Epicondylitis commonly affects the elbow medially or laterally, typically in the 4th or 5th decade of life and without predilection with regard to sex. Epicondylitis is an inflammatory process that may be more accurately described as tendinosis. In the lateral epicondylar region, this process affects the common extensor tendon; in the medial epicondylar region, the common flexor tendon is affected. The condition is widely believed to originate from repetitive overuse with resultant microtearing and progressive degeneration due to an immature reparative response. Advances in understanding of the anatomy and pathophysiology of epicondylitis have shaped current treatment practices. Conservative measures are undertaken initially, because symptoms in most patients improve with time and rest. Those who fail to respond to conservative therapy are considered for surgical treatment. When surgery is contemplated, magnetic resonance imaging or ultrasonography is useful for evaluating the extent of disease, detecting associated pathologic processes, excluding other primary sources of elbow pain, and planning the surgical approach. Familiarity with the normal anatomy, the pathophysiology of epicondylitis and its mimics, and diagnostic imaging techniques and findings allows more accurate diagnosis and helps establish an appropriate treatment plan.

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Glen Ross, MD

LEARNING OBJECTIVES

After reading this article and taking the test, the reader will be able to:

- Describe the clinical manifestations, pathophysiology, and treatment of medial and lateral epicondylitis.
- Identify the soft-tissue components in the complex anatomy of the medial and lateral epicondylar regions of the elbow.
- Select optimal MR imaging and US techniques for detecting medial and lateral epicondylitis and common coexistent conditions.

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Abbreviations: ECRB = extensor carpi radialis brevis, GRE = gradient-recalled echo, LUCL = lateral ulnar collateral ligament, MCL = medial collateral ligament, NSAID = nonsteroidal anti-inflammatory drug, RCL = radial collateral ligament, SE = spin echo, STIR = short inversion time inversion recovery

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Introduction
Lateral and medial epicondylitis are common disorders affecting the upper extremity. Epicondylitis causes pain and functional impairment and typically results from specific occupational and sports-related activities. Lateral epicondylitis, initially described by Morris as “lawn tennis elbow” in 1882 and now most commonly termed tennis elbow, may occur in patients performing any activity that involves repeated supination and pronation of the forearm with the elbow in extension (1–8). Medial epicondylitis, although commonly termed golfer’s elbow, may occur in throwing athletes, tennis players, and bowlers, as well as in workers whose occupations (eg, carpentry) result in similar repetitive motions (7,9). Lateral epicondylitis occurs with a frequency seven to 10 times that of medial epicondylitis (4,9). Both lateral and medial epicondylitis most commonly occur in the 4th and 5th decades of life, without predilection with regard to sex.

Epicondylitis represents a degenerative process involving the origin of the extensor tendons at the lateral elbow and the flexor-pronator muscle group at the medial elbow. It is thought that repetitive stress and overuse lead to tendinosis with microtrauma and partial tearing that may progress to a full-thickness tendon tear (1–3). The diagnosis of epicondylitis hinges on a careful history and physical examination. In most patients, the condition is managed conservatively with cessation of the offending activity, applications of ice, administration of a nonsteroidal anti-inflammatory drug (NSAID) or a corticosteroid injection, and use of a splint or brace (4,7). These measures are followed by a rehabilitation program aimed at gradually increasing power, flexibility, and endurance with eventual reintroduction into the implicated sport or occupational activity (7). In rehabilitation, it is important to correct any biomechanical abnormalities that may have led to the initial injury. Other treatments include injection of autologous blood or platelet-rich plasma, ultrasonographically guided tenotomy, extracorporeal shock-wave therapy, and iontophoresis and phonophoresis to obtain deep penetration of topical medications into the soft tissues (10).

Although conservative treatment is often successful, magnetic resonance (MR) imaging or ultrasonography (US) may be performed to verify the diagnosis in the presence of recalcitrant or confounding symptoms, quantify the degree of tendon injury, identify associated abnormalities, and aid in preoperative planning. The differential diagnosis for lateral elbow pain includes occult fracture, osteochondritis dissecans of the capitellum, lateral osteoarthritis, lateral ulnar collateral ligament (LUCL) instability, and radial tunnel syndrome. In cases of suspected medial epicondylitis, it is important to exclude medial osteoarthritis, medial collateral ligament (MCL) injury, and ulnar neuropathy, any of which may mimic or coexist with medial epicondylitis.

Surgery is often performed if there is no clinical response after 3 to 6 months of conservative treatment. Surgical techniques include open and arthroscopic approaches with dissection, release, and débridement of the degenerated tendon (1,4,8). We prefer a mini-open approach that allows a shorter recovery time, and we encourage early postoperative mobilization therapy. The goal in rehabilitation is the eventual reintroduction of the implicated activity with corrected biomechanics. The literature reports a high success rate for surgical procedures, with overall patient satisfaction and full return to preinjury activities (1,8–10).

