Jennifer Moriatis Wolf
Editor

Tennis Elbow

Clinical Management

Springer

Tennis Elbow

Jennifer Moriatis Wolf
Editor

Tennis Elbow

Clinical Management

Springer

Editor
Jennifer Moriatis Wolf
Department of Orthopedic Surgery
University of Connecticut Health Center
Farmington, CT, USA

ISBN 978-1-4899-7533-1 ISBN 978-1-4899-7534-8 (eBook)
DOI 10.1007/978-1-4899-7534-8

Library of Congress Control Number: 2014954810

Springer New York Heidelberg Dordrecht London

Printed on acid-free paper

Springer is part of Springer Science+Business Media (www.springer.com)

Contents

Contributors

Shrikant J. Chinchalker, M.Th.O, B.Sc. OT, OTR, CHT Hand Therapy Department, Roth-McFarlane Hand and Upper Limb Center, St. Joseph's Health Care, London, ON, Canada

Carlton M. Clinkscales, MD Department of Orthopaedics, Littleton Hospital, Centennial, CO, USA

Mark S. Cohen, MD Director, Hand and Elbow Section, Director, Orthopaedic Education, Department of Orthopedic Surgery, Rush University Medical Center, Chicago, IL, USA

Roger A. Daley, MD, PhD Department of Orthopaedic Surgery, Medical College of Wisconsin— Froedtert Memorial Lutheran Hospital, Milwaukee, WI, USA

A. Lee Dellon, MD Johns Hopkins University, Towson, MD, USA

Jonathan F. Dickens, MD Orthopaedic Sports Medicine, John A. Feagin, Jr. Sports Medicine Fellowship, Keller Army Hospital, West Point, NY, USA

Torkell Ellingsen, MD Department of Rheumatology, Odense University Hospital, Odense, Denmark

Ulrich Fredberg, MD Department of Rheumatology, Silkeborg Regional Hospital, Silkeborg, Denmark

Ruby Grewal, MD, MSc, FRCSC Division of Orthopedics, Department of Surgery, Hand and Upper Limb Center, St. Joseph's Health Center, London, ON, Canada

Barbara Haines, OTR, CHT Rehab: Hand/UE, Froedtert /Medical College of WI, Milwaukee, WI, USA

Igor Immerman, MD Department of Orthopaedic Surgery, UC Davis Medical Center, Sacramento, CA, USA

Christopher Judson, MD Department of Orthopaedic Surgery, University of Connecticut Health Center, Farmington, CT, USA

David M. Kalainov, MD Department of Orthopaedic Surgery, Chicago, IL, USA

Jay V. Kalawadia, MD Department of Orthopaedic Surgery, Northwestern University, Chicago, IL, USA

David S. Kaminski HBSc, BScOT Division of Hand Therapy, Hand and Upper Limb Center, St. Joseph's Health Care, London, ON, Canada

Thøger Persson Krogh, MD Department of Rheumatology, Silkeborg Regional Hospital, Silkeborg, Denmark

Brendan D. Masini, MD Department of Orthopaedic Sports Medicine, John A. Feagin, Jr. Sports Medicine Fellowship, Keller Army Hospital, West Point, NY, USA

Augustus D. Mazzocca, MD, MS Department of Orthopaedic Surgery, University of Connecticut Health Center, Farmington, CT, USA

David W. Meister, MD Department of Orthopaedic Surgery, Medical College of Wisconsin, Milwaukee, WI, USA

Robert P. Nirschl, M.D., M.S. Director Virginia Sportsmedicine Institute, Nirschl Orthopaedic Center, Arlington, Virginia

Brett D. Owens, MD Orthopaedic Surgery, John A. Feagin, Jr. Sports Medicine Fellowship, Keller Army Hospital, West Point, NY, USA

Joey G. Pipicelli, MScOT Division of Hand Therapy, Hand and Upper Limb Center, St. Joseph's Health Care, London, ON, Canada

David Ring, MD, PhD Department of Orthopaedic Surgery, Massachusetts General Hospital, Boston, MA, USA

Rachel S. Rohde, MD Department of Orthopaedic Surgery, William Beaumont School of Medicine, Oakland University, Michigan Orthopaedic Institute, Southfield, MI, USA

Robert M. Szabo MD, MPH Department of Orthopaedic Surgery, UC Davis Medical Center, Sacramento, CA, USA

Brian A. Tinsley Department of Orthopaedic Surgery, University of Connecticut Health Center, Farmington, CT, USA

Jonathan Winston, MD Department of Orthopaedic Surgery, University of Connecticut Health Center, Farmington, CT, USA

Jennifer Moriatis Wolf, MD Department of Orthopaedic Surgery, University of Connecticut Health Center, Farmington, CT, USA

Chapter 1
Tennis Elbow: Definition, Causes, Epidemiology

Jonathan Winston and Jennifer Moriatis Wolf

Introduction

The diagnosis of lateral epicondylitis was first made by Runge in 1873, in which the author described lateral humeral condylar tenderness and difficulty in writing [28]. In 1882, Morris coined the term "lawn tennis elbow" as he found the condition was associated with the tennis backhand stroke [17]. Over time, this entity became known as "tennis elbow" or lateral epicondylitis. It is important to realize that the term "tennis elbow" is a misnomer as golfers, baseball players, clothing pressers, salesmen carrying grips, violinists, blacksmiths, telephone operators, and homemakers are all susceptible to this condition. Patients afflicted with this condition typically experience pain at the origin of the extensor muscle, pain with resisted wrist extension, and tenderness with palpation of the tendinous origin of the muscles at the lateral humeral epicondyle. The condition can be very difficult to treat.

Definition

The elbow is a trocho-ginglymus joint with articulations between the humerus, ulna, and radius [25]. A series of musculotendinous units cross the elbow joint and function to position the arm and hand in space. The lateral epicondyle of the humerus has been described as a pyramid-shaped bony prominence from which the anco-

J. M. Wolf (✉) · J. Winston
Department of Orthopaedic Surgery, University of Connecticut Health Center, 263 Farmington Avenuen, MARB4-ORTHO, Farmington, CT, USA
e-mail: jmwolf@uchc.edu

J. M. Wolf (ed.), *Tennis Elbow,* DOI 10.1007/978-1-4899-7534-8_1

neus, extensor carpi radialis brevis (ECRB), extensor digitorum communis (EDC), and extensor carpi radialis longus (ECRL) originate [7]. Among these, the ECRB has been implicated most often in lateral epicondylitis, with additional involvement of the EDC 35–50 % of the time [26, 27]. The ECRB originates from the anterior face of the lateral epicondyle, is located deep to the other extensors, and is characteristically tendinous at this location.

The term "epicondylitis" falsely implies an inflammatory reaction. Excised ECRB tendon in patients with lateral epicondylitis has shown the normal tissue of ECRB invaded by immature fibroblasts and nonfunctional vascular buds, with disorganized surrounding and hypercellular tissue. This finding led Nirschl et al. to coin the term "angiofibroblastic tendinosis" [11, 27]. Despite the absence of inflammation, patients with lateral epicondylitis complain of pain, particularly during activities requiring wrist extension. Elevated levels of substance-P, calcitonin gene-related peptide, and glutamate have been found within the ECRB tendon in patients with chronic tennis elbow, thus offering another etiology for pain [2, 23].

Etiology

The cause of pain and disability in lateral epicondylitis is unknown. It is likely to be multifactorial with an emphasis on repetitive microtrauma and overuse in genetically predisposed individuals.

Some authors have proposed that the rate of lateral epicondylitis is lower in those with two-handed backstrokes vs. single-handed backstrokes, as the nondominant arm helps offload the forces seen by the leading arm [12]. While in theory this might make sense, no clinical studies have proven this to be true. In fact, one investigation found no difference in electromyography (EMG) profiles of ECRB activity between one- and two-handed backstrokes [12]. Similarly, grip size and the use of dampeners to reduce vibration of the strings of racquets have not been shown to affect the rate of lateral epicondylitis [15, 22, 32]. Hennig et al. did find that the more experienced tennis players experienced less vibration and had decreased EMG [16] firing in the wrist extensors during backhand stroke compared with novice players, lending credence to improper technique as a likely cause of developing lateral epicondylitis. Supporting this idea, Kelley et al. showed increased activity in the wrist extensors and pronator teres on EMG and high-speed film during ball impact and early follow-through in tennis players with lateral epicondylitis compared with the control group [18].

Lanz and Wachsmuth [21] described seven bursae, including the radiohumeral bursa located deep to the common extensor tendon and superficial to the radiohumeral joint capsule. Some authors have postulated that these bursae are a potential cause of lateral epicondylitis as repetitive wrist extension with the arm pronated inflamed these structures [8, 24].

Others have evaluated the vascularity of the lateral epicondylar region to help understand the etiology of tennis elbow. Schneeberger and Masquelet studied the

arterial anatomy of the ECRB in cadavers and consistently found an avascular zone at the undersurface of the extensor tendon origin [29]. Others noted hypovascular zones at the lateral epicondyle and 2–3 cm distal to the extensor insertion [3]. Another possible mechanism maybe the autonomic nervous system, which controls vasoconstriction and vasodilatation of the blood vessels surrounding the ECRB. Smith et al. showed abnormal sympathetic vasomotor response in 40 patients with lateral epicondylitis compared with the contralateral unaffected side using a laser Doppler flowmeter to measure dermal blood flow velocity [31].

Laban et al. stressed the importance of examining the shoulder, as unrecognized shoulder pathology may place elevated stress across the common extensor muscle group. In 19 patients with decreased internal rotation, the authors showed that they used increased wrist flexion to compensate for the loss of arc of motion in the shoulder [20].

Bunata et al. studied the anatomical relationship between the ECRB and lateral edge of the capitellum in 85 cadaveric elbows. The authors found that the ECRB undersurface is vulnerable to friction wear as the ECRL compresses the ECRB against the lateral edge of the capitellum [6].

Dellon et al. described a neuroma of the posterior cutaneous nerve of the forearm as a potential source of pain in the area of the lateral epicondyle, particularly after surgical treatment of lateral epicondylitis [9]. This is an important consideration in patients with recalcitrant pain following surgical treatment. In a small series of nine patients, Dellon reported eight patients had excellent pain relief and one patient had good pain relief when the neuroma was excised with implantation of the proximal nerve end into the brachioradialis. Additionally, Dellon showed statistically significant greater improvement in pain relief and faster return to work in patients who underwent denervation of the lateral epicondyle compared to patients who received an epicondylectomy [5].

Epidemiology

Lateral epicondylitis affects 1–3 % of adults in the general population each year [1, 34]. However, the incidence may differ depending on the population of interest as more than 50 % of amateur tennis players reported having been affected with lateral epicondylitis at some point in their career [19]. Typically, adults in the fourth or fifth decade of life are affected. Males and females are affected equally [10, 30], and oftentimes it is the dominant arm that is symptomatic.

Manual laborers, smokers, and those who repetitively bend/straighten their elbow for more than 1 h/day and have poor social support have been associated with higher rates of lateral epicondylitis [10, 14, 30, 35] (Table 1.1). The burden on the economic system is substantial, with 5 % of the affected working-age subjects reporting work absence because of elbow symptoms in the past 12 months [35]. In addition, comorbid conditions, including rotator cuff pathology, DeQuervain's disease, carpal tunnel syndrome, and oral corticosteroid therapy, have been shown

Table 1.1 Reported risk factors for lateral epicondylitis

Age 30–50 years
Manual labor
Smoking
DeQuervain's tenosynovitis
Carpal tunnel syndrome
Oral corticosteroid therapy
Repetive activities > 1 h/day
Poor social support
Poor tennis mechanics

to be independent risk factors for developing lateral epicondylitis. The exact mechanism for this is unclear [33].

The workers' compensation (WC) population can be a difficult subpopulation to treat. Balk compared surgical outcomes of ECRB tendon release between patients filing for WC and those who had no work-related issues. At a mean follow up of 51 months, both groups were found to be equally satisfied with their results and pain relief. However, fewer WC patients returned to their original or similar work status compared with non-WC patients (65% vs. 77%, respectively). Furthermore, 24% of WC patients changed jobs because of persistent symptoms, versus only 4% of non-WC patients [4]. Similarly, Grewal et al. reported on a cohort of 36 patients treated with arthroscopic release for tennis elbow, in which 23 were WC claimants. After surgery, WC patients took twice as long to return to work (24.5 weeks vs. 10.3 weeks), scored lower on American Shoulder and Elbow Society (ASES) scores, Mayo Elbow Performance index, Patient Rated Tennis Elbow Evaluation, Medical Outcomes Study 12-Item Short Form, and Work Limitation Questionnaire [13].

Within the military population, it appears that female gender, age greater than 40 years, and white race are risk factors for developing lateral epicondylitis [36].

Conclusions

Lateral epicondylitis is a condition primarily occurring in adults between the ages of 30 and 50 years. Although the name "lateral epicondylitis" implies an inflammatory reaction, histologic evidence suggests that this condition is more reflective of a chronic angiofibroblastic tendinosis. While many theories exist as to the true cause of lateral epicondylitis, no single explanation is sufficient to elucidate the true cause. There are many risk factors for developing lateral epicondylitis, and one should not falsely assume that only tennis players are susceptible.

References

1. Allander E. Prevalence, incidence, and remission rates of some common rheumatic diseases or syndromes. Scand J Rheumatol. 1974;3:145–53.
2. Alredson H, Ljung B-O, Thorsen K, et al. In vivo investigations of ECRB tendons with microdialysis technique-no signs of inflammation but high amounts of glutmate in tennis elbow. Acta Orthop Scand. 2000;71:475–9.
3. Bales CP, Placzek JD, Malone KJ, Vaupel Z, Arnoczky SP. Microvascular supply of the lateral epicondyle and common extensor origin. J Shoulder Elbow Surg. 2007;16:497–503.
4. Balk ML, Hagberg WC, Buterbaugh GA, Imbriglia JE. Outcome of surgery for lateral epicondylitis (tennis elbow): effect of worker's compensation. Am J Orthop. 2005;34(3):122–6.
5. Berry N, Neumeister MW, Russell RC, Dellon AL. Epicondylectomy versus denervation for lateral humeral epicondylitis. HAND. 2011;6(2):174–8.
6. Bunata Robert E, Brown DS, Capelo R. Anatomic factors related to the cause of tennis elbow. J Bone Joint Surg Am. 2007;89:1955–63.
7. Calfee RP, Patel A, Dasilva MF, Akelman E. Management of lateral epicondylitis: current concepts. J Am Acad Orthop Surg. 2008;16:19–29.
8. Carp L. Tennis elbow caused by radiohumeral bursitis. Arch Surg. 1932;24:905–22.
9. Dellon AL, Kim J, Ducic I. Painful neuroma of the posterior cutaneous nerve of the forearm after surgery for lateral humeral epicondylitis. J Hand Surg Am. 2004;29(3):387–90.
10. Dimberg L. The prevalence and causation of tennis elbow (lateral humeral epicondylitis) in a population of workers in an engineering industry. Ergonomics. 1987;30(3):573–9.
11. Faro F, Wolf JM. Lateral epicondylitis: review and current concepts. J Hand Surg. 2007;8:1271–7.
12. Giangarra CE, Conroy B, Jobe FW, et al. Electromyographic and cinematographic analysis of elbow function in tennis players using single-and double-handed backhand strokes. Am J Sports Med. 1993;21:394–9.
13. Grewal R, MacDermid JC, Shah P, King GJ. Functional outcome of arthroscopic extensor carpi radialis brevis tendon release in chronic lateral epicondylitis. J Hand Surg Am. 2009;34(5):849–57.
14. Haahr JP, Andersen JH. Physical and psychosocial risk factors for lateral epicondylitis: a population based case-referent study. Occup Environ Med. 2003;60:322–9. doi:10.1136/oem.60.5.322.
15. Hatch GF III, Pink MM, Mohr KJ, et al. The effect of tennis racket grip size on forearm muscle firing patterns. Am J Sports Med. 2006;34:1977–83.
16. Hennig EM, Rosenbaum D, Milani TL. Transfer of tennis racket vibrations onto the human forearm. Med Sci Sports Exerc. 1992;24:1134–40.
17. Jobe FW, Ciccotti CM. Lateral and medial epicondylitis of the elbow. J Am Acad Orthop Surg. 1994;2:1–8.
18. Kelley JD, Lombardo SJ, Pink M, Perry J, Giangarra CE. Electromyographic and cinematographic analysis of elbow function in tennis players with lateral epicondylitis. Am J Sports Med. 1994;22:359–63.
19. Kitaie E, Itay S, Ruder A, Engel J, Modan M. An epidemiological study of lateral epicondylitis (tennis elbow) in amateur male players. Ann Chir Main Memb Super. 1986;5:113–21.
20. Laban MM, Iyer R, Tamler MS. Occult periarthrosis of the shoulder: a possible progenitor of tennis elbow. Am J Phys Med Rehabil. 2005;84:895–8.
21. Lanz T, Wachsmuth W. Praktische anatomie. Berlin: Springer; 1959.
22. Li FX, Fewtrell D, Jenkins M. String vibration dampers do not reduce racket frame vibration transfer to the forearm. J Sports Sci. 2004;22:1041–52.
23. Ljung B-O, Forsfren S, Frieden J. Substance P and calcitonin gene related peptide expression at the extensor carpi radialis brevis mucle origin: implications for the etiology of tennis elbow. J Orthop Res. 1999;17:554–9.
24. McVay CB. Surgical anatomy, vol 2, 6th ed. Philadelphia: Saunders; 1984.

25. Morrey BF. The elbow and its disorders, 4th ed. Philadelphia: Saunders; 2009.
26. Nirschl RP, Ashman ES. Elbow tendi-nopathy: tennis elbow. Clin Sports Med. 2003;22:813–6.
27. Nirschl RP, Pettrone FA. Tennis el-bow: the surgical treatment of lateral epicondylitis. J Bone Joint Surg Am. 1979;61:832–9.
28. Runge F. Zur genese und behandlung des schreibekramfes. Berl Klin Wochenschr. 1873;10:245.
29. Schneeberger AG, Masquelet AC. Arterial vascularization of the prioximal extensor carpi radialis brevis tendon. Clin Orthop Relat Res. 2002;46:3–12.
30. Shiri R, Viikari-Juntura E, Varonen H, Heliovaara M. Prevalent and determinants of lateral and medial epicondylitis: a population study. Am J Epidemiol. 2006;164(11):1065–74.
31. Smith RW, Papadopolous E, Mani R, Cawley MI. Abnormal microvascular responses in a lateral epicondylitis. Br J Rtheumatol. 1994;33:1166–8.
32. Stroede CL, Noble L, Walker HS. The effect of tennis racket string vibration dampers on racket handle vibrations and discomfort following impacts. J Sports Sci. 1999;17:379–85.
33. Titchener AG, Fakis A, Tambe AA, Smith C, Hubbard RB, Clark DI. Risk factors in lateral epicondylitis (tennis elbow): a case-control study. J Hand Surg Eur Vol. 2013;38:159–64.
34. Verhaar JA. Tennis elbow: anatomical, epidemiological and therapeutic aspects. Int Orthop. 1994;18:263–7.
35. Walker-Bone K, Palmer KT, Reading I, Coggon D, Cooper C. Occupation and epicondylitis: a population-based study. Rheumatology. 2012;51:305–10.
36. Wolf JM, Mountcastle S, Burks R, Sturdivant RX, Owens BD. Epidemiology of lateral and medial epicondylitis in a military population. Mil Med. 2010;175(5):336–9 (4).

Chapter 2
Tennis Elbow in Athletes: More Than Just Tennis?

Brendan D. Masini, Jonathan F. Dickens and Brett D. Owens

Lateral Epicondylitis: Origins in Sport

From the earliest descriptions of lateral epicondylitis pathology, there has been an association with sport. These descriptions include a letter by Henry J. Morris published in *Lancet* in 1882 describing the condition of "lawn tennis arm [1]." Soon after, Major used the term "lawn tennis elbow" published in the *British Medical Journal* in 1883, to describe the painful condition of epicondylitis in participants in the newly popular game [2]. This makes it the forerunner of sport specific elbow pathology that now includes golfer's elbow, pitcher's elbow, and Little Leaguer's elbow to describe specific pathologies recognized in sport. From the time of that first description, and despite many etiologic, diagnostic, and therapeutic studies on the subject, the relationship with sport has been preserved. Although less than 10% of patients with this condition will be tennis players, or for that matter, athletes, we are compelled to further explore the relationship of this entity with sport, and to understand that tennis is not the only competitive activity among which this injury pattern can be recognized.

B. D. Owens (✉) · B. D. Masini · J. F. Dickens
John A. Feagin, Jr. Sports Medicine Fellowship, Keller Army Hospital,
West Point, NY 10996, USA
e-mail: owensbrett@gmail.com

B. D. Masini
e-mail: Brendan.D.Masini.mil@mail.mil

J. F. Dickens
e-mail: Jonathan.F.Dickens.mil@mail.mil

J. M. Wolf (ed.), *Tennis Elbow,* DOI 10.1007/978-1-4899-7534-8_2

Biomechanics of Tennis

Although lateral epicondylitis can be caused by many sporting activities, there is no other event that causes it with the frequency of tennis. It has been estimated that up to 50% of all recreational players will experience the condition at some point of their career [3]. Furthermore, the link between increased playing time and increased risk of developing the condition has also been well established with twofold or higher incidence in players with more than 2 hours of racquet time per week [4]. For this reason, tennis-specific activities have been the subject of the most intense scrutiny of sport-specific factors contributing to lateral epicondylitis.

