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Diagnosis and treatment of medial epicondylitis of the elbow

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Epicondylitis is one of the most common elbow problems in adults, occurring both laterally and medially. Medial epicondylitis of the elbow, commonly referred to as ''golfer's elbow,'' is characterized by pathologic changes to the musculotendonous origin at the medial epicondyle. Accurate diagnosis is dependent upon a complete understanding of the anatomic, epidemiologic, and pathophysiologic factors that distinguish epicondylitis from other elbow conditions.

Originally described by Henry Morris in 1882 as ''lawn-tennis elbow,'' epicondylitis has since been studied and written on extensively [1]. Lateral epicondylitis, often labeled ''tennis elbow,'' has received the majority of this attention. There exists, however, a paucity of literature regarding medial epicondylitis, likely due to its infrequent incidence of only 9.8% to 20% of all epicondylitis diagnoses [2–4]. Originally thought to be an inflammatory process, as the name suggests, epicondylitis has been shown histologically to result from tendonous microtearing, followed by an incomplete reparative response. Consequently, some physicians prefer the more accurate term, tendonosis, when describing elbow epicondylitis.

Epidemiology and etiology

Medial epicondylitis occurs much less frequently than lateral epicondylitis, which has been diagnosed seven to ten times more often [5]. Although the syndrome has been identified in patients ranging from 12 to 80 years old, it predominantly occurs in the fourth and fifth decades. Male and female prevalence

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rates are reportedly equal. Seventy-five percent of patients are symptomatic in their dominant arms.

The majority of the literature on epicondylitis suggests that the disorder's primary etiology is a repetitive stress or overuse of the flexor-pronator musculature [5–8]. Degenerative changes in the musculotendonous region of the medial epicondyle are the result of chronic repetitive concentric and eccentric contractile loading of the flexor-pronator group. Most often such changes are seen in the pronator teres and the flexor carpi radialis muscles, although larger diffuse tears can occur in the palmaris longus [5], flexor digitorum superficialis [9], and flexor carpi ulnaris. Although repetitive overuse has been identified as the primary etiology, a single traumatic event, such as a direct blow or a sudden, extreme eccentric contraction, may result in the development of epicondylitis. Medial epicondylitis has been associated with activities involving repetitive forearm pronation and wrist flexion. Although it occurs frequently in baseball pitchers, resulting from intense valgus forces on the medial elbow during the late cocking and acceleration phases of throwing [5,10], medial epicondylitis has also been related to golf, tennis, bowling, racquetball, football [11], archery, weightlifting [12], and javelin throwing [13]. This disorder, however, is not solely athletic in origin, because it is also associated with occupations such as carpentry, plumbing, and meat cutting, all of which require repetitive forearm, wrist, and hand motions [12].

Biomechanics

The biomechanics of the medial elbow and the flexor-pronator musculature are most often described in terms of the baseball pitching mechanism. The acceleration phase of pitching—from the point at which the ball has virtually no forward velocity to the point of release—may produce peak angular velocity and extreme valgus forces greater than the tensile strength of the medial ligamentous and musculotendonous structures. The forces are initially transmitted to the flexor-pronator muscle group and then to the deeper medial collateral ligament. Such repetitive activity may lead to the degenerative changes initiating epicondylitis. Similarly, in tennis, the acceleration phase of the tennis serve has been shown through electromyography by Morris et al to result in the highest muscular activity in the flexor-pronator group, primarily the pronator teres [14]. Morris et al suggested that during acceleration, the flexor pronator group provides the optimal positioning of the forearm while transferring maximum momentum and power to the ball. Hence forehands, serves, and overheads in tennis can result in the medial tension overload, which initiates epicondylitis. In golf, medial epicondylitis is attributed to incorrect technique, in which the club is ''thrown'' from the apex of the backswing down toward the ball. Appropriately termed ''hitting from the top,'' this incorrect technique creates similar valgus forces upon the medial epicondyle of the dominant arm, leading to the same tension overload pattern [15,16].