The article reviews the anatomy, pathophysiology, and clinical and imaging manifestations of epicondylitis in the lateral and medial epicondylar regions of the elbow separately. Other common conditions that may mimic or coexist with epicondylitis in these regions are considered, and indications for the use of MR imaging and US in differential diagnosis and treatment planning are described. The implications of the clinical history and imaging findings for the selection of the most appropriate medical or surgical treatment option are discussed in detail.
Lateral Epicondylitis

Normal Anatomy of the Lateral Elbow

The extensor carpi radialis brevis (ECRB), extensor digitorum communis, and extensor carpi ulnaris form a strong, discrete, conjoined tendon that is attached at the anterior aspect of the lateral epicondyle and lateral supracondylar ridge, adjacent to the origins of the brachioradialis and extensor carpi radialis longus (11). The lateral epicondyle is also the site of attachment for the extensor digiti minimi and the supinator, which merge with the ECRB, extensor digitorum communis, and extensor carpi ulnaris to form the common extensor tendon (Fig 1). The ECRB occupies the deep and anterior aspect of this common tendon and inserts at the base of the third metacarpal bone. The undersurface of the ECRB is in contact with the capitellum and slides along its lateral edge during elbow extension and flexion. Repetitive wear and abrasion due to this contact may play a role in the pathophysiology of epicondylitis (12).

Capsular injury as well as thickening and tearing of the lateral ulnar collateral ligament (LUCL) and radial collateral ligament (RCL) have been identified in association with severe lateral epicondylitis (14,15). The lateral collateral ligament complex consists of the RCL, annular ligament, accessory lateral collateral ligament, and LUCL (Fig 2). The RCL originates at the lateral epicondyle anteriorly and blends with the fibers of the annular ligament and fascia of the supinator muscle (11). The annular ligament, the primary stabilizer of the proximal radioulnar joint, tapers distally and surrounds the radial head in a funnel.
shape. Disruption of this ligament leads to radioulnar instability (11). The accessory lateral collateral ligament helps stabilize the annular ligament but is inconsistently present (11). The fibers of the accessory ligament originate from the annular ligament and insert on the supinator crest, along the lateral aspect of the ulna. The LUCL contributes to ligamentous constraint against varus stress. Originating from the lateral epicondyle as a continuation of the RCL, the LUCL runs along the lateral and posterior aspects of the radius to insert on the tubercle of the supinator crest of the ulna. Disruption of the LUCL results in posterolateral rotatory instability of the elbow (11,14).

Pathogenesis
Lateral epicondylitis is most often the result of repetitive stress injury but may result from direct trauma. The condition is common among tennis players, especially nonprofessionals, in whom poor mechanics may be an instigating factor (7). Lateral epicondylitis is caused by repeated contraction of the forearm extensor muscles, particularly at the origin of the ECRB, which results in microtearing with subsequent degeneration, immature repair, and tendinosis. In addition to the mechanical forces that lead to excessive varus stress on the ECRB, its unique anatomic position against the lateral aspect of the capitellum places the tendon at risk for repeated undersurface abrasion during elbow extension (12). The lack of vascularity at the undersurface of the tendon further contributes to degeneration and tendinosis (12).

At gross examination, the affected tendon appears gray and friable (1,7). Epicondylitis was initially believed to originate from an inflammatory process involving the radial humeral bursa, synovium, periosteum, and annular ligament (9). In 1979, Nirschl and Petrone (1) described their observation of the disorganization of normal collagen architecture by invading fibroblasts in association with an immature vascular reparative response, which they collectively termed “angiofibroplastic hyperplasia.” The same process later was described as “angiofibroplastic tendinosis” because no inflammatory cells were identified (13,16). Because inflammation is not a significant factor in epicondylitis, the term tendinosis is preferred over epicondylitis or tendinitis. Over time, scar tissue forms that is vulnerable to repetitive trauma, which leads to further tearing. Continuation of this cycle of injury and immature repair results in more substantial tears, with consequent alteration and failure of musculotendinous biomechanics and worsening of symptoms (17).

Clinical Manifestations and Diagnosis
Patients present with lateral elbow pain, which is frequently exacerbated when they grasp objects during wrist extension with resistance. A history of tennis playing or similar racket sports is some-
times elicited, but the condition often results from other athletic or occupational activities or from an unknown cause. In racket sports, the backhand swing most commonly instigates symptoms (7). With palpation during physical examination, focal tenderness is present at the origin of the ECRB, about 1 cm distal to the midportion of the epicondyle (7). Reduced strength with resisted gripping and with supination and extension of the wrist also are commonly seen. Maneuvers such as the “chair test” (in which the patient is asked to lift a chair with a pronated hand) and the “coffee cup test” (in which the patient picks up a full cup of coffee) evoke focal pain at the lateral epicondyle (7). The diagnosis of lateral epicondylitis is clinically based in most cases.