The origin of tennis elbow pathology remains unclear. Repetitive contractions have been implicated by causing microtrauma to the common extensor origin, with cumulative degeneration leading to pain and disability [5]. Morris and associates evaluated tennis players using electromyography (EMG) analysis. Healthy high-level tennis players performed groundstrokes with the findings of greatest muscle activity noted in those muscles stabilizing the wrist, specifically the extensor carpi radialis brevis (ECRB), the extensor carpi radialis longus (ECRL), and the extensor digitorum communis (EDC). Amongst these, the ECRB was noted to have the greatest activity. The authors suggest that these muscles provide optimal stability for these phases of the groundstroke by maintaining the position of the wrist in extension and radial deviation [6]. The repetitive focus of stress on the ECRB predisposes this location to injury.

Other proposed mechanisms leading to lateral epicondylitis symptoms include mechanical impingement of the ECRB tendon against the lateral edge of the capitellum during elbow motion [7]. Repetitive abrasion through the arc of motion could help explain the wide variety of sporting and occupational associations. A neurogenic cause of these symptoms has also been proposed based on the anatomy of nerve innervation to the ECRB [8]. In a cadaver study, 40.2% of specimens had either a muscular or tendinous arch around the posterior branch of the radial nerve, which the authors proposed as a potential cause of tennis elbow symptoms.

Whether traumatic, degenerative, or neurogenic, repetitive wrist extension against resistance appears to be the common pathway for development of lateral elbow pain in sports. In tennis, the backhand groundstroke is thought to be the greatest source of pain generation as it fits the requirement of an extended and radially deviated wrist contracting against the resistance of the ball strike. Adding a rotary moment to this motion to add backspin or topspin by pronating or supinating the extended wrist may serve to exacerbate the traumatic forces.

Personal factors related to the participants of sport may also predispose to developing tennis elbow. Shoulder range of motion and strength have been proposed as contributing factors. Female recreational tennis players with a diagnosis of lateral epicondylitis were found to have weaker trapezius muscle strength, weaker wrist extension strength, and higher shoulder internal to external rotation and wrist flexion to extension strength ratios [9]. This study suggests that imbalance of upper extremity muscle groups, found more commonly in amateur athletes rather than

highly trained athletes, may be a factor. As some evidence suggests, it may be the recreational athlete who is more at risk than the elite player [4].

Tennis specific suggestions for factors that may predispose to tennis elbow include racquet grip size. Nirschl proposed that appropriate grip circumference can be measured on the hand as roughly equal to the distance from the proximal palmar crease to tip of ring finger, with larger grip circumferences thought to be protective. This remains a common recommendation although an EMG study of collegiate tennis players using grips 1/4 inches above and below this guideline showed no significant differences in muscle activity [10].

Factors that serve to increase the force of resistance against the firing muscles of the forearm are also postulated to increase symptom development. In tennis, some of these studied factors include string tension with higher tension leading to greater forces acting on the extensors. This may put amateur players at greater risk as they may opt for a string tension that exceeds their training and performance. With off-center hits, increased grip tightness caused significantly more wrist extension torque which may also contribute to lateral epicondylitis pathology [11].

The string density is another racquet factor that contributes to force generation. A higher string count per unit area will also dampen forces transmitted to the arm. This is a factor related to racquet design and less amenable to aftermarket modification than grip size. Increased racquet weight requires greater force generation to support in a wrist extended position and will put greater stress on the muscles in question. In all sports that require swinging an object, choosing the appropriate size and weight equipment will be protective from injury. In addition, materials that serve to dampen vibratory forces such as graphite and epoxies will lessen the forces transmitted to the extensor origin [1].

Court surfaces have also been implicated in development of tennis elbow. Harder court surfaces conserve greater momentum of the ball, and subsequently increase the force transmitted through the racquet. These surfaces are most cost-effective to maintain in a municipal setting, and thus the most likely available to the average player. Softer court surfaces such as grass and clay courts in the specialty club environment are less accessible to amateur players.

Other Racquet Sports

The literature about tennis elbow in other racquet sports is minimal. Badminton has been reported to have a “surprisingly low incidence of tennis elbow” [12]. With extremely light racquets and projectile designs, the forces acting across the wrist extensors is likely to be much less than is experienced in other racquet sports. Squash, with a heavier ball and long moment arm acting on the racquet, has also been noted in reports of lateral epicondylitis [13]. Racquetball has been included in lists of sports at risk for development of lateral epicondylitis [1], although specific reports are rare.

Other Swinging Sports

The non-racquet sports that require swinging an object have similar risk factors to tennis, but fewer reports of epicondylitis are documented.

Golf is known primarily for its association with epicondylitis of the medial side of the elbow, with golfer's elbow, an accepted name for this tendinopathy. Interestingly, arm pain from lateral epicondylitis is the most common upper extremity injury in amateur golfers [14]. Again, these injuries have been noted as more common in amateur and female players [15, 16]. As in the tennis swing, there is vigorous contraction of the extensor muscles to stabilize the wrist. Furthermore, club impact with the ground at the end of the swing places additional stress across the extensors [17]. Like recreational tennis as opposed to baseball or cricket, the majority of participants in this sport are recreational, with a wide variety of skill levels and with varying quality of equipment.

The association of baseball and epicondylitis primarily revolves around the medial sided elbow pathology that is common in overhead throwers due to repetitive valgus loads. Concern for lateral sided elbow injuries is largely absent from the volumes of data that have been accumulated on baseball injuries, and exist primarily as theoretical injury patterns. The lead arm in the batting motion supports a heavy object with a wrist extended and radially deviated position, often with a supination motion at contact and follow-through against a heavy projectile moving with significant velocity. Furthermore, many swing patterns involve a single-handed follow-through, mimicking the single handed tennis backhand that is considered to be the greatest offender of the tennis swing for development of lateral epicondylitis. Perhaps it is because the absolute number of recreational baseball and softball players are much less than the number of recreational tennis players, or that the frequency of play and number of swings per exposure are much less that this pathology is not reported. However, by mechanism alone, the baseball swing may put a player at risk and this pathology should be suspected in the baseball or softball player who presents with lateral elbow pain.

In much the same way as baseball, the cricket swing can be expected to place forces across the wrist extensors subjecting them to injury and development of lateral epicondylitis. Cricket also has a small footprint on the lateral epicondylitis literature and receives only passing mention [8]. Unlike baseball, a cricket batsman remains at bat until retired and may be required to swing many more times than a baseball player in a match.

Olympic Sports

A consistent theme through the discovery of lateral epicondylitis in sport is that the amateur participants with less refined techniques or equipments place themselves at greater risk than the highly trained professional athlete. However, archery is one pursuit that defies this logic. Archery is an Olympic sport, although more often practiced by the amateur in the realm of hunting and outdoor activity rather than

competition. In the process of the shot, the wrist extensor musculature of the bow arm is subjected to significant force as the wrist holds an extended posture to counteract the force of string pull. The faulty technique of the amateur is to hold the bow in a flexed wrist posture, which is protective of the lateral epicondylar insertion [18]. Thus, it may be the elite competitive archers, rather than the beginners that present with this complaint. Furthermore, this may be exacerbated at higher levels of competition where string tensions may be increased.

Martial arts disciplines have also been identified as a source of tennis elbow symptoms. In the performance of these techniques, certain postures and movements demand prolonged contraction of forearm and wrist musculature. One case report detailed a fulltime karate instructor with lateral epicondylitis of both upper extremities as the result of this training [19]. This clearly represents a provocative activity with overtraining or repetition of an action beyond a physiologic threshold. This union can be extrapolated to any of the sporting activities discussed.

Swimming is an excellent form of nonimpact aerobic exercise and is recommended to many orthopaedic patients as a way to avoid injury or degeneration to weight-bearing joints. This is likely the same population who is at risk for lateral epicondylitis resulting from pool work. In swimmers, this condition is recognized in athletes who are typically greater than 30 years old and can frequently be associated with training errors or faulty stroke techniques [20]. As is the case with many other sports, proper technique and avoidance of overtraining can be preventative.

Rowing as a competitive sport has fewer overall participants; however, rowing machines are ubiquitous in gyms, and popular for cardiovascular exercise. Lateral epicondylitis has been recognized as an upper extremity complaint in rowers, and understanding of basic rowing biomechanics and techniques has been advocated for providers to more effectively recognize and treat patients in this population [21].

Other Sports

Increasing in popularity with the advance of indoor facilities is the sport of rock climbing or bouldering. This sport is uniquely demanding of upper extremity strength and endurance, and overuse tendinopathies of the elbows are common. The forces required of the muscles crossing the wrist and elbow are significant, especially for very demanding routes which may include reverse inclines or overhangs. In a survey of Austrian climbers, 29.6% of all men and 13.4% of women reported occurrence of lateral epicondylitis [22]. In fact, lateral epicondylitis trailed only annular ligament strains of the fingers for the most commonly reported injury. The investigators also noted that lateral elbow tendinopathy was more common in men with increased age and increased climbing stress.

A population that is sometimes overlooked in sport injury discussions is the disabled athlete, including the wheelchair athlete. The use of the upper extremities for force generation in a competitive setting lends itself to overuse injuries and tennis elbow is recognized as being prevalent in wheelchair users [23]. Wheelchair racing including

distance racing has been considered a high-injury risk sport and in an evaluation of the British Wheelchair Racing Association, overuse injuries were common and recurred more often than other types of injuries [24]. In a study of wheelchair fencers, elbow strains were the predominant musculoskeletal complaint [25]. With the rapidly growing availability of wheelchair sports, physicians should be aware of the associated musculoskeletal injuries and be prepared to offer appropriate treatment.

The military athlete is another population that deserves mention. The active-duty military population is known for high levels of physical activity, continuous physical fitness, and strenuous job-related demands placed on their upper extremities. In a focused study of this population, female gender, age greater than 40, and Caucasian race were found to have higher incidence risk ratios of tennis elbow [26]. As with the general and athletic populations, in the military, lateral epicondylitis was found to be more common than medial epicondylitis.

Other Recreational Pursuits

While not technically a sport, professional musicians often have demanding, repetitive motions that can be physically demanding and result in musculoskeletal injury. Percussionists, keyboard players, and stringed instrument players all have repetitive flexion and extension of the wrist, which predisposes to lateral epicondylitis. Furthermore, this is an activity that may involve hours of dedicated practice several times a week or even daily for dedicated players. While percussion or keyboard may be equilateral in terms of their presentation, string players will more commonly present with pathology in their fingering hand [27]. Musicians tend to have vague pain localized to the lateral epicondyle thought to be more typical of the occupational injury rather than the athlete; however, treatment recommendations and surgical indications are felt to be similar [28].

Treatment, and Return to Sport

Sport specific reports of treatment are rare, but there are some studies that give reference for evaluation of treatment in athletes as compared to the general population.

Counterforce bracing, and wrist extension bracing for lateral epicondylitis symptoms, are relatively simple and inexpensive methods to initiate treatment. Correct placement of the counterforce brace should be directed to the athlete. Placement just distal to the lateral epicondyle has been found to reduce loads greater than placement directly over the lateral epicondyle [29]. Wrist extension braces place the arm in a position of rest for lateral epicondylar muscles. Specific outcomes for return to activity are not well reported.

More intensive intervention may involve utilization of physical therapists or athletic trainers in the treatment of these patients. A consecutive group of nine rock

climbers presenting with lateral epicondylitis diagnoses were treated with a program of cervical spine manipulation, mobilization treatments to the elbow, manipulation of the wrist, dry needling, and kinesio tape. They were followed with measures of the patient-related tennis elbow evaluation as well as pain pressure threshold testing to ECRB and ECRL, brachioradialis and supinator muscles. At 2 and 6 months, all patients showed improvement [30]. This study suggests that a variety of nonoperative treatments should be explored and can be expected to give some improvement to patients with these conditions.

Corticosteroid injections are common in the treatment of lateral epicondylitis that is refractory to noninvasive means of treatment. The risks of steroid injection include tendon rupture, fat necrosis, and skin discoloration and should be weighed against conflicting data on efficacy [31, 32]. These risks may be more concerning in a high-level athlete or in the setting of chronic treatment.

Another nonoperative treatment option is extracorporeal shock wave treatment. This has been trialed on athletes including a study of effects in tennis players. In a study of 78 players with at least 12 months of symptoms a placebo-controlled trial was performed with weekly treatments over 3 weeks. In the treatment group, 65% of players had improved symptoms at 3 months compared with 28% in the placebo treatment group, which was statistically significant [33]. However, a similar study in a general population did not show any difference in treatment versus placebo group [34].

Surgical treatment has been well described in the patient refractory to conservative management; among athletes, open versus arthroscopic techniques have been debated. Although there are no head-to-head studies in athletes there is some consideration that in the appropriate patient arthroscopic treatment may allow more rapid return of the athlete to sport activities [35].

Although there are many excellent outcomes reported with a variety of surgical modalities, not all athletes will be able to return to their previous level of sporting competition. In a series of 19 patients treated with open extensor release and origin reattachment found that 18 of 19 patients were "better," yet six (60%) of those playing high-demand sports and two (15%) of those with high-demand employment changed sports or jobs postoperatively [36]. All athletes that are contemplating surgical intervention for this condition should be counseled that they may not return to their previous level of competition.

Summary

Lateral epicondylitis is a diagnosis that has a special relationship with sport. It derives from motion patterns that are ubiquitous in upper extremity competition whether swinging a racquet or not. The fact that it is also a source of occupational morbidity only increases the importance of physician awareness and knowledge of treatment options. Sport participation is an important link to lifetime fitness and has innumerable benefits to patient health. For many patients, return to sport may be as

important to them as return to work. For the physician practice that treats athletes of all skill levels, it is important to understand that tennis elbow is more than just tennis.

References

1. Jobe FW, Cicotti MG. Lateral and medial epicondylitis of the elbow. J Am Acad Orthop Surg. 1994;2:1–8.
2. Major HP. Lawn-tennis elbow. Br Med J. 1883;2:557.
3. Nirschl RP. Elbow tendinosis/tennis elbow. Clin Sports Med. 1992;11:851–70.
4. Gruchow HW, Pelletier D. An epidemiologic study of tennis elbow: incidence, recurrence, and effectiveness of prevention strategies. Am J Sports Med. 1979;7:234–8.
5. Riek S, Chapman AE, Milner T. A simulation of muscle force and internal kinematics of extensor carpi radialis brevis during backhand tennis stroke: implications for injury. Clin Biomech. 1999;14:477–83.
6. Morris M, Jobe FW, Perry J, et al. Electromyographic analysis of elbow function in tennis players. Am J Sports Med. 1989;17:241–7.
7. Bunata RE, Brown DS, Capelo R. Anatomic factors related to the cause of tennis elbow. J Bone Joint Surg Am. 2007;89:1955–63.
8. Nayak SR, Ramanathan L, Krishnamurthy A, et al. Extensor carpi radialis brevis origin, nerve supply and its role in lateral epicondylitis. Surg Rad Anat. 2010;32(3):207–11.
9. Lucado AM, Kolber MJ, Cheng MS, et al. Upper extremity strength characteristics in female recreational tennis players with and without lateral epicondylalgia. J Ortho Sport Phys Ther. 2012;42(12):1025–31.
10. Hatch GF III, Pink MM, Mohr KJ, et al. The effect of tennis racquet grip size on forearm muscle firing patterns. Am J Sports Med. 2006;34(12):1977–83.
11. King MA, Kentel BB, Mitchell SR. The effects of ball impact location and grip tightness on the arm, raquet and ball for one-handed tennis backhand groundstrokes. J Biomech. 2012;45(6):1048–52.
12. Hensley LD, Paup DC. A survey of badminton injuries. Br J Sports Med. 1979;13(4):156–60.
13. Verow P. Squash. Practitioner. 1989;233(1470):876–9.
14. Bayes MC, Wadsworth LT. Upper extremity injuries in golf. Phys Sport Med. 2009;37(1): 92–6.
15. Gosheger G, Liem D, Ludwig K, et al. Injuries and overuse syndromes in golf. Am J Sports Med. 2003;31(3)438–43.
16. Batt ME. A survey of golf injuries in amateur golfers. Br J Sports Med. 1992;26(1):63–5.
17. Cohn MA, Lee SL, Strauss EJ. Upper extremity golf injuries. Bull Hosp Jt Dis. 2013;71(1): 32–8.
18. Ertan H. Muscular activation patterns of the bow arm in recurve archery. J Sci Med Sport. 2009;12:357–60.
19. Halloran L. Bilateral epicondylitis in a karate instructor. Ortho Nurs. 1998;17(5):28–30.
20. MacLean I. Swimmer's injuries. Austral Fam Phys. 1984;13(7):499–502.
21. Rumball JS, Lebrun CM, Di Ciacca SR, et al. Rowing injuries. Sports Med. 2005;35(6): 537–55.
22. Pieber K, Angelmaier L, Csapo R, et al. Acute injuries and overuse syndromes in sport climbing and bouldering in Austria: a descriptive epidemiological study. Cent Euro J Med. 2012;124:357–62.
23. Fedorczyk JM. Tennis elbow: blending basic science with clinical practice. J Hand Ther. 2006;19(2):146–53.
24. Taylor D, Williams T. Sports injuries in athletes with disabilities: wheelchair racing. Paraplegia. 1995;33(5):296–9.

25. Chung WM, Yeung S, Wong AY, et al. Musculoskeletal injuries in elite able-bodied and wheelchair foil fencers-a pilot study. Clin J Sport Med. 2012;22(3):278–80.
26. Wolf JM, Mountcastle S, Burks R, et al. Epidemiology of lateral and medial epicondylitis in a military population. Mil Med. 2010;175(5)336–9.
27. Caldron P, Calabrese L, Lederman R, et al. A survey of musculoskeletal problems encountered in high level musicians. Arthritis Rheum. 1985;28(S):587.
28. Hotchkiss RN. Common disorders of the elbow in athletes and musicians. Hand Clinics. 1990;6(3):507–15.
29. Walther M, Kirschner S, Koenig A, et al. Biomechanical evaluation of braces used for the treatment of epicondylitis. J Shoulder Elbow Surg. 2002;11:265–70.
30 Gonzalez-Iglesias J, Cleland JA, del Rosario Gutierrez-Vega M, et al. Multimodal management of lateral epicondylagia in rock climbers: a prospective case series. J Manipulative Physiol Ther. 2011;34(9)635–42.
31. Altay T, Gunal I, Ozturk H. Local injection treatment for lateral epicondylitis. Clin Orthop. 2001;398:127–30.
32. Newcomer KL, Laskowski ER., Idank DM, et al. Corticosteroid injection in early treatment of lateral epicondylitis. Clin J Sport Med. 2001;11:214–22.
33. Rompe JD, Decking J, Schoellner C, et al. Repetitive low-energy shock wave Treatment for chronic lateral epicondylitis in tennis players. Am J Sports Med. 2004;32(3):734–43.
34. Haake M, Konig IR, Decker T, et al. Extracorporeal shock wave therapy in the treatment of lateral epicondylitis: a randomized multicenter trial. J Bone Joint Surg Am. 2002;84-A(11):1982–91.
35. Cohen MS, Romeo AA. Open and arthroscopic management of lateral epicondylitis in the athlete. Hand Clin. 2009;25(3)331–8.
36. Rosenberg N, Henderson I. Surgical treatment of resistant lateral epicondylitis: follow-up study of 19 patients after excision, release and repair of proximal common extensor tendon origin. Arch Orthop Trauma Surg. 2002;122:514–7.

Chapter 3
Natural History and Common Misconceptions: Treatment with Education and Empathy

David Ring

Introduction

Each of the common names for the illness addressed in this book is either inaccurate (e.g., using "itis" for a noninflammatory condition), stigmatizes arm use while inaccurately attributing etiology (e.g., tennis elbow), or is too nonspecific for such a specific disease process (e.g., lateral elbow pain). In my opinion, the best disease labels are accurate and descriptive. In this case: enthesopathy of the origin of the extensor carpi radialis brevis (eECRB).

An enthesis is an attachment point of ligament or muscle to bone. eECRB is one of the many enthesopathies that mostly arise and resolve in middle-age. The pathophysiology for these enthesopathies (as well as for tendinopathies such as trigger finger and de Quervain tendinopathy and degenerative changes in the meniscus of the knee) is myxoid degeneration [1]. Myxoid (or mucoid) degeneration is characterized by gelatinous change in connective tissue. There is an increase in extracellular matrix, fewer tenocytes, and less organized collagen. People with eECRB feel like the elbow is inflamed, but the pathophysiology is not inflammatory.

