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MEDIAL AND LATERAL EPICONDYLITIS IN THE ATHLETE

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MEDIAL EPICONDYLITIS

Literature regarding medial epicondylitis has been scant in comparison to lateral epicondylitis. This is likely due to its decreased incidence, which is reported to be between 9.8% and 20% of all epicondylitis diagnoses[2, 27, 44] when compared with lateral epicondylitis. Medial epicondylitis, or "golfers' elbow," refers to an overuse syndrome of the flexor pronator mass. The muscles involved include the pronator teres, flexor carpi radialis, palmaris longus,[26] and on rare occasion, the flexor carpi ulnaris[26] and flexor digitorum superficialis.[4] Ulnar nerve symptoms have been reported in up to 60% of cases.[13, 34] Medial epicondylitis has been reported to occur concurrently with ipsilateral lateral epicondylitis.[47]

Etiology

The typical patient is between 21 and 65 years of age,[4, 26, 47] is male,[32, 47] and has injured the dominant arm.[47, 51] Tennis has been more commonly implicated[9, 19, 26, 34, 47, 51]; however, medial epicondylitis also has been reported with golf, bowling,[47] archery, baseball, weightlifting,[14] javelin throwing,[29] racquetball,[47] and football.[47] Repetitive activities not

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commonly performed by the patient, such as hammering or use of a screwdriver, also can cause medial epicondylitis.[14] During all these activities, the elbow is subject to overuse. The muscle often fatigues, and with muscle weakness, the flexor pronator mass fails with eccentric muscle contractions.[14] The result is repetitive microtrauma followed by chronic inflammation.

Recent histologic studies, although performed after at least one steroid injection had been given in the affected area, revealed torn tendon margins with scar tissue, cystic degeneration, round cell infiltrate, and occasional fibrinoid degeneration.[4] Although Goldie's[18] studies revealed free nerve endings in the granulation tissue at the site of lateral epicondylitis, he did not perform a similar study for the medial counterpart. Nirschl[31] described the histopathologic appearance as angiofibroblastic tendinosis with vascular and fibrous granulation tissue. Essentially, studies have shown a chronic rather than an acute process, which results in an abnormal tendon histology.

Diagnosis

Diagnosis includes a careful history and physical examination as well as radiographic and imaging studies. Electromyography (EMG) is indicated in those patients with neurologic changes, and laboratory studies are performed when rheumatologic disorders are suspected.

History

Patients typically report a history of chronic medial elbow pain accompanying activities involving wrist flexion and pronation, such as serving, overhead and forehand strokes in tennis. Baseball players experience pain during pitching.[3] The weekend handyman may complain of pain while hammering.[14] Muscle rupture can occur acutely[26]; however, most often there is not a specific inciting event. Patients also report weak grasp and pain, which may radiate to the forearm.

Physical Examination

Palpation anterior to the medial epicondyle will elicit pain as will resisted wrist flexion and forearm pronation. There also may be swelling and warmth in this area.[4] Elbow range of motion is assessed; a flexion contracture may be present in those patients with chronic symptoms.[32] The ulnar collateral ligament is examined by applying a valgus stress to the slightly flexed elbow carefully, with the wrist in flexion and the forearm pronated. If the patient has a chronic ulnar collateral ligament strain, he/she will have pain, and the examination may elicit laxity.[26]

Neurologic examination may reveal a Tinel's sign of the ulnar nerve.

The location in relation to Nirschl's ulnar nerve zones is important for determining the cause of the nerve compression[32] (Fig. 1). Tinel's sign in zone 1 implies congenital ulnar nerve subluxation, but a Tinel's sign in zone 2 may be due to compression from loose bodies, spurs, or rheumatoid synovitis. A zone 3 Tinel's sign indicates nerve compression as the ulnar nerve passes through the two heads of the flexor carpi ulnaris.

Grip strength may be decreased compared with the contralateral side. The significance of this decrease may be underestimated considering the high incidence of medial epicondylitis in the dominant extremity and the fact that grip strength testing has shown the dominant extremity to have 3% to 7% greater strength.[43, 45]

The physical examination is concluded with a cervical spine examination to ensure a proper diagnosis.

Radiographs and Imaging

Standard radiographs may show calcifications adjacent to the medial epicondyle in 20% to 30% of patients with medial epicondylitis.[2, 47] In a study by Baumgard and colleagues, all calcifications resorbed within

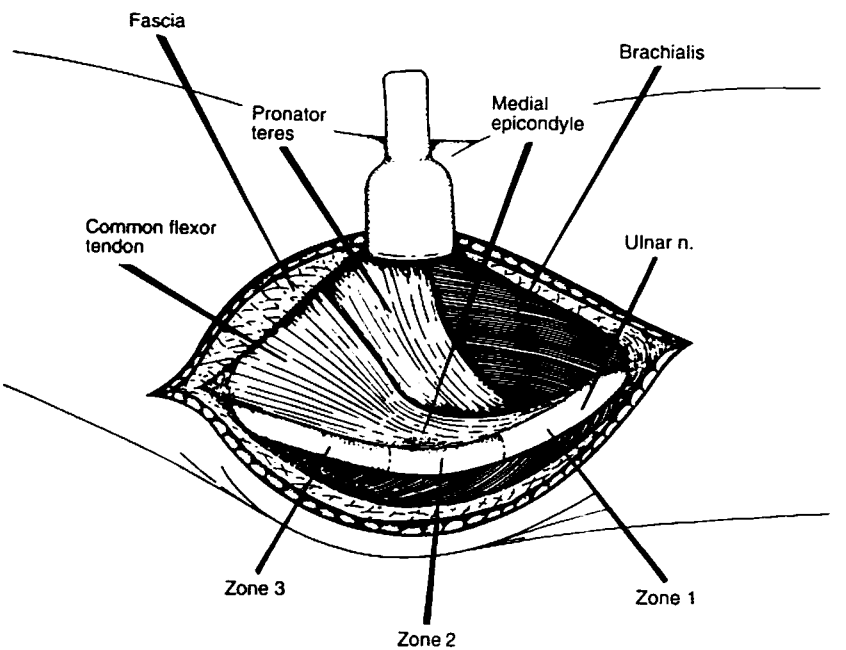

Figure 1. Ulnar nerve zones at the medial elbow. Surgical decompression is advocated for zone 3 compression. (Courtesy of Joanne Halbrecht, MD, and Kevin D. Plancher, MD, MS.)

6 months postoperatively.[2] Radiographs of the cervical spine may be indicated as well as an MR imaging or CT scan of the elbow to rule out other intra- or extra-articular pathologic conditions.

Electrodiagnostic Studies

EMG and nerve conduction studies are helpful to rule out a proximal radiculopathy or neurapraxia if the patient displays neurologic symptoms.