However, the differential diagnosis of lateral elbow pain is broad (Table 2), and imaging often is necessary when refractory or confounding symptoms are present. It has been reported that 5% of those with an initial diagnosis of lateral epicondylitis have radial tunnel syndrome (18). Radial tunnel syndrome involves entrapment of the posterior interosseous nerve (a deep branch of the radial nerve) within the radial tunnel. The radial tunnel is bounded medially by the brachialis muscle and anterolaterally by the brachioradialis, extensor carpi radialis longus, and ECRB. Posteriorly, the radial tunnel is delineated at its proximal end by the capitellum and at its distal end by the distal aspect of the supinator muscle. Patients present with insidious pain along the proximal radial aspect of the forearm, without motor deficit, and, typically, without localizability to a specific nerve distribution. Many patients with this condition report a history of activity involving repetitive forearm supination and pronation. Physical examination with palpation at the radial tunnel or resisted supination of the forearm and extension of the middle finger produces pain. The most common MR imaging finding of radial tunnel syndrome is denervation edema or atrophy within the muscles innervated by the posterior interosseous nerve (Fig 3) (18).

Role of Diagnostic Imaging
Imaging is not routinely indicated for the diagnosis of lateral epicondylitis but typically is performed in recalcitrant or complicated cases to allow evaluation of the extent of disease and exclusion of other pathologic processes that cause lateral elbow pain. Imaging also plays an important role in preoperative planning. MR imaging is the most widely used modality, although US also may be performed. In a study by Miller et al (19), the sensitivity of US for the detection of both lateral and medial epicondylitis ranged from 64% to 82%, whereas that of MR imaging ranged from 90% to 100%. Elbow radiography often is negative but may show calcium deposition adjacent to the lateral epicondyle and may help exclude other pathologic processes (20).

MR Imaging Technique and Findings—Proper patient positioning and sequence selection are essential for accurate MR imaging of the elbow. We perform all elbow MR imaging examinations by using a 1.0-T extremity magnet (ONI Medical Systems, Wilmington, Mass) with the following sequences: coronal two-dimensional gradient-recalled echo (GRE), coronal proton density-weighted fat-saturated fast spin echo (SE), coronal short inversion time inversion-recovery (STIR) fast SE, axial T1-weighted fast SE, axial T2-weighted fast SE, sagittal T1-weighted fast SE, and sagittal STIR fast SE (Table 3). The patient is imaged while reclining with the arm abducted, elbow extended, and wrist supinated.

### Table 3
Protocol for MR Imaging of the Elbow with a 1.0-T Extremity Magnet

<table>
<thead>
<tr>
<th>Plane</th>
<th>Sequence</th>
<th>TE (msec)</th>
<th>TR (msec)</th>
<th>ETL</th>
<th>Matrix</th>
<th>BW (Hz)</th>
<th>FOV (mm)</th>
<th>Section Thickness (mm)</th>
<th>Gap (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coronal</td>
<td>2D GRE*</td>
<td>18</td>
<td>510</td>
<td>1</td>
<td>300 × 192</td>
<td>30</td>
<td>123</td>
<td>2.5</td>
<td>0.3</td>
</tr>
<tr>
<td>Coronal</td>
<td>PD FS fast SE</td>
<td>15</td>
<td>3100</td>
<td>10</td>
<td>288 × 192</td>
<td>40</td>
<td>140</td>
<td>2.5</td>
<td>1.0</td>
</tr>
<tr>
<td>Coronal</td>
<td>STIR fast SE</td>
<td>15</td>
<td>3700</td>
<td>8</td>
<td>288 × 192</td>
<td>35</td>
<td>150</td>
<td>3.0</td>
<td>0.5</td>
</tr>
<tr>
<td>Axial</td>
<td>T1 fast SE</td>
<td>16</td>
<td>800</td>
<td>2</td>
<td>288 × 224</td>
<td>40</td>
<td>120</td>
<td>3.0</td>
<td>1.0</td>
</tr>
<tr>
<td>Axial</td>
<td>T2 fast SE</td>
<td>80</td>
<td>3500</td>
<td>10</td>
<td>260 × 240</td>
<td>35</td>
<td>120</td>
<td>3.0</td>
<td>1.0</td>
</tr>
<tr>
<td>Sagittal</td>
<td>T1 fast SE</td>
<td>15</td>
<td>650</td>
<td>2</td>
<td>288 × 192</td>
<td>35</td>
<td>150</td>
<td>4.0</td>
<td>0.5</td>
</tr>
<tr>
<td>Sagittal</td>
<td>STIR fast SE</td>
<td>15</td>
<td>4100</td>
<td>10</td>
<td>260 × 192</td>
<td>35</td>
<td>150</td>
<td>4.0</td>
<td>0.5</td>
</tr>
</tbody>
</table>

*Flip angle for the GRE pulse sequence is 25°.

Note—BW = bandwidth, ETL = echo train length, FOV = field of view, FS = fat saturated, PD = proton density-weighted, TE = echo time, T1 = T1-weighted, TR = repetition time, T2 = T2-weighted, 2D = two-dimensional.
along the anterior surface of the condyles in the axial plane, and the sagittal plane is perpendicular to that coronal plane.