A useful summary of this disease that is easily understood by most patients is as follows:

1. This disease arises for no rhyme or reason in healthy middle-aged people doing healthy things.
2. It lasts about a year and leaves no trace.
3. We have been working on this for years, but have not found a way to change the course of the disease [2, 3]. Our treatments are palliative at best—they may decrease symptoms while we wait for the disease to resolve.

D. Ring (✉)
Department of Orthopaedic Surgery, Massachusetts General Hospital, 55 Fruit Street Yawkey 2100, Boston, MA 02114, USA
e-mail: DRING@PARTNERS.ORG

J. M. Wolf (ed.), *Tennis Elbow,* DOI 10.1007/978-1-4899-7534-8_3

I suspect that most caregivers—let alone patients—find this "best evidence" summary unexpected, counterintuitive, and unbelievable—at least to some degree. It does not fit our "experience." We have met patients that have had eECRB for 10 years. We have seen people get better from corticosteroid injections. Many of us have personal experience with eECRB—experience that does not mesh with this best evidence summary.

I cannot give caregivers a "magic bullet" for their patients, but I am confident that I can explain the range of debate and emotion on these issues. There are simple things that we can do better—starting with how we conceive of this disease, what we tell our patients, and which coping strategies we directly or indirectly reinforce with our language and behavior. At a minimum, eECRB is a great paradigm for becoming familiar with the wonderful complexity of the human illness experience.

Science

The best healer for patients with eECRB may be curiosity. The best attitude of caregivers is also curiosity. Most of us are familiar with the selective attention test where 50 % of us miss the gorilla while counting the number of times players pass a basketball. Magicians routinely fool even the most intelligent people using this type of misdirection and sleight of hand. In fact, magicians prefer an intelligent audience because the intelligent are easier to fool. Human intelligence is rooted in rationalization and pattern formation—it is built to get it wrong sometimes in order to come to a decision quickly in an emergency. That is why humans invented science. Be ready for your first impressions to be wrong.

eECRB is a paradigm for conditions that require strict science. Subjective, benign, and self-limited, it is difficult to be sure that anything we do for eECRB is superior to the natural history of untreated disease, the tendency of symptoms to wax and wane and regress to the mean, and the placebo effect.

Symptoms and Disability

With all the room for debate in eECRB, there is one thing that is so consistently observed, I think it qualifies as a fact: symptom intensity (e.g., pain) and magnitude of disability correlate most strongly with ineffective coping strategies [4, 5]. Pathophysiology as measured with magnetic resonance imaging or operative findings has little or no correlation with symptoms and disability to date [6].

This may amount to common sense. Humans are built to respond to symptoms—pain in particular—by feeling protective and preparing for the worst. Psychologists measure this tendency in the negative as catastrophic thinking ("Every time I cause pain, I'm keeping it from healing"; "If I don't do something it will always hurt

and I will never be able to depend on my arm") and in the positive as self-efficacy ("I'll be fine"; "I've had pains like this in the past and it always works out"; "I can achieve my goals even with these symptoms") [7].

High self-efficacy is the best pain medicine and the best enabler across all hand and arm conditions. Good health seems less a matter of having a certain type of body (e.g., painless) and more a matter of being able to depend on the body you have. Self-efficacy is something that can be learned and practiced. As caregivers, everything we do and say should be calculated to increase peace of mind and confidence.

Cognitive Behavioral Therapy

It sounds complex and technical, but the methods for optimizing self-efficacy amount to little more than "reading the human instruction manual." We need to learn how our machine operates and learn to make it work as well as possible for ourselves. The placebo effect can be considered an external influence that helps us bring out our confidence and sense of well-being; our "inner healer." Cognitive behavioral therapy and similar approaches help us learn, practice, and excel at using our "inner healer" independently, without external influence such as a pill, shot, or authority [7]. The healthiest among us do this naturally. All of us can learn and improve. Techniques for optimizing thoughts (cognitions) and behaviors in response to symptoms are common to many cultures going back thousands of years [8].

Common Misconceptions

There are several common misconceptions (psychologists would call them "cognitive errors" [8]) among patients with eECRB and—quite often—their caregivers. These misconceptions are common to all painful conditions.

1. eECRB is an injury
2. Hurt = harm
3. Without treatment, eECRB will never go away

Pain feels like damage, and continued pain seems like continued damage. It feels like something needs to be done. This is expected and understandable, because it is how our machine works. It is an evolutionary advantage to respond to pain by preparing for the worst and avoiding pain (being protective). But the best evidence regarding eECRB is that it is a benign self-limiting, atraumatic enthesopathy with no reliable disease-modifying treatments. In other words, the facts are opposite to our natural first impressions.

Getting Past First Impressions

The patients (and caregivers) that seem to have the most trouble with this divide between first impressions and best evidence are very intuitive. They are used to trusting their gut feelings and going with their first impressions. Feelings are facts. It has served them well. Very intuitive people do not like magic. They would not pay to see a magic show. Why? They are very uncomfortable when things are not as they seem.

In eECRB, things are not as they seem. It is important to be curious and to be ready to go beyond one's first impressions. Nobel Prize winner Daniel Kahneman simplifies this into system 1 and system 2 [9]. System 1 is the immediate impressions that we form—our gut feelings or intuition. Two times two? Four. This requires no thought or energy—it is immediately available to us. Seventeen times 28? For most of us, this requires concentration and effort. We need to bring in our analytical system 2.

Kahneman was awarded the Nobel Prize in economics for demonstrating the importance of having a readily available and hearty system 2 to keep our system 1 in check [9]. I like to think of this as curiosity. We need to value our first impressions, but be curious enough to put them to the test. In my opinion, this is measurable as "self-efficacy" and it seems to be the most important element of good health.

Recovery from illness and injury can be quite counterintuitive. What do you do when you fracture your radial head and it is very uncomfortable to move your elbow? You stretch it and get it moving. It is not surprising that many of us find that very difficult to believe and put into practice. Do I need to limit painful activities when I have eECRB? There is no evidence that remaining active during eECRB affects the course of the disease, which is very counterintuitive and for many patients, difficult to accept.

Elements of Care

For my patients with eECRB, I wish I had a shot or a pill that would take their pain away for good. The biomedical treatments described for eECRB range from steroids to botulinum toxin to shock waves to blood to platelet rich plasma. So far, none have shown consistent and reliable outcomes in the long-term compared to no treatment [2, 3]. Let us keep looking, but humbly and with strict science.

In the meantime our patients need care. They come to us because they are not sure they will be able to depend on their arm. I think we can help even if our treatments to date are palliative at best.

The key seems to be empathy—something many of us are not naturally skilled at. But take it from me—it is possible to learn and practice empathy. I work on it every day. Acknowledge how difficult it can be to depend on one's arm when it hurts, and that our "best evidence" is unexpected and counterintuitive. Acknowledge the frustration and disbelief, but stick with the evidence: "It gets better, it just takes

way too long." Provide accurate information. Your own biases and doubts about the evidence aside, patients deserve to know the full range of debate and the weight of the evidence to date.

Do not reinforce common misconceptions and ineffective coping strategies. If you insist that eECRB is an injury and you demand rest, you will increase disability. Beware of stress and emotion contagion. You may find yourself feeling protective and uncertain just as your patient feels. It might lead you to encourage overprotectiveness and order tests or treatments that are not likely to be helpful.

Do not try to convince the patient when best evidence is counterintuitive. Just plant the seeds for health and wellness and acknowledge that it is unexpected and difficult to believe. Do not allow a patient to try to convince you or draw you into an argument—it will not be productive. Just acknowledge how convincing it is that things will not get better. Caregivers should remain curious and open to being wrong without giving in to a temptation to utilize low yield interventions. Offer to monitor patients to be sure nothing is overlooked. If you sense inadequate trust, offer to work as a team with your partners and colleagues. Suggest, "Why don't we have one of my colleagues put their head to this? Together we may come up with something creative."

Do not make promises or guarantees. Active deception is unethical. For instance, you cannot promise that steroids are effective when a growing number of placebo injection controlled trials indicate that they are not [10]. Patients deserve to be informed about best evidence in a dispassionate way.

Finally, do not medicalize what seems to be a rite of passage through middle age. In other words, do not reinforce a patient's sense that the answer is outside themselves; that they need you. Best evidence suggests that Voltaire's dictum still applies to eECRB: Medicine is what the doctor does to entertain the patient while nature cures the disease. Encourage patients to be healthy, independent of specific palliative treatments provided by caregivers.

References

1. Chard MD, Cawston TE, Riley GP, Gresham GA, Hazleman BL. Rotator cuff degeneration and lateral epicondylitis: a comparative histological study. Ann Rheum Dis. 1994;53(1):30–4. PubMed PMID: 8311552; PubMed Central PMCID: PMC1005239.
2. Bernstein J, Wolf JM. Autologous blood and platelet-rich plasma injections for enthesopathy of the extensor carpi radialis brevis origin. J Hand Surg Am. 2013;38(5):992–4. doi:10.1016/j.jhsa.2013.01.001. Epub 2013 Feb 27. PubMed PMID: 23455408.
3. Szabo RM. Steroid injection for lateral epicondylitis. J Hand Surg Am. 2009;34(2):326–30. doi:10.1016/j.jhsa.2008.10.011. Epub 2008 Dec 20. PubMed PMID: 19097708.
4. Lindenhovius A, Henket M, Gilligan BP, Lozano-Calderon S, Jupiter JB, Ring D. Injection of dexamethasone versus placebo for lateral elbow pain: a prospective, double-blind, randomized clinical trial. J Hand Surg Am. 2008;33(6):909–19. doi:10.1016/j.jhsa.2008.02.004. PubMed PMID: 18656765.
5. Das De S, Vranceanu AM, Ring DC. Contribution of kinesophobia and catastrophic thinking to upper-extremity-specific disability. J Bone Joint Surg Am. 2013;95(1):76–81. doi:10.2106/JBJS.L.00064. PubMed PMID: 23283376.

6. van Kollenburg JA, Brouwer KM, Jupiter JB, Ring D. Magnetic resonance imaging signal abnormalities in enthesopathy of the extensor carpi radialis longus origin. J Hand Surg Am. 2009;34(6):1094–8. doi:10.1016/j.jhsa.2009.02.023. Epub 2009 Jun 5. PubMed PMID: 19501476.
7. Vranceanu AM, Barsky A, Ring D. Psychosocial aspects of disabling musculoskeletal pain. J Bone Joint Surg Am. 2009;91(8):2014–8. doi:10.2106/JBJS.H.01512. Review. PubMed PMID: 19651964.
8. Harrington A. The cure within: history of mind-body medicine. New York: W.W. Norton; 2008.
9. Kahneman D. Thinking fast and slow. New York: Farrar, Straus, and Giroux; 2011.
10. Coombes BK, Bisset L, Brooks P, Khan A, Vicenzino B. Effect of corticosteroid injection, physiotherapy, or both on clinical outcomes in patients with unilateral lateral epicondylalgia: a randomized controlled trial. JAMA. 2013;309(5):461–9. doi:10.1001/jama.2013.129. PubMed PMID: 23385272.

Chapter 4
Chronic Lateral Epicondylitis—New Perspectives on Injection Strategies and Ultrasound Evaluation

Thøger Persson Krogh, Ulrich Fredberg and Torkell Ellingsen

Lateral epicondylitis (LE) is a common disorder of the extensor origin at the lateral humeral epicondyle, first described by Runge in 1873 [42, 43]. The annual incidence rate of LE is 1.0 per 100 workers [16]. Clinically, the patient with LE often presents with a painful elbow, defined as pain on the lateral side of the elbow aggravated by direct palpation of the lateral epicondyle and during resisted dorsiflexion of the wrist [37, 42].

Etiology

A few years ago, the pain associated with chronic tendon overuse was believed to be due to a chronic inflammatory process, but as no inflammatory cells could be demonstrated, there was a change in definition away from inflammation ("tendinitis") toward degeneration ("tendinosis") [20].

In general, "tendinitis" is used primarily as a histopathologic term that describes a condition in which the primary site of involvement is the tendon and in which an inflammatory response is seen within the tendon. "Tendinosis" is used primarily to describe a histopathologic finding that consists of intratendinous degeneration without signs of inflammation and without correlation with clinical symptoms.

T. Ellingsen (✉)
Department of Rheumatology, Odense University Hospital, Søndre Boulevard, 5000 Odense, Denmark
e-mail: torkell.ellingsen@rsyd.dk

T. P. Krogh · U. Fredberg
Diagnostic Centre, Silkeborg Regional Hospital, Falkevej 1-3, 8600 Silkeborg, Denmark
e-mail: thoger.krogh@hotmail.com

U. Fredberg
e-mail: ulrifred@rm.dk

J. M. Wolf (ed.), *Tennis Elbow,* DOI 10.1007/978-1-4899-7534-8_4

"Tendinopathy" is used to signify the combination of tendon pain and impaired performance often associated with swelling of the tendon and intratendinous changes on evaluation with US or MRI. The diagnosis of tendinopathy can, in contrast to tendinitis and tendinosis, be made clinically without histopathologic examination. Lateral epicondylopathy could therefore be a more neutral term than LE. The exact pathogenesis of chronic tendinopathy remains largely unknown, but seems to be a multifactorial process with a wide range of suggested intrinsic and extrinsic etiological factors that are assumed to be the mechanisms of tendinopathy. The scientific background for most of these suggestions is lacking, and they must be characterized as nonproven theories. The traditional view of tendinopathy is a tendon injury associated with overuse due to repetitive mechanical loads, microtears, and acute and then chronic phases of inflammatory "tendinitis" that lead to tendon degeneration ("tendinosis"). The existing data indicate that the initiators include traumatic events or a prolonged repetitive motion injury that induces the production of many proinflammatory agents. Mechanical strain theory at the moment is the most accepted theory to explain the injury mechanisms of tendon overload. The tendon cells can produce these proinflammatory agents when subjected to cyclic stress, and in animal studies these inflammatory agents can be used to produce experimental chronic tendinopathy. Furthermore, many of the proinflammatory mediators and neuropeptides are also found in chronic tendinopathy. The debate on inflammation versus degeneration is still open [12, 21, 28, 30, 31, 41].

Ultrasonography

US is an important diagnostic tool in sports medicine and rheumatology, and a common treatment measure in clinical trials [11, 14, 17]. US is a reliable, noninvasive, widely available, and inexpensive imaging technique for assessing tendon pathology [15, 17]. The high acoustic contrast with the surrounding tissue makes tendons particularly suitable for ultrasonographic examination [15]. US findings in tendinopathy are generally characterized by increased tendon Doppler activity, irregularity of the fibrillar appearance, focal hypoechoic areas, and calcifications [1, 19, 32, 48]. Several studies have described these ultrasonographic features in patients with LE: increased tendon thickness, increased Doppler activity (Fig. 4.1), intratendinous and peritendinous elastographic alterations, bony spurs, tendon calcifications and bone cortex abnormalities, fibrillar disruption, tears, detachment from the bone, focal hypoechoic and hyperechoic regions, and diffuse heterogeneity (see Fig. 4.1; [1, 5, 8, 18, 19, 26, 29, 32, 33, 38, 39, 48]). The focal hypoechoic areas in the deep part of the extensor carpi radialis brevis component of the common extensor tendon correspond histopathologically with collagen degeneration with fibroblastic proliferation ([5]; Fig. 4.1)

To perform an ultrasonographic evaluation of the common extensor tendon, patients are examined in a sitting position with the elbow flexed to 90°, the wrist

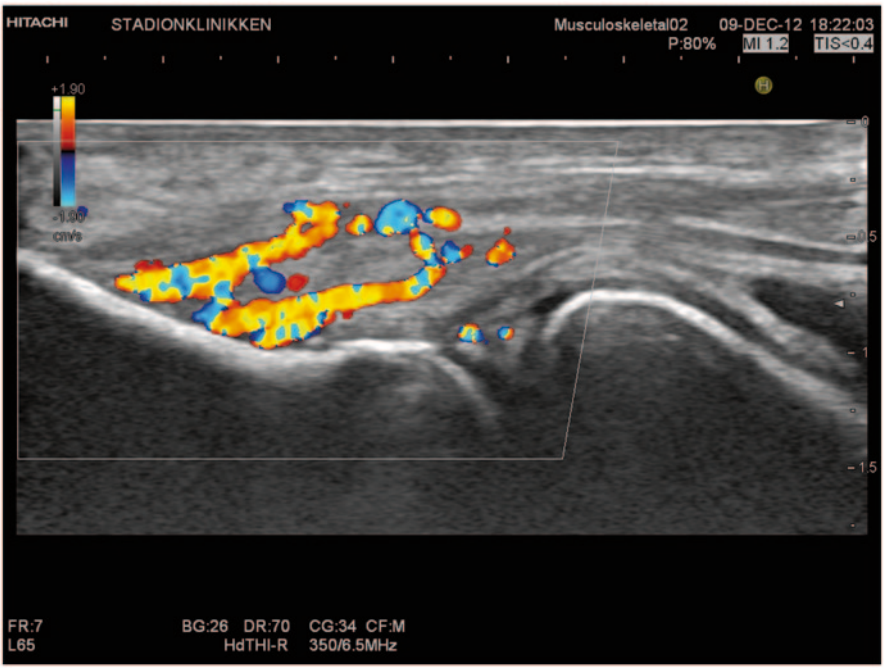

Fig. 4.1 Ultrasonography of the common extensor tendon in a patient with lateral epicondylitis. The tendon thickness is increased, and the Doppler flow is also illustrated

pronated, and the arm resting on a table. The transducer is aligned with the long axis of the radius over the common extensor tendon (See Fig. 4.2).

Tendon Thickness Measurement of the thickness of the common extensor tendon can be performed in different ways. In a study by Krogh et al. [23] two different techniques are described, and the intra- and inter-observer variation is excellent with both methods. Method 1, labeled "1-cm measure," measures tendon thickness 1 cm distal from the insertion of the common extensor tendon (on top of the lateral epicondyle), perpendicular to the length of the tendon (Fig. 4.3a and c). Method 2, labeled "plateau measure," measures tendon thickness at an anatomical landmark at the horizontal bony surface of the lateral epicondyle, which is referred to as "the plateau." "The plateau" is a flat aspect of the capitellum of the lateral epicondyle located between the insertion of the tendon and the radio-humeral joint. Tendon thickness is measured from "the plateau" to the tendon surface perpendicular to the length of the tendon (Fig. 4.3b and c). Other authors have used similar methods for tendon thickness measurement [18, 25].