Differential Diagnoses

Differential diagnoses include several entities (Table 1). The aforementioned studies and physical examination can be used to make the appropriate diagnosis. As discussed earlier, medial epicondylitis can occur on rare occasions in conjunction with lateral epicondylitis and ulnar nerve symptoms.[47]

Treatment

The duration of symptoms is related directly to the response from rehabilitation; those whose symptoms have persisted for a long time demonstrate a slower recovery.[14] Initial treatment is aimed at diminishing inflammation through activity modification, rehabilitation, and the use of ice and nonsteroidal anti-inflammatory drugs.[14, 26] Activity modification includes performance of activities within a limited or pain-free range.[14, 26] If pain increases, the activity should be stopped immediately.[26] Ice is applied to the affected area for 10 to 20 minutes before and after exercises.[14, 26] Passive range-of-motion exercises are performed as tolerated. The effect of bracing on muscle contraction is controversial.

Table 1. DIFFERENTIAL DIAGNOSES

Arthritis
Cervical radiculopathy
Chondromalacia
Cubital tunnel syndrome
Fibrosis
Joint laxity
Loose bodies
Olecranon/coronoid impingement
Osteophytes
Referred pain from biceps insertion, brachialis, or lacertus
Tardy ulnar nerve palsy
Ulnar collateral ligament instability
Ulnar trochlear synovitis

One study has shown no effect,[19] whereas another has demonstrated some effect on muscle contraction.[17] Nonsteroidal anti-inflammatory medications are given for 2 to 3 weeks, although no study has shown their usefulness. If there is no pain relief with this regimen, corticosteroid injection into the area of maximal tenderness is given. In addition to their anti-inflammatory effects, steroids can inhibit tendon healing and weaken the tendons[49]; therefore, no more than two or three steroid injections should be given into the inflamed area. The use of splints is encouraged to avoid any tendon rupture, especially when steroids are injected into the arm.

Once the inflammation is reduced, treatment is expanded to include stretching and strengthening. (For the specific protocol on medial and lateral epicondylitis, see the article by Thomas et al in the April 1995 issue of *Clinics in Sports Medicine*, pp 459–477.) Some authors advocate the use of electrical stimulation,[4] iontonophoresis, high-voltage galvanic stimulation, phonophoresis,[4, 14] and ultrasonography. These authors have not found any of these modalities useful in practice. Ultrasonography should not be used in children, as its effect on open growth plates is not well known.[14] Strengthening is performed through resistive wrist flexion and forearm pronation.[14, 26]

The final phase of treatment consists of functional rehabilitation.[14] This involves a gradual return to exercise through technique modification or retraining. Medial epicondylitis, like lateral epicondylitis, often results from improper technique while performing sports activities. Ilfeld[22] showed that patients with medial epicondylitis who played tennis often had an incorrect serve and forehand stroke. These patients had strokes that consisted of hitting the ball late, with the head of the racquet behind the elbow when contact was made with the ball, all leading to elbow problems. The use of graphite frames, a racquet strung less tightly, and playing on slower surfaces all can diminish vibration transmission to the extremity.[21, 26] Use of a larger racquet head also will provide a larger area within which to hit the ball centrally on the racquet strings, thus preventing an off-center hit and valgus stress to the elbow.[26]

Prevention

Instruction on proper technique in performing activities that involve repetitive wrist flexion and forearm pronation as well as stretching, strengthening, and warm-up exercises are important components of a prevention program.

Surgical Treatment

If a 6- to 12-month trial of conservative treatment fails, surgery is considered. Nirschl[32] approaches the flexor pronator mass through a 7-cm incision just posterior to the medial epicondyle (Fig. 2). The tendons are incised longitudinally beginning at the origin and extending for

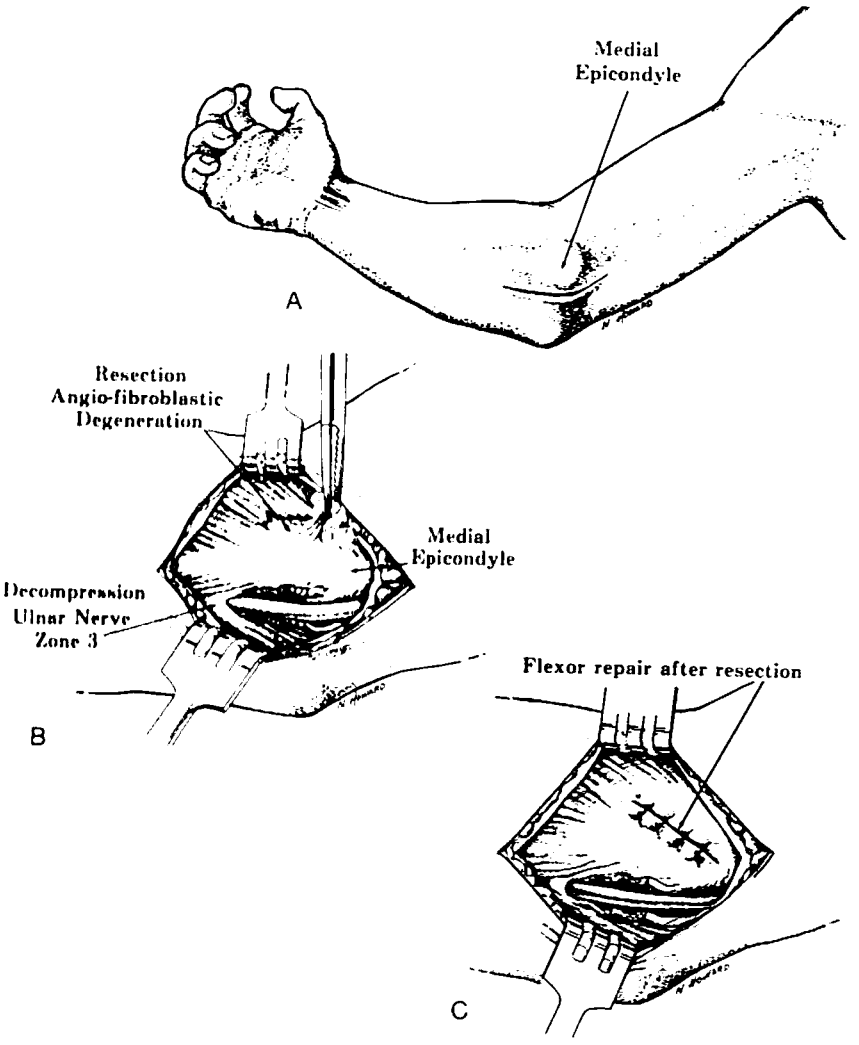

Figure 2. Nirschl's technique for medial release and ulnar nerve decompression in medial epicondylitis with ulnar neurapraxia. *A*, Incision is made as shown. *B*, Resection of angiofibroblastic tendinosis. The pathologic tissue is removed in longitudinal and elliptic fashion, leaving attachments of normal tissue intact. In 60% of cases, dysfunction of the ulnar nerve has been noted clinically; decompression of the ulnar nerve in zone 3 of the medial epicondylar groove is done. *C*, Repair of medial tennis elbow. Repair of the common flexor origin is undertaken. Note that the medial epicondylar attachments of normal tissue are not disturbed. (*From* Nirschl RP: Elbow tendinosis/tennis elbow. Clin Sports Med 11:851–870, 1992.)