Anatomy

The flexor-pronator group comprises the musculotendonous structures of the medial elbow. From the radial to the ulnar aspects of the forearm, the musculature includes the pronator teres, the flexor carpi radialis, the palmaris longus, the flexor digitorum superficialis, and the flexor carpi ulnaris. The pronator teres and flexor carpi radialis both attach to the anterior aspect of the medial epicondyle. These tendons are stretched during the acceleration phases of throwing and swinging. Thus, the pronator teres and the flexor carpi radialis are most often the muscles afflicted with these alterations. Professional throwing athletes are often noted to sustain hypertrophy of the humerus and flexor-pronator forearm muscles, 50% of which have a flexion contracture and 30% of which have an increased valgus angle in comparison with the nondominant arm [11,17,18]. Though these changes have been identified in this particular athlete group, they have never been directly correlated with the occurrence of medial epicondylitis.

The close proximity of the ulnar nerve to the medial epicondyle can result in concomitant pathology. When stretched, the nerve can sustain direct injury with an inflammatory response. This can lead to neuritis as well as entrapment or compression. The ulnar nerve passes through the medial intermuscular septum into a groove on the medial head of the triceps. Here the nerve passes through the arcade of Struthers, a fascial layer extending from the medial head of the triceps to the medial intermuscular septum, which is present in 70% of the population. The nerve then descends posteriorly to the medial epicondyle to pass through the fibro-osseous cubital tunnel. This cubital tunnel is bound by the medial epicondyle anteriorly, the ulnar collateral ligament laterally, and the fibrous arcade formed by the two heads of the flexor carpi ulnaris posteromedially. For simplicity, Nirschl divided this medial epicondylar groove into three main zones, the first of which is proximal to the medial epicondyle [19]. The second is at the medial epicondyle, and the third zone is distal to the medial epicondyle. In patients with symptomatic concomitant ulnar nerve compression undergoing surgical treatment for medial epicondylitis, Nirschl has identified zone 3 to be the most common site for nerve compression, as the ulnar nerve enters the flexor carpi ulnaris arcade [19].

When evaluating medial elbow pain, one must also consider medial elbow instability. The stability of the elbow is provided primarily by the congruous bony articulation between the olecranon of the ulna and the trochlea of the humerus. Soft-tissue structural stability is provided primarily by the medial collateral ligament, but also to a lesser extent by the anterior capsule, lateral collateral ligament, and the flexor and extensor muscles masses. Cadaveric studies by Morrey and An have established that the medial collateral ligament is composed of three bands: anterior, posterior and transverse [20]. It is the anterior band of this ligament that stabilizes the elbow during valgus stress. Muscle strain can be caused by poor mechanics, improper conditioning, lack of flexibility, or fatigue—all of which may lead to increased transmission of forces to the medial collateral ligament. Repetitive and excessive stress upon the medial collateral ligament can

lead to microscopic damage, and ultimately to ligamentous insufficiency and elbow instability.