Doppler Activity It has been shown to have a good sensitivity and specificity as a diagnostic tool for patients with LE [9, 47, 49]. The color Doppler activity is usually seen in an area limited proximally by the tip of the lateral epicondyle and distally by the humeroradial joint space. The outer border is the most superficial

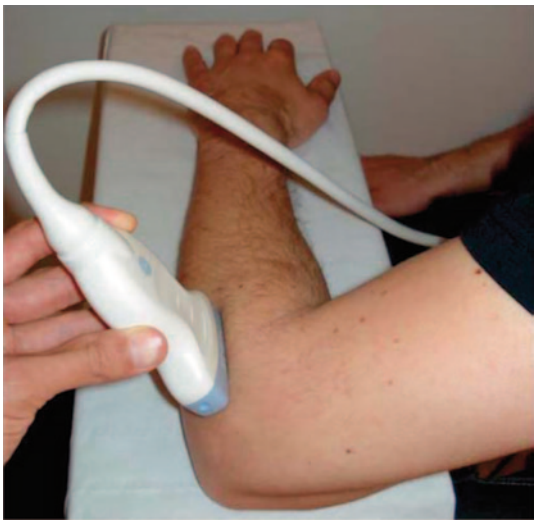

Fig. 4.2 Transducer location on testing for tennis elbow. The transducer is aligned with the long axis of the radius over the common extensor tendon

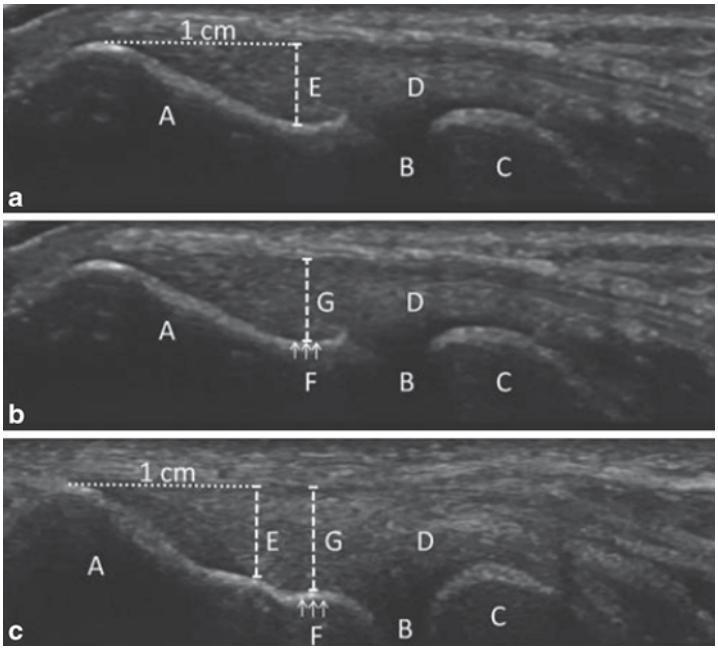

Fig. 4.3 Measuring the tendon thickness by ultrasound. Longitudinal sonogram illustrating two different methods (**a** and **b**) for measuring the thickness of the common extensor tendon. Labels: Lateral epicondyle (*A*), radiohumeral joint (*B*), radial head (*C*), common extensor tendon (*D*), tendon thickness 1 cm distal form the attachment (E), and tendon thickness at "the plateau" (*G*). *Arrows* indicate "the plateau" (*F*)

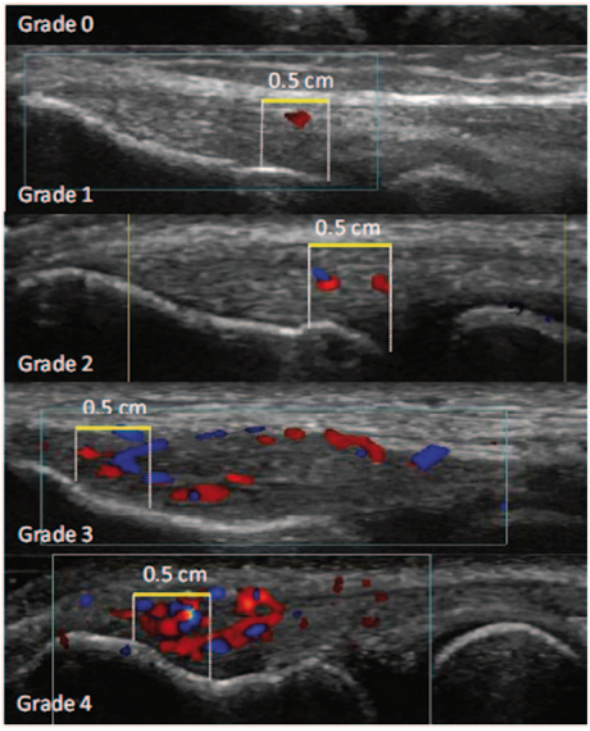

Fig. 4.4 Measurement of Doppler activity (grade 0–4). Longitudinal ultrasonogram of the common extensor tendon illustrating grading of color Doppler activity from grade 0–4. The *horizontal yellow line*, measuring 0.5 cm, marks the superficial border of the ROI, *white vertical lines* mark the proximal and distal borders, and the bone surface marks the deeper borders

fibers, and the deep border is the bone (Fig. 4.1). There are several ways to assess Doppler activity, but as yet no consensus has been reached regarding which method to use. In newer studies [23, 39] color Doppler activity is graded in a new ranking scale from grade 0–4 (see Fig. 4.4). This modulation can be used both in scientific studies and in daily clinical work. The grading is estimated in a 0.5-cm longitudinal part of the tendon with the maximal Doppler activity (region of interest, ROI). The scale is as follows: Grade 0: no activity, grade 1: single vessel, grade 2: Doppler activity in less than 25% of the region of interest, grade 3: Doppler activity in 25–50% of the region of interest, and grade 4: Doppler activity in more than 50% of the region of interest. Regarding color Doppler activity, the results showed an excellent correlation between the observers and an overall satisfactory agreement [23]. However, factors including probe position, probe pressure, and equipment settings including gain, wall filter, color priority, and pulse repetition frequencies can influence the outcome.

Several other methods for assessing Doppler activity have been suggested, e.g., as a binary outcome (negative/positive), where grade 0 and 1 would count as negative Doppler activity and grade 2 or more as positive Doppler activity. In conclusion, it is difficult to compare the results of Doppler activity across studies because of the use of various methods.

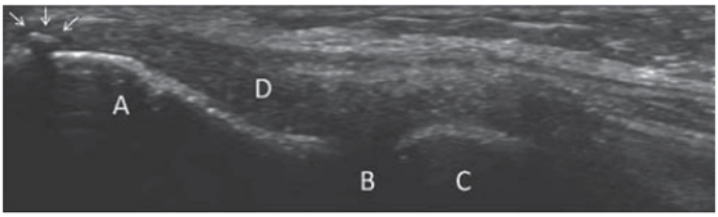

Fig. 4.5 Detection of bony spurs by ultrasonography. *Arrows* indicating the bony spurring at the top of the lateral epicondyle (**a**), radiohumeral joint (**b**), radial head (**c**), and common extensor tendon (**d**)

Bony Spurs Bony spurs (enthesophytes) were initially described on conventional radiographs [3, 40]. Bony spurring is defined as a bony outgrowth arising at the insertional site of the common extensor tendon (see Fig. 4.5). The bony spurs are easily seen on US, and several ultrasonographic studies in patients with LE have dealt with the observation of bony spurs [5, 9, 26, 39, 46]. However, the clinical significance of bony spurs is unclear.

Treatment

The treatment of LE varies widely from "watchful waiting" to nonsteroidal anti-inflammatory drugs, physical therapies including exercise, bracing, injection therapies, and, as a last option, surgery. Glucocorticoid injections have been used since the 1950s, and for many years have been the treatment of choice [6]. However, despite a well-documented short-term effect, several studies have shown no long-term effect [22, 27, 36, 45], and some studies have even shown that long-term benefit after corticosteroid injections is less than after other treatments. Whether this is due to an adverse effect of the corticosteroids or whether there is a marked short-term effect, after which the patients resumes the harmful overload without adequate rehabilitation, is so far unclear.

In a newer study of patients with LE by Krogh et al. [24], US of the elbow demonstrated a reduction in tendon thickness following injection of glucocorticoid and the same was observed regarding color Doppler activity. The reduction in tendon thickness observed after glucocorticoid injection goes well in hand with a study [13] that showed a reduction in tendon thickness in both patellar and Achilles tendons.

During the past 10 years, several new therapies have become available that focus on the use of growth factors (GFs), among others, as a stimulant of tendon repair. Platelet-rich plasma (PRP) is blood plasma with an increased concentration of autologous platelets. PRP is now being used as a part of wound treatment, bone healing, alloplastic surgery, and muscle/tendon damage [2, 34, 44]. PRP can potentially enhance tendon healing and tissue regeneration by delivering various growth factors and cytokines, thereby effecting cell proliferation, chemotaxis, cell differentia-

tion, and angiogenesis. The theory is that application of PRP intratendinously will stimulate the repair mechanism and promote tendon healing [4, 7, 10].

In conclusion, we can say that the role of injection strategies in LE is doubtful, considering the long-term outcome. Ultrasound improves the diagnostic algorithm and can probably be supportive as a future outcome measure. Consensus in terms of outcome measures in clinical trials needs to be established. Despite the development of many new therapies, the most well-documented treatment is relief from the harmful activity and slow rehabilitation below the pain threshold, as recommended in *The Lancet* in 1882 [35].

References

1. Allen GM, Wilson DJ. Ultrasound in sports medicine-a critical evaluation. Eur J Radiol. 2007;62(1):79–85.
2. Aspenberg P, Virchenko O. Platelet concentrate injection improves Achilles tendon repair in rats. Acta Orthop Scand. 2004;75(1):93–9.
3. Benjamin M, Toumi H, Ralphs JR, Bydder G, Best TM, Milz S. Where tendons and ligaments meet bone: attachment sites ('entheses') in relation to exercise and/or mechanical load. J Anat. 2006;208(4):471–90.
4. Borzini P, Mazzucco L. Tissue regeneration and in loco administration of platelet derivatives: clinical outcome, heterogeneous products, and heterogeneity of the effector mechanisms. Transfusion. 2005;45(11):1759–67.
5. Connell D, Burke F, Coombes P, McNealy S, Freeman D, Pryde D, et al. Sonographic examination of lateral epicondylitis. AJR Am J Roentgenol. 2001;176(3):777–82.
6. Cyriax J, Troisier O. Hydrocortone and soft-tissue. Br Med J. 1953;2(4843):966–8.
7. de Mos M, van der Windt AE, Jahr H, van Schie HT, Weinans H, Verhaar JA, et al. Can platelet-rich plasma enhance tendon repair? A cell culture study. Am J Sports Med. 2008;36(6):1171–8.
8. De ZT, Lill SR, Fink C, Feuchtner GM, Jaschke W, Bellmann-Weiler R, et al. Real-time sonoelastography of lateral epicondylitis: comparison of findings between patients and healthy volunteers. AJR Am J Roentgenol. 2009;193(1):180–5.
9. duToit C, Stieler M, Saunders R, Bisset L, Vicenzino B. Diagnostic accuracy of power Doppler ultrasound in patients with chronic tennis elbow. Br J Sports Med. 2008;42(11):872–6.
10. Foster TE, Puskas BL, Mandelbaum BR, Gerhardt MB, Rodeo SA. Platelet-rich plasma: from basic science to clinical applications. Am J Sports Med. 2009;37(11):2259–72.
11. Fredberg U, Bolvig L. Significance of ultrasonographically detected asymptomatic tendinosis in the patellar and achilles tendons of elite soccer players: a longitudinal study. Am J Sports Med. 2002;30(4):488–91.
12. Fredberg U, Stengaard-Pedersen K. Chronic tendinopathy tissue pathology, pain mechanisms, and etiology with a special focus on inflammation. Scand J Med Sci Sports. 2008;18(1):3–15.
13. Fredberg U, Bolvig L, Pfeiffer-Jensen M, Clemmensen D, Jakobsen BW, Stengaard-Pedersen K. Ultrasonography as a tool for diagnosis, guidance of local steroid injection and, together with pressure algometry, monitoring of the treatment of athletes with chronic jumper's knee and Achilles tendinitis: a randomized, double-blind, placebo-controlled study. Scand J Rheumatol. 2004;33(2):94–101.
14. Grassi W, Cervini C. Ultrasonography in rheumatology: an evolving technique. Ann Rheum Dis. 1998;57(5):268–71.
15. Grassi W, Filippucci E, Farina A, Cervini C. Sonographic imaging of tendons. Arthritis Rheum. 2000;43(5):969–76.

16. Herquelot E, Gueguen A, Roquelaure Y, Bodin J, Serazin C, Ha C, et al. Work-related risk factors for incidence of lateral epicondylitis in a large working population. Scand J Work Environ Health. 2013;39(6):578–88.
17. Jacobson JA. Ultrasound in sports medicine. Radiol Clin North Am. 2002;40(2):363–86.
18. Jaen-Diaz JI, Cerezo-Lopez E, Lopez-de CF, Mata-Castrillo M, Barcelo-Galindez JP, De la Fuente J, et al. Sonographic findings for the common extensor tendon of the elbow in the general population. J Ultrasound Med. 2010;29(12):1717–24.
19. Joshua F, Edmonds J, Lassere M. Power Doppler ultrasound in musculoskeletal disease: a systematic review. Semin Arthritis Rheum. 2006;36(2):99–108.
20. Khan KM, Cook JL, Kannus P, Maffulli N, Bonar SF. Time to abandon the "tendinitis" myth. BMJ. 2002;324(7338):626–7.
21. Kraushaar BS, Nirschl RP. Tendinosis of the elbow (tennis elbow). Clinical features and findings of histological, immunohistochemical, and electron microscopy studies. J Bone Joint Surg Am. 1999;81(2):259–78.
22. Krogh TP, Bartels EM, Ellingsen T, Stengaard-Pedersen K, Buchbinder R, Fredberg U, et al. Comparative effectiveness of injection therapies in lateral epicondylitis: a systematic review and network meta-analysis of randomized controlled trials. Am J Sports Med. 2013;41(6):1435–46.
23. Krogh TP, Fredberg U, Christensen R, Stengaard-Pedersen K, Ellingsen T. Ultrasonographic assessment of tendon thickness, doppler activity and bony spurs of the elbow in patients with lateral epicondylitis and healthy subjects: a reliability and agreement study. Ultraschall Med. 2013;34:468–74.
24. Krogh TP, Fredberg U, Stengaard-Pedersen K, Christensen R, Jensen P, Ellingsen T. Treatment of lateral epicondylitis with platelet-rich plasma, glucocorticoid, or saline: a randomized, double-blind, placebo-controlled trial. Am J Sports Med. 2013;41(3):625–35.
25. Lee MH, Cha JG, Jin W, Kim BS, Park JS, Lee HK, et al. Utility of sonographic measurement of the common tensor tendon in patients with lateral epicondylitis. AJR Am J Roentgenol. 2011;196(6):1363–7.
26. Levin D, Nazarian LN, Miller TT, O'Kane PL, Feld RI, Parker L, et al. Lateral epicondylitis of the elbow: US findings. Radiology. 2005;237(1):230–4.
27. Lindenhovius A, Henket M, Gilligan BP, Lozano-Calderon S, Jupiter JB, Ring D. Injection of dexamethasone versus placebo for lateral elbow pain: a prospective, double-blind, randomized clinical trial. J Hand Surg Am. 2008;33(6):909–19.
28. Ljung BO, Lieber RL, Friden J. Wrist extensor muscle pathology in lateral epicondylitis. J Hand Surg Br. 1999;24(2):177–83.
29. Maffulli N, Regine R, Carrillo F, Capasso G, Minelli S. Tennis elbow: an ultrasonographic study in tennis players. Br J Sports Med. 1990;24(3):151–5.
30. Maffulli N, Khan KM, Puddu G. Overuse tendon conditions: time to change a confusing terminology. Arthroscopy. 1998;14(8):840–3.
31. Maffulli N, Testa V, Capasso G, Ewen SW, Sullo A, Benazzo F, et al. Similar histopathological picture in males with Achilles and patellar tendinopathy. Med Sci Sports Exerc. 2004;36(9):1470–5.
32. Martinoli C, Bianchi S, Derchi LE. Tendon and nerve sonography. Radiol Clin North Am. 1999;37(4):691–711.
33. Miller TT, Shapiro MA, Schultz E, Kalish PE. Comparison of sonography and MRI for diagnosing epicondylitis. J Clin Ultrasound. 2002;30(4):193–202.
34. Molloy T, Wang Y, Murrell G. The roles of growth factors in tendon and ligament healing. Sports Med. 2003;33(5):381–94.
35. Morris H. THE RIDER'S SPRAIN. Lancet. 1882;120(3074):133–4.
36. Newcomer KL, Laskowski ER, Idank DM, McLean TJ, Egan KS. Corticosteroid injection in early treatment of lateral epicondylitis. Clin J Sport Med. 2001;11(4):214–22.
37. Pienimaki T, Tarvainen T, Siira P, Malmivaara A, Vanharanta H. Associations between pain, grip strength, and manual tests in the treatment evaluation of chronic tennis elbow. Clin J Pain. 2002;18(3):164–70.
38. Poltawski L, Jayaram V, Watson T. Measurement issues in the sonographic assessment of tennis elbow. J Clin Ultrasound. 2010;38(4):196–204.

39. Poltawski L, Ali S, Jayaram V, Watson T. Reliability of sonographic assessment of tendinopathy in tennis elbow. Skeletal Radiol. 2011;41(1):83–9.
40. Pomerance J. Radiographic analysis of lateral epicondylitis. J Shoulder Elbow Surg. 2002;11(2):156–7.
41. Rees JD, Maffulli N, Cook J. Management of tendinopathy. Am J Sports Med. 2009;37 (9):1855–67.
42. Regan W, Grondin P, Morrey BF. Lateral epicondylitis (tennis elbow). In: DeLee J, Drez D, Miller M, editors. DeLee and Drez's orthopaedic sports medicine. 3rd ed. Elsevier; 2009. p. 1197–204.
43. Runge F. Zur Genese und Behandlung des Schreibekrampfes. Berliner Klinische Wochenschrift. 1873;10:245–8.
44. Sampson S, Gerhardt M, Mandelbaum B. Platelet rich plasma injection grafts for musculoskeletal injuries: a review. Curr Rev Musculoskelet Med. 2008;1(3–4):165–74.
45. Smidt N, Van Der Windt DAWM, Assendelft WJJ, Deville WLJM, Korthals-De Bos IBC, Bouter LM. Corticosteroid injections, physiotherapy, or a wait-and-see policy for lateral epicondylitis: a randomised controlled trial. The Lancet. 2002;359(9307):657–62.
46. Struijs PA, Spruyt M, Assendelft WJ, van Dijk CN. The predictive value of diagnostic sonography for the effectiveness of conservative treatment of tennis elbow. AJR Am J Roentgenol. 2005;185(5):1113–8.
47. Torp-Pedersen T, Torp-Pedersen S, Bliddal H. Diagnostic value of ultrasonography in epicondylitis. Ann Intern Med. 2002;136(10):781–2.
48. Wakefield RJ, Balint PV, Szkudlarek M, Filippucci E, Backhaus M, D'Agostino MA, et al. Musculoskeletal ultrasound including definitions for ultrasonographic pathology. J Rheumatol. 2005;32(12):2485–7.
49. Zeisig E, Ohberg L, Alfredson H. Extensor origin vascularity related to pain in patients with Tennis elbow. Knee surgery sports traumatology. Arthroscopy. 2006;14(7):659–63.

Chapter 5
Medical Management: Role of NSAIDs/Topical Medications

Rachel S. Rohde

Introduction

Oral and topical anti-inflammatory medications are commonly used in the management of lateral epicondylitis. Studies regarding treatment patterns suggest that the use of nonsteroidal anti-inflammatory drugs (NSAIDs) is one of the most frequently prescribed modalities of general practitioners as well as orthopedic specialists [15, 20]. They have been referred to as the "medication of choice" [12]. However, the effectiveness of NSAID use for lateral epicondylitis management is not well established (Table 5.1).

Oral Agents

Several oral agents have been suggested as treatments for lateral epicondylitis. An early study described benoral tablets—the active ingredients of which are vitamin B_1 and B_6—as an effective treatment for what was considered "chronic non-articular rheumatism" [22]. To the author's knowledge, further studies regarding the benefit of therapeutic vitamins for tennis elbow have not been reported.

Initial studies regarding the use of NSAIDs (tenoxicam, piroxicam, and flurbiprofen) in lateral epicondylitis demonstrated improvement in pain and decreased limitations, but at the expense of mild but common adverse gastrointestinal reactions [23, 27]. Similar pain relief was noted with the use of oral diclofenac vs. placebo, but no objective functional improvement was observed and significant adverse gastrointestinal events were noted [13].

R. S. Rohde (✉)
Department of Orthopaedic Surgery, William Beaumont School of Medicine, Oakland University, Michigan Orthopaedic Institute, 26025 Lahser Road, Southfield, MI 48033, USA
e-mail: rohders@gmail.com

J. M. Wolf (ed.), *Tennis Elbow,* DOI 10.1007/978-1-4899-7534-8_5

Table 5.1 Studies describing the effect of topical and oral anti-inflammatory medication on lateral epicondylitis symptoms

Topical agents	Author	Year	Follow up (days)	Partici-pants	Type	Comparison
Diclofenac 10 mg/g gel BID × 30 days vs. 2–4 medicinal leeches applied once	Bäcker et al. [2]	2011	45	40	RCT	Leech therapy
Diclofenac gel TID × 21 days	Burnham and Oner [4]	1998	21	14	RCT	–
Difflam (benzydamine) cream 5× daily × 21 days	Burton [5]	1988	21	33	RCT	Manual therapy
Diclofenac vs. salicylate iontophoresis QD, 5×/week, up to 18 days	Demirtas et al. [6]	1998	25	40	RCT	Iontophoresis of two topical agents
Diclofenac tissuegel patch × 14 days	Jenoure et al. [11]	1997	28	85	RCT	–
Diclofenac diethylamine salt (gel) QID × 14 days	Schapira et al. [26]	1991	14	32	RCT	–
Diclofenac epolamine 1.3 % gel 5 g TID × 10 days	Spacca et al. [28]	2005	10	40	RCT	–
Indomethacin 1 % ointment TID-QID × 14 days	Tsuiyama et al. [32]	1979	14	187	RCT	–
–	–	–	–	–	–	–
Oral agents	–	–	–	–	–	–
Diflunisal 1000 mg PO then 500 mg PO BID × 15 days vs. Naproxen 500 mg PO then 250 mg PO Q6–8 H PRN × 15 days	Adelaar et al. [1]	1987	15	22	RCT	Oral NSAID comparison
Acemetacin 90 mg PO QD	Erturk et al. [7]	1997	21	36	RCT	Steroid injection
Naproxen 500 mg PO BID × 14 days	Hay et al. [10]	1999	365	164	RCT	Steroid injection
Naproxen 500 mg PO BID × 14 days	Hay et al. [10]	1999	365	164	RCT	Placebo
Diclofenac sodium 75 mg PO bid × 28 days	Labelle and Guibert [13]	1997	28	129	RCT	Placebo, both w/long arm cast
Naproxen 750 mg PO QD × 10 days vs. betahistine dihydrochloride 48 mg PO QD × 10 days	Polat et al. [21]	2011	182	55	RCT	Betahistine dihydrochloride (vasodilator)
Naproxen 500 mg then 250 mg PO BID × 14 days	Saartok and Eriksson [25]	1986	14	21	RCT	Steroid injection
Diflunisal 1000 mg PO then 500 mg PO BID × 15 days vs. Naproxen 500 mg PO then 250 mg QID x 15 days	Stull and Jokl [29]	1986	15	40	RCT	Oral NSAID comparison

The most extensively investigated NSAID for the treatment of lateral epicondylitis is oral naproxen. Initially, naproxen was demonstrated in smaller studies to relieve pain as well as comparable NSAIDs such as diflunisal [29] or a betamethasone injection [25]. However, a later multicenter study showed that 2 weeks of naproxen (500 mg twice daily) resulted in 57% improvement in 4 weeks compared to 50% treated by placebo and 92% after methylprednisolone injection [10]. Interestingly, all three groups reported 82–85% improvement in 1 year.