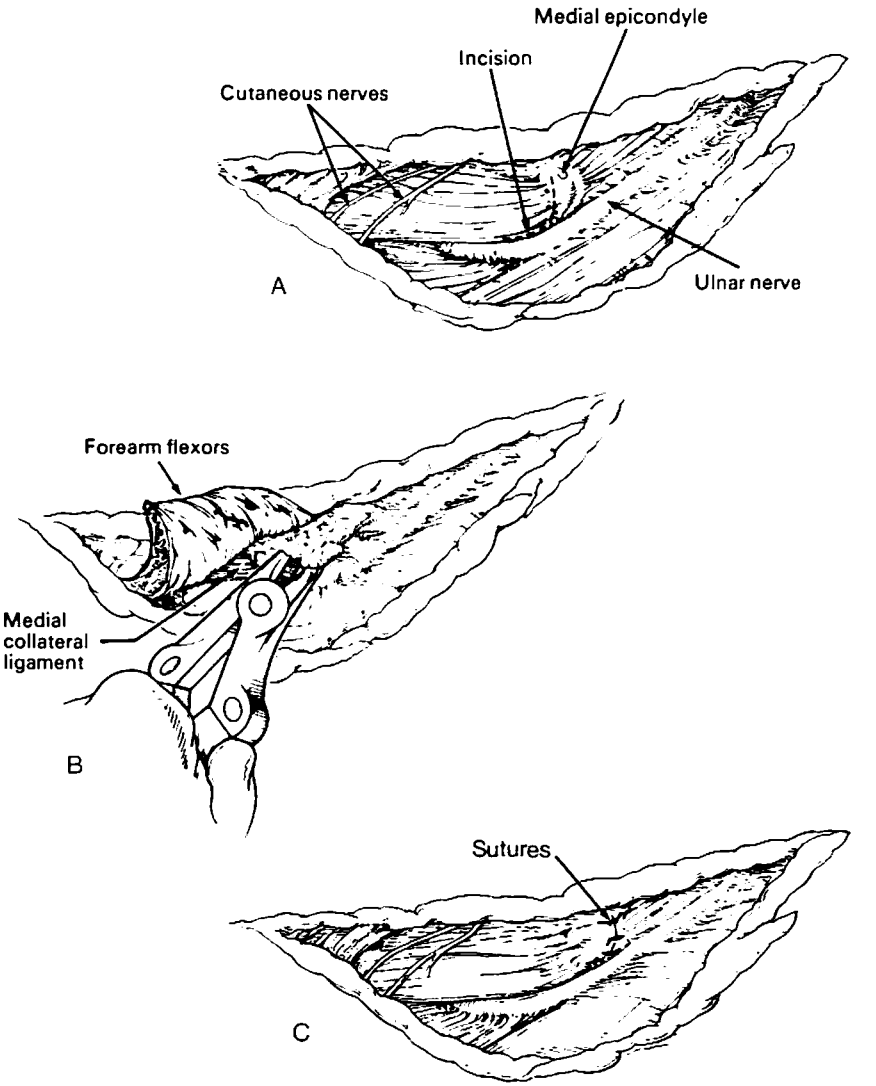

Figure 3. Vangsness and Jobe release for medial epicondylitis. *A*, Wound open and deep structures visualized. *B*, Reflection of the common flexor origin and excision of the degenerative tissue with a rongeur. *C*, Reattachment of the forearm flexors. (*From* Vangsness C, Jobe FW: Surgical treatment of medial epicondylitis: Results in 35 elbows. J Bone Joint Surg Br 73:409–411, 1991; with permission.)

approximately 5 cm distally. When the tendons are spread, the area of pathologic tissue is visualized and excised. The defect is repaired with absorbable suture. Subcuticular ulnar nerve transposition is performed as dictated for exposure in those patients displaying ulnar nerve symp-

toms, valgus angulation, valgus ligamentous instability, or nerve subluxation/dislocation. The patient is splinted at 90 deg postoperatively in a counterforce elbow immobilizer, and exercises begin on postoperative day 3.

Vangsness and Jobe[47] approach the flexor origin through a 10- to 12-cm incision centered on the medial epicondyle (Fig. 3). The flexor origin is detached and the pathologic tissue is excised. Soft tissue is removed from the medial epicondyle, and several small drill holes are made. The flexor tendons are then reattached to their origin. Ulnar nerve transposition is performed in those patients with ulnar nerve involvement. The patient is splinted at 90 deg in a posterior splint that ends proximal to the wrist. Resistive wrist flexion and forearm pronation are delayed for 6 to 8 weeks. In this series of 35 patients, 88% had good to excellent results.

Wilhelm and Gieseler[50] incise the common flexor origin and perform a denervation of the medial epicondyle by dissection of the surrounding tissue. Good to excellent results were obtained in 14 of 17 patients in a series performed by Wittenberg et al.[51] Baumgard and Schwartz[2] had 83% excellent results in their small series (six patients) with percutaneous release of the common flexor origin (Fig. 4). They describe an incision that extends from the proximal anterior aspect to the distal inferior aspect of the medial epicondyle. The common flexor origin is released by sharply dissecting it off of the bone.

Complications

Regardless of the procedure, care must be taken to avoid the medial antebrachial cutaneous nerve, the ulnar nerve, and the ulnar collateral ligament.

Described surgical complications include hematoma,[47] persistent ulnar nerve symptoms,[47] wound infection,[32, 51] and decreased sensation over the scar.[51] Other potential complications are laceration of the ulnar collateral ligament, necessitating repair or reconstruction; ulnar or me-

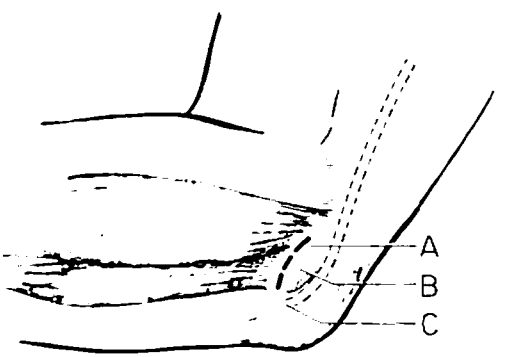

Figure 4. Baumgard and Schwartz percutaneous release for medial epicondylitis. Incision (A) on the anterior aspect of the medial epicondyle (B). Course of the ulnar nerve (C). (*From* Baumgard SH, Schwartz DR: Percutaneous release of epicondylar muscles for humeral epicondylitis. Am J Sports Med 10:233–236, 1982; with permission.)

dial antebrachial cutaneous nerve laceration; neuroma; and fistula formation.

Full return to sports can be anticipated within 4 to 6 months postoperatively, although it may take some patients up to 1 year to return to their sport.[32, 47]

LATERAL EPICONDYLITIS

Lateral epicondylitis has remained an important diagnosis when discussing overuse injuries of the elbow and forearm. It was first described in the English literature more than 100 years ago in *The Lancet*.[30a] This entity still poses many diagnostic, therapeutic, and rehabilitative challenges to the orthopedic surgeon. This section discusses the pertinent anatomy, proposed pathophysiology, epidemiology, diagnosis, nonsurgical and surgical treatment, and the rehabilitative protocols and complications of lateral epicondylitis.

