The pUCL of the index MCP joint originates at the dorsoulnar MCP head (one-third of the way down) (L) and inserts on the proximal volar aspect of the proximal phalanx (one-quarter of the distance from volar to dorsal) (M). H, J, and K reflect the dimensions of the origin and insertion of the collateral ligament relative to its position. G, width of the origin of the collateral ligament; P, distance of the edge of the insertion of the collateral ligament to the joint surface; N, distance of the origin of the collateral to the joint surface. (Reprinted with permission from Dy CJ, Tucker SM, Kok PPL, Hearns KA, Carlson MG. Anatomy of the radial collateral ligament of the index metacarpophalangeal joint. *J Hand Surg Am.* 2013;38[1]:124–128.[5])

but it does not touch the volar plate at all because there is a bare area between the 2 structures**.** Deeper fibers are more linear but travel in a more oblique direction volarly toward its insertion. With motion, the pCL pivots around the origin. If the pCL originated from the fovea, the range of motion (ROM) would be less. With flexion, the dorsal fibers of the pCL ride over the volar fibers because there is differential gliding of the 2 bundles relative to each other. The accessory collateral ligament (aCL) fibers are flimsy and lie between the pCL and the volar plate. Oriented dorsal to volar, they insert into the dorsolateral volar plate deep to and contiguous with the transverse retinacular ligament. The aCL suspends and stabilizes the volar plate.[7]

PHYSIOLOGY OF THE COLLATERAL LIGAMENTS

Ligaments are covered by an epiligament often indistinguishable from the ligament itself.[8] This layer contains sensory and proprioceptive nerves that are more concentrated closer to the ligament-bone interface.[9] When ligaments are strained, these nerves send out signals either activating or inhibiting surrounding muscle contraction around the joint to protect it from overstrain by modifying the forces across the ligament. This occurs in a finely tuned feedback loop. Ligaments passively stabilize joints by preventing excessive motion and guide the joints through their normal motion under tensile load, distributing those loads so that joints maintain their physiological motion patterns. Under the microscope, the collagen fibers of ligaments appear crimped. When tension is applied across the ligaments, they elongate and "uncrimp" until all the fibers are all nearly linear, giving the ligament its maximum stiffness. Thus, ligaments can undergo viscoelastic elongation without damaging its microstructure. Further stress will disrupt the tissue, which will undergo failure through tearing or plastic deformation, both of which can render the joint unstable.

Chikenji et al in 2011[10] reported on the distribution and function of nerve endings (mechanoreceptors) in human DIP and PIP joint ligaments. These nerve endings have been recognized for decades, but it has become evident only recently that these nerve receptors play an integral part in joint proprioception and injury prevention by activating skeletal muscle contraction to dampen forces around the primary restraints.[11]

Chikenji in 2010[12] reported on encapsulated nerve endings in the PIP joint. Using immunohistochemical staining, they found that, in the PIP joint, there is a high concentration of type 1 endings in the proximal volar plate. These are Ruffini stretch receptors responsible for position sense. Type 2 Paccini nerve endings, which sense pressure and vibration, fire action potentials at the beginning and end of a pressure stimulus[13] but are silent when the stimulus is constant in intensity. These receptors are specialized in the detection of motion. They primarily congregate in the proximal radial-ulnar side of the volar plate, C1 pulley insertion on the volar plate, and aCL.

In the DIP joint, there is increased density of type 2 fibers in the proximal ulnar and radial side of the volar plate where the C5 pulley and aCLs insert into the volar plate. There are relatively few type 2 nerve endings at the distal end of the volar plate and

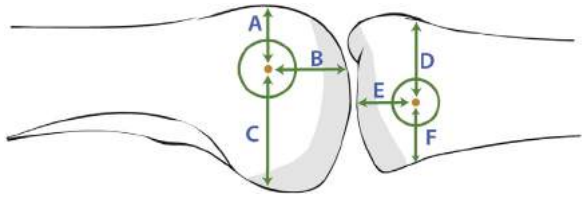

FIGURE 3: Relative distances of the origin and insertion of the pRCL of the index MCP joint to the bony margins of the metacarpal head and proximal phalanges. The most dorsal part of the attachment on the proximal phalanx is 20% volar to the dorsal cortex (A). The center of the insertion of the pRCL is 46% dorsal to the volar cortex of the proximal phalanx (B). Note that the distance from the center of the origin to the center of the insertion is greater in MCP joint flexion (C + E) than in extension (B + E). D, distance from the center of the collateral ligament insertion to the dorsal cortex; F, distance of the collateral ligament insertion to the volar cortex. (Reprinted with permission from Dy CJ, Tucker SM, Kok PPL, Hearns KA, Carlson MG. Anatomy of the radial collateral ligament of the index metacarpophalangeal joint. *J Hand Surg Am.* 2013;38[1]:124–128.[5])

ligaments. Some type 1 fibers are seen in the dorsal capsule and volar plate but not in high concentrations.

