# Tendinopathies of the wrist and hand

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## INTRODUCTION

A large proportion of hand and wrist tendinopathies occur in individuals who perform highly repetitive and forceful jobs (Elder & Harvey 2005). The Department of Labor, Bureau of Labor Statistics (1999), reported incidence of hand and wrist tendinitis (tendinopathy not...
DEFINITION OF TENDINOPATHY

The lack of more positive results with conservative treatment may be due to mislabeling tendinosis as tendinitis (Khan et al 2000). Tendinitis must be qualified. Studies are now consistently showing what was normally diagnosed as tendinitis may represent only one classification of tendinopathy (Futami & Itoman 1995). Tendinopathy represents histological findings that differ significantly from the generally accepted condition of tendinitis. This is due primarily to the lack of evidence of inflammatory precursors and cells in the tendon itself (Gabel et al 1994, Yuan et al 2003, Curwin 2005, Fredberg & Stengaard-Pedersen 2008). Khan et al (2006) supported Bonar’s classification of tendinopathy, which defined four classifications, each with distinct histological findings. Clinicians have yet to apply this knowledge to support specific conservative treatment use (Cannon 2001). A fourth edition manual of upper extremity rehabilitation printed in 2001 did not use the words tendinosis or tendinopathy, but used the terms tendinitis and paratendinitis for all related conditions of upper extremity pain caused by tendon pathology (Cannon 2001).

One reason for the continued consideration of tendon pathologies as tendonitis may be the initially positive outcomes with corticosteroids in symptomatic tendons (Fredberg & Stengaard-Pedersen 2008). The presence of an -itis or inflammation in the form of neurogenic inflammation may also support persistence of the old terminology. Fredberg & Stengaard-Pedersen (2008) concluded that some combination of classic inflammation and neurogenic inflammation does mean that tendonitis is not a complete misnomer. It is the histological difference in tendinopathies stemming from tendinitis, tendinosis, and paratendinitis that may dictate different treatments; particularly in manual therapy.

There continue to be areas of needed research into this subject. Findings from animal studies and from tendon studies performed on other areas of the body will be used in this chapter to provide data which may be extrapolated to apply to the hand and wrist, despite differences between weight bearing versus positional tendons (Smith et al 1997).

AETIOLOGY

Researchers report that knowledge of the aetiology of tendinopathy is evolving (Sharma & Maffulli 2005, Fredberg & Stengaard-Pedersen 2008). Many factors contribute to tendinopathy, both intrinsic and extrinsic (Riley 2004). Renstrom & Hach (2005) summarized extrinsic factors as: malalignments, reduced flexibility, muscle weakness or imbalance, overuse and excessive body weight. Hart et al (2005) added genetics, gender, and fitness level, while Hammer (2007a) reported biomechanical faults. Intrinsic factors that affect apoptosis can lead to tendon degeneration. This process of programmed cell death can be exacerbated by intrinsic oxidative or mechanical stresses (Yuan et al 2003, Sharma & Maffulli 2005). Theories on tendon rupture have been separated into two categories: vascular and mechanical (Riley 2004). The reader is encouraged to read the work of Riley (2004) to explore this topic further.

ANATOMY OF THE TENDON

Basic components

The tendon is the attachment site of a muscle to bone. It is designed to transfer tension from the muscle to the bone, thereby causing motion to take place (Kannus 2000).
The basic building block of the tendon, *tropocollagen*, is formed by fibroblasts (O’Brien 2005). These are assembled into *fibrils* which are arranged into *fibres*, which are organized into *fascicles* and bound together with a loose connective tissue called *endotendon* (endotenon) (Kannus 2000, Sharma & Maffulli 2005). The endotendon is the pathway for blood vessels, nerves, and lymphatics (Riley 2004). Bundles of fascicles are bound together by another layer of connective tissue called the *epitendon* (epitenon) which is continuous with the endotendon (Kannus 2000) (Fig 26.1).

Synovial tendon sheaths, also called *paratendon* (paratenon), are found in areas subjected to increased mechanical stress, such as the tendons of the hands and feet, where efficient lubrication is required (Sharma & Maffulli 2005). Fibre bundles are predominantly aligned with the long axis of the tendon and these are responsible for the tensile strength of the tendon (Riley 2004). A small proportion of fibres run transversely, and there are even spirals and plait-like formations (Kannus 2000). This complex ultrastructure provides resistance against transverse, shear, and rotational forces acting on the tendon (Riley 2004).

**Blood and nerve supply**

Tendon vascular support comes from three sources: at the myotendinous junction, the osteotendinous junction, and the extrinsic system through the paratendon (Benjamin & Ralphs 1996, Sharma & Maffulli 2005, Scott et al 2007). Innervation accompanies vascular pathways through the paratenon (Hart et al 2005). The nerve receptors that supply tendons can terminate in the vicinity of mast cells, where neuropeptides are involved in normal tendon regulatory control (Hart et al 2005).

**Patho-anatomy**

Tenocytes and *tenoblasts* are the cells involved in tendon healing (Sharma & Maffulli 2005, 2006). Tenocytes are sparse in tendon tissue but have extensions that create an extensive network inside the matrix (O’Brien 2005). They are responsible for maintenance of matrix and collagen (Harley & Bergman 2008). Tenocytes are crucially responsive to environmental conditions. Mechanical demands placed on tendon tissue will promote changes in the microarchitecture of the tissue (Magra et al 2007). Strain applied to a tendon can change its structure; these changes can be damaging or they can be reparative if appropriately and purposefully applied in treatment.