Lateral epicondylitis, commonly known as tennis elbow, is seen in the athletic enthusiast involved in racquet sports. This syndrome occurs in a variety of other sporting activities but is also found in various individuals who do not participate in sports.[6] Coonrad et al,[9] in assessing more than 1000 patients, reported that less than 5% of golf or tennis players have lateral epicondylitis.

Anatomy

The primary pathologic tissue of lateral epicondylitis involves the origin of the extensor carpi radialis brevis, and, less commonly, the long extensor and anterior aspect of the extensor digitorum communis tendon.[35] There is some controversy as to whether lateral epicondylitis is an inflammatory or a degenerative process. In a cadaveric study by Regan et al, evidence of vascular proliferation and hyaline degeneration was observed.[39]

The musculotendinous structures of concern arising from the lateral epicondyle include the extensor carpi radialis longus (ECRL), the extensor carpi radialis brevis (ECRB), the extensor digitorum communis (EDC), and the extensor carpi ulnaris (ECU)(Fig. 5). The anatomic origin of the ECRB is complex and has contributions from the common extensor tendon at the lateral epicondyle, lateral collateral ligament, the annular ligament, the overlying fascia, and the intramuscular septum. The ECRL arises from the distal aspect of the lateral supracondylar ridge, taking origin above the elbow joint with the ECRB underneath the ECRL. The understanding of this anatomy is essential if a surgeon elects to use the treatment regimen recommended by Nirschl.[31, 35] The close relationship of the ECRB to the lateral collateral and annular ligaments of the elbow helps to support Bosworth's[5] earlier proposed surgical procedures for resection of the annular ligament for treatment of this condition, al-

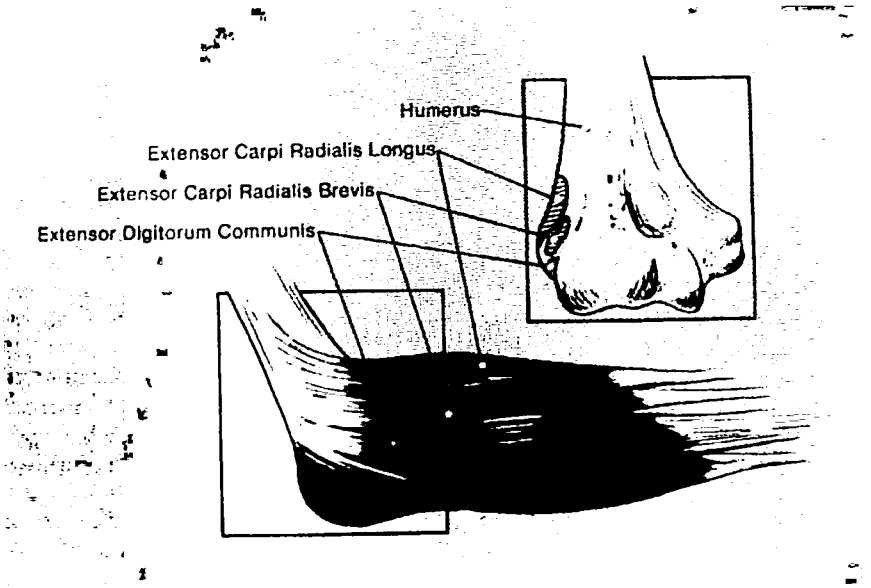

Figure 5. Anatomy of the musculotendinous structures arising from the lateral epicondyle. (Courtesy of The Indiana Hand Center, Indianapolis, Indiana.)

though the authors would not recommend this surgical procedure as an option in 1995.

Wadsworth believes that lateral epicondylitis is caused by a partial tear of the musculotendinous unit of the ECRB. His manipulation (Mills maneuver) places high forces on the ECRB, resulting in its complete rupture. He believes that by completing the tear of the ECRB, the patient's symptoms will be alleviated. This therapeutic action has not received great support in the United States.[48]

Etiology and Epidemiology

Many sports and occupational activities that require repetitive forearm motion have been implicated in the development of lateral epicondylitis.[26] Studies that use cohorts of sports enthusiasts differ in the patient profiles from those using individuals who develop lateral epicondylitis from occupational activities, although the causes of the proposed pathophysiology remain similar.

Occupational activities that require forceful and repetitive forearm dorsiflexion, radial deviation, and supination (as is seen in meat cutters, plumbers, and weavers) can cause increased stress on the wrist extensors, resulting in degenerative changes, most commonly within the extensor carpi radialis brevis. The same biomechanic overload has been found in tennis players.

Jobe and others, using an EMG technique, studied the activity of the muscles stabilizing the elbow while playing tennis. They found the greatest activity during ground strokes to be in the musculotendinous unit stabilizing the wrist and in decreasing order: the ECRB, followed by the ECRL and the EDC. The ECRB showed the greatest contraction of all tested musculotendinous units during both the acceleration and follow-through phases. These data have helped to support the theory that the ECRB, either by overuse in sports or occupational activities, is the musculotendinous unit most prone to be affected in this condition.[24]

Gruchow reported on the epidemiologic profile of lateral epicondylitis in tennis players. He found in more than 500 tennis players that age and playing time per day were the most important contributing factors in the development of symptoms. He noted that the risk in developing symptoms in players completing more than 2 hours of racquet time per week was four times greater than in those playing less than this limit. In individuals over the age of 40, he noted that men and women had a two and four times greater chance, respectively, of developing lateral epicondylitis.[20]

Several studies have shown that suboptimal mechanics in hitting the tennis ball, improper grip size, racquet weight, and tension of the strings, all have been implicated in causing tennis elbow. Tennis-playing surfaces that generate greater velocity to the ball require increased force in striking the ball; this generates increased stress to the extensors. Studies seem to agree that lateral epicondylitis occurs frequently in both men and women in their 40s and 50s equally; the dominant arm is involved in more than three fourths of the cases.[24]

Athletic and nonathletic patients present with a similar profile in which the underlying cause is the repetitive forearm and wrist motion that puts the extensor carpi radialis brevis at its greatest use or, perhaps, overuse.

Pathophysiology

In 1936, Cyriax described more than 29 causes of lateral epicondylitis contributing to the vast confusion of the pathophysiology for this diagnosis.[10] Runge in 1873 described in the German literature "writer's cramp," and since that time, numerous anatomic aspects of the lateral aspect of the elbow have been implicated in the pathophysiology of lateral epicondylitis.[41] A radial humeral bursitis was described by Carp[7] in 1932. A synovial fringe with inflammation was described by Trethowan in 1929.[45a] Bosworth in 1955 implicated trauma with fibrosis of the annular ligament as the cause of lateral epicondylitis.[5] Garden in 1961 implicated repeated trauma with periostitis of the ECRB from increased wrist dorsiflexion and forearm supination.[15] Neural inflammation led the way for Kaplan's[25] proposed denervation of the elbow joint in 1959. Early degenerative osseous changes of the radial capitellar joint have been described as contributing to lateral epicondylitis.[24] Roles described

entrapment of the posterior interosseous nerve as a contributing and sole cause of lateral epicondylitis.[23, 40] Sanders theorizes that entrapment of the posterior interosseous nerve is the problem. His treatment relies on the posterior interosseous nerve release during surgery.[42]

These are only a fraction of the many proposed theories, but current thinking usually supports Nirschl's theory that lateral epicondylitis begins as an injury to the origin of the ECRB.[31, 35] The injury begins as a microtear, which can enlarge to involve the extensor digitorum communis or even the ECRL (Fig. 6). The characteristic grayish friable tissue so often termed *angiofibroblastic hyperplasia* has been well documented histologically.[35] Nirschl has reported that over 97% of patients in his series have this characteristic finding.[31, 35] Almost one third of patients demonstrating these gross findings also showed gross tendon rupture.