COLLATERAL LIGAMENT BIOMECHANICS AND EXPERIMENTAL LIGAMENT RUPTURE MODELS

The collateral ligaments of the MCP joint: general principles

The ROM and stability of the MCP joint of the thumb differ from the fingers in that[14]:

1. The arc of flexion of the thumb MCP is less than that of the fingers.
2. The finger MCP joints stabilize one another by their proximity to one another and the deep transverse metacarpal ligaments.
3. Finger MCP joints are lax in extension and stable in flexion for grasp.
4. Thumb MCP joints must maintain stability in its full range of flexion and extension to allow pinch and grasp.
5. The main stabilizer to abduction and adduction stress is the pRCL and pUCL.
6. The pRCL and pUCL are the static restraints when the MCP is in flexion. In contrast, the aCL and volar plate become taut and are the static restraint when the MCP joint is in extension.
7. Because ligamentous collagen fibers are oriented in the line of force, the direction of joint movement and the bony geometry of joint surfaces will determine which fibers within a particular ligament will undergo tension.[2]
8. Sesamoids stabilize the thumb in extension.[15] In that area, there are 2 volar ligaments: MCP ligament and sesamoid metacarpal ligament. When the sesamoid metacarpal ligament is sectioned, the MCP joint is unstable in extension with hinging. If the pCL is sectioned as well, total rotational instability of the MCP joint results.
9. Thumbs must be evaluated for adduction (ulnar deviation) laxity with the MCP joint in neutral.[16] If the MCP joint is examined in pronation, one may get a false-negative, and if examined in supination, a false-positive examination may be the result.[4]

NORMAL COLLATERAL LIGAMENT LAXITY

The MCP joint

The cam shape of the metacarpal head results in a tightening of the collateral with MCP joint flexion and results in increased joint stability. Lutsky et al[17] found a steady increase in tautness of both the RCL and the UCL as the angle of flexion of the MCP joint increased from 0° to 30° to 90°. Their conclusion was that all collateral ligaments should be evaluated at 0° and 90°. Both hands have the same stability and laxity at all angles of flexion, so one hand can be used for a control assessment of the other hand.

The collateral ligaments of the MCP joint of the index finger have the greatest amount of obliquity in the sagittal plane and that diminishes in the ulnar digits where the pCL is more in line with the long axis of the proximal phalanx. Also, in the index and middle fingers, the shape of the metacarpal head, although circular in the sagittal plane, is trapezoidal in the coronal plane.[18] This means that, as the MCP joint goes into more flexion, the collateral ligament becomes more taut as the distance between the 2 ends of the ligament increases as it passes over the condyles of the metacarpal head. In the ring and little finger, the distance between the 2 ends stays the same so that there is no increase in tightness of the collateral ligaments with MCP flexion.[19] The clinical implication is that the time-honored tradition of using orthotics in fourth or fifth metacarpal neck fractures in extreme MCP joint flexion to prevent an extension contracture may be unnecessary. Another implication is that testing for collateral ligament instability should be made with the MCP joint in flexion.[20] In flexion of the MCP joint, the dorsal fibers of the pRCL and pUCL are taut while the volar fibers and the aCL are lax, whereas in extension, the reverse occurs.

The PIP joint

The PIP joint is designed to undergo between 100° and 110° of motion in flexion and extension and represents 85% of total finger motion. It must do so while

maintaining near-maximum rigidity to radial-ulnar deviation stress in all angles of flexion, unlike the MCP joint. This stability is critical to the normal functioning of the PIP joint. The volar plate is a fibrocartilage thickening of the joint capsule preventing PIP joint hyperextension. The checkrein ligaments are proximal extensions of the volar plate and are attached on the sides to the aCL.

Chen et al[21] studied the changes in length of pCL and aCL during PIP flexion and extension. The dorsal portion of radial and ulnar pCL lengthens in flexion by up to 2 mm at 90° flexion. This is believed due to the passing of the ligament over the condylar tubercles of the proximal phalanx head. The volar portion of the pCL lengthens in extension and shortens up to 2.6 mm moving from extension to full flexion. This could be due to the shape of the condyles and reflects the need to maintain added stability to allow pinch.

The average length of the radial and ulnar aCL shortens 3 mm as the finger goes from full extension to full flexion (like the volar pCL.) The proximal and middle portions of the aCL do not change throughout the ROM of the PIP joint.

EXPERIMENTAL LIGAMENT RUPTURE MODELS

Minamikawa et al[22] conducted serial sectioning studies of the PIP collateral ligament to determine the relative importance of various structures around the PIP joint in maintaining lateral stability.

Lateral stress of the intact collateral produced 5° of adduction and 9° of supination throughout the entire arc of flexion/extension. The PIP remained stable to lateral abduction when up to half of the collateral ligament was sectioned. The fully sectioned collateral ligament causes 20° of lateral abduction and should be considered a third-degree sprain when examined clinically. In sectioning studies, it was revealed that, even if the collateral ligaments are totally sectioned, a remaining intact aCL, central slip, lateral band, and volar plate will prevent lateral angulation under load. However, if all of these are sectioned first, cutting the collateral ligaments will then completely destabilize the joint. They also found that, as long as half of the pCL is still intact, sectioning of all the accessory stabilizing structures mentioned previously will not destabilize the PIP joint.