The normal MR imaging appearance of the common extensor tendon is that of a vertically oriented structure that originates from the lateral epicondyle. The tendon should show uniform low signal intensity, regardless of the imaging sequence used (Fig 4). The ECRB is the deepest and most anterior component of the common extensor tendon. Tendon morphology is best assessed on coronal and axial images. Like the common extensor tendon, the lateral ligaments exhibit uniform low signal intensity with all sequences. The LUCL is seen as a low-signal-intensity band medial to the common extensor tendon. It originates from the lateral epicondyle and, after coursing posterior to the radial head, inserts on the tubercle of the supinator crest of the ulna. The RCL, which is located immediately anterior to the LUCL, also originates from the lateral epicondyle (Fig 5). The fibers of the RCL course distally along the long axis of the radial head to blend with the fibers of the annular ligament. A small region with the signal intensity of fluid is often seen partially undercutting the RCL at its radial head attachment and is considered normal (21,22). Coronal images are
Figure 7. Moderate lateral epicondylitis. (a) Proton density–weighted fat-saturated MR image obtained in a 60-year-old man depicts a region of slightly increased signal intensity due to fluid accumulation within the superficial fibers of the common extensor tendon, a finding suggestive of a small partial-thickness tear (arrow). (b) Sagittal STIR MR image shows a central region with the signal intensity of fluid in the proximal common extensor fibers, with a surrounding rim of intermediate signal intensity (arrow), findings consistent with a partial-thickness tear and tendinosis. ANT = anterior, ECRL = extensor carpi radialis longus, ECU = extensor carpi ulnaris.

best for evaluating the RCL and LUCL, but the entire LUCL is not likely to be seen on a single coronal image because of its oblique course.

Tendon and ligament abnormalities are best identified on proton density–weighted and T2-weighted fast SE images (with or without fat saturation). The MR imaging findings of tendinosis on both T1- and T2-weighted images include intermediate signal intensity within the substance of the tendon—most commonly, the ECRB—with or without tendon thickening (15,19,23). Partial-thickness tears are seen as a region with the signal intensity of fluid extending partway across the tendon with diffuse tendon thinning. A full-thickness tear appears as a fluid-signal-intensity gap across the substance of the tendon or between the proximal tendon and its attachment to the lateral epicondyle (11). The histologic and surgical findings correlate well with the MR imaging features of tendon degeneration and the degree of tendon tear (15). However, an MR imaging– or US-based grading system that is clinically, surgically, and outcome relevant has yet to be developed.

We therefore grade lateral epicondylitis as mild (tendinosis or low-grade partial tear), moderate (intermediate-grade partial tear), or severe (high-grade partial tear or full-thickness tear). Mild epicondylitis is characterized by tendon thickening and increased internal signal intensity. In moderate epicondylitis, there is a partial-thickness tear with thinning and focal disruption that does not extend across the full thickness of the tendon. Severe epicondylitis consists of a near-complete or complete tear, characterized as a fluid-filled gap separating the tendon from its origin at the lateral epicondyle. Low-grade tears are those affecting less than 20% of the tendon thickness; intermediate tears, 20% to 80%; and high-grade tears, more than 80% (Figs 6–8).
It is important to evaluate the LUCL, RCL, extensor muscles, synovium, cartilage, and subchondral bone for coexistent abnormalities that may require a modification of surgical management. In particular, associated intramuscular edema may be seen in the common extensor muscles (Fig 9). The LUCL should be carefully evaluated. Bredella et al (14) showed that lateral epicondylitis is frequently associated with thickening and tears of the LUCL. In addition, an acute injury of the LUCL may occur in association with an injury of the common extensor tendon (Fig 10). Rupture of the LUCL may result in posterolateral rotatory instability, and surgical release of the extensor tendon may lead to further destabilization of the elbow (14). The radial
collateral ligament should be assessed to detect periligamentous edema or frank tearing. The radiocapitellar and ulnohumeral joints should be examined for focal chondral defects and signs of secondary osteoarthrosis.

**US Technique and Findings.**—US is an excellent option for diagnostic imaging evaluation of lateral epicondylitis, with a reported sensitivity of approximately 80% and specificity of approximately 50% (17,19,24). The lateral region of the elbow is best scanned in both transverse and longitudinal planes with a variable-high-frequency linear-array transducer (5–12-MHz or higher) and with the elbow flexed (Fig 11). US allows visualization of the entirety of the common extensor tendon, from the musculotendinous junction to the site of origin on the lateral epicondyle. The common extensor tendon origin is seen as a continuous band of longitudinally oriented fibers (Fig 12). The ECRB constitutes the most anterior aspect of the common extensor tendon and the major portion of its attaching surface (11,14). Fibers from the RCL and LUCL, located deep to the common extensor tendon, also can be evaluated with US. Tendinosis appears as tendon enlargement and heterogeneity, and tendon tears are depicted as hypoechoic regions with adjacent tendon discontinuity. Surrounding fluid and calcification also may be seen. Levin et al (17) found a statistically significant relationship between clinical symptoms of lateral epicondylitis and US findings of intratendinous calcification, tendon thickening, bone irregularity, focal hypoechogenicity, and diffuse heterogeneity. However, given its high false-positive rate, real-time US may be most useful for determining the extent of tendon damage in patients who are symptomatic (17). We use the same system at US as at MR imaging to grade lateral epicondylitis as mild, moderate, or severe (Figs 13–15).
Figure 15. Severe epicondylitis. Longitudinal US image of the common extensor tendon origin in a 64-year-old man reveals a large hypoechoic region at the tendon origin, a finding indicative of a near-full-thickness tear. The mildly retracted tendon (*) has a markedly heterogeneous appearance characteristic of tendinopathy. A small focus of calcium deposition (arrow) is seen adjacent to the lateral epicondyle.