Scott’s research (Scott 2007) evidenced that it is stimulation of the tenocyte that is associated with tendinosis, rather than intrinsic inflammation. Alterations in cell activity lead to tendon changes from mechanical stress rather than the converse (Riley 2004). The local stimulation of tenocytes, which is a load-driven cellular response, rather than inflammation or apoptosis, is the true mechanism in tendinosis (Scott et al 2007). Apoptosis plays a role later in the tendinopathic process (Scott et al 2007). Localized hypoxia from vigorous exercise can lead to tenocyte death (Sharma & Maffulli 2005) and tendinopathic changes.

Tenocyte metabolism is regulated partly by mechanical stimulation (Maeda et al 2009). Maeda et al (2009) showed that cyclic strain will change gene expression in tendon cells. Force applied to a tendon changes cellular process via mechano-transduction, the process in which a cell converts biomechanical stimuli into chemical signals (Maffuli & Longo 2008). Mechano-transduction utilizes gap junctions, stretch activated channels (Wall & Banes 2005), voltage operated calcium channels (VOCC) and tandem pore domain potassium channels (TPDPC).
to communicate with adjoining tenocytes (Wall & Banes 2005, Magra et al 2007). Tension on surface proteins, called integrins, embedded in the cell membrane is transmitted to the cell’s cytoskeleton. This force is transmitted via the intracellular network to the nucleus of the cell and can alter protein expression (Chiquet 1999). Huang et al (2004) observed that mechanical loading is essential for homeostasis of the bone, cartilage, and skin. Additionally, external forces are capable of producing changes in intracellular reactions. Tenocytes are responsible for changing structure in response to demand by altering, ‘gene expression patterns, protein synthesis and cell phenotype’ (Maffulli & Longo 2008). This alteration is suspected of being the link to overuse and tendinopathic changes (Scott et al 2007). Importantly from a manual therapy perspective, Maffulli & Longo (2008) supported that an alteration of mechanical forces may augment the healing process. Conversely, understimulation can cause tendinopathic changes (Arnoczky et al 2006).

The tendon matrix is responsible for maintenance of the tendon. Its damage, according to Riley (2004), is the leading event in tendinopathy. The ground substance of the extracellular matrix network surrounding the collagen and the tenocytes contains proteoglycans, glycosaminoglycans, glycoproteins, as well as several other small molecules (O’Brien 2005). Water makes up 60–80% of the ground substance (O’Brien 2005). Proteoglycans are strongly hydrophilic, enabling rapid diffusion of water-soluble molecules and the migration of cells (Sharma & Maffulli 2005). They, along with glycoproteins, have a role in organization of collagen into fibrils and fibres (O’Brien 2005). When repetitive damage becomes extensive it overwhelms the ability to heal (Riley 2004). Arnoczky et al (2007) credited extracellular matrix degeneration as a precursor of tendon weakness. Riley (2004) described the possibility that changes in cellular activity in the matrix due to mechanical strain can influence the structural properties of tendons.

Tendon injury

Riley summarized overuse tendinopathy as the phenomenon caused by repeated strains below the failure threshold that outstrips the cell’s ability to heal (Riley 2004). Tissue injury from repetitive strain is thought to be a cellular event (Arnoczky et al 2006). Recent studies in animal modeling have produced results of tendinopathy that correspond those found in non-experimental tendinopathies in humans. Soslowsky’s model of repetitive motion identified tendinopathic changes in supraspinatus tendons in rats (Soslowsky et al 2000). These changes mimic what has been found in idiopathic tendinopathies in humans, including reduced mechanical properties (Lavagnino et al 2006, Arnoczky et al 2007). Glazebrook et al (2007) found similar changes in rats after overuse induced by repetitive running. Backman et al (2005) produced similar results with rabbits.

Post-injury disuse of a tendon, through immobilization or compensation, can also have detrimental effects. The concept of stress shielding can be applied to tendons. An example of this in terms of bone is application of Wolff’s law with reduced bone density following fracture immobilization. Woo et al (1981) observed that after fracture healing, reapplied weight bearing will increase bone density. Kannus & Jozsa (1991) showed that under-stimulation of tendon cells post-injury produced degenerative findings in investigation of tendinopathy. DeBoer et al (2007) supported this with his demonstration of tendon protein synthesis rates decreasing progressively through 10 days of immobilization. Lavagnino et al (2006) induced mechanical injury in rat tail tendons, followed by immobilization, which revealed an upregulation of collagenase mRNA and protein synthesis in this damaged area. Even undamaged fascicles showed similar upregulation during the immobilization portion of this study. In an earlier study they found that these adverse effects could be controlled, in vitro, with cyclic stretching (Lavagnino et al 2003). Screen et al (2005) reported similar results with cyclic stretching in non-injured tendon fascicles. In regard to treatment of tendinopathy, attempting to immobilize a tendinosis via splinting or casting thus appears to be detrimental.

Tendon healing

The phases of tendon healing following injury resemble that of other connective tissues in the body. The phases are: (1) acute inflammatory, lasting 1–2 days; (2) repair-regeneration or proliferative phase, lasting up to 6 weeks; and (3) maturation or remodelling phase, lasting 3 weeks to a year (Leadbetter 1992, Sharma & Maffulli 2005). Each of these phases in tendinopathy has unique cellular progression that should be considered when preparing a treatment plan. Tenocytes begin new collagen synthesis around day 5 post-injury and continue synthesis for 5 weeks (Maffulli & Moller 2005). Intrinsic tenocytes begin proliferating at week 4 and are involved in remodelling through week 8 (Maffulli & Moller 2005). Applying standard but specific treatments in a global fashion to all tendinopathies without addressing the stage of healing could be ineffective. Cook & Purdam (2009) recommended that interventions should be tailored to the suspected pathology.