Pathophysiology

Since Morris' first description of epicondylitis in 1882, a vast amount of literature has been dedicated to the pathophysiology of this disorder. Early descriptions postulated an inflammatory process involving the radial humeral bursa, periosteum, synovium, and annular ligament [21–24]. These theories, however, have recently been discounted by the histologic analysis of Nirschl and Pettrone [6], and Regan et al [25]. Their studies revealed that the normal collagen architecture is disrupted by a fibroblastic and immature vascular response, an incomplete reparative process, and a notable paucity of acute and chronic inflammatory cells. In its earliest stages, epicondylitis may display inflammatory or synovitic characteristics; its later stages demonstrate evidence of microtearing, characterized by tendon degeneration, with or without calcification, and an aborted, incomplete neurovascular response. Grossly, the pathologic tissue appears gray and friable. These structural alterations have been termed "angiofibroblastic hyperplasia" by Nirschl and Pettrone and can occur medially or laterally [6]. Nirschl went on to propose four descriptive stages of epicondylar tendonosis [26]. Stage 1 exhibits generalized inflammation, which may recede. Stage 2 injury is characterized by the pathologic tissue alterations of angiofibroblastic degeneration. Structural failure is the hallmark of stage 3. Stage 4 injury includes the components of stages 2 or 3, but is also accompanied by fibrosis or calcification. The usage of exact terminology in the description of epicondylitis is essential in understanding the nature of the disorder and establishing goals of therapeutic intervention. Although the precise universal pathophysiology of epicondylitis has yet to be established, it is generally accepted that the injury results from microtearing of the tendon origin at the epicondyle. This progresses to a failed reparative response and subsequent tendon degeneration that ultimately alters the typical musculotendonous biomechanics of the elbow.

Diagnosis

The diagnosis of medial epicondylitis requires a careful patient history and physical examination, and radiographic and imaging studies, to distinguish it from other possible etiologies of medial elbow discomfort, such as ulnar collateral ligament instability or ulnar neuritis.

Medial epicondylitis is characterized by pain of insidious onset along the medial elbow, which is worsened by resistance to forearm pronation and wrist flexion. Tenderness to palpation usually occurs over the pronator teres and the flexor carpi radialis, and maximally at 5 mm to 10 mm distal and anterior to the

midpoint of the medial epicondyle. The severity of pain may vary, but is most often present and acute during the offending activity. Local swelling and warmth may also exist [9]. Initially the range of motion of the afflicted extremity can be full, but over time it may become limited and lead to a flexion contracture, which is commonly noted in the throwing athlete [27,28]. King et al observed 50% of professional baseball pitchers to have a flexion contracture in their elbow [18].

When examining for medial epicondylitis, it is also essential to consider ulnar neuritis and ulnar collateral ligament instability, especially in the overhead athlete. These may coexist, often consequent to the excessive valgus forces imparted to the medial elbow with throwing. Ulnar neuritis is identified by a positive Tinel's sign, as indicated by local pain and numbness or tingling radiating distally with the direct compression of the ulnar nerve at the elbow. A Tinel's sign in zone 1 may indicate congenital ulnar nerve subluxation; in zone 2, a Tinel's sign may be due to compression caused by osteophytes, loose bodies, or rheumatoid synovitic changes. A zone 3 Tinel's sign implies compression as the ulnar nerve passes through the two heads of the flexor carpi ulnaris [29]. The elbow flexion test for ulnar neuritis is performed by placing the elbow in maximum flexion, the forearm in pronation, and the wrist in extension for approximately 30 to 60 seconds. If neuritis is present, the patient will experience medial elbow pain, as well as numbness or tingling in the ring and little fingers. Ulnar collateral ligamentous instability is best identified by applying a 30° valgus stress test, or by the milking test, which is performed by pulling on the thumb with the elbow flexed and the forearm supinated. Both of these elicit focal pain along the ulnar collateral ligament.

The radiographs of affected elbows are generally normal, although 20% to 25% of patients can have soft-tissue calcification in proximity to the epicondyle [27]. Throwing athletes may have medial ulnar traction spurs and medial collateral ligament calcification [28]. Electromyography (EMG) is indicated in patients with neurologic alterations. Laboratory studies can be helpful in patients with suspected rheumatoid disorders. An MRI or dynamic ultrasonography may be useful in the diagnosis of throwing athletes with confounding medial symptoms by providing more precise evaluation of the ulnar collateral ligament [30]. MRI and ultrasonography also aid in determining traumatic tears to the flexor pronator origin at the epicondyle.