Figure 16. Intraoperative photograph, obtained during a modified Nirschl procedure for treatment of lateral epicondylitis, shows a portion of the torn ECRB tendon origin (arrow) within the forceps. The gray-white discoloration of the tendon is indicative of degeneration.

Treatment
Initial treatment is typically conservative and may include the application of cold packs (for local vasoconstriction and analgesia), rest, oral NSAID therapy, corticosteroid injections, splinting, and physical therapy. Some advocate the use of prolotherapy and extracorporeal shock wave lithotripsy (7). Patients with lateral epicondylitis that is unresponsive to conservative treatment after 6 to 9 months are referred for imaging and may eventually require surgery.

Our preferred surgical procedure is a modified Nirschl technique with a mini-open approach. This procedure does not allow access to the joint as arthroscopy would, but it is easier to perform, takes less time, and is less costly. First, the ECRB is accessed by splitting the extensor carpi radialis longus and the extensor digitorum brevis (Fig 16). The degenerated portions of the ECRB and the leading edge of the extensor digitorum brevis are then excised. The ECRB does not require reattachment because it is supported by adjacent fascial attachments that prevent its distal retraction (1,12). Next, holes are drilled in the epicondyle, and any traction spurs are removed. The extensor carpi radialis longus and extensor digitorum communis are then repaired, and the wound is closed. Patients can quickly return to activities of daily living with a full range of motion and can resume sports activities in 3 to 4 months after this procedure.

Figure 17. Drawing shows the musculotendinous anatomy of the medial aspect of the elbow. FCR = flexor carpi radialis, FCU = flexor carpi ulnaris, FDS = flexor digitorum superficialis, PL = palmaris longus, PT = pronator teres.

Figure 18. Drawing shows the ligamentous anatomy of the medial aspect of the elbow. AL = annular ligament, ant = anterior band, post = posterior band, trans = transverse band.
Medial Epicondylitis

Normal Anatomy of the Medial Elbow
The muscles of the flexor-pronator group include the pronator teres, flexor carpi radialis, palmaris longus, flexor digitorum superficialis, and flexor carpi ulnaris (Table 4). The flexor carpi radialis, palmaris longus, and flexor carpi ulnaris form the common flexor tendon. The pronator teres and flexor carpi radialis (together termed the flexor-pronator mass) attach to the anterior aspect of the medial epicondyle and are most commonly injured in medial epicondylitis (9,11) (Fig 17).

Owing to the valgus stress produced by overhand throwing, these muscles are typically hypertrophied in professional throwing athletes (9). The stability of the medial elbow is supported mainly by the articulation of the olecranon of the ulna and the trochlea of the humerus. The flexor and extensor muscles, joint capsule, MCL, and LUCL conjointly provide elbow stabilization.

Injury to any one of these structures leads to increased stress on the others. For these reasons, all of these structures are evaluated in patients with medial elbow pain.

The MCL, also known as the ulnar collateral ligament, comprises three ligamentous bands: the anterior bundle, posterior bundle, and an oblique band termed the transverse ligament. These three bands form a triangular shape along the medial aspect of the elbow, deep to the pronator mass (Fig 18). The posterior and transverse ligaments form the floor of the cubital tunnel just deep to the ulnar nerve. The anterior bundle extends from the inferior aspect of the medial epicondyle and inserts on the sublime tubercle (medial aspect of the coronoid process) and provides the primary constraint against valgus stress (7,11,22). MCL