TENDINOPATHY CLASSIFICATION

Tendinopathy actually represents several different, mixed and sometimes overlapping degenerative processes. Histologically there are mixed findings. Absence of inflammatory cells, increased ground substance, increased...
vascularity and cellularity with collagen disorganization, are evidenced (Khan et al 2006). Each of these can disrupt some tendon fibres and weaken the remaining fibres (Maffulli & Moller 2005). The role of the tenocyte in tendon changes has already been discussed. Murrell (2002) stated that apoptosis, or programmed cell death, may have a roll in tendinopathy. Oystein et al (2007) showed apoptosis was enhanced in patellar tendinopathy biopsies compared to controls.

Inflammation is partially controlled by a neurogenic process. Substance P and calcitonin-related gene peptide (CRGP) are sensory neuropeptides (Hart et al 2005). These, among other substances, are found in symptomatic tendons (Andersson et al 2008) and directly stimulate nociceptor endings (Ueda 1999). Hart et al (2005) hypothesized that neuropeptides are involved via mast cells in tissue in normal tendon regulatory control; also, a dysfunctional regulatory loop produces an inadequate repair response. This differs from classic inflammation. Riley (2004) observed, ‘…nerve endings and mast cells may function as units to modulate tendon homeostasis and mediate adaptive responses to mechanical strain. He also stated that ‘excessive stimulation as a result of overuse may result in pathological changes to the tendon matrix’ (Riley 2004, p 137). There is a growing body of evidence that pain associated with tendinopathy may be neurogenic.

Tendinopathy severity is graded according to histological features distinguished under light microscopy (Maffulli et al 2008). Various scales have been proposed. Two early scales were originally developed for lower extremity research. The Mover scale and the Bonar scale have since development each been applied successfully to research of the upper extremity (Maffulli et al 2008). Each scale considers the microscopic appearance of five to seven factors; each factor is given a grade ranging from lowest number (normal tendon), to highest number (markedly abnormal tendon). The sample is graded cumulatively with combined scores from each factor (Maffulli et al 2008). Scott et al (2007) used a modified Bonar scale to specifically assess tendinosis. The modified scale considers five histological changes: (1) tenocyte morphology; (2) tenocyte proliferation; (3) collagen changes; (4) glycosaminoglycans (GAG), and (5) neovascularization (Scott et al 2007).

A lack of common description of these histological tissue changes which vary from scale to scale to modified scale has limited a clear classification and understanding of tendinopathy with its underlying causes. Kahn et al (2006) cited Clancy as having initially made a classification of tendinopathy types that was later modified by Bonar and now includes: tendinosis, tendinitis (tendinitis) or partial rupture, paratendinitis (paratendonitis/paratendinitis/tenosynovitis/tenovaginitis) and paratendinitis with tendinosis. The following sections provide details.

**Tendinosis**

Tendinosis is defined by Maffulli et al (2003a) as intratendinous degeneration typical with aging or devascularization. It is characterized by fibre disorientation, hypercellularity, and focal necrosis and calcification (Maffulli et al 2003a). Kraushaar & Nirschl (1999) defined the three findings in tendinosis as fibroblastic hyperplasia, hyper-vascularity, and abnormal collagen production with the former being the first response. Kannus & Jozsa (1991) examined 891 spontaneously ruptured tendons in the upper and lower extremity. Histopathologic examination showed that 97% of these had degenerative changes. These were sub-classified into hypoxic degeneration (44%), mucoid degeneration (21%), tendolipomatosis (8%), and calcific tendinopathy (5%) (Kannus & Jozsa 1991) There is multiple cell/tissue involvement and this may be difficult to discern from other classifications.

**Tendinitis**

Tendinitis and partial rupture are grouped together in this classification. An active inflammatory response, symptomatic degeneration and true vascular disruption are characteristic findings (Kahn et al 2006). Lymphocytes and neutrophils are seen (Kraushaar & Nirschl 1999). It has similar characteristics to tendinosis but histopathologically will also demonstrate fibroblastic proliferation, haemorrhage and granulation tissue (Maffulli et al 2003a). Hammer (2007a) stated that isolated active inflammation is not common but is usually associated with some degree of rupture, which implies that this classification is falsely over-diagnosed.

**Paratendinitis**

Paratendinitis, also termed tenosynovitis, is evidenced as frank inflammation of the outer layer of the tendon (Kahn et al 2006). Microscopically this will reveal infiltrate possibly including fibrin deposition, exudate, and areolar tissue degeneration which could explain the palpable crepitation at certain stages of its progression (Kahn et al 2000, Maffulli et al 2003a).

**Combined paratendinitis and tendinosis**

This fourth classification (Kahn et al 2006), originally described by Clancy, includes characteristics of both tendinosis with an overlying paratendinitis as described above. Most clinicians, including primary care physicians, would not be able to differentiate which of these were most prominent in a patient presenting with general hand/wrist pain, as the signs and symptoms are similar to isolated paratendinitis.
Of the above categories, only tendinitis and paratendinosis have an inflammatory component and would conceivably respond to an anti-inflammatory regimen, and likewise would logically not respond to deep tissue friction massage.

**EXAMINATION AND DIAGNOSIS**

A comprehensive assessment is the most important step to determining appropriate treatment of most musculoskeletal disorders. History, clinical tests, and imaging will contribute to a differential diagnosis. The reader is referred to Chapters 2–4 for discussion of history taking, physical examination, and pertinent imaging. Clinical testing for tendinopathy can include palpation, selective tissue testing, and provocation testing. It is theorized that clinical tests will help to differentiate the structure involved, yet reliability and validity are still in research. Indeed, this is only one part of the diagnostic equation. Identifying the type of tendon involvement and stage of pathology is another factor of greater difficulty.