The clinical implication is that, in dealing with PIP joint contractures, complete excision of the aCL and palmar half of the pCL will enhance ROM of the joint without destabilization. This procedure has been described in the treatment of PIP joint contractures.[23] In all the sectioning tests, maximal lateral laxity occurs at about 50° of flexion.

Bowers et al[24] considered the collateral ligament to be ruptured when there is lateral instability at 90° flexion. Kiefhaber et al[25] defined 20° of PIP angulation in extension to be diagnostic of a complete tear of the collateral ligament.

Minamikawa et al,[22] based on their cadaver study, felt that 10° of lateral angulation in extension and 20° of lateral angulation in 30° of flexion is indicative of a complete pCL rupture. More than that indicates an additional rupture of the aCL and secondary restraints.

Rhee et al[26] studied loaded PIP collateral ligaments to failure and discovered 4 distinct rupture patterns:

1. Midsubstance tear
2. Proximal detachment
3. Distal detachment
4. Bony avulsion fracture

Low-speed forces will commonly produce a midsubstance tear, although such tears may be seen at every force level. At low speeds, the ligaments may stretch considerably prior to rupture and will require higher forces to rupture than a rapidly applied force. High-speed forces produce distal detachments, tears at the pCL/aCL junction and avulsion fractures at the insertion of the palmar plate.

INJURY TO THE COLLATERAL LIGAMENT: CLINICAL ASPECTS

Patterns of injury

Edelstein et al[2] studied injury patterns of the RCL of the thumb MCP joint.

Radial collateral ligament injuries of the MCP joint of the thumb are much less commonly reported than UCL injuries, comprising 10% to 40% of all injuries (Fig. 4). Ulnar collateral ligament MCP joint injuries affect pinch and power grasp whereas RCLs are important for axial thumb tip pressure stability such as pushing a button or radial -sided forces such as closing a door. A complete RCL tear with an intact UCL will result in thumb pronation instability as the proximal phalanx rotates around the thumb metacarpal around the axis of the UCL.

Stener lesions occur mostly on the ulnar side of the MCP joint of the thumb (Fig. 5), although they can occur on the radial side and even in a finger but those are much less common. On the ulnar side of the thumb MCP joint, the adductor is palmar to the axis of rotation so that, when the UCL avulses off the proximal phalanx, it will drift dorsally and proximally allowing the ligament to flip backward and become superficial to the adductor aponeurosis. On the radial side of the thumb MP joint, however, the abductor aponeurosis lies more dorsal and more

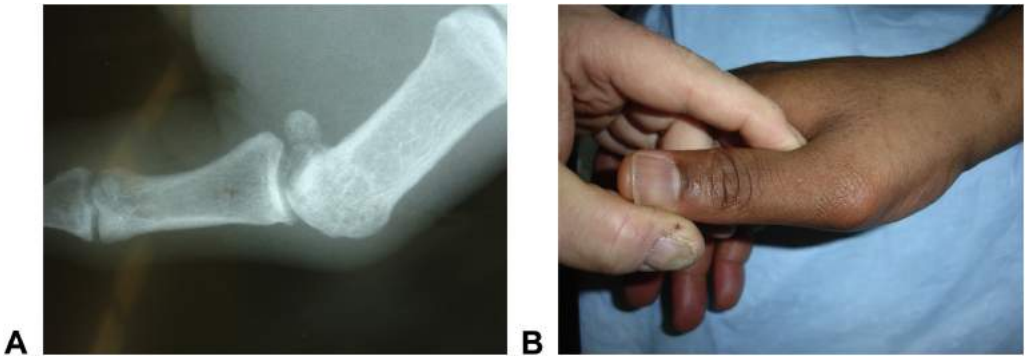

FIGURE 4: **A** Third-degree RCL injury to the thumb MCP joint. Note that the head of the metacarpal is uncovered 50% at rest. **B** Stress maneuver to detect an RCL injury to the thumb MCP joint.

completely covers the RCL so it is much harder for the RCL to flip out from underneath the aponeurosis and get trapped proximally and superficially.

When the RCL of the thumb MCP joint is completely ruptured, the proximal phalanx will subluxate volarly and ulnarly relative to the metacarpal head, pivoting on the ulnar situated adductor aponeurosis. This will cause dorsal radial prominence of the metacarpal head.

The UCL usually avulses distally while the RCL tears in midsubstance (16%), off the metacarpal head (55%) or distal (29%). The insertion of the RCL is often wider than the origin making rupture there less likely.[27]

Gaston et al[28] reviewed injury patterns of the RCL of the index MCP joint and found that chronic RCL deficiency will result in pronation of the proximal phalanx and scissoring under the middle finger. This was corroborated in a sectioning study by Hseih et al.[29] In cadaveric sectioning studies, they showed that transection of the RCL alone resulted in ulnar deviation and volar translation of the proximal phalanx on the metacarpal head. If the RCL, volar plate, and dorsal capsule were sectioned, this resulted in pronation and ulnar shift of the proximal phalanx as it pivoted on the remaining healthy UCL.

Smith[30] showed that sectioning of the thumb UCL resulted in volar rotation of the proximal phalanx on the ulnar side of the MCP joint pivoting on the healthy RCL, resulting in a supination deformity of the thumb.