Nonsurgical treatment

Nonsurgical treatment is the cornerstone of care for both medial and lateral epicondylitis. The objective of such conservative care is to relieve pain and reduce inflammation, allowing sufficient rehabilitation and return to activities. Although this treatment has been described as highly successful, there remains a lack of information concerning the long-term outcome of nonsurgical treatment. The available literature suggests that 5% to 15% of patients suffer recurring symptoms, but the majority of these relapses are due to incomplete rehabilitation

or premature discontinuation of the suggested preventative measures [28]. In a prospective review of the nonoperative treatment, Binder and Hazleman saw 26% of patients experience a recurrence of symptoms, and over 40% had prolonged minor discomfort [31]. Thus, the previously reported success rates of 85% to 90% for nonsurgical treatment may be overly optimistic, with symptoms recurring more often than thought. Nonetheless, nonsurgical treatment, which can be divided into three phases, remains the mainstay of treatment for medial epicondylitis.

Phase I

Phase I begins with the immediate, temporary cessation of offending activities. Complete immobilization or inactivity is not recommended; this is to avoid muscular atrophy, which can hinder rehabilitation efforts. The affected elbow is iced for 15 to 20 minutes, three to four times per day. This is recommended for its local vasoconstrictive and analgesic effects. Oral nonsteroidal anti-inflammatory medication may be administered for a 10- to 14-day course, provided the patient has no medical contraindications. Patients with improved but lingering symptoms may benefit from a second course of medication after a brief period of abstinence. Because epicondylitis is believed to be a degenerative process, the benefits of anti-inflammatory medications are thought to stem from their ability to relieve pain associated with the accompanying synovitis.

If the patient does not respond to these measures, a period of night splinting is appropriate, with local corticosteroid injection around the affected tendon insertion. As with the anti-inflammatory medication, the corticosteroid aids in relieving the accompanying synovitis. The choice and dose of the corticosteroid injection are at the discretion of the physician, because no carefully controlled prospective study comparing the different agents exists in the current literature [32]. The appropriate injection technique for administering the corticosteroid requires instilling the agent into the fatty subaponeurotic recess deep to the flexor pronator mass. Care must be taken to avoid injecting the mixture into superficial tissues, which may cause subcutaneous atrophy, or into the tendon, which may result in irreversible ultrastructural tendon alterations. Patients with more darkly pigmented skin ought to be warned about the risk of depigmentation after local corticosteroid injection. The short-term efficacy of such corticosteroid injections has been documented in several prospective, randomized studies [33,34]. Stahl and Kaufman noted a significant decrease in pain at 6 weeks after the injection; however, there was no difference from preinjection pain and pain at 3 months and 1 year [33]. Price et al identified pain relief in 55% to 89% of patients; however of those who initially experienced relief, 18% to 54% suffered a recurrence of symptoms [32].

Ultrasound and high-voltage galvanic stimulation, among other physical therapy modalities, have also been suggested as relieving the pain of epicondylitis. Though literature promoting the success of such modalities exists, no prospective, randomized studies have been conducted to determine their efficacy.

Krishek et al demonstrated only a 28% success rate of shock-wave therapy at 1 year [35]. These modalities are often recommended at the initiation of a nonsurgical program; however, if pain relief is not achieved soon after initiation, they should be discontinued. Topical medications, such as dimethylsulfoxide, provided no greater relief than placebos in a randomized, double-blind, prospective study [36].

Counterforce bracing can aid athletes with symptoms in daily life during the first phase of nonsurgical treatment. Additionally, it can be helpful in returning the athlete to sport. By limiting full contractile expansion of the musculotendonous unit, a counterforce brace decreases intrinsic muscle force; however, it must be noted that compression of the anterior interosseous nerve and symptoms of posterior interosseous nerve entrapment have been reported with counterforce bracing [37,38]. Thus, it is essential to instruct the athlete on the proper application of the brace. Should symptoms of epicondylitis fail to improve, counterforce bracing should be discontinued.