Topical Agents

Topical agents have become more available in recent years. Although the adverse effects of oral NSAIDs include significant gastrointestinal issues, those of topical agents generally are limited to local cutaneous reactions [8, 30]. This makes topical use a potentially favorable alternative to oral administration.

Early topical application of dimethyl sulfoxide (DMSO) showed no benefit in a double-blinded controlled study [19]. Attention since then has turned to the topical NSAIDs such as naproxen gel (10%); this has been studied only in the acute treatment phase (up to 7 days) and has proved to be more effective than placebo for symptom improvement [31].

A similar NSAID, topical diclofenac (2%), has proved to be better than placebo at reducing pain and weakness associated with lateral epicondylitis [4]. A randomized controlled study involving 158 patients with shoulder arthritis or tennis elbow confirmed that a 10 day course of treatment three times daily is more effective than placebo for pain relief and restoring the ability to perform activities of daily living [28]. Currently, diclofenac sodium topical gel (1%)—Voltaren Gel (Endo Pharmaceuticals)—is indicated only for treatment of arthritis-associated pain in the hands and knees, but a future indication might include lateral epicondylitis if further evidence corroborates these findings.

More recently, promising results in wound and fracture care in animal models have spawned consideration of topical nitric oxide use. Nitric oxide is generated in tendinopathy [3] and has been shown to enhance wound and tendon healing [34]. The mechanism by which nitric oxide affects healing is unknown, but might involve enhanced collagen synthesis. A randomized prospective controlled study of 86 patients performing rehabilitation with either glyceryl trinitrate patch or placebo patch showed that the transdermal patch improved symptoms and functional outcome [17]. This was confirmed in a follow up study by the same group but no additional treatment effects were noted with increasing the dose [18]. This benefit appears to be short-term only, as no difference was noted 5 years following treatment [3, 14].

Although iontophoresis and phonophoresis will be addressed in the therapy chapter, several studies have compared the use of various topical agents during these therapeutic modalities. It has been demonstrated that sodium diclofenac was more effective than sodium salicylate [6] and then placebo [33] during iontophoresis.

Iontophoresis with dexamethasone also was more effective than with placebo in the short-term [16], while this was not corroborated by a contemporaneous study [24].

Summary

Systematic review of trials regarding the use of oral or topical NSAIDs has been limited by study design [9]. Although oral and topical NSAIDs appear to have some value in controlling the pain associated with lateral epicondylitis, they likely do not modify the progression of disease. There appears to be more evidence to support the use of topical NSAIDs than oral NSAIDs, at least for short-term relief. Use of these pharmacologic agents to mitigate the pain of lateral epicondylitis as needed should be tailored to the patient with specific attention paid to the potential adverse effects.

References

1. Adelaar R, Maddy L, Emroch L. Diflunisal versus naproxen in the management of mild to moderate pain associated with epicondylitis. Adv Ther. 1987;4(6):317–27.
2. Bäcker M, Ludtke R, Afra D, Cesur O, Langhorst J, Fink M, et al. Effectiveness of leech therapy in chronic lateral epicondylitis: a randomized controlled trial. Clin J Pain. 2011;27(5):442–7.
3. Bokhari AR, Murrell GA. The role of nitric oxide in tendon healing. J Shoulder Elbow Surg. 2012;21(2):238–44.
4. Burnham R, Gregg R, Healy P, Steadward R. The effectiveness of topical diclofenac for lateral epicondylitis. Clin J Sport Med. 1998;8(2):78–81.
5. Burton A. A comparative trial of forearm strap and topical anti-inflammatory as adjuncts to manual therapy in tennis elbow. Manual Med. 1988;3:141–3.
6. Demirtaş RN, Oner C. The treatment of lateral epicondylitis by iontophoresis of sodium salicylate and sodium diclofenac. Clin Rehabil. 1998;12(1):23–9.
7. Erturk H, Celiker R, Sivri A, Cetin A, Cindas A. The efficacy of different treatment regiments that are commonly used in tennis elbow. J Rheum Med Rehab. 1997;8:298–301.
8. Gómez A, Florido JF, Quiralte J, Martín AE, Sáenz de San Pedro B. Allergic contact dermatitis due to indomethacin and diclofenac. Contact Dermat. 2000;43(1):59.
9. Green S, Buchbinder R, Barnsley L, Hall S, White M, Smidt N, Assendelft W. Non-steroidal anti-inflammatory drugs (NSAIDs) for treating lateral elbow pain in adults. Cochrane Database Syst Rev. 2002;(2):CD003686.
10. Hay EM, Paterson SM, Lewis M, Hosie G, Croft P. Pragmatic randomised controlled trial of local corticosteroid injection and naproxen for treatment of lateral epicondylitis of elbow in primary care. BMJ. 1999;319(7215):964–8.
11. Jenoure P, Rostan A, Gremion G, Meier J, Grossen R, Bielinki R, et al. Multi-centre, double-blind, controlled clinical study on the efficacy of diclofenac epolamine tissugel plaster in patients with epicondylitis. Medicina Dello Sport. 1997;50:285–92.
12. Kumar S, Stanley D. Controversial topics in surgery: current thoughts on the treatment of tennis elbow. Ann R Coll Surg Engl. 2011;93:432–6.
13. Labelle H, Guibert R. Efficacy of diclofenac in lateral epicondylitis of the elbow also treated with immobilization. The University of Montreal Orthopaedic Research Group. Arch Fam Med. 1997;6(3):257–62.

14. McCallum SD, Paoloni JA, Murrell GA. Five-year prospective comparison study of topical glyceryl trinitrate treatment of chronic lateral epicondylosis at the elbow. Br J Sports Med. 2011;45(5):416–20.
15. Nilsson P, Lindgren EC, Månsson J. Lateral epicondylalgia. A quantitative and qualitative analysis of interdisciplinary cooperation and treatment choice in the Swedish health care system. Scand J Caring Sci. 2012;26(1):28–37.
16. Nirschl RP, Rodin DM, Ochiai DH. Maartmann-Moe C; DEX-AHE-01-99 study group. Iontophoretic administration of dexamethasone sodium phosphate for acute epicondylitis. A randomized, double-blinded, placebo-controlled study. Am J Sports Med. 2003;31(2):189–95.
17. Paoloni JA, Appleyard RC, Nelson J, Murrell GA. Topical nitric oxide application in the treatment of chronic extensor tendinosis at the elbow: a randomized, double-blinded, placebo-controlled clinical trial. Am J Sports Med. 2003;31(6):915–20.
18. Paoloni JA, Murrell GA, Burch RM, Ang RY. Randomised, double-blind. placebo-controlled clinical trial of a new topical glyceryl trinitrate patch for chronic lateral epicondylosis. Br J Sports Med. 2009;43(4):299–302.
19. Percy EC, Carson JD. The use of DMSO in tennis elbow and rotator cuff tendonitis: a double-blind study. Med Sci Sports Exerc. 1981;13(4):215–9.
20. Peterson M, Elmfeldt D, Svärdsudd K. Treatment practice in chronic epicondylitis: a survey among general practitioners and physiotherapists in Uppsala County, Sweden. Scand J Prim Health Care. 2005;23(4):239–41.
21. Polat A, Ekinci O, Terzioglu B, Canbora M, Muftuoglu T, Gorgec M. Treatment of lateral epicondylitis using betahistine dihydrochloride. J Musculoskel Pain. 2011;19(4):201–6.
22. Ronn HH. A follow-up study of the use of benoral tablets in non-articular rheumatism. J Int Med Res. 1977;5(1):48–52.
23. Rosenthal M. The efficacy of flurbiprofen versus piroxicam in the treatment of acute soft tissue rheumatism. Curr Med Res Opin. 1984;9(5):304–9.
24. Runeson L, Haker E. Iontophoresis with cortisone in the treatment of lateral epicondylalgia (tennis elbow)-a double-blind study. Scand J Med Sci Sports. 2002;12(3):136–42.
25. Saartok T, Eriksson E. Randomized trial of oral naproxen or local injection of betamethasone in lateral epicondylitis of the humerus. Orthopedics. 1986;9(2):191–4.
26. Schapira D, Linn S, Scharf Y. A placebo-controlled evaluation of diclofenac diethylamine salt in the treatment of lateral epicondylitis of the elbow. Curr Ther Res. 1991;49(2):162–8.
27. Schorn D. Tenoxicam in soft-tissue rheumatism. S Afr Med J. 1986;69(5):301–03.
28. Spacca G, Cacchio A, Forgács A, Monteforte P, Rovetta G. Analgesic efficacy of a lecithin-vehiculated diclofenac epolamine gel in shoulder periarthritis and lateral epicondylitis: a placebo-controlled, multicenter, randomized, double-blind clinical trial. Drugs Exp Clin Res. 2005;31(4):147–54.
29. Stull PA, Jokl P. Comparison of diflunisal and naproxen in the treatment of tennis elbow. Clin Ther. 1986;(9 Suppl C):62–6.
30. Sullivan WJ, Panagos A, Foye PM, Sable AW, Irwin RW, Zuhosky JP. Industrial medicine and acute musculoskeletal rehabilitation. 2. Medications for the treatment of acute musculoskeletal pain. Arch Phys Med Rehabil. 2007;88(3 Suppl1):S10–3.
31. Thorling J, Linden B, Berg R, Sandahl A. A double-blind comparison of naproxen gel and placebo in the treatment of soft tissue injuries. Curr Med Res Opin. 1990;12(4):242–8.
32. Tsuyama N, Nichikawa K, Tsujimoto M, Miyanaga Y, Mizushima H, Nakajima A. Clinical evaluation of nonsteroidal anti-inflammation/analgesics for external use-indomethacin ointment in the orthopaedic field. Clin Evaluation. 1979;7:285–309.
33. Vecchini L, Grossi E. Ionization with diclofenac sodium in rheumatic disorders: a double-blind placebo-controlled trial. J Int Med Res. 1984;12(6):346–50.
34. Witte MB, Barbul A. Role of nitric oxide in wound repair. Am J Surg. 2002;183(4):406–12.

Chapter 6
Role of Therapy and Rehabilitation

Ruby Grewal, Joey G. Pipicelli, David S. Kaminski and Shrikant J. Chinchalker

Introduction

Tennis elbow is a disabling musculoskeletal condition leading to pain and/or tenderness around the lateral aspect of the elbow. Nonoperative rehabilitation should be considered the "first line" of treatment as it has been suggested that greater than 85 % of patients respond to this approach, however, some series do suggest more modest results [9, 21]. There are many treatment options proposed in the literature; however, many of these studies are flawed [22].

Tennis elbow is a term commonly used when referring to lateral epicondylosis (LE), a degenerative condition of the common extensor origin at the lateral aspect of the elbow. LE is believed to result from repetitive overuse, which leads to tendinosis with microtrauma at the common extensor tendon origin. This process mainly involves the extensor carpi radialis brevis (ECRB) origin [61, 63, 93]. Nirschl was the first to describe the histopathology of this disorder and coined the term "angiofibroblastic hyperplasia" to describe the disorganization of normal collagen architecture by invading fibroblasts, blood vessels, and collagen. Nirschl also noted the absence of inflammatory cells in histologic specimens [30, 63, 93]. Because inflammation is not a significant component of the pathophysiology, the term tendinosis is used to describe the condition rather than tendonitis [73, 93]. Histologic examination

R. Grewal (✉)
Division of Orthopedics, Department of Surgery, Roth and MacFarlane Hand and Upper Limb Center, 268 Grosvenor St, London, ON N6A-4L6, Canada
e-mail: rgrewa@uwo.ca

J. G. Pipicelli · D. S. Kaminski · S. J. Chinchalker
Division of Hand Therapy, Roth and MacFarlane Hand and Upper Limb Center, 268 Grosvenor Street, London, Ontario N6A4V2, Canada
e-mail: Joey.Pipicelli@hotmail.com

D. S. Kaminski
e-mail: dave.kaminski@sjhc.londong.on.ca

J. M. Wolf (ed.), *Tennis Elbow,* DOI 10.1007/978-1-4899-7534-8_6

Table 6.1 [48] Pathological stages of tendinosis

Stage 1	Characterized by peritendinous inflammation and crepitus may be palpable over the common extensor origin
Stage 2	Inflammatory response ends, as angiofibrotic changes beginning to occur, leading to degradation of the origin of the ECRB tendon
Stage 3	Pathologic changes leading to a structural failure and rupture along with tear of ECRB tendon
Stage 4	Continued tendon degradation and structural failure, but also with other changes such as fibrosis, soft matrix calcifications, and hard osseous calcification

of the diseased tissues also reveals the following findings: tenocyte hyperplasia; endothelial cell hyperplasia; microvascular thrombosis; hyaline, fatty, mucoid, calcified, fibrous infiltrates within the tendon substance; and cell necrosis [30]. Four stages of tendinosis have been described [48, 62]. (See Table 6.1) *Stage* 1 consists of peritendinous inflammation; crepitus is usually palpable over the common extensor tendon. *Stages* 2–4 refer to the presence of angiofibroblastic degeneration. As the fibrosis worsens, the scar tissue that forms can tear with further repetitive trauma, and associated tendon ruptures are seen in *Stage* 3**.** *Stage* 4 can be associated with calcification. [30, 48]. The pathological stages of tendinosis as a cellular response to overload injuries and microtears were outlined by Kraushaar and Nirschl [48].

The repetitive overuse that results in LE is also due in part to the underlying anatomy. The deep surface of the ECRB is in close contact with the capitellum, and repetitive wear and abrasion at this site of contact is felt to play a role in the development of LE [17]. It is felt that repeated undersurface abrasion against the capitellum with elbow extension puts the tendon at risk for microtears and degeneration. These continued repetitive forces lead to further degeneration because of the poor underlying vascular anatomy. Studies have shown that the undersurface of the extensor tendon origin is macroscopically avascular [17, 29], reducing the healing potential, making this area more vulnerable to injury. There are two main hypovascular zones, at the lateral epicondyle and 2–3 cm distal to the extensor insertion [5, 29]. It has also been shown that there may be an imbalance between vasoconstrictor and vasodilator innervation in this area, further contributing to LE and exacerbating the healing difficulty already present in this watershed area [29, 51, 81]. Some feel that the abnormal vascularity contributes to pain mediation in chronic tendinopathies [30].

Neurogenic Inflammation

Pain is a chief complaint of this condition despite the absence of inflammation. The presence of neurochemicals within the involved tissue has been identified. It is believed that these neurochemicals, such as glutamate, substance P, and calcitonin gene-related peptide may be responsible for pain mediation [30]. These substances

have been identified in cases of chronic tennis elbow and provide a possible mechanism for pain mediation in this and other chronic tendinopathies [30].

Therapeutic Rehabilitation

Evidence-based medicine is the use of current best evidence in making treatment decisions about the care of individual patients. Both research and clinician expertise are important components of evidence-based practice. Evidence suggests that LE is one of the most commonly seen upper extremity conditions. However, gaps exist between common treatment approaches, clinical practice guidelines, and available evidence. Recent systematic reviews related to LE indicate that the results of therapeutic interventions vary, there is a lack of scientific evidence to draw conclusions about optimal therapeutic interventions, and conservative management is multimodal [6, 12, 47, 80]. Many studies have found that conservative treatment is effective in the treatment of LE [24, 55, 63, 70] and many authors have indicated that only about 10 % of patients with LE undergo operative management. Fedorczyk [31] suggests that therapeutic intervention is effective, however, therapists have failed to report their outcomes in peer-reviewed literature. We feel that satisfactory outcomes are to be expected if quality rehabilitation is instituted which incorporates therapeutic interventions that promote the modulation of pain, tissue healing and regeneration, muscular conditioning, patient education, and worksite and sporting modifications.

Phases of Rehabilitation

Three phases of rehabilitation have been proposed for LE. The **acute phase** is commonly characterized by moderate pain at rest, which can be easily provoked with light functional activity. Typical interventions in this phase are aimed at pain control and activity modification. The **restorative phase** begins when minimal resting pain is present and minimal provocation of symptoms with active range of motion (ROM) or light functional activity. The third phase, **maintenance phase,** involves the workplace, equipment, and technique modification for work and sport and continued conditioning to prevent recurrence.

Over 40 different treatment techniques have been reported in the literature with varying levels of scientific evidence [76]. MacDermid et al. [54] conducted a survey of expert opinion and practice patterns of the management of LE by hand therapists. These authors found that hand therapists consider the most essential components of rehabilitation to include patient education, stretching, activity modification, strengthening, pain management, and use of orthoses/splints. However, it is imperative when implementing the various treatment techniques that they are applied based on the current phase of rehabilitation and stage of tendon healing.

The acute phase of rehabilitation is characterized by symptoms that include tenderness over the lateral aspect of the elbow with moderate pain at rest which can be easily provoked with ROM and light functional activity. Patients often report discomfort with elbow extension, forearm pronation, and upon resisted wrist extension or firm gripping [78]. There may be swelling and/or crepitus over the lateral elbow; however, in clinical practice this is most often not present unless the patient has sought medical attention within the first 6 weeks of onset [45]. If swelling is present, the patient would be categorized as a Stage 1 based on the Kraushaar and Nirschl classification system. Therapy during the acute stage is directed at controlling pain and activity modification. Furthermore, therapists attempt to minimize the progression of LE into the latter stages of the Kraushaar and Nirschl classification system.

Patient Education

In order to progress through the phases of rehabilitation, efforts must be taken to modify behaviors and activities as well as to refrain from aggravating activities. Clinicians should provide the patient with education regarding how to apply ergonomic principles during aggravating activities as well as to decrease load and repetition during activity. Emphasis should be placed on avoiding prolonged or repetitive wrist flexion, extension, and radial deviation as these positions increase tension on the muscle–tendon unit [56, 94]. Also, forceful gripping, repetitive finger use, and repetitive pronation and supination can similarly lead to pain and microtrauma of the common extensor tendon. For the computer-based worker, education in optimal hand, wrist, and elbow positions while seated as well as changes to the angle of tilt of the keyboard and position of the keys relative to the worker can be beneficial. (i.e., split keyboard, negative tilt, gel wrist support bar, elbow at approximately 90°, forearms supported). For others, such as factory workers or sports-minded individuals, larger handled tools that allow a wider grip, soft-handled tools that reduce grip effort, and ergonomically designed tools that are lighter and place the wrist in neutral to slight extension during use have been suggested as effective strategies to begin controlling LE symptoms. The literature indicates that ergonomic interventions are subjectively effective, but that minimal change occurs in overall symptom severity [34].

Pain Control Techniques

Rest

The term rest is a misnomer, because cessation from the offending activity is the goal. However, we do not want complete inactivity or immobilization as this can lead to disuse atrophy and compromise the later stages of rehabilitation [20]. Rest with intermittent graded activity should assist with reducing pain. Intermittent graded activity allows for intermittent loading of the involved structures preventing atrophy while promoting gradual, progressive vascularization of the tendon during

the initial stages of healing. Thus, activity restriction opposed to complete rest is recommended with restriction from repetitive lifting, gripping, and pronation/supination of the affected arm [18].

Ice

Cryotherapy is a modality that should be introduced at the onset of LE symptoms. It can be applied after activity with the intent to minimize post-activity soreness and to reduce inflammation if present. It is generally applied for 5–15 min a few times per day [76]. Cryotherapy will provide short-term symptom relief [55].

Medication

Medication taken orally (such as antiinflammatories, NSAIDS, etc) or injected (cortisone, lidocaine) does not directly promote recovery [12, 35, 79, 88]. However, it may provide pain control allowing the patient to progress through the phases of rehabilitation. During the acute phase, oral medication may provide symptom control if taken during the first 10–14 days from the onset of injury [20]. If symptoms have been present for many weeks, oral medications will likely provide minimal effect. For a detailed review of medical management and steroid injections, we refer you to Chaps. 4 and 7, respectively.