Injury to the index RCL ligament is more common because it is a border digit and lacks the stabilizing influence of a deep transverse metacarpal ligament on the radial side. However, it is physically buttressed by the presence of the other 3 digits.[31] There is also the stabilizing influence of the first dorsal interosseous. A stable RCL of the index is critical for key pinch. Other stabilizers are the aCL, dorsal capsule, and volar plate. A 43-kg load is needed to rupture the RCL.[20] The

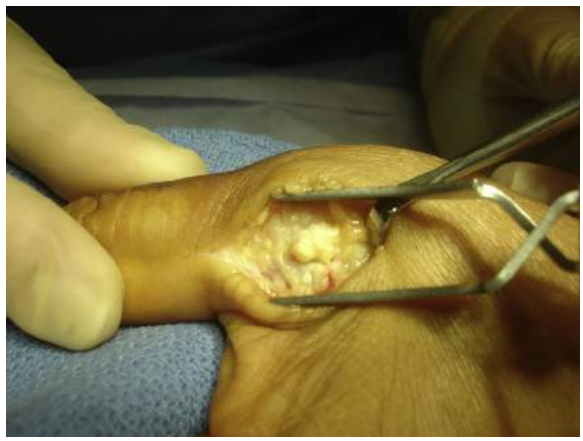

FIGURE 5: Stener lesion in a third-degree injury to the UCL of the MCP joint of the thumb. The ligament is avulsed distally from the proximal phalanx before coming out the proximal margin of the adductor aponeurosis.

RCL is thicker, wider, stronger, and more oblique than the UCL so that it is more taut in flexion.

DIAGNOSIS

An injured thumb should be inspected in full extension (aCL) and 30° flexion (pCL). Instability greater than 30° or 15° more than the contralateral side denotes a complete rupture of the collateral ligament.[2] Frequently, stress testing of collateral ligaments is very painful and requires a local anesthetic into the injured ligament in order to stress the MCP joint to a potential end point in abduction or adduction. The maneuver is repeated in full extension and 30° of MCP flexion to relax the aCLs. Great care needs to be exercised in abduction to prevent the iatrogenic development of a Stener lesion. One can avoid that by grasping the metacarpal tightly and gently angling the proximal phalanx into abduction. If there is no

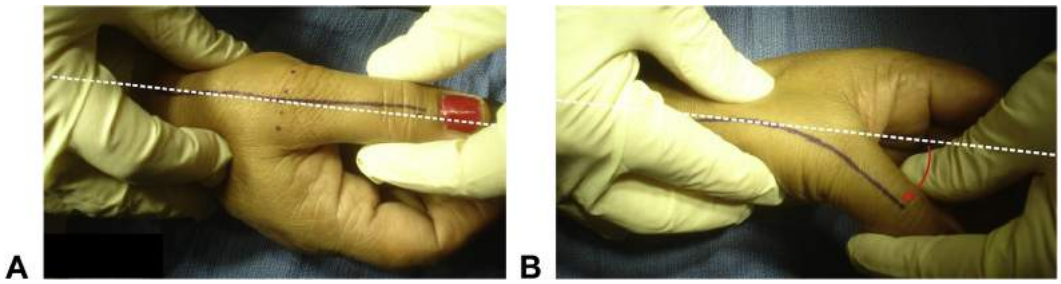

FIGURE 6: **A** Draw a line along the long axis of the thumb at rest. If the collateral ligaments are intact, the entire axis will shift in the direction of the stress and the line will remain straight. **B** If the UCL is completely torn, the "axis" line will bend in the direction of stress with no perceptible end point. Care must be taken not to overstress for fear of creating a Stener lesion.

end point at 30° of abduction, one would then assume that there is a third-degree UCL rupture (Fig. 6).

Radiographically, more than 3 mm palmar subluxation and more than 30° deviation instability with stress denotes complete collateral ligament rupture. Internal rotation and supinated oblique images solve the problem of overlap on lateral views and demonstrate volar, ulnar subluxation of the proximal phalanx.[32] A solid end point is more important than measuring the degree of laxity between injured and uninjured side radiographs.

Ultrasound imaging, in a study by Melville et al,[33] showed 76% sensitivity and 81% specificity to diagnose complete MCP ligament tears.[34] Absence of a normal UCL and a heterogeneous mass proximal to the metacarpal tubercle show a 100% sensitivity and specificity for a ligament rupture. Physiological activity in a ligament is translated into detectable changes in echodensity.[4] Ultrasound allows for dynamic evaluation. Midsagittal views show avulsion and proximal migration of the volar plate, joint effusion, and collateral ligament injuries. Sprains appear as a diffusely swollen hypoechoic ligament with loss of normal ligament fibrous structure.