Table 4
Anatomy of the Muscles of the Medial Compartment of the Elbow

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Function</th>
<th>Origin</th>
<th>Insertion Site</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pronator teres</td>
<td>Pronates the forearm</td>
<td>Humeral head: medial epicondyle; ulnar head: coronoid process</td>
<td>Lateral surface of the radius</td>
</tr>
<tr>
<td>Flexor carpi radialis</td>
<td>Flexes and abducts the wrist</td>
<td>Common flexor tendon from the medial epicondyle of the humerus</td>
<td>Volar aspect of the base of the second and third metacarpal bones</td>
</tr>
<tr>
<td>Palmaris longus</td>
<td>Flexes the wrist and tightens the palmar aponeurosis</td>
<td>Common flexor tendon from the medial epicondyle of the humerus</td>
<td>Flexor retinaculum and palmar aponeurosis</td>
</tr>
<tr>
<td>Flexor carpi ulnaris</td>
<td>Flexes and adducts the hand</td>
<td>Humeral head: common flexor tendon from the medial epicondyle; ulnar head: olecranon and dorsal aspect of the proximal ulna</td>
<td>Pisiform, hamate, and base of the fifth metacarpal bones</td>
</tr>
<tr>
<td>Flexor digitorum supercili</td>
<td>Flexes the middle phalanges and MCP joint</td>
<td>Humeral head: medial epicondyle; ulnar head: coronoid process; radial head: oblique line of the radius</td>
<td>Palmar aspect of the middle phalanges of the second through fifth digits</td>
</tr>
<tr>
<td>Flexor digitorum profundus</td>
<td>Flexes the distal phalanges at the DIP joints; assists in flexion of the wrist and proximal phalanges</td>
<td>Anterior and medial aspect of the ulna and interosseous membrane</td>
<td>Dorsal aspect of the distal phalanges of the second through fifth digits</td>
</tr>
<tr>
<td>Flexor pollicis longus</td>
<td>Flexes the thumb</td>
<td>Volar aspect of the mid radius, interosseous membrane and medial aspect of the coronoid process of the ulna</td>
<td>Palmar aspect of the base of the distal phalanx of the thumb</td>
</tr>
<tr>
<td>Brachioradialis</td>
<td>Flexes the elbow</td>
<td>Lateral supracondylar ridge of the humerus</td>
<td>Lateral aspect of the radial styloid process</td>
</tr>
</tbody>
</table>

Source.—Adapted from reference 11.
Note.—DIP = dorsal interphalangeal, MCP = metacarpophalangeal.
injury, specifically anterior band injury, is included in the differential diagnosis of medial elbow pain, and therefore the MCL must be evaluated. The MCL is also prone to concurrent injury with medial epicondylitis (9,11,22).

Given its location in the medial elbow, the ulnar nerve should be evaluated in all patients with medial elbow pain. The ulnar nerve is located within the cubital tunnel and may be injured in association with medial epicondylitis from chronic stretching and irritation or from direct injury (9,11). The cubital tunnel is bounded by the medial epicondyle anteriorly, the MCL laterally, and the flexor carpi ulnaris posteromedially.

**Pathogenesis**

Valgus forces transmitted to the medial elbow during forearm pronation and wrist flexion may exceed the strength of the muscles, tendons, and supporting ligaments. In golfers and throwing athletes, the strain produced by these forces is exacerbated by poor technique. The result may be medial epicondylitis, a condition that is primarily due to repetitive stress or overuse of the flexor-pronator musculature, just as cumulative stress or overuse of the common extensor mechanism results in lateral epicondylitis. Strain caused by poor mechanics, poor conditioning, limited flexibility, or fatigue leads to increased transmission of both concentric and eccentric contractile loading forces (9). These forces lead to degenerative changes at the musculotendinous junction of the flexor-pronator muscle group. In medial epicondylitis, the flexor-pronator mass (pronator teres and flexor carpi radialis) is most commonly injured, followed by the palmaris longus (9,11). The MCL is the ligament most commonly involved (11). The pathologic features of medial epicondylitis are similar to those of lateral epicondylitis and include degeneration, angiofibroblastic change, and an inadequate reparative response, leading to tendinosis and tearing (1–3,9).

**Clinical Manifestations and Diagnosis**

Patients with medial epicondylitis typically present with medial elbow pain, which often develops insidiously (except in acute trauma). Symptoms of weakness in grip strength are also common. Patients may offer a history of sports activities, including golf, overhead throwing sports, and racket sports, with difficulty in initiating the serve and executing the forehand stroke (7,9). Tenderness is elicited by palpation of the insertion of the flexor-pronator mass (5–10 mm distal and anterior to the middle aspect of the medial epicondyle) (9). In addition, pain is exacerbated by resisted wrist flexion and forearm pronation at an angle of 90°. Flexion contractures may develop in professional athletes because of muscular hypertrophy (9). Because of common symptoms and associated valgus forces, ulnar neuritis and MCL instability, as well as other causes of medial elbow pain, should be considered in the differential diagnosis (Table 5). The Tinel sign (distal pain and tingling during direct compression of the nerve at the elbow), among other findings at physical examination, is helpful for establishing the diagnosis of ulnar neuritis (7,9). MCL stability may be evaluated by applying a valgus stress or by performing the “milking test” (pulling on the thumb with the elbow in flexion and the forearm in supination) (9). A positive result of both of these tests is defined as elicitation of focal pain along the MCL.

**Role of Diagnostic Imaging**

As in lateral epicondylitis, imaging is not always essential in the initial evaluation of medial epicondylitis. However, with a confounding clini-
cal picture or with refractory cases, imaging is recommended. Both MR imaging and US may be used in the evaluation of medial epicondylitis. If there are signs of ulnar neuritis and medial instability, MR imaging is preferred. Radiographs often appear normal but may show calcification adjacent to the medial epicondyle (Fig 19) (9). In chronic cases, traction spurs and medial collateral ligament calcification may be seen, as well.