Diagnoses

The differential diagnoses of a patient presenting with pain on the lateral side of the elbow should be assessed carefully by the physician before diagnosing this pain as lateral epicondylitis. The vast spectrum of proposed pathophysiologic causes of lateral epicondylitis should alert the physician to the different sources of lateral epicondylar pain. Entrapment of the musculocutaneous nerve as it exits between the biceps and the brachialis, a phenomenon seen often in tennis players, can present with pain over the lateral epicondylar region. Entrapment of the posterior interosseous nerve resulting in radial tunnel syndrome also can be a source of lateral epicondylar pain; although some authors have tended to support its coexistence, it still can exist separately and needs to be

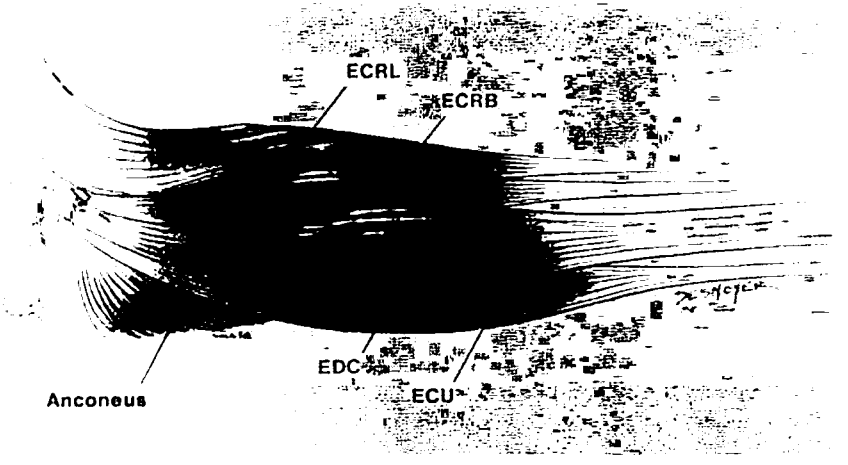

Figure 6. Zone of injury to the origin of the ECRB. (Courtesy of The Indiana Hand Center, Indianapolis, Indiana.)

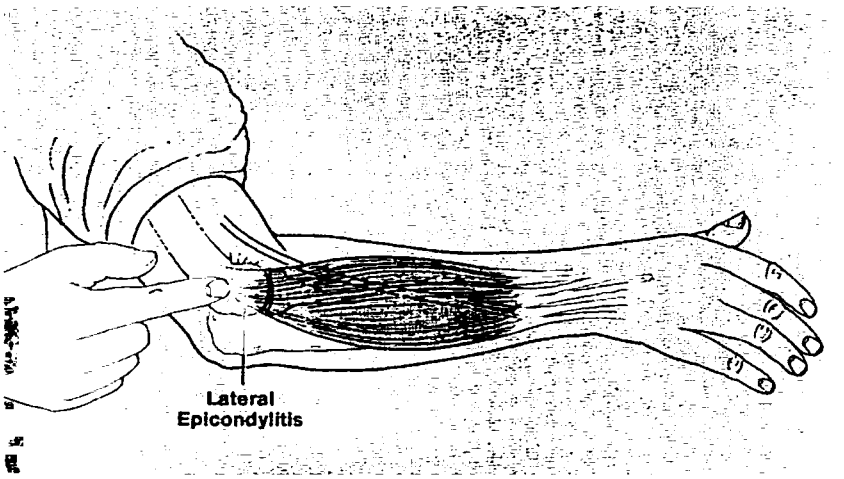

Figure 7. Physical examination demonstrating tenderness over the common extensor origin often localized to the ECRB. (Courtesy of The Indiana Hand Center, Indianapolis, Indiana.)

ruled out. Radial capitellar degenerative changes can be ruled out by physical examination and a proper radiographic examination. More recently, a chronic compartment syndrome involving the anconeus muscle has been implicated as the cause of lateral elbow pain with intracompartmental pressure measurements documenting its existence.[1] O'Driscoll et al[36] have described pain in this region secondary to posterolateral rotatory instability, and they think this diagnosis should be ruled out. Cervical degenerative disc disease must be ruled out as patients with a C7 radiculopathy may present with pain over the lateral epicondylar area.

Even with myriad diagnoses of patients presenting with pain of the lateral epicondylar area, it is the history of repetitive overuse of the forearm and wrist that needs to be well documented.

Physical Examination

Physical examination will confirm tenderness over the common extensor tendon origin often localized to the ECRB (Fig. 7). Although mostly insidious at onset, acute cases can present and sometimes will reveal tenderness proximally along the lateral supracondylar ridge, which may confirm involvement of the ECRL. The area of maximal discomfort most commonly is located up to 5 mm distal and anterior to the lateral epicondyle. Increased discomfort with resisting wrist and finger extension with the elbow in full extension will exacerbate the pain. The "chair test," which involves asking the patient to raise the back of a chair with the elbow in full extension, the forearm pronated,

and the wrist dorsiflexed, many times will generate an apprehensive facial expression prior to the attempt, a reaction which has been accurate in documenting the patient's symptoms. Volitional weakness in wrist extension because of pain may be present, although a neurovascular examination should be normal.

Radiographs should be ordered as studies have shown that up to 25% of patients may show calcification in the soft-tissue area surrounding the lateral epicondyle.[35] It has not been shown that this calcification has any relationship to prognosis.

Treatment

Nonsurgical Treatment

Most studies document the overwhelming success of nonsurgical treatment in lateral epicondylitis. In a series of 1000 patients, Coonrad reported greater than an 82% success rate in conservative treatment.[9] Nirschl's series had a 93% success rate with nonoperative treatment.[35] These studies attest to the overall nonsurgical treatment of this entity and dictate its importance prior to the consideration of surgical treatment.

Almost all treatment modalities involve cessation of the offending activity, rest, splint immobilization, and nonsteroidal anti-inflammatory medication with consideration for a steroid injection. When symptoms subside, gradual rehabilitation with strengthening and either avoidance or modification of activities by selected improvement in athletic mechanics should be used.