Magnetic resonance imaging (MRI) sensitivity is 75% and specificity is 98% for ligament tears about the MCP joint.[35] False-negative[36] and false-positive imaging of ligaments may occur. The normal recess at the base of the dorsal capsule may be mistaken for a tear.[8] Although MRI may be useful for detecting ligaments that are torn, it may not detect ligaments that are lax or stretched, even twice normal length, because the MRI shows tissue contrast not quality.[14] Coronal views will show tear and severe stretch injuries (Fig. 7). Axial images visualize the dorsal capsule and the volar plate. Sagittal images show joint alignment and volar plate. Chronic ligament injuries are seen as ligament thickening and fibrosis. This has led some to conclude that, when the MRI is normal, high clinical suspicion and a skilled clinical examination are more reliable, and inexpensive tests in the clinic can allow treatment to proceed rapidly and in the most economical manner without the routine use of MRI.[36]

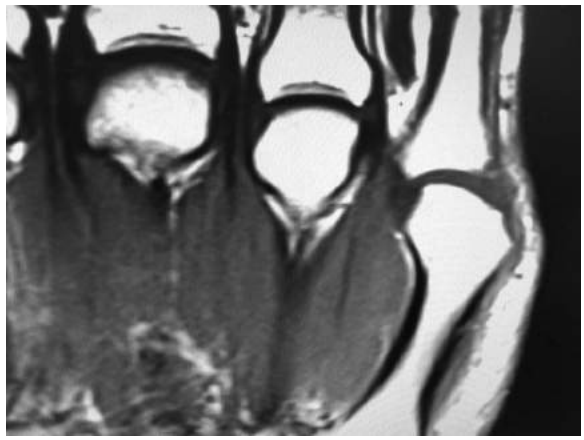

FIGURE 7: MRI of the third, fourth, and fifth MCP joints. Note the complete tear of the UCL of the fifth MCP joint with uncovering of the MCP head and radial deviation of the proximal phalanx.

Grades of ligament injuries as a guide to treatment

Gaston et al[28] studied RCL injuries to the index MCP joint and classified them as follows:

Grade 1, tender RCL. Ligaments become stretched with a few torn fibers, no laxity if seen early. Treated with 4 to 6 weeks of orthosis wear with the MCP joint 30° to 45° flexed and in slight radial deviation, followed by 3 to 6 weeks buddy taping with excellent results.

Grade 2, tender RCL. Greater number of torn fibers, with more pain and swelling and laxity than normal but with a definite end point. Treated with 3 weeks casting, 3 weeks of protective orthosis

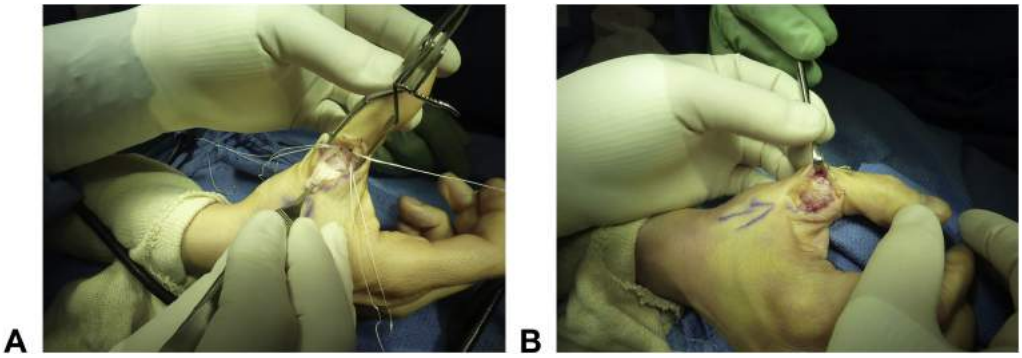

FIGURE 8: A After opening the adductor aponeurosis and reduction of the Stener lesion, the ligament is fully mobilized, pulled out to length, and reinserted onto the proximal phalanx and secured with bone anchors. **B** The ligament has been reattached and the MCP secured with a Kirschner wire. The aponeurosis is closed.

wear. Patient should be advised of surgical possibility.

Grade 3, tender RCL. All fibers torn, laxity without end point. Patients can present acutely or late secondary to failed conservative treatment or noncompliance. Nonsurgical treatment uniformly has poor results. Untreated grade 3 injuries result in chronic swelling, pain, and instability leading to severe dysfunction. Treatment must include reattachment of the ligament (Fig. 8).

Ligament response to injury

When a ligament is overloaded and the fibers tear, the immune system responds in a sequence of distinct and overlapping events[37]:

1. Acute inflammatory phase: 0 to 72 hours

Bleeding and platelet-rich fibrin clot formation growth factors (such as platelet-derived growth factor and vascular endothelial growth factor) attract immune cells, promote neovascularity, and stimulate the growth of fibroblasts. Phagocytes remove debris and damaged tissue.

2. Proliferative/regenerative phase 3 to 28 days

Immune cells release growth factors and cytokines that initiate fibroblast proliferation to rebuild ligament tissue matrix. Initially disorganized scar tissue forms containing blood vessels, fat cells, fibroblasts, and inflammatory cells. Over the next several weeks, collagen, proteoglycans, and glycoproteins form that become aligned in the direction of stress. However, the newly formed collagen fibrils are abnormal and smaller in diameter than normal ligament tissue.[9]

3. Remodeling: Greater than 28 days…months…years

After 4 weeks, collagen maturation begins, which lasts for months and years after the insult. The tissue matrix begins to resemble normal ligament tissue, but differences persist with higher cell and matrix turnover, flaws between fibers, nonparallel collagen fibers, higher cell/matrix ratio, and immature type III collagen cross-linking. The biomechanical strength and elasticity of individual fibers is inferior to native ligament. This makes healed ligaments less efficient in maintaining loads across joints. This may explain the long-term increased thickness of collateral ligaments after major injury.