**MR Imaging Technique and Findings.**—MR imaging protocols are the same as those described earlier for lateral epicondylitis (Table 3). The common flexor tendon origin is seen at the anteromedial aspect of the medial epicondyle. It courses distally, parallel to the long axis of the ulna, appearing as a low-signal-intensity band on MR images obtained with any sequence (Fig 20). The common flexor tendon is medial and proximal to the MCL, and the pronator teres is seen just anterior to the common flexor tendon. The three bands of the MCL are most reliably identified in the coronal plane, with the anterior band coursing from the anteroinferior medial epicondyle to the sublime tubercle of the ulna (Fig 21). The anterior band demonstrates low signal intensity on MR images obtained with any sequence, and it should be firmly attached to the sublime tubercle. On axial images, the ulnar nerve within the cubital tunnel is depicted as a smooth round structure surrounded by fat, which has signal isointense to that of muscle on T1-weighted images and iso- or hyperintense to that of muscle on T2-weighted images (11,25).

MR imaging findings of medial epicondylitis range from tendinosis, which is indicated by intratendinous thickening and increased signal intensity on images obtained with any sequence, to complete rupture (11,23). A tendon tear is identifiable as a fluid-signal-intensity gap between the tendon and the epicondyle or by interdigitation of fluid with the tendon or muscle fibers. We grade medial epicondylitis in the same way described earlier for lateral epicondylitis (Figs 22–24). However, as noted in the earlier discussion of lateral epicondylitis, an MR imaging– or US-based grading system with clinical, surgical, and outcome relevance has yet to be developed.

**Figure 20.** Normal medial elbow. Axial T2-weighted fast SE (a) and sagittal STIR (b) MR images obtained in a 30-year-old man demonstrate a normal appearance of the common flexor tendon (arrow), which originates as a band with uniformly low signal intensity on the anteromedial aspect of the medial epicondyle. ANT = anterior.

**Figure 21.** Normal medial elbow. Coronal GRE MR image obtained in a 43-year-old man depicts a normal appearance of the MCL (arrow) at its insertion on the sublime tubercle of the ulna (*).
Figure 22. Mild medial epicondylitis. Axial T2-weighted fast SE MR image obtained in a 52-year-old man shows a linear wisp of fluid signal intensity at the undersurface of the common flexor tendon origin (arrow), a finding indicative of a small partial-thickness tear.

Figure 23. Moderate medial epicondylitis. Coronal STIR MR image obtained in a 57-year-old man demonstrates a large region with the signal intensity of fluid at the undersurface and within the substance of the common flexor tendon origin (arrow), a finding indicative of an intermediate-grade partial-thickness tear.

Figure 24. Severe medial epicondylitis. Coronal proton density-weighted fat-saturated MR image obtained in a 48-year-old woman depicts a large area of fluid signal intensity at the origin of the common flexor tendon (arrow), a finding indicative of a high-grade partial-thickness tear, with retraction of the torn fibers (*).

Figure 25. Severe medial epicondylitis. Axial T2-weighted fast SE MR image obtained in a 48-year-old man demonstrates prominent regions of intermediate to high signal intensity within the flexor digitorum superficialis (black arrow), flexor carpi radialis (white arrow), and pronator teres (arrowhead), findings indicative of muscle strain associated with medial epicondylitis.
Figure 27. Severe medial epicondylitis and ulnar neuritis. (a) Coronal STIR MR image obtained in a 49-year-old man depicts the signal intensity of fluid throughout the insertional fibers of the common flexor tendon with an adjacent region of intermediate signal intensity (arrow), findings indicative of a high-grade partial-thickness tear and associated muscle strain. (b) Axial T2-weighted fast SE MR image demonstrates increased signal intensity in the ulnar nerve with associated loss of normal signal in the surrounding fat (arrow), findings indicative of ulnar neuritis.

In addition to findings in the flexor-pronator mass, abnormalities may be seen in the MCL, ulnar nerve, and other muscles in the medial elbow. In severe cases, muscle strain is commonly seen in the palmaris longus and flexor digitorum superficialis (Fig 25). An MCL sprain can be seen as high signal intensity of the ligament on proton density–weighted fat-saturated fast SE images. Associated full- and partial-thickness MCL tears may be seen in severe medial epicondylitis or in the setting of acute trauma to the common flexor tendon (Fig 26) (11). Associated ulnar neuritis, which typically affects the nerve within or just distal to the cubital tunnel (11,25), is identified as thickening and increased signal intensity of the nerve on T2-weighted or proton density–weighted fat-saturated fast SE images (Fig 27).
US Technique and Findings.—At US, the medial epicondylar region is best scanned in the transverse and longitudinal planes with a variable-high-frequency linear-array transducer (5–12-MHz or higher) and with the patient’s arm in extension and the forearm in supination (Fig 28). US images should be obtained to depict the entirety of the common flexor tendon, from the musculotendinous junction to the tendon origin at the medial epicondyle. At its origin, the normal common flexor tendon appears as a continuous band of longitudinally oriented fibers with uniform echogenicity (Fig 29). The appearance of the common flexor tendon is similar to that of the common extensor tendon, but its attachment is less broad based. Medial epicondylitis may be identified as outward bowing, heterogeneous echogenicity, or thickening of the common tendon, with subjacent fluid collection and intratendinous calcification (17,19). Discrete tears appear as hypoechoic regions with adjacent tendon discontinuity. For US-based grading of medial epicondylitis, we use the same system described earlier for US-based grading of lateral epicondylitis (Figs 30–32).