The first phase of nonsurgical treatment involves gaining symptomatic pain improvement with the hope of resultant reduction of inflammation. The offending activity, when identified, is reduced as some feel complete cessation should be avoided to prevent disuse atrophy. Ice should be applied to decrease the ensuing inflammatory cascade. Nonsteroidal anti-inflammatory medication usually is started for up to a 2-week period. Ultrasonography and other modalities have been reported to be useful and should remain in the armamentarium of the clinician. Their efficacy, however, remains suspect as no well-controlled prospective study has documented their effectiveness. The use of a short arm splint in this phase can be a useful adjunct with the wrist in slight dorsiflexion. Theoretically, the splint may decrease the stress on the wrist extensors and help symptoms. A forearm support band, such as that described by Froimson, placed just below the elbow may alter the stress biomechanically on the origin of the wrist extensors and relieve discomfort.[13] The patient should be educated in avoiding placing the support band excessively tight, as compression of the anterior interosseous nerve with a complete anterior interosseous nerve syndrome has been reported.[11]

Recently, an air-filled bladder has been introduced in lieu of the

forearm support band. Snyder-Mackler, using indwelling EMG recordings, confirmed significant reduction in ECRB and EDC muscle activity when using this type of "aircast," giving support to the statement that bracing inhibits full muscular contraction and decreases the force imparted on partially injured musculotendinous units (Fig. 8).[46]

If the patient does not respond dramatically to these initial therapeutic measures, a corticosteroid injection can be used. Different preparations exist and are used according to the individual surgeon's preference. Day,[10,1] in his double-blind study, showed cortisone was better than saline and lidocaine. Price and others showed no definite increased efficacy of one type of steroid over the other.[38] Most studies do document the importance of the proper instillation of the dose in the area deep to the ECRB, slightly anterior and distal to the lateral epicondyle, where a

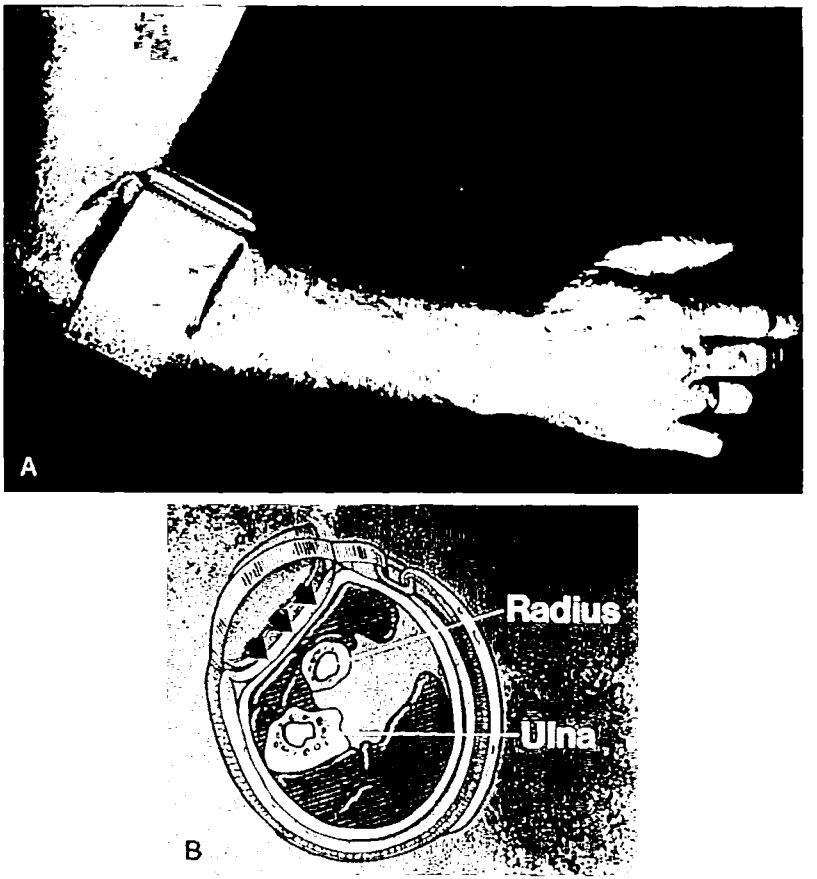

Figure 8. *A*, Air-filled forearm support. *B*, Cross-sectional illustration demonstrating Aircast (Aircast, Summit, NJ), giving support to the lateral epicondylar region. (Part *B* courtesy of The Indiana Hand Center, Indianapolis, Indiana.)

palpable recess usually is found. Inadvertent injection superficially can result in subcutaneous atrophy and hypopigmentation and should be avoided and discussed with the patient. Pain relief with a properly given injection has been described in up to 90% of patients.[38] Rehabilitative protocols involving stretching with strengthening supervised by therapists are helpful in returning the individual to the predisease state. (See article by Thomas et al in the April 1995 issue of *Clinics in Sports Medicine*, pp 459–477.) Modification of poor mechanics in either sports or occupational activities should be performed at this point. In the tennis player, mechanics that allow the athlete to make contact with the ball in front of the body with the elbow and wrist extended will decrease the load on the wrist extensors. On the backhand side, maintaining wrist extension at the point of contact will help to decrease the forces on the lateral epicondyle. As improvement in flexibility, endurance, and power is documented, the patient is allowed to return to his or her previous activity.

Nirschl has advocated that proper grip- and racquet-size measurements be taken prior to playing tennis. His method for proper grip size measures from the proximal palmar crease to the radial aspect of the tip of the ring finger.[31, 35] Decreasing string tension, as already mentioned for medial epicondylitis has been helpful in dispersion of the forces. The use of lighter racquets, low-vibration materials, and playing on softer surfaces, such as clay, are also important in preventing recurrence.

Most studies have shown that up to 10% of patients will have recurrence of symptoms with conservative treatment. Surgical treatment may be indicated if symptoms persist for 6 months to 1 year after presentation. Other indications for surgery include muscle weakness, incapacitating pain, limitations in sports, and special circumstances involving professional athletes.

Surgical Treatment

Jobe has summarized the various surgical options into four general categories: (1) extra-articular procedures addressing the common extensor origin with release; (2) intra-articular procedures removing synovial fringes and parts or all of the annular ligament; (3) extra-articular procedures that address the ECRB tendon and areas remote from the origin, specifically distally; and (4) extra-articular procedures that excise the characteristic pathologic degenerated tissue with reattachment of the origin of the ECRB.[24]

Procedures in the first group include a fasciotomy of the extensor aponeurosis as described by Michele in 1956.[28] A long-term follow-up study by Posch in 1978 confirmed satisfactory results with this technique.[37] Recent application of this extra-articular technique with release of the fascia and common extensor origin have included percutaneous release under local anesthesia in the office. Baumgard, Yerger, and others all have reported satisfactory results.[2, 52] There is some concern about overzealous release of the common extensor tendon, which may