Phase II

As soon as symptoms are improved by Phase I treatment, a guided rehabilitation program should be initiated. Establishing full, painless, wrist and elbow range of motion is the first goal, soon followed by stretching and progressive isometric exercises. Initially the elbow should be flexed during these exercises to minimize pain, but as the patient progresses, greater elbow extension should be continuously achieved. As preinjury flexibility and strength return, concentric and eccentric resistive exercises are added to the Phase II program. The achievement of greater-than-preinjury strength is the ultimate goal, because preinjury muscle strength proved vulnerable to tension overload [19]. As soon as a patient can perform sprint repetitions to fatigue without significant discomfort, a sports or occupational simulation is staged. If satisfactorily completed, the patient is reinitiated into the sport or occupation with a gradual increase of exposure and intensity. The importance of a maintenance exercise program for the shoulder and elbow musculature to maintain optimal strength and flexibility is especially stressed to the athlete.

Phase III

When the athlete returns to sport, it is critical that the athlete, coaches, and trainers identify any inadequacies in equipment or technique that may contribute to a recurrence of symptoms. Equipment properly sized to the athlete is essential, especially in racquet sports, to prevent subsequent bouts of epicondylitis. Correct grip size is calculated by measuring from the proximal palm crease to the tip of the ring finger, along its radial border. Lighter graphite frames, racquets less tightly strung, racquets with higher string count per unit area, and playing on slower surfaces will all minimize vibration transmission to the medial elbow [5,39]. A larger racquet head provides a larger area to hit the ball centrally on

the strings, therefore preventing off-center contact, which is a source of valgus stress to the elbow. In golf, clubs of proper weight, length, and grip are similarly important and can significantly reduce the injurious forces generated within the elbow. Examination of technique to identify faults allows a safe return, reducing the risk of recurrence. Ilfield showed that patients with medial epicondylitis who regularly played tennis often exhibited an improper serve and forehand stroke [40]. These incorrect strokes included hitting the ball late, hitting the ball with the head of the racquet behind the elbow as contact is made, and hitting the ball off-center on the racquet, all of which contribute to elbow problems. Continued conditioning of the entire body along with the affected extremity is vital. Conditioning, including flexibility, strength, and endurance, is best performed with a slow, structured interval program, monitored by coach, trainer, and physician to ensure a successful return without symptom recurrence.

Surgical treatment

If a patient fails to respond to a disciplined, 3- to 6-month nonoperative program and all other possible pathologic causes for the pain of epicondylitis have been excluded, surgical treatment is recommended. In elite throwing athletes, operative treatment can be undertaken sooner if physical examination and imaging studies indicate tendon disruption. At this stage, nonoperative treatment will most likely prove insufficient to return the high-level athlete to peak performance.

Historical treatment

The history of surgical management of epicondylitis spans nearly three quarters of a century. Although much literature discusses operative treatment of lateral epicondylitis, little exists concerning similar treatment of medial epicondylitis. Various procedures ranging from percutaneous epicondylar release to epicondylectomy have been used, without a single widely accepted technique [28]. Currently, however, the standard surgical treatment consists of: (1) excision of the pathologic portion of the tendon, (2) enhancement of local vascularity to stimulate a healing response, (3) firm reattachment of any elevated tendon origin back to the epicondyle, (4) repair of the resultant defect, and (5) management of any concurrent ulnar nerve or ulnar collateral ligament pathology.

Surgical technique

With the patient lying supine, a tourniquet is applied and the arm is placed on an arm board. A 5-cm to 7-cm oblique incision is made just anterior to the medial epicondyle. The surgeon must take care to identify the medial antebrachial cutaneous nerve during the approach through the subcutaneous tissue. Proximal extension of the incision may be necessary when considering anterior transpo-