Orthosis Application

Two popular methods of orthotic intervention to provide pain control include a forearm counterforce strap and a wrist extension orthosis. The forearm counterforce strap is placed around the muscle bellies of the wrist extensors just distal to the elbow joint (Fig. 6.1). During the acute phase of rehabilitation, a wrist extension orthosis may be helpful to allow the wrist extensors to rest. These orthoses can be custom fabricated or prefabricated (Fig. 6.2). Jensen et al. [43] investigated the amount of electrical activity in the wrist extensors by electromyography during activity with and without the wrist orthosis. These authors found that the application of a wrist orthosis in 15° extension reduces muscle activity during lifting activi-

Fig. 6.1 Forearm counterforce bracing is the application of a nonelastic strap to prevent full muscular expansion of the proximal forearm. This acts to reduce force transmission across the proximal portion of the muscle–tendon unit which may diminish pain with gripping activities

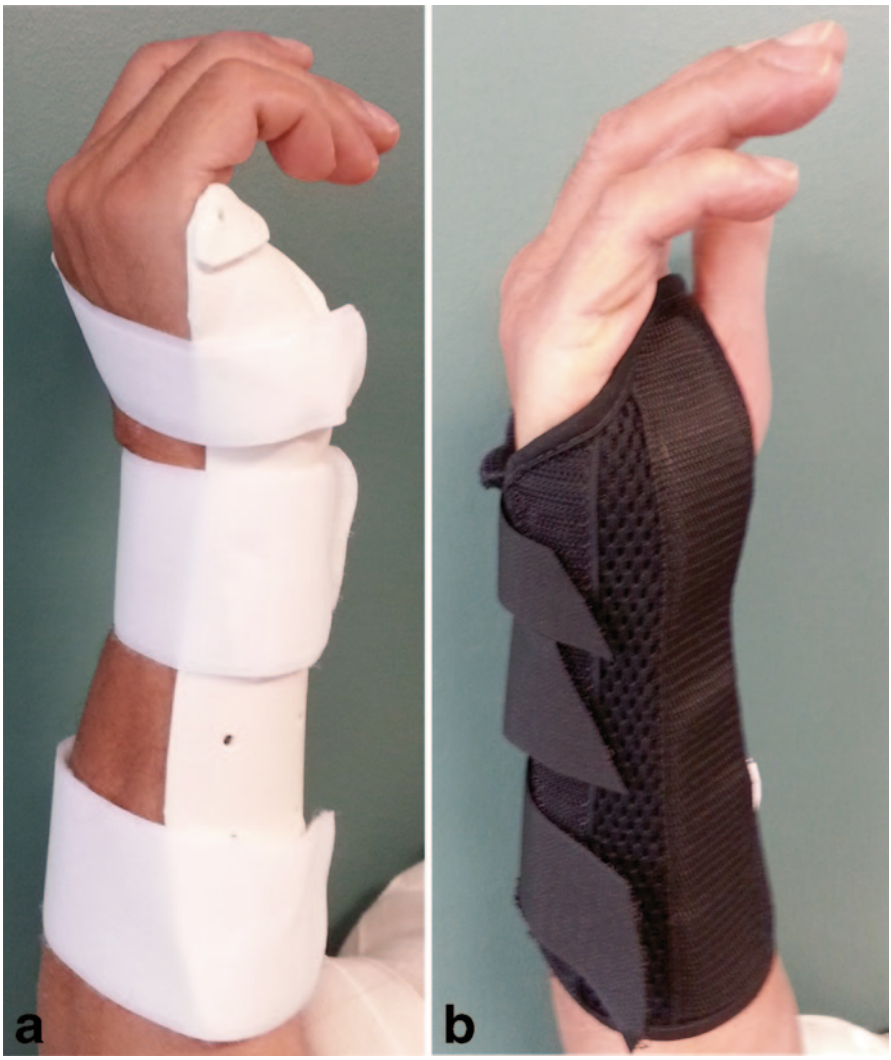

Fig. 6.2 **a** Custom fabricated wrist extension orthosis positioning the wrist in approximately 30–40° of wrist extension to adequately unload the wrist extensors. Five degrees of wrist extension

ties, and therefore, assumed that there was decreased tension placed on the tendon. In the acute phase of rehabilitation, our preference is to use a custom-made wrist extension orthosis positioning the wrist in approximately 30–40° of extension to adequately unload the wrist extensors. This position has been shown to optimize grip strength [66], therefore, splinting the wrist in this position should allow for optimal hand function while providing rest to the wrist extensors.

Counterforce bracing is the application of a nonelastic strap to prevent full muscular expansion of the proximal forearm [42, 60]. The therapeutic effect of this form

of orthotic application lies in the compressive force applied just distal to the origin of the ECRB. This acts to reduce force transmission across the proximal portion of the muscle–tendon unit which may diminish pain with gripping activities. This form of bracing may also promote rest to the injured structures. Counterforce bracing may be used during the acute, restorative, or maintenance phase of rehabilitation [59, 62, 71]. Snyder-Macker and Epler [82] found a decrease in ECRB and EDC muscle force recruitment with counterforce brace application when compared to no counterforce brace applied as measured by electromyography. It is hypothesized that by inhibiting muscle expansion, the counterforce strap decreases the magnitude of the muscle contraction reducing tension placed to the common extensor origin.

Orthotic application has been the subject of a Cochrane Review [87]. Only five studies met the inclusion criteria for the review and no definitive conclusion could be drawn regarding the effectiveness of orthotic interventions. However, a recent prospective randomized study comparing counterforce bracing to a prefabricated wrist extension orthosis concluded that the orthotic application provides for greater pain relief [33]. These authors postulated that this improvement in pain may be a result of the greater immobilization provided to the wrist extensor muscles in the orthosis. In clinical practice, our approach is to conduct a trial for both orthotic applications and have the patient continue with the device that provides the greatest pain relief.

Exercise: Stretching

Progressive stretching exercises are one of the most commonly used treatments in the management of LE [36]. The purpose is to allow elongation of the muscle–tendon unit in an effort to reduce pain and stiffness. The patient is instructed to perform passive wrist flexion exercises with variable amounts of elbow extension to maintain length of the musculotendinous unit (Fig. 6.3). The stretch is enhanced by progressing to full elbow extension with the forearm pronated and combined with passive wrist flexion. The patient should be instructed to hold this stretch gently for 15–30 s to prevent forceful vigorous stretching which may contribute to worsening of their symptomology. This form of stretching assists with pain reduction [70].

Ultrasound

Therapeutic ultrasound is a high frequency sound wave used to stimulate tissue beneath the skin's surface. Ultrasound is performed to deliver heat to deep musculoskeletal tissues such as tendon, muscle, and joint structures [40]. Essentially, the theorized mechanism of clinical utility with this modality is a stimulation of blood flow and soft tissue extensibility which may have a positive effect of tendon healing as well as decreasing pain. Ultrasound can be performed at different frequencies for various durations. The clinician has the option to select continuous or pulsed-wave treatments. With pulsed-wave treatments, there is a periodical interruption in the intensity applied to the targeted tissue so that no ultrasound energy is produced during the off time within the application. This will produce nonthermal effects of ultrasound. Continuous wave ultrasound can be applied which will produce thermal as well as nonthermal effects.

Ultrasound may be useful during the acute stages of rehabilitation to assist with pain reduction. However, long-term application during the various phases of rehabilitation is likely of minimal benefit. Various authors have recommended that

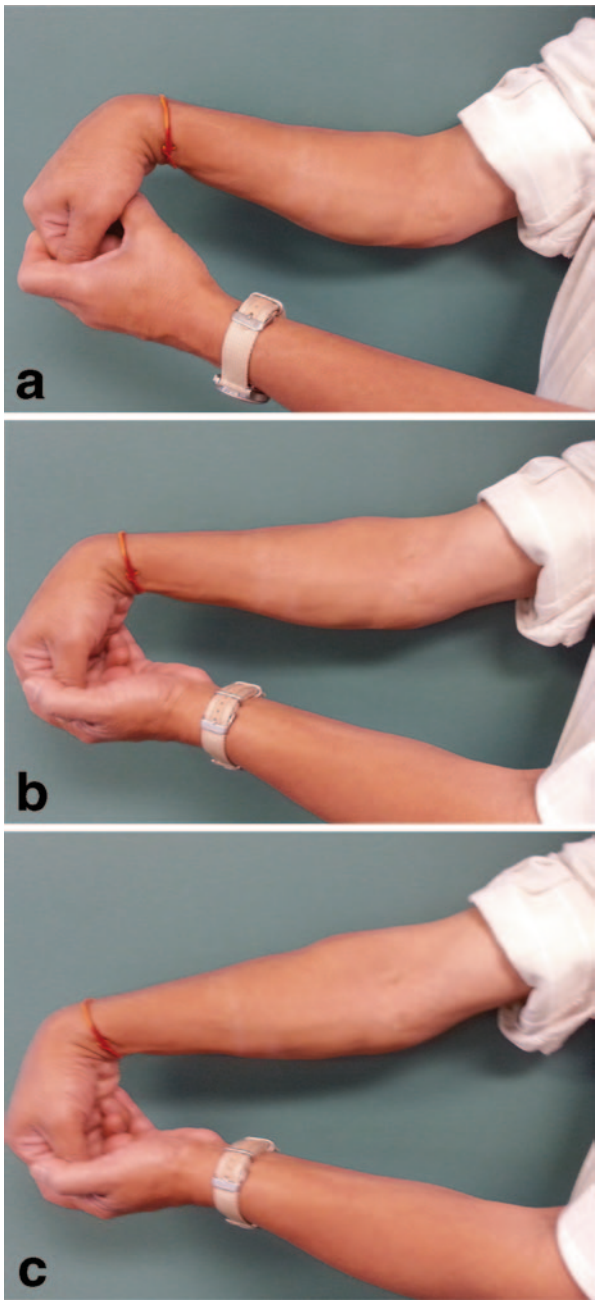

Fig. 6.3 Prefabricated wrist extension orthosis positioning the wrist in approximately 10–15° of wrist extension

ultrasound be applied along with various other therapeutic interventions such as stretching, activity modification, orthotic application, and progressive resistance exercise. [10, 38, 53].

Phonophoresis and Iontophoresis

Iontophoresis and phonophoresis are used to deliver analgesics and/or antiinflammatory agents transdermally. Iontophoresis is the delivery of ionizable substances through the skin driven by an electric field by using a direct current application. Phonophoresis is the use of ultrasound to enhance the delivery of topically applied medications.

The hypothesized benefit of such applications is to provide patients with a higher concentration of medication within the target tissue without exposing the patient to the risks associated with injections. Although injections will provide a higher concentration of medication to the target tissue, these are invasive procedures. The literature reports that phonophoresis does not appear to be superior to ultrasound [41, 46]. However, iontophoresis with dexamethasone sodium phosphate may assist with short-term pain relief during the acute stage of rehabilitation [65].

Low Level Laser Therapy

Low Level Laser Therapy (LLLT) is believed to reduce pain by modulating tissue neuronal activity and inflammation by suppressing inflammatory enzymes that create swelling, redness, pain, and heat [11, 89]. The effect depends on the application of the correct wavelength and density of light delivered to the target tissues for an appropriate period of time (typically between 30 and 60 s). Pulsed treatment can improve tissue repair and antiinflammatory effect; analgesia is best achieved with a continuous beam.

The literature is inconclusive on the effects of LLLT and LE. Some authors report that LLLT provides pain control, however, other papers found no evidence of long-term relief when compared with placebo [13, 19, 37, 49, 52, 84, 92].

Extracorporeal Shock Wave Therapy

Extracorporeal Shock Wave Therapy (ESWT) is reported to encourage tendon healing by disrupting avascular tissue, promoting vascularization, and the release of local growth factors. The literature indicates that ESWT as a treatment for LE provides little or no benefit with regards to pain relief, thus, there is no evidence to support its use [16, 74].

Restorative Phase

The restorative phase should begin when minimal resting pain is present and symptoms cannot be provoked with ROM or light functional activity. During this phase components of treatment used in the acute phase will persist, though the emphasis will move toward resistance exercise, progression to a home exercise program, and continued activity modification.

Progressive Resistance Exercise

Isometric and Concentric Contractions

Once the patient is nearly pain-free at rest and has only minimal pain with light functional activity, they should begin a progressive resistance exercise program. This program should begin with multiangle isometric strengthening of the wrist extensors, wrist flexors, forearm rotators, and digital flexors and extensors [27]. Once tolerance is exhibited with such exercises with minimal increase in symptomology, the patient is progressed to concentric wrist extension as well as isotonic contractions beginning with low weight and low repetitions. The patient should be instructed to begin with five repetitions gradually increasing to 20–30 repetitions for 1–3 sets, 2–3 times per day.

Eccentric Exercise

Eccentric exercise has been advocated to resolve pain associated with chronic tendinopathies. Eccentric strengthening loads the musculotendinous unit to induce hypertrophy and increased tensile strength, reducing the strain on the tendon during movement [2, 83]. Eccentric loading may provide a greater stimulus for collagen produced within the tendon to withstand greater force than encountered during aggravating and provocative activity [26, 83]. It is theorized that eccentric exercise reduces neovascularization within the affected tendon which is believed to be a causative factor in painful tendinopathies [1, 67].

Eccentric strength training has been shown to be effective for treating Achilles [2, 28, 44, 77], patellar [69, 72], and shoulder tendinopathies [95]. More recently, eccentric training has been applied to LE. Crossier et al. [23] demonstrated improvement in pain after eccentric exercise performed on an isokinetic dynamometer which necessitated patients going to a clinic for treatments. Isokinetic dynamometers are expensive and not widely available, therefore it is not a viable treatment option for most patients with lateral epicondlyosis. Tyler et al. [91] conducted a prospective randomized, controlled trial which compared standard treatment (stretching, ultrasound, cross-friction massage, heat and ice) to standard treatment plus eccentric loading. These authors used an inexpensive rubber bar (FlexBar, Thera-Band; Hydenic Corporation, Akron, OH) to perform eccentric exercises performing 3 sets of 15 repetitions daily for approximately 6 weeks. They found an improvement in the eccentric resistance group in all outcome measures utilized (including visual analog scale, Disability of the Arm, Shoulder, and Hand (DASH) Score, and strength) compared to the traditional group. Although further study is needed on the long-term effectiveness of eccentric loading to LE, evidence exists for the short-term benefits of this form of exercise. At our center, we advocate that chronic cases follow a home exercise program which includes a combination of isotonic contractions performed in

stages and stretching exercises. This program is a modification of an exercise program advocated by Nirschl and Sobel [64] and consists of three stages of self-progression, which is based on the patient's response to the program. Please see Fig. 6.4 for program details.

Equipment or Aspect of Equipment	Suggested Modification	Explanation
Racquet	Midsize frame with medium flexibility 90–95 square inches	The greater the stiffness, the greater the shock on the arm on impact with ball, particularly with off-center impact; midsize frames have less stiffness
String tension	Lower the string tension by a few pounds	Creates a greater postimpact ball velocity, which produces greater power with less stroke effort
Proper grip size	Larger handle preferred	Larger grip will require less effort to hold on to racquet
Tightness of grip	Use submaximal grip strength	Elite players use submaximal grip tightness to minimize wrist stiffness and fatigue; avoid "deathgrip" common in novice players
Grip bands	Apply to handle	May reduce impact vibration and prevent slippage, allowing a looser grip
Tennis court	Play on slower court surfaces such as clay or asphalt	Slows ball velocity, which will decrease impact forces
Tennis balls	Use new tennis balls	Less impact force and less power required to hit them
Stroke modification	Use correct stroke mechanics; seek professional instruction	Hitting the ball incorrectly has been associated with increased incidence of elbow pain

Fig. 6.4 Progressive stretching exercises allow elongation of the muscle–tendon unit in an effort to reduce pain and stiffness. Patients are instructed to perform passive wrist flexion exercises with variable amounts of elbow extension to maintain length of the musculotendinous unit. Figures **a–c** are demonstrated passive wrist flexion exercises with variable amounts of elbow extension

Soft Tissue Mobilization

Soft tissue mobilization has been advocated for use with tendinopathies in order to attempt to reduce pain and to promote tissue healing. Deep transverse friction massage, a treatment advocated by Cyriax, has been the subject of a Cochrane review [15]. Only two randomized controlled trials met sufficient quality standards and were included in the review, however, no conclusion on effectiveness could be drawn. Other soft tissue mobilization techniques are popular in clinical practice and it is postulated that such techniques increase blood flow in the vicinity of the injured tendon which is thought to promote healing. Furthermore, massage techniques promote muscle relaxation through passive movement of the tissue. However, clinical trials with adequate sample sizes and strong methodology are lacking.

Maintenance Phase: Work and Sports Modifications

Somewhat of a misnomer, "tennis elbow" actually occurs in fewer than 5–10% of tennis players [4, 21]. Repetitive work activities that may lead to the development of LE include such activities as repetitive gripping and twisting, pulling, and static postures of the upper extremity. LE is associated with jobs that require repeated or forceful movements of the fingers, wrist, and forearm. It can develop as a result of too much force exerted at once or small amounts of microtrauma over a prolonged period. When the therapist identifies which work activities may be related to the cause of lateral elbow pain, they should suggest the patients discuss with their employer the need for an ergonomic evaluation. Suggestions can be provided to the patient in the interim such as: performing gripping activities with the wrist in self-selected comfortable positions; enlarged tool handles to match the size of the individual's hand; office keyboard and workstation setup; the need for frequent rest periods to make lengthy tasks smaller and more manageable.

Sports Modifications

As the patient gradually attempts to return to sport and other recreation activities, the general practitioner and therapist should provide some general information and guidelines for the patient to consider. For example, the patient's return to recreational activity should be slow and gradual (i.e. 15–30 min initial maximum duration, gradually increased by 15 min intervals as tolerated). We also initially advise 2–3 days of rest between activity days. With regards to racquet sports and specific equipment and stroke modifications, the patient should be encouraged to seek the advice of a tennis or other racquet sports professional. General practitioners and therapists should be aware of some general considerations for stroke and equipment modifications with various racquet sports. See table given below for general considerations for stroke and equipment modification.

Outcome Measures

The reported effectiveness of a therapeutic intervention is dependent on the outcome measure used. When reviewing the literature to determine effectiveness of rehabilitation interventions, there is considerable variation among studies in part due to the lack of standardized outcome measures. This makes generalization of the effectiveness of rehabilitation difficult. Clinicians should become comfortable with using outcome measures at regular intervals during active rehabilitation in order to objectively evaluate interventions. However, with this patient population we must select measures that are reflective of their chief complaints, which typically include pain, loss of function, and muscle weakness. When clinicians and researchers are selecting an outcome measure to use, it is important to use measures that have been validated and proven to be responsive in the target population. We recommend that the following components be evaluated: pain, strength, and function.

Furthermore, we should administer the outcome measures at baseline, frequent intervals during rehabilitation (i.e., 2, 6, and 12 weeks), at discharge, and at long-term follow-up (such as 6 months–1 year).

In the early phases of rehabilitation, the visual analog scale or numeric pain rating scale should be used to assess pain at rest as well as pain with activity. Also, the Patient-Rated Tennis Elbow Evaluation (PRTEE) can be used to measure pain as it includes a subscale for pain and function [58, 68]. The PRTEE has been shown to be the most responsive outcome measure in this patient population [3, 14, 75] and should be used as the primary outcome measure in the research of LE [75]. (See Appendix A) The DASH [8] is one of the most commonly used outcome measures in the upper extremity, however it may not be sensitive to change in any one particular portion of the upper extremity such as the lateral epicondyle. Therefore, the PRTEE is a more appropriate choice in our opinion and more likely to detect change.

To measure strength and muscle irritability, pain-free grip strength testing should be used. Pain-free grip has been shown to be more sensitive to change compared to maximal grip [85]. Pain-free grip can be assessed by using a hand dynamometer (Jamar, Preston Rolyan, Bolingbrook, IL). Dorf et al. [25] found that extended elbow grip strength testing compared to flexed elbow grip strength testing caused an increase in lateral elbow pain and decreased strength. Thus, it can be extrapolated that patients should be instructed to grip the instrument with the elbow extended, forearm in neutral rotation with the shoulder adducted versus the method advocated by the American Society of Hand Therapists [32], which positions the patient with the elbow at 90° of flexion. The patient should be instructed to grip the instrument, stopping immediately when pain is first sensed in the lateral elbow. The average of three trials should be recorded.

To assess function and the resumption of meaningful activity, the clinician and researcher have a few choices. The PRTEE can be used, as it has a subscale for function. The Canadian Occupational Performance Measure is an individualized outcome measure designed to detect change in a patient's self-perception of resumption of meaningful activity [50]. The Patient Specific Functional Scale has been found to be a valid, reliable, and responsive measure for detecting a meaningful change in function for patients with a musculoskeletal dysfunction affecting the upper extremity [39].

Summary

Therapeutic management of LE requires a comprehensive rehabilitation approach. This chapter provides the reader with a review of various therapeutic techniques typically applied. Furthermore, an evidence-informed algorithmic approach to rehabilitation is provided based on the stage of healing. There is a need for randomized, controlled clinical trials to confirm which rehabilitation techniques produce optimal outcomes.