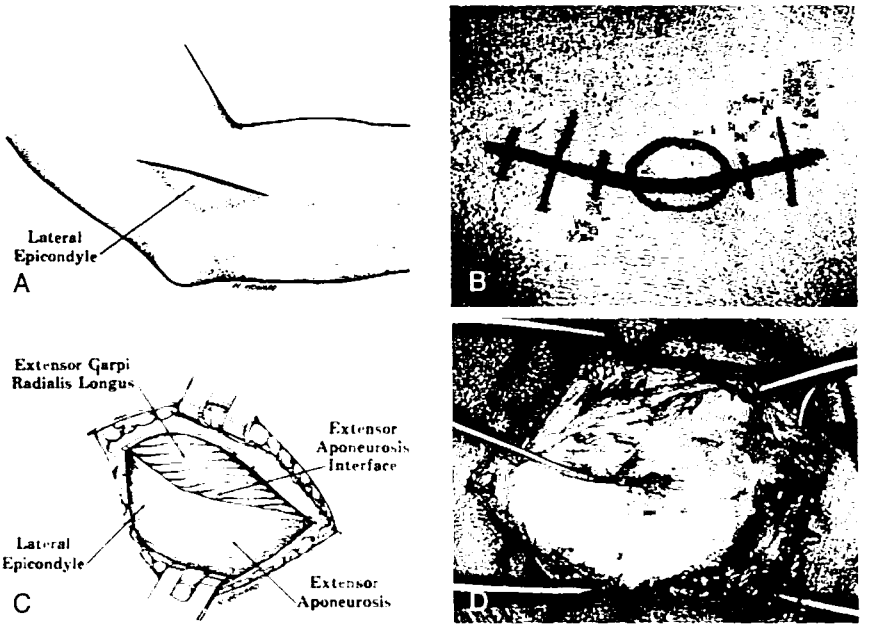

Figure 9. *A*, Curved incision drawn out approximately 8 cm in length over the lateral epicondyle. *B*, Clinical photograph demonstrating incision pictured in *A*. *C*, Incision made in the interface between the ECRL and the common extensor aponeurosis. *D*, Photograph demonstrating actual clinical dissection.

Illustration continued on following page

involve inadvertent release of the ECRL, which, in turn, may increase postoperative weakness.

The intra-articular procedures have been reported extensively by Bosworth.[5] The most popularized procedure by Bosworth involved release of the annular ligament in varying degrees with a high success rate. (This procedure did include debridement of the ECRB origin, which may have been the most important factor in pain relief seen in patients.)

Garden's procedure, first described in 1961, implicated the ECRB as the pathologic offender in this condition and proposed reducing tension by a disease Z-plasty lengthening.[15] Initial reports by Garden of success rates up to 100% have not been duplicated, and Carroll has proclaimed that only one in five patients will benefit from this operation.[8, 12]

The most popular surgical treatment involves the Nirschl extra-articular technique, in which the pathologic portion of the ECRB tendon origin is excised with the defect repaired and resutured back to the epicondylar site of origin.[31, 35] The patient is placed in a supine position, a sterile tourniquet is used, and adequate regional anesthesia is administered. A curved incision of approximately 8 cm in length is made proximally to distally to the lateral epicondyle in a slightly anterior fashion (Fig. 9*A* and *B*). An incision is made in the interval between the

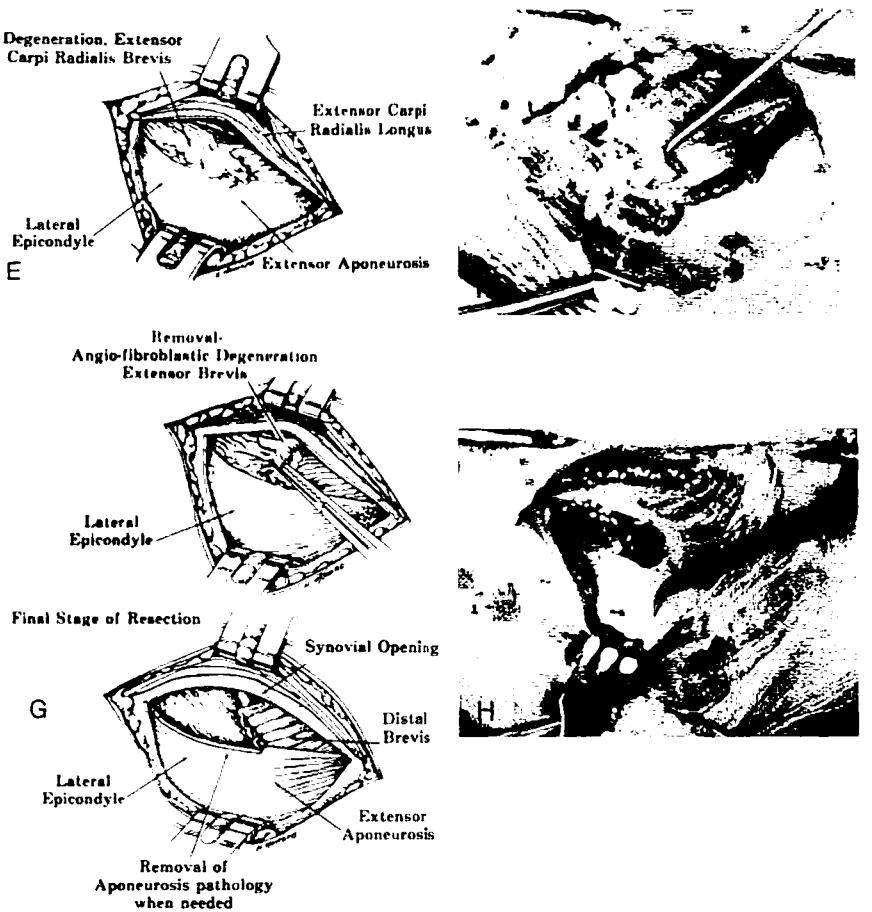

Figure 9 *(Continued). E*, ECRL is retracted anteriorly, exposing the origin of the ECRB. *F*, Clinical photograph demonstrating the origin of the ECRB with anatomic probe in place. *G*, Angiofibroblastic response in the ECRB identified and excised. *H*, Clinical photograph with anatomic probe pointing to the angiofibroblastic response at the origin of the ECRB.

Illustration continued on opposite page

ECRL and the common extensor aponeurosis (Fig. 9C and *D*). The ECRL is retracted anteriorly, exposing the origin of the ECRB (Fig. 9*E* and *F*). The typical angiofibroblastic response is identified and excised (Fig. 9*G* and *H*). In up to one third of cases, some changes may be seen in the anterior edge of the extensor aponeurosis, which should be resected along with any epicondylar exophytic reaction (seen in 25% of the cases).