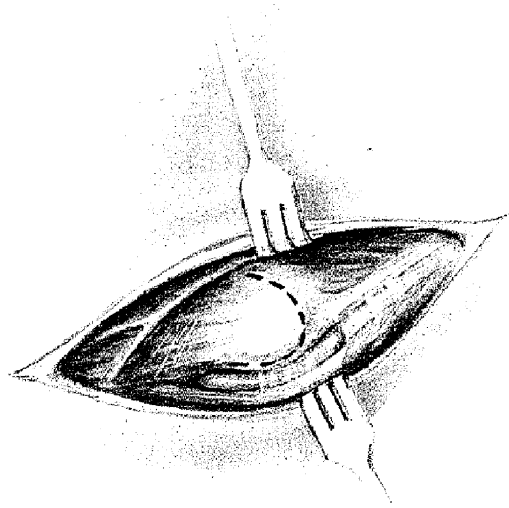

Fig. 1. Skin incision overlying the medial epicondyle with exposed cutaneous and ulnar nerves; intended incision of common flexor-pronator mass. (*From* Ciccotti MG, Lombardo SJ. Lateral and medial epicondylitis of the elbow. In: Jobe FW, Pin MM, Glousman RE, editors. Operative techniques in upper extremity sports injuries. St. Louis (MO): Mosby-Year Book; 1996. p. 443; with permission.)

sition of the ulnar nerve. The common flexor-pronator origin is identified, along with the ulnar nerve, which is protected within the ulnar groove. Depending upon the location and nature of the pathologic tissue, an incision of the pronator teres-flexor carpi radialis interval is developed either longitudinally or transversely (Fig. 1). If the pathology is focal, a longitudinal split and excision of the abnormal tissue is performed. If the pathology is diffuse, which is less commonly reported, a transverse incision is performed and the abnormal tissue is completely excised (Fig. 2). Deep to the flexor-pronator mass, the ulnar collateral ligament must also be evaluated for possible concurrent pathology. Surgical management of the ulnar nerve or ulnar collateral ligament is performed when necessary. Preparation of the medial epicondyle for reattachment of the common flexor-pronator origin includes rongeuring any fibrous tissue and drilling multiple small holes in the surface to create a sufficient vascular bed. The flexor-pronator origin is then reattached to the bleeding surface with interrupted sutures, either through drill holes or through the attached adjacent flexor-pronator origin (Fig. 3). Finally, routine subcutaneous and skin closures are performed. A posterior plaster splint is applied to the elbow and the wrist, with the elbow at 90° of flexion and the forearm in neutral rotation.

Though arthroscopic debridement has been proposed for the surgical management of lateral epicondylitis, the close proximity of both the ulnar nerve and the ulnar collateral ligament make such risks prohibitive for medial epicondylitis.

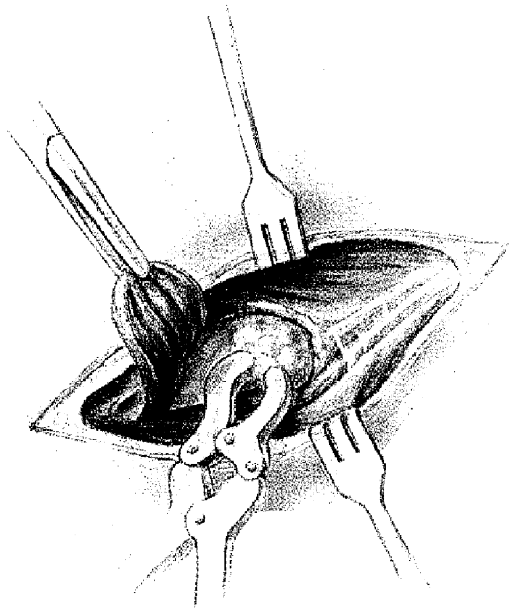

Fig. 2. Distal reflection of the common flexor-pronator origin with excision of pathologic tissue. (*From* Ciccotti MG, Lombardo SJ. Lateral and medial epicondylitis of the elbow. In: Jobe FW, Pin MM, Glousman RE, editors. Operative techniques in upper extremity sports injuries. St. Louis (MO): Mosby-Year Book; 1996. p. 444; with permission.)