The diagnosis of tendinopathy will be the result from a comprehensive examination, but distinguishing between tendinosis and tendinitis can be difficult (Khan et al 2000). Maffulli et al (2003a) identified tendinopathy clinically as localized tendon swelling and pain with impaired function. Curwin (2005) stated that we must assume the level of tendon involvement can be correlated with the level of dysfunction and pain. Per Curwin (2005), the degree of injury cannot be ascertained acutely. Elder & Harvey (2005) maintained that acutely the specific area is usually easy to isolate. Leadbetter (1992) defined acute injury as having a sudden specific onset followed by gradually decreasing pain. Identifying an acute onset during history taking should help differentiate a current stage of acute or subacute inflammatory process from a chronic stage when inhibiting pain occurs during activity or afterward (Leadbetter 1992), and will help guide treatment.

One complicating factor in isolating a specific involved structure is that most tendons to be identified will have anatomic variations or supernumerary insertions. These vary too much for inclusion here. Another complication is the possibility that a trigger point is responsible for all or a portion of the symptoms. Trigger points in the upper quarter can refer pain to the wrist area. The subscapularis, biceps brachii and brachialis are some of the muscles that can refer pain to the wrist (Finando & Finando 2005). Lack of clearing these points/areas of potential contribution will delay appropriate treatment. It cannot be overemphasized that suspected trigger points should be cleared as part of the initial examination. The reader is referred to Chapter 32 of this text for additional information on referred pain from muscle/myofascial trigger points (TrPs) in arm pain syndromes.

**Clinical tests**

Regarding general palpation, oedema and hyperaemia of the paratendon may be evidenced clinically. A fibrinous exudate accumulates within the tendon sheath, and crepitus may be felt on clinical examination (Kahn et al 2000, Sharma & Maffulli 2005). This may be important in differentiating paratendinitis from tendinopathy; however, the presence of crepitus to palpation does not prove that paratendinitis is present (Kahn et al 2000, Sharma & Maffulli 2005).

Palpation for tenderness is a common tool for clinical diagnosis and differential testing in tendinopathy. Cook et al (2001) assessed the value of palpation to identify patellar tendinopathy in a group of 326 young athletes. Intra-rater reliability was good at 82%. Palpation of tendons in patients with symptoms resulted in sensitivity of 68% and specificity of 9% (Cook et al 2001). However, applicability to the wrist is limited since the patellar tendon is larger than those of the hand/wrist.

Maffulli et al (2003b) found a high positive predictive value in palpation, when combined with the Royal London Hospital test and a painful arc sign to determine Achilles tendinopathy. The painful arc sign is theorized to differentiate pathology within the tendon itself versus pathology of the paratendon. If the pathology is confined to the tendon structure, a palpable area of thickness and tenderness will move with the tendon as the ankle is moved; if the painful, thickened area stays in a fixed position regardless of ankle movement then the pathology is within the paratendon (Easley & Le 2009). The sensitivity and specificity of this test was 52% and 83% (Maffulli et al 2003b). The Royal London Hospital test identifies tendinopathy by eliciting local tenderness with palpation of the tendon in neutral or slightly on slack. The test is positive if the tenderness decreased significantly or disappears with the tendon on stretch. The sensitivity and specificity of this test was 54% and 91%. The sensitivity and specificity of direct palpation was 58% and 74%. When the three tests were combined, sensitivity was 58% and specificity was 83% (Maffulli et al 2003b). There is a dearth of evidence-based research application of these clinical tests to tendons of the wrist and hand.

Cyriax supported selective tissue tension testing (STT) (Hammer 2007b). Selective tissue tension testing is utilized to compare non-contractile to contractile tissue involvement (Hammer 2007b). The tendon is isolated as much as possible based on planes of motion performed, either isolated or overlapped with other tendons. The examiner attempts to administer a minimal isometric force to the tendon/muscle while the patient resists. Elicitation of pain is a positive test (Hammer 2007b). Hancock et al (2005) found agreement (0.71–0.79, kappa and 95% confidence interval) among Cyriax-trained assessors using STT combined with clinical history when assessing tendinopathy of the rotator cuff. Reliability has yet to
be established for any upper extremity tendon application (Stasinopoulos & Johnson 2007).

Provocation tests (special tests) are used with varying evidence-based support of reliability, sensitivity, and specificity. These tests are included, as available and relevant, in the outlined discussion of tendinopathies unique to specific tendons of the wrist and hand following diagnostic imaging and invasive testing.

Diagnostic imaging and invasive testing

Due to the difficulty in reliably diagnosing tendinopathy, Fredberg & Stengaard-Pedersen (2008) recommended ultrasound (US) or magnetic resonance imaging (MRI) if there is no response to conservative treatment or if radicular pain is present. Fredberg & Stengaard-Pedersen (2008) described the efficacy of ultrasound versus MRI. These include more detailed visualization of tendon microstructure, better tendon border definition, and its interactive nature. A focal thickening, visualized with ultrasound, is associated with tendonitis in tendons without sheaths. This may correspond to angiofibroblastic areas associated with micro-ruptures (Daenen et al 2003). Furthermore, the tendon or tendon sheath, as viewed via ultrasound, will be thickened on more chronically involved tendons (Daenen et al 2003). Ultrasound can be performed directly over the subjectively painful area and even during range of motion (Fredberg & Stengaard-Pedersen 2008). McNee & Teh (2007) considered ultrasound the ‘investigation of choice’ in tendon pathology. Beddi & Bagga (2007) stated that ultrasound is the gold standard for tendon examination.

Isolated identification of the involved structure can be assessed by removing sensation from specific areas, continuing until the patient’s symptoms are resolved. Selective anaesthetic injections, usually with lidocaine, are supported by Elder & Harvey (2005) as ‘the best diagnostic test’ for tendinopathy of the hand and wrist, but they offer no studies to back up this recommendation.

TENDINOPATHIC ENTITIES OF THE HAND AND WRIST

This section will describe common areas of tendon pain in the wrist. Areas of rare involvement are not included.