Joint hypermobility and instability secondary to ligament incompetence is a substantial risk factor for degenerative arthritis.

Traditional therapies for grade 1 and grade 2 ligament injuries

Healing ligaments are dramatically affected by the presence or absence of joint motion. Although traditionally rest or immobilization has been prescribed, immobilization increases synovial adhesion, collagen breakdown, and diminished collagen synthesis, creating more disorganized collagen fibrils. Immobilization also promotes the development of disorganized ligament matrix tissue that lacks stiffness, orientation, and strength.[38] It will also diminish the strength of the bone-ligament interface.[39]

Many studies done on knee and ankle injuries show that functional training of injured ligaments allowed earlier return to work and sports than those treated with immobilization. There was more objective joint stability on testing with less pain, swelling, and stiffness in the long term.[40]

At a tissue level, early controlled activity will enhance proliferative cellular activity with increases

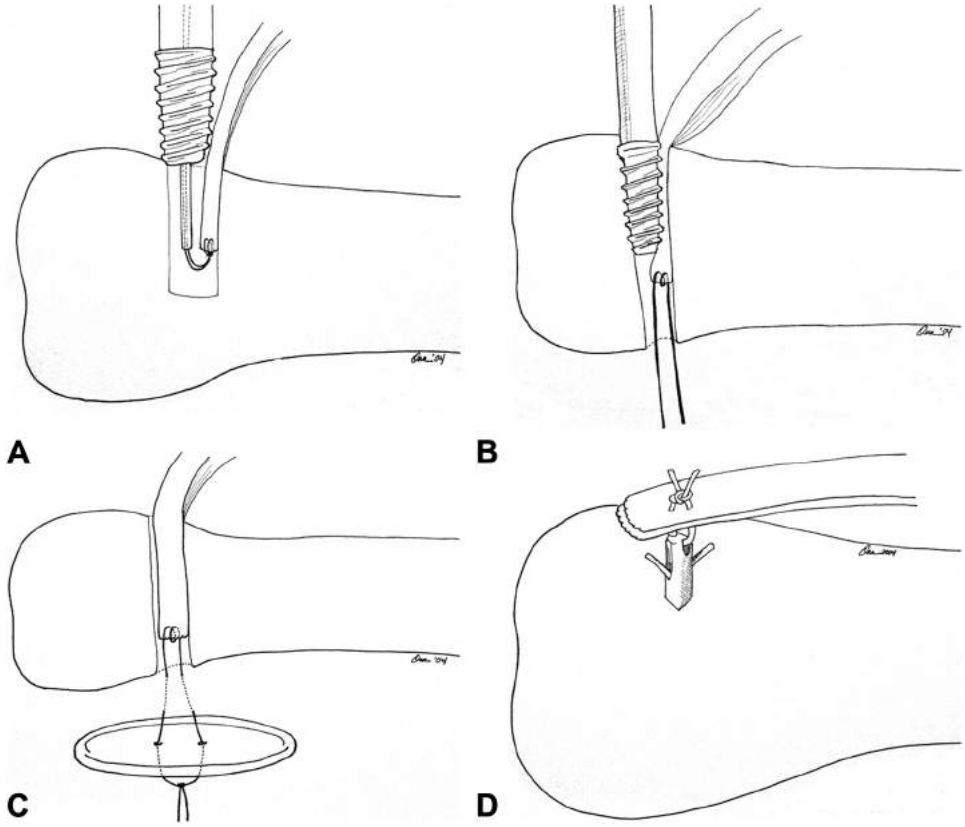

FIGURE 9: Four types of tendon attachment to bone: Large **A** and small **B** interference fit screws; bone anchor **C**; pull-out button **D**. (Reprinted with permission from Lee SK, Kubiak EM, Liporace FA, Parisi DM, Lesaka K, Posner MA. Fixation of tendon grafts for collateral ligament reconstructions; a cadaveric biomechanic study. *J Hand Surg Am.* 2005;30[5]:1051−1055.[45])

in tissue mass and strength improvements in matrix organization and more normalized levels of collagen content. Motion increases blood flow to the affected joint, providing the damaged ligament tissue with nutrients and metabolites necessary for ligament healing.

Nonsteroidal anti-inflammatory drugs (NSAIDs) have been the mainstay of treatment of ligament injuries but may hinder the prostaglandin-based inflammatory cascade necessary to initiate the first stages of ligament healing. These may inhibit the recruitment of phagocytes needed to clear debris and initiate the influx of fibroblasts.[41] Numerous studies show that NSAIDs can inhibit healing of ligaments leading to ligaments with impaired mechanical strength.[42] NSAIDs should be used cautiously in acute ligament injuries and for only short periods.