Postoperative management

At 7 to 10 days postoperatively, the splint and skin sutures are removed. At this point, gentle passive and active hand, wrist, and elbow exercises are begun. Gentle isometrics are undertaken at 3 to 4 weeks postoperatively, with more rigorous, resistive exercise, including wrist flexion and forearm pronation, beginning at 6 weeks. A progressive strengthening program follows. Generally, a patient will return to activities by 3 to 6 months postoperatively.

Surgical results

Vangsness and Jobe reviewed 35 patients with recalcitrant medial epicondylitis who underwent surgical treatment with a standard technique employing the aforementioned surgical principles [11]. They reported 88% good to excellent results after an average follow-up of 6 years. Fourteen percent required ulnar nerve submuscular transposition; however, none of these patients suffered from persistent symptoms. Nineteen of twenty athletes returned to their previous level of sport. Fourteen percent of patients noted some loss of endurance or experienced limitations with heavy lifting. Isokinetic and grip strength testing demonstrated no significant side-to-side differences. Gabel and Morrey reviewed their

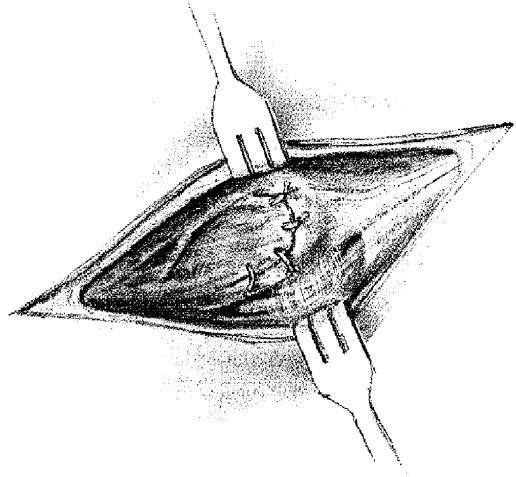

Fig. 3. Reattachment of the common flexor-pronator origin to the medial epicondyle. (*From* Ciccotti MG, Lombardo SJ. Lateral and medial epicondylitis of the elbow. In: Jobe FW, Pin MM, Glousman RE, editors. Operative techniques in upper extremity sports injuries. St. Louis (MO): Mosby-Year Book; 1996. p. 444; with permission.)

results in 26 patients (30 elbows) using several different surgical techniques to manage medial epicondylitis, including partial and complete elevation of the flexor pronator mass, with or without complete reattachment [41]. They reported 87% good to excellent results after an average follow-up of 7 years, and 96% good to excellent results in patients with absent to mild ulnar nerve symptoms. Their surgical management of ulnar neuropathy also employed various techniques, dependent on preoperative symptoms and intraoperative findings. More than 50% of patients with preoperative ulnar nerve symptoms had a less favorable outcome. Wittenberg et al noted good to excellent results in 14 of 17 patients with medial epicondylitis [42]. Baumgard and Schwartz reported 83% excellent results in a small series of 6 patients, having used a technique of percutaneous release of the common flexor-pronator origin [2]. Despite the limited literature on medial epicondylitis, surgical treatment appears to generally result in reliable pain relief and high patient satisfaction. Some residual objective strength deficits in the involved extremity may occur, but these do not seem to interfere with functional activities.

Summary

Although limited literature exists on medial epicondylitis of the elbow, this disorder is an injury affecting many professionals and athletes at every level, especially throwing athletes. Care must be taken in diagnosing medial epicondylitis to distinguish it from other possible pathologies of the medial elbow, which

may exist concurrently. The large majority of patients diagnosed with medial epicondylitis will respond to a well-structured, nonsurgical program; however, patients with persistent or recurring symptoms can be treated surgically, which yields high patient satisfaction and ultimately a reliable return to preinjury levels of activity.

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