Flexor carpi ulnaris

Pathology of the flexor carpi ulnaris (FCU) muscle (Fig 26.2) may include tendinitis, tendinosis, or a combination of these two. This is the most common wrist flexor tendinopathy (Elder & Harvey 2005) and often occurs in those who play racquet sports and golf (Rettig 2001). The FCU inserts into the pisiform, the hook of the hamate, and the fifth metacarpal (Moore 1992). It is not held in place by the flexor retinaculum, but instead relies on its own tendon sheath (Elder & Harvey 2005).

Testing

- Characterized by painful palpation of the pisiform and the FCU tendon, presence of angiofibroblastic hyperplasia is often evidenced by palpable swelling and thickening in symptomatic FCU tendons (Budoff et al 2005)
- Pain with resisted wrist flexion and ulnar deviation
- Shuck test if pisotriquetral involvement is suspected (Rettig 2001)
- Passive wrist extension and radial deviation will provoke symptoms (Elder & Harvey 2005)

Differential diagnosis

Rettig (2001) recommended the pisotriquetral grind test to implicate the pisotriquetral joint pain over a FCU tendinopathy. Campbell (2001) and Burke (1996) describe the test as grasping the pisiform and compressing it onto the triquetrum and rotating the pisiform under pressure. Palpation alone may implicate the tendon with pain and crepitus, whereas pain with compression implicates the pisotriquetral joint. Pisotriquetral compression syndrome (Rettig 2001), arthritis, calcific tendinitis and ulnar neuritis, pisiform ligament complex syndrome, pisotriquetral arthropathy (Rayan 2005), and Guyon’s canal syndrome (Elder & Harvey 2005) are additional differential diagnoses.

Extensor carpi ulnaris

Tendinopathy of the extensor carpi ulnaris (ECU) (Fig 26.3) may commonly include a tendinitis, tendinosis, or
these in combination. It is also subject to subluxation (Elder & Harvey 2005). Activities such as racquet sports and baseball batting will cause rapid and repetitive supination, flexion, and ulnar deviation, which have been cited as promoting factors (Elder & Harvey 2005, Hammer 2007c). Rettig (2001) noted that ECU tendinopathy often involved the non-dominant hand in tennis players who used a two-handed backhand stroke. Futami & Itoman (1995) found that of 155 patients with dorsal wrist pain, 53 had pain possibly caused by tenovaginitis (paratendinitis) of the ECU induced by overuse. Bencardino & Rosenburg (2006) associated sub-luxations of the ECU with tenosynovitis and recommended testing supination and volar flexion for ulnar sub-luxation of the tendon. Montalvan et al (2007) studied 28 clinical cases of ECU related pain with three clinical patterns described: (a) acute traumatic instability of the ECU in the fibro-osseus groove (12 cases); (b) tendinopathy (14 cases); and (c) complete ECU rupture (4 cases).

### Testing

- Symptoms are provoked by combined active supination and wrist extension (Elder & Harvey 2005) and combined resisted ulnar deviation and extension (Elder & Harvey 2005, Young et al 2007)
- Dislocation can be reproduced by a clicking on supination and extension actively, not passively (Elder & Harvey 2005)
- Tenderness to palpation over sixth dorsal compartment (Rettig 2001) at the ECU tendon and ulnar head (Elder & Harvey 2005)

### Differential diagnosis

- Rupture, sub-luxation, dislocation, triangular fibro-cartilage complex (TFCC) pain, triquetrum-lunate ligament lesion, pisiform-lunate joint pain, and fractures of the lunate, triquetrum and pisiform are differential diagnoses (Futami & Itoman 1995). Additional diagnoses to be excluded are extensor digiti minimi tenosynovitis, TFCC tears (Elder & Harvey 2005), and stenosing tenosynovitis in the ulnar wrist (Rettig 2001).

### Extensor carpi radialis longus and brevis (distal tendons)

A common combined pathology of the extensor carpi radialis longus (ECRL) and brevis (ECRB) tendons (Fig 26.3) distally is known as intersection syndrome. This is also termed peritendinitis crepitans (Young et al 2007), crossover tendinitis, and squeaker’s wrist (Rettig 2001). The syndrome may include tendinitis, tendinosis, and/or bursitis. It is common among racquet players, weight lifters, and canoeists (Hammer 2007c). Ski pole and hammer usage can also provoke this particular syndrome (Elder & Harvey 2005).

Intersection syndrome is associated with friction from the crossing of the first dorsal compartment abductor pollicis longus (APL) and the extensor pollicis brevis (EPB) over the second dorsal compartment (ECRL and ECRB) (Young et al 2007). It is a paratendinitis (tenosynovitis) that can result in stenosis of the affected tendons.

Cvitanic (2007) noted a natural foramen between the extensor pollicis longus (EPL) and ECRB in cadavers at the site of the intersection. This could explain the areas of multiple symptoms in the dorsal forearm and could make differential diagnoses more complicated. Inflammatory conditions of the ECRB and ECRL at their insertions may be associated with bony protuberances at the capitare, second or third metacarpals, or trapezoid (Daenen et al 2003).

### Testing

- Pain to palpation and visible swelling may be evidenced in the tendons proximal to the first compartment (Plancher et al 1996, Benicardino & Rosenberg 2006)
- Thickening and interstitial fluid collection around both tendons approximately 4 to 6 to 8 cm proximal to Lister’s tubercle will show on MRI (Benicardino & Rosenberg 2006, Plancher et al 1996)
- Crepitation between the APL/EPB and ECRL/ECRB with wrist flexion or extension may be palpable (Elder & Harvey 2005)

### Differential diagnosis

Finkelstein’s test will be positive but in a more proximal region of the dorsal forearm than would be the case in...
DeQuervain’s tenosynovitis (Elder & Harvey 2005, Young et al 2007).