Although steroid injections have been shown to be effective in reducing inflammation and pain in ligament injuries for 6 to 8 weeks, they too will inhibit the inflammatory mediators such as cytokines needed to promote accumulation and function of neutrophils, macrophages, and fibroblasts. They may even cause collagen breakdown at the injection site.[43] Steroid-injected ligaments are smaller in cross-sectional areas and show diminished tensile strength and load to failure. Many now caution against the use of cortisone injection in acutely injured ligaments.[44] Although they may relieve pain, they can be detrimental to ultimate healing.

Grade 3 ligament injuries

Usually grade 3 ligament injuries require surgery because they rarely respond to the conservative measures already described. Failure to treat effectively may result in chronic pain, swelling, joint instability, and dysfunction in routine activities. Many types of repairs and late reconstructions have been described, utilizing sutures, bone anchors, tendon weaves, and various muscle-tendon advancements (Fig. 9).

Lee et al[45] conducted a study to determine the relative strength of 4-tendon graft reconstructions for UCL injuries of the thumb (Fig. 10).

1. Triangular apex proximal
2. Triangular apex distal
3. Figure of eight
4. Parallel configuration

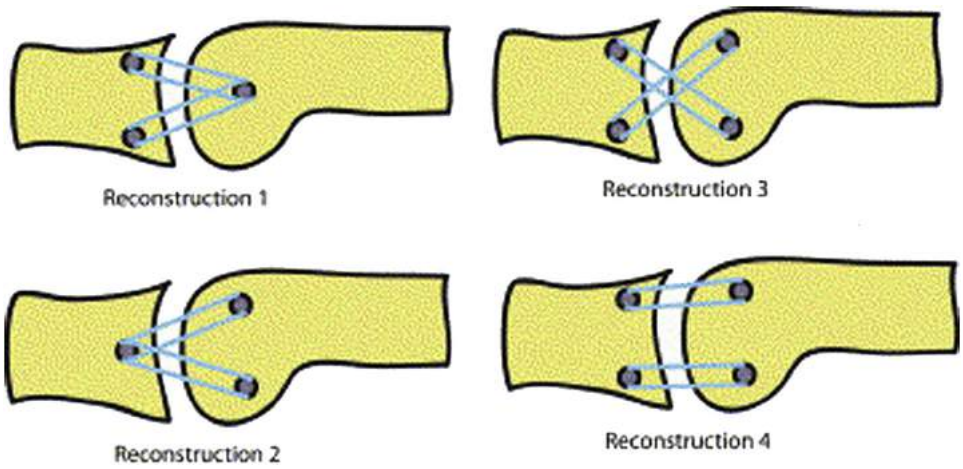

FIGURE 10: Four configurations of tendon weaving for late tendon reconstruction of MCP joint laxity: 1, triangular apex proximal; 2, triangular apex distal; 3. figure of eight; 4. parallel configuration. (Reprinted with permission from Lee SK, Kubiak EN, Lawler E, Lesaka K, Liporache FA, Green SM. Thumb metacarpophalangeal ulnar collateral ligament injuries: a biomechanic simulation study of four static reconstructions. *J Hand Surg Am.* 2005;30[5]:1056−1060.[46])

All 4 repairs were compared with normal and the fully sectioned ligament measuring valgus angulation to a fixed load with MCP joint on 0° and 30°. All 4 reconstructions were significantly stronger than the fully sectioned ligament and were not significantly different from one another, but only the apex proximal configuration approximated the ROM of the intact ligament. The other configurations had significantly decreased range than the intact ligament.

In another study by Lee et al,[46] they compared 4 methods of fixation of tendon grafts in collateral ligament reconstructions with regard to pull-out strength. This attempted to determine which method would most readily allow early postoperative ROM. There were 4 types of fixation (Fig. 9).

1. Bone tunnel with a 4-mm Arthrex (Arthrex Inc., Naples, FL) biotenodesis interference fit screw
2. Bone tunnel with a 3.3-mm Accutrak (Acumed LLC, Hillsboro, OR) tenodesis interference fit screw
3. Bone tunnel with a pull-out button
4. At the side of the metacarpal neck with a mini-Mitek (DePuy Synthes Sports Medicine, Raynham, MA)

The differences between each technique were significant: strongest to weakest, $1 > 2 > 3 > 4$.

Modes of failure

1. Interference fit screws: tendon pulled out of bone tunnels.
2. Suture button: rupture at suture button interface or suture pulled out of tendon.
3. Bone anchor: rupture at suture-anchor interface. Suture anchors and interference fit screw have superior strength and stiffness compared with suture fixation to a bone anchor.

Dzwierzynski et al[47] and others compared the strength of the intact ligament with any of the repair techniques: suture repair, pull-out wire repair, and Mitek anchor repair. The intact ligament strength to failure far exceeded any of the repair techniques. The strongest repair was suture anchor followed by pull-out wire; suture repair was weakest. Failure of the repair occurred by 3 means: breakage of the suture, pulling out of the repair material from the ligament, or out of the proximal attachment.