Appendix A:

PATIENT RATED TENNIS ELBOW EVALUATION

1. PAIN	*Rate the average amount of pain in your arm over the past week by circling the number that best describes your pain on a scale from 0-10. A zero (0) means that you did not have any pain and a ten (10) means that you had the worst pain you have ever experienced or that you could not do the activity because of pain.*

Sample scale	0 1 2 3 4 5 6 7 8 9 10 No Pain Worst Ever
RATE YOUR PAIN:	No Pain Worst ever
When you are at rest	0 1 2 3 4 5 6 7 8 9 10
When doing a task with a repeated arm movement	0 1 2 3 4 5 6 7 8 9 10
When carrying a plastic bag of groceries	0 1 2 3 4 5 6 7 8 9 10
When it is at its least	0 1 2 3 4 5 6 7 8 9 10
When your pain was at it's worst	0 1 2 3 4 5 6 7 8 9 10

A. SPECIFIC ACTIVITIES

*Rate the **amount of difficulty** you experienced performing each of the items listed below over the past week, by circling the number that describes your difficulty on a scale of 0-10. A **zero** (0) means you did not experience any difficulty and a **ten** (10) means it was **so difficult you were unable to do it at all**.*

Sample scale 0 1 2 3 4 5 6 7 8 9 10

No Difficulty **Unable To Do**

Turn a door knob or key	0 1 2 3 4 5 6 7 8 9 10
Carry a grocery bag or briefcase by the handle	0 1 2 3 4 5 6 7 8 9 10
Lift a full coffee cup or glass of milk to your mouth	0 1 2 3 4 5 6 7 8 9 10
Open a Jar	0 1 2 3 4 5 6 7 8 9 10
Pull up pants	0 1 2 3 4 5 6 7 8 9 10
Wring out a washcloth or wet towel	0 1 2 3 4 5 6 7 8 9 10

B. USUAL ACTIVITIES

*Rate the **amount of difficulty** you experienced performing your **usual** activities in each of the areas listed below, over the past week, by circling the number that best describes your difficulty on a scale of 0-10. By usual activities, we mean the activities you performed **before** you started having a problem with your arm. A **zero** (0) means that you did not experience any difficulty and a **ten** (10) means it was so difficult you were unable to do any of your usual activities.*

Personal care activities (dressing, washing)	0 1 2 3 4 5 6 7 8 9 10
Household work (cleaning, maintenance)	0 1 2 3 4 5 6 7 8 9 10
Work (your job or usual everyday work)	0 1 2 3 4 5 6 7 8 9 10
Recreational activities or sporting activities	0 1 2 3 4 5 6 7 8 9 10

References

1. Alfredson H. Chronic midportion Achilles tendinopathy: an update on research and treatment. Clin Sports Med. 2003;22:727–41.
2. Alfredson H, Pietila T, Jonsson P, Lorentzon R. Heavy-load eccentric calf muscle training for the treatment of chronic Achilles tendinosis. Am J Sports Med. 1998;26:360–6.

3. Altan L, Ercan I, Konur S. Reliability and validity of Turkish version of the patient rated tennis elbow evaluation. Rheumatol Int. 2010;30:1049–54.
4. Assendelft WJ, Hay EM, Adshead R, Bouter LM. Corticosteroid injections for lateral epicondylitis: a systematic overview. Br J Gen Pract. 1996;46:209–16.
5. Bales CP, Placzek JD, Malone KJ, Vaupel Z, Arnoczky SP. Microvascular supply of the lateral epicondyle and common extensor origin. J Shoulder Elbow Surg. 2007;16(4):497–501.
6. Barr S, Cerisola FL, Blanchard V. Effectiveness of corticosteroid injections compared with physiotherapeutic interventions for lateral epicondylitis: a systematic review. Physiotherapy. 2009;95(4):251–65.
7. Basford JR, Sheffield CG, Cieslak KR. Laser therapy: a randomized, controlled trial of the effects of low intensity Nd:YAG laser irradiation on lateral epicondylitis. Arch Phys Med Rehabil. 2000;81:1504–10.
8. Beaton DE, Katz JN, Fossel AH, Wright JG, Tarasuk V, Bombardier C. Measuring the whole or the parts? Validity, reliability, and responsiveness of the disabilities of the arm, shoulder and hand outcome measure in different regions of the upper extremity. J Hand Ther. 2001;14(2):128–46.
9. Binder Al, Hazlernan BL. Lateral humeral epicondylitis—a study of natural history and the effect of conservative therapy. Br J Rheumatol. 1983;22:73–6.
10. Binder A, Hodge G, Greenwood AM, Hazleman BL, Thomas DPP. Is therapeutic ultrasound effective in treating soft tissue lesions? BMJ. 1985;290:512–4.
11. Bingol U, Altan L, Yurtkuran M. Low-power laser treatment for shoulder pain. Photomed Laser Surg. 2005;23:459–64.
12. Bisset L, Paungmali A, Vicenzino B, Beller E. A systematic review and meta-analysis of clinical trials on physical interventions for lateral epicondylalgia. Br J Sports Med. 2005;39(7):411–22.
13. Bjordal JM, Lopes-Martins RA, Joensen J, Couppe C, Ljunggren AE, Stergioulas A, Johnson MI. A systematic review with procedural assessments and meta-analysis of low level laser therapy in lateral elbow tendinopathy (tennis elbow). BMC Musculoskelet Disord. 2008;9:75.
14. Blanchette MA, Normand MC. Cross-cultural adaptation of the patient-rated tennis elbow evaluation to Canadian French. J Hand Ther. 2010;23(3):290–9.
15. Brosseau L, Casimiro L, Milne S, et al. Deep transverse friction massage for treating tendinitis (Cochrane Review). Cochrane Database Syst Rev. 2002;4:CD003528.
16. Buchbinder R, Green SE, Youd JM, Assendelft WJ, Barnsley L, Smidt N. Systematic review of the efficacy and safety of shock wave therapy for lateral elbow pain. J Rheumatol. 2006;33:1351–63.
17. Bunata RE, Brown DS, Capelo R. Anatomic factors related to the cause of tennis elbow. J Bone Joint Surg Am. 2007;89(9):1955–63.
18. Calfee RP, Patel A, DaSilva MF, Akelman E. Management of lateral epicondylitis: current concepts. J Am Acad Orthop Surg. 2008;16(1):19–29.
19. Chang WD, Wu JH, Yang WJ, Jiang JA. Therapeutic effects of low-level laser on lateral epicondylitis from differential interventions of Chinese-Western medicine: systematic review. Photomed Laser Surg. 2010;28(3):327–36.
20. Ciccotti MG, Ramani MN. Medial epicondylitis. Techn Hand Upper Extrem Surg. 2003;7(4):190–6.
21. Coonrad RW, Hooper WR. Tennis elbow: its course, natural history, conservative and surgical management. J Bone Joint Surg Am. 1973;55:1177–82.
22. Cowan J, Lozano-Calderon S, Ring D. Quality of prospective controlled randomized trials: analysis of trials of treatment for lateral epicondylitis as an example. J Bone Joint Surg Am. 2007;89:1693–9.
23. Croisier JL, Foidart-Dessalle MI, Tinant F, et al. An isokinetic eccentric programme for the management of chronic lateral epicondylar tendinopathy. Br J Sports Med. 2001;41:269–75.

24. Croisier JL, Foidart-Dessalle M, Tinant F, Crielaard JM, Forthomme B. An isokinetic eccentric programme for the management of chronic lateral epicondylar tendinopathy. Br J Sports Med. 2007;41(4):269–75.
25. Dorf ER, Chhabra AB, Golish SR, McGinty JL, Pannunzio ME. Effect of elbow position on grip strength in the evaluation of lateral epicondylitis. J Hand Surg Am. 2007;32(6):882–6.
26. el Hawary R Stanish WD Curwin SL. Rehabilitation of tendon injuries in sport. Sports Med. 1997;24:347–58.
27. Ellenbecker TS, Mattalino AJ. The elbow in sport: injury, treatment, and rehabilitation. Champaign: Human Kinetics; 1997.
28. Fahlstrom M, Jonsson P, Lorentzon R, Alfredson H. Chronic Achilles tendon pain treated with eccentric calf-muscle training. Knee Surg Sports Traumatol Arthrosc. 2003;11:327–33.
29. Faro F, Wolf JM. Lateral epicondylitis: review and current concepts. J Hand Surg Am. 2007;32(8):1271–9.
30. Fedorczyk JM. Tennis elbow: blending basic science with clinical practice. J Hand Ther. 2006;19(2):146–53.
31. Fedorczyk JM. Elbow tendinopathies: clinical presentation and therapist's management of tennis elbow. In: Skirven TM, Osterman AL, Fedorczyk JM, Amadio PC, editors. Rehabilitation of the hand and upper extremity. 6th edn. St. Louis: C.V. Mosby; 2011. pp. 1098–108.
32. Fess EE, Moran C. Clinical assessment recommendations. Indianapolis: American society of hand therapists monograph; 1981.
33. Garg R, Adamson GJ, Dawson PA, Shankwiler JA, Pink MM. A prospective randomized study comparing a forearm strap brace versus a wrist splint for the treatment of lateral epicondylitis. J Shoulder Elbow Surg. 2010;19(4):508–12.
34. Gerr F, Marcus M, Monteilh C, Hannan L, Ortiz DJ, Kleinbaum D. A randomized control trial of postural interventions for prevention of musculoskeletal symptoms among computer users. Occup Environ Med. 2005;62(7):478–87.
35. Pattanittum P, Turner T, Green S, Buchbinder R. Non-steroidal anti-inflammatory drugs (NSAIDs) for treating lateral elbow pain in adults. Cochrane Database Syst Rev. 2013;(5):CD003686. doi: 10.1002/14651858.CD003686.pub2.
36. Greenfield C, Webster V. Chronic lateral epicondylitis: survey of current practice in outpatient departments in Scotland. Physiotherapy. 2002;88(10):578–94.
37. Haker E, Lundeberg T. Is low-energy laser treatment effective in lateral epicondylalgia? J Pain Symptom Manage. 1991;6:241–6.
38. Halle JS, Franklin Orthop Sports Phys Ther. 1986;8:62–9.
39. Hefford C, Abbott JH, Arnold R, Baxter GD. The patient-specific functional scale: validity, reliability, and responsiveness in patients with upper extremity musculoskeletal problems. J Orthop Sports Phys Ther. 2012;42(2):56–65. doi: 10.2519/jospt.2012.3953. Epub 2012 Feb 1.
40. Holdsworth LK, Anderson DM. Effectiveness of ultrasound used with a hydrocortisone coupling medium or epicondylitis clasp to treat lateral epicondylitis: pilot study. Physiotherapy. 1993;79:19–24.
41. Hoppenrath T, Ciccone CD. Is there evidence that phonophoresis is more effective than ultrasound in treating pain associated with lateral epicondylitis? Phys Ther. 2006;86(1):136–40.
42. Ilfeld FW, Field SM. Treatment of tennis elbow. Use of a special brace. JAMA. 1966;195:67–70.
43. Jansen CW, Olson SL, Hasson SM. The effect of use of a wrist orthosis during functional activities on surface electromyography of the wrist extensors in normal subject. J Hand Ther. 1997;10:283.
44. Jonsson P, Wahlstrom P, Ohberg L, Alfredson H. Eccentric training in chronic painful impingement syndrome of the shoulder: results of a pilot study. Knee Surg Sports Traumatol Arthrosc. 2006;14:76–81. doi:10.1007/~00167–004-0611-8.
45. Khan KM, Cook JL, Bonar F, Harcourt P, Astrom M. Histopathology of common tendinopathies: update and implications for clinical management. Sports Med. 1999;27(6):393–408.
46. Klaiman MD, Shrader JA, Danoff JV, et al. Phonophoresis versus ultrasound in the treatment of common musculoskeletal conditions. Med Sci Sports Exerc. 1998;30:1349–55.

47. Kohia M, Brackle J, Byrd K, et al. Effectiveness of physical therapy treatments on lateral epicondylitis. J Sport Rehabil. 2008;17(2):119–36.
48. Kraushaar BS, Nirschl RP. Tendinosis of the elbow (tennis elbow). Clinical features and findings of histological, immunohistochemical, and electron microscopy studies. J Bone Joint Surg Am. 1999;81(2):259–78.
49. Lam LK, Cheing GL. Effects of 904-nm low-level laser therapy in the management of lateral epicondylitis: a randomized control trial. Photomed Laser Surg. 2007;25:65–71.
50. Law M, Baptiste S, McColl MA, Opzoomer A, Polatajko H, Pollock N. The Canadain occupational performance measure: an outcome measure for occupational therapy. Can J Occup Ther. 1990;57:82–7.
51. Ljung BO, Forsgren S, Friden J. Substance P and calcitonin gene-related peptide expression at the extensor carpi radialis brevis muscle origin: implications for the etiology of tennis elbow. J Orthop Res. 1999;17(4):554–9.
52. Lundeberg T, Haker E, Thomas M. Effect of laser versus placebo in tennis elbow. Scand J Rehabil Med. 1987;19:135–8.
53. Lundeberg T, Abrahamsson P, Haker E. A comparative study of continuous ultrasound, placebo ultrasound and rest in epicondylalgia. Scand J Rehabil Med. 1988;20:99–101.
54. MacDermid JC, Wojkowski S, Kargus C, Marley M, Stevenson E. H and therapist management of the lateral epicondylosis: a survey of expert opinion and practice patterns. J Hand Ther. 2010;23(1):18–29.
55. Manias P, Stasinopoulos D. A controlled clinical pilot trial to study the effectiveness of ice as a supplement to the exercise programme for the management of lateral elbow tendinopathy. Br J Sports Med. 2006;40(1):81–5.
56. Marcus M, Ger F, Monteilh C, Ortiz DJ, Gentry E, Cohen S, Edwards A, Ensor C, Kleinbaum D. A prospective study of computer users: postural risk factors for musculoskeletal symptoms and disorders. Am J Ind Med. 2002;41:236–49.
57. Martinez-Silvestrini JA, Newcomer KL, Gay RE, Schaefer MP, Kortebein P, Arendt KW. Chronic lateral epicondylosis: comparative effectiveness of a home exercise program including stretching alone versus stretching supplemented with eccentric or concentric strengthening. J Hand Ther. 2005;18:411–9. (quiz 420).
58. Newcomer KL, Martinez-Silvestrini JA, Schaefer MP, Gay RE, Arendt KW. Sensitivity of the Patient-rated Forearm Evaluation Questionnaire in lateral epicondylitis. J Hand Ther. 2005; 18:400–6.
59. Ng GY, Chan HL. The immediate effects of tension of counterforce forearm brace on neuromuscular performance of wrist extensor muscles in subjects with lateral humeral epicondylosis. J Orthop Sports Phys Ther. 2004;34(2):72–8.
60. Nirschl RP. Tennis elbow. Orthop Clin North Am. 1973;4:787–800.
61. Nirschl RP. Prevention and treatment of elbow and shoulder injuries in the tennis player. Clin Sports Med. 1988;7(2):289–308.
62. Nirschl RP. Elbow tendinosis/tennis elbow. Clin Sports Med. 1992;11(4):851–70.
63. Nirschl RP, Pettrone FA. Tennis elbow. The surgical treatment of lateral epicondylitis. J Bone Joint Surg Am. 1979;61:832–9.
64. Nirschl RP, Sobel JB. Rehabilitative exercise program. In: Arm care: a complete guide to prevention and treatment of tennis elbow. Arlington: Medical Sports, Inc; 1996. p. 39–64.
65. Nirschl RP, Rodin DM, Ochiai DH, Maartmann-Moe C. Iontophoretic administration of dexamethasone sodium phosphate for acute epicondylitis. Am J Sports Med. 2003;31(2):189–95.
66. O'Driscoll SW, Horii E, Ness R, et al. The relationship between wrist position, grasp size, and grip strength. J Hand Surg Am. 1992;17A:169–77.
67. Ohberg L, Alfredson H. Effects on neovascularisation behind the good results with eccentric training in chronic mid-portion Achilles tendinosis. Knee Surg Sports Traumatol Arthrosc. 2004;12:465–70.
68. Overend TJ, Wuori-Fearn JL, Kramer JF, MacDermid JC. Reliability of a patient-rated forearm evaluation questionnaire for patients with lateral epicondylitis. J Hand Ther. 1999;12:31–7.

69. Panni AS, Tartarone M, Maffulli N. Patellar tendinopathy in athletes: outcome of nonoperative and operative management. Am J Sports Med. 2000;28:392–7.
70. Pienimaki TT, Tarvainen TK, Siira PT, Vanharanta H. Progressive strengthening and stretching exercises and ultrasound for chronic lateral epicondylosis. Physiotherapy. 1996;82(9): 522–30.
71. Plancher KD, Halbercht J, Lourie GM. Medial and lateral epicondylitis in the athlete. Clin Sports Med. 1996;15:283–305.
72. Purdam CR, Jonsson P, Alfredson H, Lorentzon R, Cook JL, Khan KM. A pilot study of the eccentric decline squat in the management of painful chronic patellar tendinopathy. Br J Sports Med. 2004;38:395–7.
73. Regan W, Wold LE, Coonrad R, Morrey BF. Microscopic histopathology of chronic refractory lateral epicondylitis. Am J Sports Med. 1992;20(6):746–9.
74. Rompe JD, Maffulli N. Repetitive shock wave therapy for lateral elbow tendinopathy (tennis elbow): a systematic and qualitative analysis. Br Med Bull. 2007;83:355–78.
75. Rompe JD, Overend TJ, MacDermid JC. Validation of the patient-rated tennis elbow evaluation questionnaire. J Hand Ther. 2007;20(1):3–10.
76. Sevier TL, Wilson JK. Treating lateral epicondylitis. Sports Med. 1999;28(5):375–80.
77. Shalabi A, Kristoffersen-Wilberg M, Svensson L, Aspelin P, Movin T. Eccentric training of the gastrocnemius-soleus complex in chronic Achilles tendinopathy results in decreased tendon volume and intratendinous signal as evaluated by MRI. Am J Sports Med. 2004;32:1286–96.
78. Smidt N, van der Windt DA. Tennis elbow in primary care. BMJ. 2006;333:927–8.
79. Smidt N, Assendelft WJ, van der Windt DA, Hay EM, Buchbinder R, Bouter LM. Corticosteroid injections for lateral epicondylitis: a systematic review. Pain. 2002;96(1–2):23–40.
80. Smidt N, Assendelft WJ, Arola H, Malmivaara A, Greens S, Buchbinder R, van der Windt DA, Bouter LM. Effectiveness of physiotherapy for lateral epicondylitis: a systematic review. Ann Med. 2003;35:51–62.
81. Smith RW, Papadopolous E, Mani R, Cawley MI. Abnormal microvascular responses in a lateral epicondylitis. Br J Rheumatol. 1994;33(12):1166–8.
82. Snyder-Mackler L, Epler M. Effect of standard and aircast tennis elbow bands on integrated electromyography of forearm musculature proximal to the bands. Am J Sports Med. 1989;17:278–81.
83. Stanish WD, Rubinovich RM, Curwin S. Eccentric exercise in chronic tendonitis. Clin Orthop Rel Res. 1986;208:65–8.
84. Stergioulas A. Effects of low-level laser and plyometric exercises I the treatment of lateral epicondylitis. Photomed Laser Surg. 2007;25:205–13.
85. Stratford PW, Levy DR. Assessing valid change over time in patients with lateral epicondylitis at the elbow. Clin J Sport Med. 1994;4:88–91.
86. Stratford PW, Levy DR, Gauldie S, Miseferi D, Levy K. The evaluation of phonophoresis and friction massage as treatments for extensor carpi radialis tendonitis: a randomized controlled trial. Physiother Can. 1989;41:93–8.
87. Struijs PAA, Smidt N, Arola H, van Dijk CN, Buchbinder R, Assendelft WJ. Orthotic devices for the treatment of tennis elbow. Cochrane Database Syst Rev. 2002;(1):CD001821.
88. Tonks JH, Pai SK, Murali SR. Steroid injection therapy is the best conservative treatment for lateral epicondylitis: a prospective randomised controlled trial. Int J Clin Pract. 2007;61(2):240–6.
89. Tuner J, Hode L. Low Level Laser Therapy. Prima Books, Stockholm, Sweden. 1999
90. Tuner J, Hode L. Laser therapy—clinical practice and scientific background. Grängesberg: Prima Books AB; 2002.
91. Tyler TF, Thomas GS, Nicholas SJ, McHugh MP. Addition of isolated wrist extensor eccentric exercise to standard treatment for chronic lateral epicondylosis: a prospective randomized trial. J Shoulder Elbow Surg. 2010;19(6):917–22.
92. Vasseljen O Jr., Hoeg N, Kjeldstad B, Johnsson A, Larsen S. Low level laser versus placebo in the treatment of tennis elbow. Scand J Rehabil Med. 1992;24:37–42.

93. Walz DM, Newman JS, Konin GP, Ross G. Epicondylitis: pathogenesis, imaging, and treatment. Radiographics. 2010;30(1):167–84.
94. Werner RA, Franzblau A, Gell N, Hartigan A, Ebersole M, Armstrong TJ. Predictors of persistent elbow tendonitis among auto assembly workers. J Occup Rehabil. 2005;15(3):393–400.
95. Woodley BL, Newsham-West RJ, Baxter GD. Chronic tendinopathy: effectiveness of eccentric exercise. Br J Sports Med. 2007;41:188–98.