**Extensor indicis proprius**

Pain and swelling over the fourth dorsal compartment is the most common finding in extensor indicis proprius (EIP) (Fig 26.3) syndrome (Plancher et al 1996). This syndrome involves an irritation of the tenosynovium near the extensor retinaculum (Elder & Harvey 2005). Plancher et al (1996) attributed symptoms of EIP tendinopathy to overuse hypertrophy, or to synovitis secondary to overuse. The former could lead to the latter if symptoms were not addressed in a timely fashion. Anatomic variations (75%) are common, complicating the exact structure involved (Plancher et al 1996, Soejima et al 2002).

**Testing**

- Pain and swelling are evidenced in fourth dorsal compartment distal to ulnar head with supination (Plancher et al 1996)
- Resisted index extension (Hammer 2007c) with wrist fully flexed (Elder & Harvey 2005) provokes symptoms

**Differential diagnosis**

Extensor digitorum communis (EDC) or extensor pollicis longus (EPL) tenosynovitis, dorsoradial ganglion, Klenebck’s disease, extensor digitorum communis tendinopathy, and fourth-compartment syndrome are diagnoses to be excluded (Elder & Harvey 2005).

**Extensor digiti minimi**

The extensor digiti minimi (EDM) (Fig 26.3) occupies the fifth dorsal compartment. The pathology most often occurring here is a tenosynovitis (Elder & Harvey 2005). Duplication of the tendon is common complicating implication of the proper structure (Young et al 2007). Elder & Harvey (2005) stated that continuous hand usage such as handwriting will provoke symptoms. Hammer (2007c) reported a lack of pain with resisted testing, which is unusual for tendinopathy, but no reason for this phenomenon was given.

**Testing**

- Grip is painful (Elder & Harvey 2005)
- Limitation in fifth digit extension is seen (Elder & Harvey 2005)
- Wrist flexion after fist closure or flexing a fist is painful (Elder & Harvey 2005)
- Tenderness to palpation is present just distal to ulnar head (Plancher et al 1996)

**Differential diagnosis**

Extensor carpi ulnaris (ECU) tenosynovitis, TFCC pathology, ulnar impaction should be ruled out (Elder & Harvey 2005).

**Abductor pollicis longus and extensor pollicis brevis**

Together the abductor pollicis longus (APL) and extensor pollicis brevis (EPB) (Fig 26.4) contribute to De Quervain’s tenosynovitis. These tendons normally pass together through a single fibro-osseous tunnel to insert on the first metacarpal and first proximal phalanx respectively (Plancher et al 1996). De Quervain’s tenosynovitis often results from excessive pinching or radial deviation (Hammer 2007c). This syndrome is a common occurrence in golf, racquet sports and fishing (Rettig 2001).

**Testing**

- Finkelstein’s test (Fig 26.5A). Ahuja & Chung (2004) detailed the true test and variations, as the test is misrepresented vigorously in the literature. The original test Finkelstein (1930) described was completely passive: the clinician grasps the patient’s thumb and quickly pulls the wrist into ulnar deviation via the thumb. A positive result is reproduction of pain at the ulnar styloid. The surgeon Eichhoff described a test for de Quervain’s disease that is often mistaken for Finkelstein’s test (Fig 26.5B). His test consisted of the patient actively placing the thumb into the palm and folding the fingers down, holding the thumb in place while the clinician passively moves the wrist into ulnar deviation. A positive test is the same as described for Finkelstein’s test. This test, which many believe to be the Finkelstein test, has been criticized as giving false-positive results. Brunelli described a test in 2003 that he claimed was more accurate than the true Finkelstein’s test. Brunelli criticized Finkelstein’s test for false-positive results due to the
A stretch of the radial collateral ligament, the scaphotrapezial ligament, or the thumb carpometacarpal ligament caused by the APL and EPB tendons being moved away from the pulley. Brunelli described a test in which the wrist is held in radial deviation while forcibly abducting the thumb (Ahuja & Chung 2004). Psychometric properties of these tests have not been established (Elder & Harvey 2005).

The EPB entrapment test identifies separate compartments and resulting stenosis; this test was reported to have sensitivity of 81% and specificity of 50% (Alexander et al 2002).

Tenderness on palpation and swelling over the radial styloid (Elder & Harvey 2005) and first dorsal compartment (Rettig 2001) are present.

Resisted thumb extension is painful (Elder & Harvey 2005).

**Differential diagnosis**

Intersection syndrome (Elder & Harvey 2005), scaphoid fracture, flexor carpi radialis (FCR) tendinopathy, first carpometacarpal (CMC) joint arthritis, and Wartenburg’s syndrome (Plancher et al 1996) are differential diagnoses.

**Extensor pollicis longus**

The extensor pollicis longus (Fig 26.4) often exhibits a tenosynovitis common to racquet sports players. History of repetitive trauma like racquet sports, pain, crepitus, and swelling around Lister’s tubercle will narrow the list of suspected diagnoses (Plancher et al 1996). Triggering of the thumb may be seen in severe cases.

**Testing**

- Pain, swelling, and crepitus along the EPL tendon at the third dorsal compartment (Plancher et al 1996), and at Listers tubercle (Elder & Harvey 2005) are evidenced.
- Pain is elicited with resisted thumb extension or passive flexion (Elder & Harvey 2005).
- Passive interphalangeal joint flexion can reproduce the pain (Elder & Harvey 2005).

**Differential diagnosis**

Differential diagnoses have not been established as necessary for this tendon pathology.