Dy et al[48] compared the strength of fixation of a 4-tunnel technique with 2 bicortical tunnels fixed by 2 interference fit screws for MCP fixation (Fig. 11). Bicortical tunnels were used to prevent bunching of the grafts in the tunnels and to facilitate tensioning of the graft. Grafts were tensioned in 30° of flexion. If the graft was positioned with the MCP joint in extension, the proximal phalanx subluxated dorsally. Interference fit fixation was seen to be technically much more easy to perform than 4-tunnel fixation and did not compromise fixation. Four-tunnel fixation increased the risk of bone fracture.[29]

There was no significant difference in terms of stability to ulnar deviation stress between 4-hole tunnel reconstruction and interference fit fixation at 90° flexion. There were significant differences in terms of stability to ulnar deviation stress between 4-tunnel reconstruction and interference fixation at 0° flexion with the screw construct being more stable.

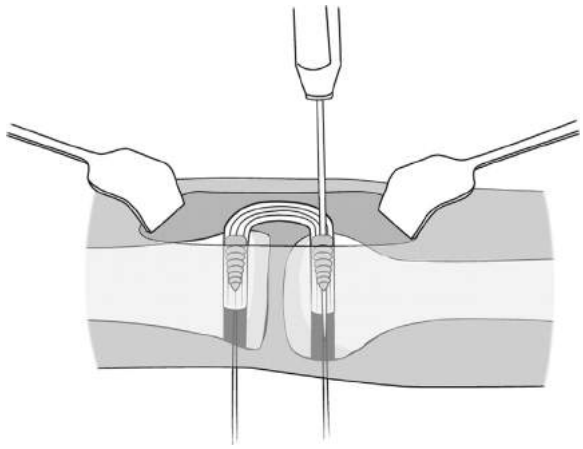

FIGURE 11: Tendon bone fixation using a slip of tendon passed through 2 bone tunnels fixed by 2 biotenodesis screws. (Reprinted with permission from Dy CJ, Tucker SM, Hearns KA, Carlson MG. Comparison of in vitro motion and stability between techniques for index metacarpophalangeal joint radial collateral ligament reconstruction. *J Hand Surg Am*. 2013;38[7]:1324−1330.[48])

A V-shaped tendon configuration is best to restore ROM with the apex on the metacarpal head at the site of the collateral ligament origin because this created the most isometric construct (Fig. 10). This differs from earlier reports that recommended putting the proximal hole at the center of rotation. The recommended tensioning of the repair is 30° flexion of the MCP joint.

In a clinical study, Smith[30] felt that, if acute ligament repairs were to be done, they were best performed before 3 weeks. After 3 weeks, tendon reconstruction would yield superior long-term results.

Melone et al[49] studied 100 collateral ligaments (60 UCL, 40 RCL) repaired with ligament advancement or tendon reconstruction and reported 95% good-excellent results at final follow-up.

Catalano et al[50] had a large study comparing acute repairs (< 2.5 weeks) and chronic reconstructions (> 7 months). At 5-year follow-up, there were no differences in results. They felt that one needed to repair the RCL of the thumb because the intact extensor pollicis longus and adductor would ulnarly deviate the thumb.

Kang et al,[31] in a clinical retrospective study, looked at repair of ruptures of the radial collateral ligament of the MCP joint of the index finger. In 12 patients, 3 acute and 9 that failed conservative treatment for more than 12 weeks presented with persistent pain, swelling, tenderness, and instability to ulnar deviation stress at 60° flexion. All were treated with suture anchors. Direct repair of the ligament was done using suture anchors. The RCL was reattached with joint in 45° of flexion. At 23 months' follow-up, all patients had resolution of pain with return to activities of daily living. The ROM was 0° to 80°. No extensor lag or stress instability was seen. Grip strength was 111% of normal and lateral pinch was 112% of normal.

A thorough understanding of the anatomy, physiology, and injury patterns of finger collateral ligaments will optimize the treatment of these common but frequently misunderstood injuries.

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EDITOR'S SUGGESTIONS FOR MORE INFORMATION

The Editor chose to include these references and videos to provide readers with additional information.

a. Mahmood B, Hammert W. Thumb MCP UCL repair. Presented at: American Society for Surgery of the Hand Annual Meeting Video Theater, September 10–12, 2015, Seattle, WA. Also available on Hand-e: http://www.assh.org/hand-e. Video A (available on the *Journal*'s Web site at www.jhandsurg.org).

b. Baratz M. Thumb MCP joint UCL reconstruction. Presented at: American Society for Surgery of the Hand Annual Meeting, September 10–12, 2015, Seattle, WA. Also available on Hand-e: http://www.assh.org/hand-e. Video B (available on the *Journal*'s Web site at www.jhandsurg.org).

c. Sweet S. Ulnar collateral ligament injuries of the thumb. Presented at: American Society for Surgery of the Hand and American Association for Hand Surgery Specialty Day, March 23, 2013, Chicago, IL. Also available on Hand-e: http://www.assh.org/Hand-e. Video C (available on the *Journal*'s Web site at www.jhandsurg.org).

d. Khouri JS, Hammert WC. Repair of long finger metacarpophalangeal joint radial collateral ligament avulsion. Presented at: American Society for Surgery of the Hand Annual Meeting Video Theater, September 10–12, 2015, Seattle, WA. Also available on Hand-e: http://www.assh.org/hand-e. Video D (available on the *Journal*'s Web site at www.jhandsurg.org).