Treatment

Initial clinical management of medial epicondylitis involves cessation of the provocative activity, application of cold packs to the elbow, and oral NSAID therapy. If these measures fail to bring relief, nighttime use of a splint and one or more local corticosteroid injections may be necessary (7,9). Other treatment options include the application of ultrasound waves or high-voltage galvanic stimulation (9). These therapies are followed by a guided rehabilitation program in which the intensity and frequency of activity is gradually increased, with the eventual goal of reinitiation into full participation in the suspended sporting or occupational activity. During rehabilitation, sporting equipment and technique are reevaluated and modified if necessary; for example, older golfing irons might be replaced with lighter graphite clubs. The success rates for nonsurgical treatments of medial epicondylitis vary across the literature, ranging from 26% to 90% (9). The use of MR imaging is therefore more commonly indicated in medial epicondylitis than in lateral epicondylitis.

If the condition fails to respond to a disciplined nonsurgical treatment regimen of 3 to 6 months’ duration, surgery is recommended. For professional athletes, earlier surgery may be indicated if there is evidence of tendon disruption at physical examination and imaging evaluation. Various surgical procedures have been employed for medial epicondylitis as for lateral epicondylitis. The surgical technique that we prefer begins with a curvilinear posterior incision to spare the medial cutaneous nerve. Care must be taken to
protect the ulnar nerve, as well (9). Degenerated peritendinous tissue in the interval between the pronator teres and the flexor carpi radialis is removed with aggressive débridement. Multiple holes are then drilled into the exposed medial epicondyle to enhance local vascularity and promote a more robust healing response. Unlike the procedure used to treat lateral epicondylitis, this procedure includes firm reattachment of the flexor-pronator tendon to its origin at the medial epicondyle (9) (Fig 33). An abnormality of the ulnar nerve or MCL, if present, may be treated surgically at the same time. Because of the close proximity of the nerve and ligament, aggressive tendon débridement is not performed for medial epicondylitis (9). Immediately after surgery, with the elbow in flexion at 90° and the forearm in neutral position, a posterior plaster splint is applied. Early postoperative mobilization is followed by strengthening exercises at 6–8 weeks and full activity at 4–5 months after surgery (9). Although the literature about surgical treatment of medial epicondylitis is limited, good to excellent results are reported, with 85% of patients returning to preinjury activity levels and reporting overall satisfaction (9).

**Summary**

Epicondylitis of the medial or lateral elbow is a common source of pain among professional and recreational athletes. Epicondylitis represents a degenerative process that, in its initial stages, is characterized by tendinosis and partial tearing with an immature reparative response. MR imaging and US have proved effective for diagnosing and characterizing these and other abnormalities, but optimal imaging protocols are essential for effective differentiation of pathologic conditions of the elbow. Knowledge of the typical imaging features of epicondylitis and associated injuries, as well as those of other common sources of elbow pain, allows the radiologist to accurately characterize the pathologic process and guide the referring clinician toward an appropriate treatment plan. Although symptoms may resolve after a few months of conservative therapy, surgery in severe, recalcitrant, or complicated cases typically brings excellent results with relatively minimal recovery time.
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References

This article meets the criteria for 1.0 credit hour in category 1 of the AMA Physician’s Recognition Award. To obtain credit, see accompanying test at http://www.rsna.org/education/rg_cme.html.
Although conservative treatment is often successful, magnetic resonance (MR) imaging or ultrasonography (US) may be performed to verify the diagnosis in the presence of recalcitrant or confounding symptoms, quantify the degree of tendon injury, identify associated abnormalities, and aid in preoperative planning.

The essential and universal lesion of lateral epicondylitis involves the ECRB, followed by the extensor digitorum communis and, to a lesser extent, other muscles and tendons of the lateral compartment.

In 1979, Nirschl and Pettrone described their observation of the disorganization of normal collagen architecture by invading fibroblasts in association with an immature vascular reparative response, which they collectively termed “angiofibroblastic hyperplasia.” The same process later was described as “angiofibroblastic tendinosis” because no inflammatory cells were identified.

MR imaging findings of tendinosis on both T1- and T2-weighted images include intermediate signal intensity within the substance of the tendon—most commonly, the ECRB—with or without tendon thickening.

The pronator teres and flexor carpi radialis (together termed the flexor-pronator mass) attach to the anterior aspect of the medial epicondyle and are most commonly injured in medial epicondylitis.