**Flexor carpi radialis**

Flexor carpi radialis (FCR) (Fig 26.2) tendinopathy is common in people who play racquet sports, golf and baseball (Rettig 2001). Elder & Harvey (2005) reported an often insidious onset without known trauma. A primary symptom is pain near the proximal aspect of the trapezium (Gabel et al 1994). This is often a result of overuse with repeated flexion of the wrist, of complication after scaphoid fracture or distal radius fracture, or of other direct trauma (Gabel et al 1994). The FCR is subject to traumatic injury due to its position. The FCR lies in direct contact with the roughened surface of the trapezium. Its insertion onto the trapezium is only 20% of the entire insertion. Additional insertions include the second and third metacarpals (Bishop et al 1994) and the joint capsule of the trapezio-scaphoid joint itself (Schmidt 1987). The tendon occupies 90% of the fibro-osseous tunnel, making it vulnerable to compression (Bishop et al 1994, Elder & Harvey 2005). FCR tendinopathy is also associated with scaphotrapezial joint osteoarthritis, malunion of the trapezium, or scaphoid cyst (Soejima et al 2002).

**Testing**

- Symptoms are exacerbated by resisted flexion and radial deviation of the wrist (Elder & Harvey 2005, Rayan 2005) and with resisted flexion (Rettig 2001); wrist hyper-extension or resisted wrist flexion with radial deviation can reproduce symptoms (Young et al 2007).
- Pain and notable swelling are evidenced at the level of the distal wrist crease along its course (Elder & Harvey 2005) and near the fibro-osseous tunnel (Young et al 2007).

**Differential diagnosis**

Differential diagnoses include osteoarthritis of the first CMC joint, scaphoid cysts, fractures, ganglion cysts,
DeQuervain’s syndrome, and Lindburg’s syndrome (Elder & Harvey 2005).

TREATMENT AND PROGNOSIS

Conservative treatment

Conservative treatment for tendinopathy includes modalities such as ultrasound, electric stimulation, ice, and laser (Curwin 2005); as well as injections and splinting (Plancher et al 1996). Konijnenberg et al (2001) attempted a meta-analysis of outcomes of repetitive strain injuries. Many body areas were included in the analysis. They found no strong evidence for any conservative treatment option (Konijnenberg et al 2001). Conservative treatments included physiotherapy involving multiple types of interventions, but none included the hand or wrist.

Manual therapy, in particular deep tissue friction massage (DTFM), is a conservative treatment for tendinopathy that is utilized by some clinicians; however efficacy has not been proven. This could be due at least in part to study design. DTFM for tendon pain was first popularized by James Cyriax. Cyriax did not perform outcome studies, but studies done by Stasinopoulos & Johnson (2007) concluded that effectiveness of DTFM for lateral epicondylitis could not be assessed from the studies they reviewed. Stasinopoulos did not study outcomes of the wrist.

Cyriax techniques of DTFM for treatment of soft tissue lesions are performed with direct pressure on the painful area. The clinician’s finger rubs firmly transversely to the fibres of the tissue, which includes tendon. Recommended duration and frequency are 20-minute sessions for 6–12 treatments with at least 48 hours between treatments (Cyriax 1983). Cyriax (1983) theorized that the treatment eroded scar tissue between muscle fibres via abrasive contact; in tenosynovitis the rolling was theorized to smooth roughened synovial surfaces. More recent work has specified the optimal time of application for tendon strain based on the previously discussed stage of pathology. Research by Zeichen et al (2000) subjected fibroblasts to strain for varying times, monitoring for proliferation of fibroblasts as a response to a biaxial strain over subsequent hours. The results showed that 15 minutes of strain resulted in increased proliferation over controls at 6 and 24 hours (Zeichen et al 2000).

The 48 hours of recommended minimum accepted time frame between treatments roughly equals the ending of the acute stage of inflammation, when remodelling begins (Leadbetter 1992). The harder pressure, as recommended by Cyriax (1983) may be justified by a Gehlsen et al study (1999) showing that firmer pressure had more positive effects.

Hammer (2007d) applied soft tissue mobilization with greater precision regarding stage of tendon pathology. While he generally concurred with treatment in the 5–15 minute duration twice/week lasting 2 weeks to 2 months, Hammer (2007d) recommended no manual treatment until the proliferative phase, which was described as 7–14 days after original injury. Treatment during acute phase when rest is recommended should be light, ‘aiding fibroblastic proliferation and breaking down of immature collagen’. The maturation phase could be treated more vigorously to reduce fibrosis (Hammer 2007c).

Kahn (2009) and Kraushaar & Nirschl (1999) theorized that mechanical disruption may transform a failed intrinsic healing into a therapeutic extrinsic healing mechanism. Brousseau et al’s (2002) research on DTFM and tendinitis (not tendinosis) considered cross friction treatment as only cross friction and not other techniques, including a stroke along the muscle. This could be one explanation for the lack of more positive outcomes: improper or non-uniform direction of force. Another reason could be lack of proper selection of subcategory of tendinopathy as classified earlier in this section. Some classifications, such as acute inflammation, theoretically cannot be affected by manipulation.

Despite the lack of randomized controlled trials of tendon pain and DTFM, other research is emerging. These studies provide, on a small scale, a patho-anatomic link between manual therapy and reversal of tendinopathic changes. Meltzer & Standley (2007) demonstrated that a modelled indirect osteopathic manipulative technique (IOMT) significantly reduced pro-inflammatory secretions compared to controls 24 h after application, concluding that the modelled IOMT can reverse some of the effects of repetitive strain (Meltzer & Standley 2007). Standley & Meltzer (2008) studied the effect of modelled manual therapy on cellular response. Improved range of motion, reduced analgesic requirements and decreased oedema post-myofascial release was theorized as a result of anti-inflammatory cytokines from strain inducement of myofascial release (Standley & Meltzer 2008).