Decortication of the lateral condylar area is performed with an appropriate drill to enhance vascular supply (Fig. 9*I*). Some surgeons prefer to perform a lateral epicondylectomy (Fig. 10). Finally, the ECRL is repaired to the anterior margin of the extensor aponeurosis, which

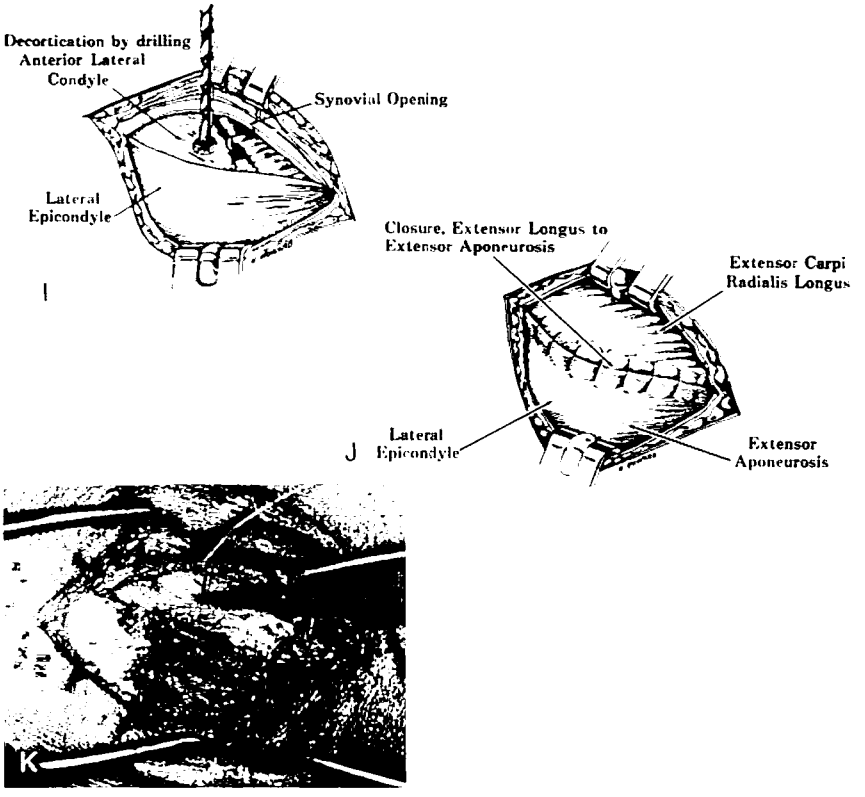

Figure 9 *(Continued). I,* Decortication of the lateral epicondylar area with a drill. *J,* Repair of the ECRL to the anterior margin of the extensor aponeurosis. *K,* Clinical photograph demonstrating surgical repair. (*Parts A, C, E, G, I, and J* from Nirschl RP: Elbow tendinosis/ tennis elbow. Clin Sports Med 11:851–870, 1992; *Parts B, D, and K* courtesy of Kevin D. Plancher, MD, MS.)

will allow the ECRB (still attached to the ECRL) to be repaired in turn (Fig. 9*J* and *K*). Use of commercial bone screws or anchors may allow better approximation of this tendinous repair to bone when needed. The subcutaneous tissues and skin are closed, and a long-arm posterior splint is applied for 7 to 10 days, at which time the patient should begin protective range-of-motion exercises and intermittent splinting.

Strengthening exercises are begun at 4 weeks with progression by 6 weeks and return to activities without restrictions at 3 months. Most studies indicate that this surgical treatment, when performed properly, yields an 85% to 90% success rate with relief of symptoms and return to activities. In addition, 10% to 12% in Nirschl's and others' series have shown improvement in symptoms, but still experience discomfort with heavy activities.[31, 35] Unfortunately, 2% to 3% of patients have shown no improvement but, fortunately, no worsening symptoms with surgery.

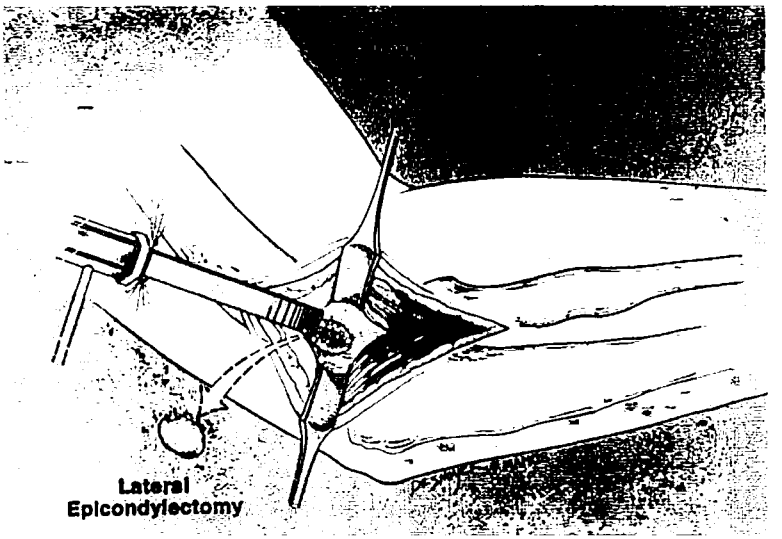

Figure 10. Osteotome used to perform lateral epicondylectomy of the elbow. (Courtesy of The Indiana Hand Center, Indianapolis, Indiana.)

Jobe and coworkers[24] have reviewed their experience, which again parallels the current consensus that 95% of patients diagnosed with lateral epicondylitis were treated successfully with nonsurgical treatment. The 5% who were unresponsive to nonsurgical treatment underwent repair with a 94% improvement in symptoms subjectively. Objectively, however, up to 36% had limitations with heavy lifting, and 15% showed weakness in grip strength with the entire series showing differing degrees of deficits in isokinetic testing. These authors conclude that a subjectively excellent result does not have to be associated with accompanying objective findings but do caution that these residual deficits in symptoms still paint a picture of concern with the current management of lateral epicondylitis.

Complications

Complications exist both in the nonsurgical and surgical treatment of epicondylitis. One must first make the correct diagnosis, which involves ruling out the differential diagnoses with appropriate radiographs and other diagnostic studies as discussed earlier. Complications associated with the use of nonsteroidal anti-inflammatory medications must be assessed along with the associated problems with corticosteroid injections. Surgical treatment as stated can result in marked weakness in forearm and wrist extensors along with synovial fistula and ganglion

formation.[3] Overzealous lateral epicondylar release involving the lateral collateral ligament can result in instability.[3]

Failure to release the posterior interosseous nerve, if involved, may result in persistent discomfort. Morrey has looked at the reoperation for failed surgical treatment of refractory lateral epicondylitis and has classified these patients into two groups.[30] The first group presented postoperatively with symptoms similar to those they experienced preoperatively, whereas the second group presented postoperatively with a new set of symptoms.

In the first group, there were failures because of inadequate release of the extensor musculotendinous unit or an incorrect preoperative diagnosis. Entrapment of the posterior interosseous nerve was found commonly at the time of reoperation.

In the second group, the different symptoms postoperatively were most often due to ligamentous insufficiency caused by the treatment itself. Morrey recommends that once the cause of failure has been ascertained, a treatment plan must be initiated. If the patient falls into the first group, the patient should be re-evaluated for up to a year, and if they are without improvement, re-exploration should be undertaken. If the patient falls into the second group, an appropriate diagnosis has to be made and a different surgical plan should be addressed for the patient's newly diagnosed problem.

SUMMARY

An appropriate diagnosis must be made after carefully excluding all other options in treating the patient with lateral epicondylitis. The majority of these patients will do well with nonsurgical treatment; however, if unresponsive to this regimen, a carefully selected patient will have a successful result with surgical reconstruction (85%–95% of patients). Attention to detail both pre- and postoperatively will help to create a successful result in a disease entity that plagues a large proportion of our athletic population.

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