Eccentric exercise is a more recently applied form of conservative treatment with the theory of reversing degeneration via specific load application. This treatment has shown positive outcomes (Ohberg et al 2004). Eccentric exercise involves contraction of a muscle to control or decelerate a load while the muscle and tendon are lengthening or in a lengthened position. Eccentric exercises have been proven effective at changing ultrasonic findings on involved Achilles tendons within 12 weeks (Ohberg et al 2004). Follow-up showed reduction in tendon diameter and return of normal tendon structure in a majority (19 of 26) of tendons. The unchanged tendons had undefined residual defects (Ohberg et al 2004).

Woodley et al (2006) reviewed 11 studies of eccentric exercises that met inclusion criteria of methodological quality and levels of evidence. They covered both upper and lower extremity tendinopathies. Eccentric exercise was more effective than other treatments that included frictions, stretching, splinting and ultrasound in treating...
tendon pain and improving patient satisfaction and return to work outcomes (Woodley et al 2006).

Curwin (2005) outlined an eccentric programme that consisted of warm up activities, stretching, 3 sets of 10 eccentric exercises, repeated stretching, and icing. This was continued for 6 weeks unless symptoms resolved first. The protocol was performed by 200 patients with chronic tendinopathy that failed conservative therapy. Marked or complete relief of symptoms was reported in 90% of patients who completed the programme. Despite the large sample size there was no control group or randomization (Curwin 2005).


The research by Kannus & Josza (1991) illuminated how stress reduction can lead to degenerative changes in tendon including reduction in mechanical properties. That may be why eccentric exercises are effective in some cases in reducing the effects of immobilization. Soft tissue mobilization along the tendon could also reduce the effects of immobilization, but only to a localized portion of that tendon. Any force, including eccentrics, will not affect the tendon equally. Undamaged fascicles will accept and transmit that force normally, while damaged fascicles, according to Arnoczky et al (2007), will not transfer that force to all fascicles, which leads to degeneration of the involved fascicles. This will be a necessary subject of future studies. An algorithm (Fig 26.6) outlines a

Figure 26.6 Selected treatment algorithm.
proposed pathway of manual treatment and eccentric in tendinopathy.

**Non-conservative treatment**

Reviewing the recent literature on surgeries for wrist tendinopathy reveals a consistent use of the terms tendinitis and tenosynovitis in surgical cases. This use is therefore continued in the report of surgical interventions.

DeQuervain’s tenosynovitis that does not respond to conservative therapy may undergo surgery. This involves decompression of the first dorsal compartment and is not without risks (Plancher et al 1996). Rettig (2001) reported that after 7–10 days of splinting, return to sports can be expected in 6–9 weeks.

Flexor carpi ulnaris surgery often involves excision of the pisiform (Rettig 2001). The expected return to sports averages 8 weeks (Rettig 2001).

As the flexor carpi radialis occupies 90% of the available space in its synovial tunnel, surgery here involves decompression of the tunnel (Plancher et al 1996).

Plancher et al (1996) stated that extensor carpi ulnaris sub-luxation does not always respond to conservative care. The sixth dorsal compartment is released in extensor carpi ulnaris tendinitis. Due to chance of sub-luxation some authors recommend release of the fibro-osseous tunnel it occupies (Plancher et al 1996). Rettig (2001) reported that after 4–6 weeks of casting return to sports required a minimum of 8 weeks.

Surgery for intersection syndrome, according to Plancher et al (1996), involves release of the second compartment with synovectomy. Rettig (2001) mentioned bursectomy between the involved tendons. Release of the third dorsal compartment is also performed on non-responsive cases of extensor pollicis longus tendinitis.

**Prognosis**

Prognosis with conservative treatment for specific tendinopathies or syndromes is not widely available in evidence-based studies. Regarding conservative treatment for DeQuervain’s tenosynovitis, Harvey et al (1990) reported 80% resolution of symptoms with injections alone. Lane et al (2001) concluded that classifying these patients before conservative treatment was initiated improved outcomes; 17 of 18 patients with classified mild symptoms improved with splinting and non-steroidal anti-inflammatory medications. Patients with symptoms classified as moderate to severe responded most favourably to injections (76%) (Lane et al 2001). Richie & Briner (2003) reviewed seven descriptive studies comparing conservative treatments for DeQuervain’s tenosynovitis. They reported an overall cure rate of 83% for injection alone, 61% for injection and splint, 14% for splint alone, and 0% for treatments that consisted solely of non-steroidal anti-inflammatory medications and rest. Tendinopathies of the other structures of the wrist have not been as widely studied.

**CONCLUSION**

While much more is now known about tenocyte and matrix dysfunction, successful application of these concepts for treatment of tendinopathy in the wrist and hand has not been proven. Studies continued to be hampered by sample size, lack of meaningful outcomes, small population selection, and lack of randomization. Adequate studies regarding conservative treatment of non-inflammatory tendinopathies do not appear to exist currently.

Eccentrics show promise for treating tendinopathy in a majority of weight bearing tendons. Successful application of this into tendinopathies of the upper extremity has yet to be accomplished.

Manual therapy’s place in treatment of wrist tendinopathy has not been established. If the efficacy of manual applied eccentrics or what some authors call ‘active release’ could be likewise established in the upper extremity, they could easily be performed adapting Curwin’s (2005) guidelines for eccentric exercises. A study that reveals the effects of manual therapy on tendinopathy will require the following: (1) selection of appropriate tendon pathology, which will pose its own difficulties; (2) soft tissue mobilization such as ‘active release’ along the tendon fibres; (3) continued self-range of motion routinely for 48 hours, to reduce the effect of immobilization; and (4) the repetition of criteria (2) every 48 hours for up to 6 weeks until function has returned. A design incorporating these factors may be able to discern the true worth of DTFM in tendinopathy.

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