Chapter 7
Steroid Injections

Igor Immerman and Robert M. Szabo

Introduction

Since the introduction of corticosteroids for the treatment of rheumatoid arthritis in 1949, they have become ubiquitous in musculoskeletal care [1]. The first use of steroids in lateral epicondylitis (LE) was described in 1953 by Hollander, and already at that time LE was recognized as a self-limited condition [2]. Currently, a simple search in PubMed for "lateral epicondylitis" and "corticosteroid" results in 40 scientific articles in the past 10 years, a testament to the ongoing interest in this treatment modality. Patients often request corticosteroids, or they present to the hand surgeon's practice having already received one or several injections from their primary care provider. The aim of this chapter is to describe the hypothesized mechanisms of action for corticosteroids in LE, and to summarize the recent scientific and clinical evidence on the topic.

Pathology and Mechanism of Action

Corticosteroids exhibit anti-inflammatory actions via their effects on inflammatory cytokines, thereby reducing immune function. They inhibit fibroblast proliferation, angiogenesis, and formation of granulation tissue. They also interfere with collagen precursor ground substance sulfation and collagen repair [1, 3]. The application of corticosteroids in LE stems from the theorized inflammatory component of the condition. Studies using laser and color doppler, as well as MRI, have demonstrated

R. M. Szabo (✉) · I. Immerman
Department of Orthopaedic Surgery, UC Davis Medical Center, 4860 Y Street, Suite 3800, Sacramento, CA 95817, USA
e-mail: robert.szabo@ucdmc.ucdavis.edu

I. Immerman
e-mail: Igor.immerman.md@gmail.com

J. M. Wolf (ed.), *Tennis Elbow,* DOI 10.1007/978-1-4899-7534-8_7

increased blood flow and edema at the lateral epicondyle in a majority of patients, consistent with an inflammatory process [4]. Patients undergoing injections of prednisone in the region of the lateral epicondyle had decreased hyperemia on Doppler ultrasound which correlated with symptom relief, supporting the anti-inflammatory effects of steroids in LE [4]. In another study, triamcinolone injections have been demonstrated to significantly decrease common extensor tendon thickness and color doppler activity in patients with LE when compared with saline controls or platelet-rich plasma (PRP) injections [5].

The most current histologic evidence suggests that LE may be a degenerative and not an inflammatory condition. The histopathology of LE is similar to that seen in other chronic tendinoses, such as Achilles tendinitis and patellar tendinosis [6]. Histologic studies of patients undergoing surgery for LE fail to demonstrate inflammation as a key feature, and the levels of prostaglandin E2 are not elevated compared to controls [7]. Rather, the process termed angiofibroblastic degeneration, with immature fibroblastic and vascular infiltration, and disorganized collagen, has been implicated in the pathogenesis of LE [4, 8–12]. However, it is possible that inflammation is present in the acute stages of the condition, and progresses to a degenerative process as the disease evolves. Surgical treatment is rarely done in the acute stages of the LE, and therefore surgical biopsies of chronic cases would unlikely demonstrate inflammation, a limitation of the currently available histological data. This concept seems to agree with the common finding of short-term efficacy of corticosteroids (presumably in the inflammatory stage), and poor long-term efficacy, as they have no known beneficial effects on degenerative tissues.

Others have suggested a neurogenic cause for pain in tendinopathy, based on a study that showed presence of nerve fibers with reactivity to substance P and calcitonin gene-related peptide (CGRP) in LE; corticosteroids may also act by altering the levels of these substances [13]. It has also been postulated that the short-term pain relief associated with corticosteroid injections may be related to blockade of glutamate and glutamate receptors, elevated levels of which have been associated with pain generation in various enthesopathies, including LE [7, 14].

In summary, the weight of the evidence suggests LE to be a degenerative process rather than an inflammatory condition. The precise mechanism of action of corticosteroid injections in LE is still undefined, and research is ongoing.

Corticosteroid Formulations and Injection Techniques

Many formulations of injectable corticosteroids are currently on the market, and the literature on LE describes the use of triamcinolone (10–20 mg), methylprednisolone (20–40 mg), betamethasone (6 mg), dexamethasone (4–10 mg), and hydrocortisone (25 mg) [8, 14–22]. These compounds vary in their active ingredient, fat- and water-solubility, duration of action, and concentration. Dexamethasone has a relatively long biologic half-life (between 36 and 72 h), and is followed in decreasing order by betamethasone, triamcinolone, methylprednisolone, and hydrocortisone.

Formulations with corticosteroids esters are not soluble in water and form crystalline suspensions, which theoretically have a longer onset and longer duration of action. Others, such as dexamethasone, are freely soluble in water and therefore are more bioavailable. Betamethasone (Celestone Soluspan, marketed by Merck & Co, Whitehouse Station, NJ) is a combination of both suspensions, which theoretically provides both rapid onset and longer duration of action. Another consideration is the propensity of the steroid to form particulate aggregates: triamcinolone has the highest, followed by betamethasone, while dexamethasone remains soluble after injection [3, 23]. However, despite theoretical differences, clinical outcomes are essentially the same. In a systematic review, Barr et al. compared studies with triamcinolone 10 mg and prednisolone 40 mg, and did not detect any clinical differences [8]. Similarly, in a study that compared injections with triamcinolone to injections with dexamethasone, no clinical differences were noted at 8 weeks or 6 months [14]. The choice of steroid may theoretically affect tendon strength postinjection, as discussed in more detail in the section on complications. Longer-acting, less soluble preparations have been suggested to carry a higher risk of skin atrophy [3, 21]

The number of injections that have been attempted in the course of treatment varies amongst studies, with the majority allowing for an additional one or two injections following the initial treatment, usually with a 2 week interval, although some series have reported as many as 20 injections in some patients [1, 10]. An increased short-term benefit has been observed with increasing number of injections in some studies, but this is not a consistent finding [8, 24]. It is not clear from the literature whether the rate of complications increases with an increasing number of injections, but because of the concerns of tendon rupture, soft tissue atrophy, and other complications most physicians limit the frequency and number of injections.

The typical injection consists of a combination of a corticosteroid and local anesthetic. The typical volume of the injection varies between 1 and 3 ml, with 1–2 ml of local anesthetic. Lidocaine (1–2 %, without epinephrine) is used most commonly, but prilocaine use has also been reported. There do not appear to be any notable differences in outcomes based on the type or concentration of local anesthetic used, so its choice remains up to the practitioner.

The most common site for the injection is at the most tender spot over the lateral elbow, with some studies specifying the injection aimed at the common extensor origin, or the muscle itself, with no apparent difference in outcomes [8, 20, 25]. One study reported on a technique of triple injections: the first injection of 1 ml volume was directed into the radial side of the annular ligament, the second 0.5 ml into the common extensor origin over the lateral epicondyle, and the third 0.5 ml, without withdrawing the needle, into the radial collateral ligament [9]. Ultrasound has been used to guide the injection material into the common extensor tendon [4]. Overall, there is no strong evidence to recommend any specific technique.

The use of a single needle puncture has been compared to a peppering technique of multiple passes (usually more than 30) of the needle during one injection. Bellapianta et al. performed a prospective randomized study comparing the two methods, and it appeared that single-injection outperformed the peppered technique; however the study was underpowered, was not blinded, and had a high loss of follow up [26].

This is weighed against other prospective randomized trials showing improved disabilities of the arm, shoulder, and hand (DASH) scores, pain levels, and clinical assessment scores in groups treated with a peppering injection technique [19, 27]. The hypothesized mechanism of action of the peppering technique involves the stimulation of bleeding and creation of multiple channels in the degenerated myxoid tissue at the lateral epicondyle, in order to stimulate healing. This concept is somewhat supported by findings that peppering with lidocaine alone, with or without corticosteroids, may have equally beneficial effects [28].

Postinjection recommendations usually include rest from strenuous activity and gradual return to normal activity over 1–2 weeks, with avoidance of aggressive activities. No studies have explicitly compared postinjection protocols [15, 16].

Efficacy of Corticosteroids

Multiple prospective randomized trials, of variable quality, have been performed over the recent years evaluating the efficacy of corticosteroid injections for the treatment of LE. The injections have been compared to physical therapy, observation, and various other treatment modalities. In the next section we describe the recent published evidence comparing corticosteroid injections to other treatment options.

Corticosteroids vs. Placebo

Lindenhovius et al. performed a level 1 study with a total of 66 patients randomized to an injection of dexamethasone with lidocaine vs. lidocaine alone. The injection was performed at the point of maximal tenderness, and spread out over multiple points, with two patients in the steroid and four in the lidocaine groups receiving a repeated injection. There were no significant differences between the groups in the grip strength, pain, or the DASH at 1- and 6-month follow up. This study was limited by a significant loss to follow up [18].

Newcomer et al. randomized 39 patients to receive either a betamethasone with bupivacaine or a bupivacaine-only injection; all patients received physical therapy as part of the treatment. Patients were evaluated at 4 weeks, 8 weeks, and 6 months, and there were no differences between the groups in terms of pain or grip strength. The only statistical significant difference was the larger magnitude of improvement in the Visual Analogue Scale (VAS) between 8 weeks and 6 months seen in the corticosteroid group, which could reflect the slightly higher pain observed at 8 weeks in the corticosteroid group [22].

Altay et al. compared 60 patients treated with lidocaine alone to 60 patients treated with triamcinolone and lidocaine; all injections were done using a peppering technique. The patients were evaluated at 2, 6, and 12 month intervals, and there were no clinical outcome differences between the groups at any time point [28].

Corticosteroids vs. Physical Therapy vs. "Wait and See"

Bisset et al. randomized 198 patients with 6 weeks of symptoms into one of three treatment approaches, consisting of a single triamcinolone injection, physical therapy, or a "wait and see" approach. At the 6-week interval, injections were superior to "wait and see" and physical therapy in pain-free grip strength, assessor severity rating, pain intensity, and elbow disability, with 78% treatment success rate (measured by a patient-rated global improvement scale) in the injection group. However, at 52 weeks, the injection group was significantly worse than physical therapy by all of the above outcomes. In comparison to "wait and see," patients treated with corticosteroid injections tended to have worse results in all outcomes, but statistical significance was reached only for the assessor's severity scale and patient-rated global improvement scale. There was a high recurrence rate in the injection group, with 72% deteriorating after 3–6 weeks, while the majority of patients in the other two groups demonstrated continuous improvement with recurrence rates of 8% in physical therapy and 9% in the "wait and see" groups. After 1 year, the absolute rates of "success," as determined by the patient-rated global improvement scale, were 68% in the injection group, compared to 90% in the "wait and see" and 94% in the physical therapy groups [29].

A similar comparison by Smidt et al. randomized 185 patients to one of three treatment approaches with an intention-to-treat analysis. At 6 weeks, patients receiving corticosteroids were significantly better than the other groups in terms of general improvement, symptom severity, pain, elbow disability, and satisfaction. By 52 weeks, however, the corticosteroid group experienced a high-recurrence rate, and the long-term success rate was only 69%, compared to 91% for physical therapy and 83% for "wait and see" groups [30].

The short-term efficacy of corticosteroids is further confirmed by Tonks et al., who performed a randomized study comparing "wait and see," physiotherapy, steroids, and physiotherapy combined with steroids [20]. Patients with symptoms for 6 months without treatment were included and randomized to one of four groups, but the study was limited by poor methodology, relatively low patient numbers, and a high drop-out rate. Patients in the injection-only group did significantly better for all outcome measures at 7 weeks, whereas no significant differences from baseline were observed in PT and PT+ injection groups.

Finally a recent controlled, injection-blinded study randomized 165 patients into 4 groups: corticosteroid injection, placebo injection, corticosteroid injection plus physical therapy, and placebo injection plus physical therapy [15]. At 4 weeks, there was a clear benefit to patients receiving physical therapy, but no difference in improvement between patients treated with steroid injection alone or steroid injection with physical therapy (71% vs. 68%). Moreover, at 1 year, patients treated with a corticosteroid injection reported lower recovery and improvement (83% vs. 96%), and higher recurrence (54% vs. 12%) than patients treated with a placebo injection, regardless of physical therapy [15].

The above data confirm the short-term efficacy of steroid injections, but demonstrate the long-term better results of physical therapy, and even watchful waiting, compared to steroid injections. Subgroup analysis of patients undergoing treatment for LE demonstrates that physical therapy alone is not as beneficial in the short-term for patients with higher baseline pain, which supports the initial use of pain-relief modalities in these patients, including possibly injections. In addition, the employment status may have an effect on the long-term outcomes of steroid injections. Specifically at 1 year, manual laborers had no demonstrable difference between injection, PT, or "wait and see" groups, whereas in nonmanual workers, steroids were worse than both "wait and see" and physical therapy [31].

Corticosteroids vs. Prolotherapy

A randomized, double-blinded study compared prolotherapy with corticosteroid injections. The study was underpowered, but the results indicated improvement in pain and the DASH at both 3 and 6 month intervals compared to baseline, with no differences between the two experimental groups [9].

Corticosteroids vs. Autologous Blood Injections

A single-blinded, prospective randomized study was reported by Kazemi et al. and compared injection of 20 mg of methylprednisolone to a 2 ml injection of autologous blood, both mixed with 1 cc of 2% lidocaine. Measured outcomes included VAS, pain-free functional questionnaire, pain in maximum grip, Quick DASH questionnaire, modified Nirschl score, maximum grip strength, and pressure pain thresholds. At 8 weeks the corticosteroid group demonstrated improvement from baseline only in limb pain, and actually showed decreased grip strength, whereas the autologous blood group demonstrated improvement in all outcome measures. Between-group comparisons demonstrated improved outcomes in the autologous blood group at both 4 and 8 weeks [17].

A level 2 study by Wolf and colleagues randomized 34 patients to receive a single injection of lidocaine mixed with either saline, autologous blood, or a corticosteroid, injected with a peppering technique. There was a progressive improvement in the DASH in all groups up until the 6 months follow up, when the difference from baseline reached statistical significance. Similar patterns were seen for pain and function scores, with no benefit or disadvantage for the steroid group [32]. The above two studies further support the concept of inducing a hematoma at the lateral epicondyle, and further point out the short duration of the positive effects of the corticosteroid injection.

Corticosteroids vs. Extracorporeal Shock Wave Therapy (ESWT) vs. Physical Therapy Modalities

Gunduz et al. randomized 59 consecutive patients to one of three treatments: ESWT; physical therapy consisting of ultrasound, hot pack, and massage; and a single injection of 20 mg dexamethasone and 1 mg of prilocaine. At 1, 3, and 6 months, all patients improved in pain as measured by VAS score, with no significant differences between the groups. Grip strength was improved only at the 1 month point in the injection group, but at 1, 3, and 6-month visits in the ESWT group; however, direct comparisons between the study groups failed to demonstrate any statistically significant differences. In addition, ultrasound measurements of the tendon thickness or echogenicity were unchanged throughout the study, with no differences between the study arms [33].

Corticosteroids vs. Botulinum Toxin (Botox) Injections

A small study randomized patients to receive a single injection of 40 mg of triamcinolone or 50U botulinum toxin type A. After 4 weeks, the reduction in pain was significantly greater in the steroid group, but by 12 weeks the results equalized. An improvement in grip strength was seen at 4 weeks in the corticosteroid group, but by 12 weeks there was no statistically significant difference [25].

Corticosteroids vs. PRP Injections

Two recent Level 1 studies have compared the effects of steroid and PRP injections [5, 34]. Krogh et al. randomized 60 patients to one of three injections: 20 patients received 40 mg of triamcinolone mixed with 20 mg of lidocaine via an ultrasound-guided injection into the common extensor tendon; the other two groups received either 3 ml of saline or 3 ml of PRP, both injected with a peppering technique. All patients were advised to take acetaminophen as needed for pain, and perform a standard elbow stretching and training program. At 1 month, the steroid injection group had significantly lower pain and disability scores than either the PRP or the saline groups, but this difference was no longer present at 3 months. This study was limited by the relatively small groups as well as high drop-out rate after 3 months. In addition, 60% of the patients in the study had previously failed corticosteroid injections [5].

Gosens et al. compared 100 patients randomized to either a PRP or a corticosteroid injection using a peppering technique. After 2 years, both groups demonstrated significant improvement over baseline in both VAS and DASH scores. However, patients treated with PRP had significantly better DASH and VAS scores than the corticosteroid group at the 2-year follow up [34].

Systematic and Meta-Analysis Review of the literature

A systematic review of ten trials by Krogh et al. demonstrated that steroid injections were no more effective than placebo beyond 8 weeks, but overall quality of studies was low, and only one study was considered to have a low risk of bias [35].

Similarly, a meta-analysis by Coombes et al. noted that corticosteroid injections had short-term efficacy in terms of pain-reduction, functional improvement, and overall improvement. In the long-term, however, corticosteroids were associated with worse symptoms than no intervention, physical therapy, and PRP. There were no detectable differences between the type of steroid (triamcinolone or hydrocortisone), or between high- and low-dosage injections. Further, they found that the use of corticosteroids in the treatment of LE was associated with a 63% risk of symptom recurrence, as well as 21% reduction in the relative risk of improvement at 1 year. Moreover, patients with multiple injections experienced a 57% reduction in rate of treatment success compared to no intervention [24, 36].

Barr et al. performed a systematic review of five randomized controlled trials comparing corticosteroid injections to physical therapy, and their conclusions support the short-term benefit of steroid injections. At mid- and long-term intervals, however, physical therapy interventions were more effective than injections [8].

Cost-Effectiveness Analysis

The costs associated with corticosteroid injections have been compared to PRP injections in the randomized study mentioned above. The authors performed an analysis taking into account the initial treatment costs plus the costs of re-intervention, including surgery. Over a 2 year follow up, corticosteroids were slightly more cost-effective (88 units of money) than the PRP group (117 units of money) [34].

Another randomized controlled trial of 185 patients was used to compare costs between corticosteroid injections, physical therapy, and "wait and see" treatments. The direct health-care costs were highest in the physical therapy group and lowest in the "wait and see" group. The total costs (which included indirect costs such as absenteeism from work) were lowest in the corticosteroid group (430 €), vs. 631 € in the "wait and see" and 921 € in the physical therapy group. Nonetheless, the cost-effectiveness analysis failed to demonstrate the superiority of any one treatment approach [37].

Complications

Administration of corticosteroids, while relatively safe, is not completely without risks. The side effects can be classified as systemic or local. Systemic adverse effects include hypertension, glucose intolerance, Cushing's habitus, adrenal suppression,

and cataract formation. Repeated soft-tissue or intra-articular injections have been associated with intra-articular calcifications, osteonecrosis, and articular cartilage damage [1, 3]. These serious side effects are extremely rare, and have not been reported in any of the prospective studies examining the use of corticosteroids for LE.

It is much more, to encounter adverse reactions at the site of injection, with postinjection pain being the most common in studies of LE. A review of the literature on the use of corticosteroid injections for athletic injuries identified a complication rate 22.6% (244 out of 1078), but these numbers include series reporting exclusively on complications. Out of studies examining the efficacy of steroids, there was a 15.2% complication rate. Postinjection pain was seen in 9.7%, skin atrophy in 2.4%, skin depigmentation in 0.8%, localized erythema/warmth in 0.7%, and facial flushing in 0.6% of patients [1].

Tendon rupture due to an intra-tendinous injection is a particularly unwanted local complication. In a systematic review of prospective, randomized trials on the use of corticosteroid injections for tendinopathies, tendon rupture was seen in only one case out of 991 patients, in a patient treated for patellar tendinitis [36]. These numbers are contrasted by studies that exclusively examine complications of corticosteroid injections in the treatment of tendinopathies and other musculoskeletal conditions, where 53.7% of complications are plantar fascia ruptures, followed by a 9.5% rate of patellar/quadriceps tendon ruptures, 8.4% rate of Achilles tendon ruptures, and 7.4% rate of subcutaneous atrophy [1]. These studies suffer from an inclusion bias, and therefore the actual rate of serious complications, as evident by prospective trials, is lower. The choice of steroid may affect tendon strength postinjection, with more mechanical structural defects and higher rupture rate associated with triamcinolone vs. methylprednisolone, betamethasone, or hydrocortisone. There may also be a dose effect, as well as mechanical disruption of the tendon due to the injection technique [1]. Nonetheless, while it is likely that the majority of injections for LE are into or around the common extensor origin, there has been only one reported case of common extensor origin rupture [38].

Skin depigmentation and subcutaneous atrophy have been reported in a number of articles, in one case requiring surgical excision of the subcutaneous injection material deposits (in a case of medial epicondylitis) [1, 15, 18, 20, 21, 29, 39]. The overall rate appears to be in the 1.5–5% range, depending on the series. Relative risks for the subcutaneous atrophy and depigmentation have been reported as 1.77 and 0.53, respectively, and in both cases 95% confidence intervals included 1.0, suggesting no statistical difference from placebo [18, 36]. In one study, triamcinolone was associated with a higher risk of skin atrophy when compared to hydrocortisone, and the effect appeared to be dose-related, although statistical significance was not reached [21]. In general, this complication becomes evident after 8–12 weeks, and resolves spontaneously by 26 weeks postinjection [15].

Author's General Guidelines

In our practice, patients are educated about the natural course of the condition, and are prescribed physical therapy as the primary treatment. Patients are discouraged from receiving a steroid injection, particularly if they have long-standing symptoms. However, it is our experience that corticosteroids can sometimes be beneficial in the acute setting, with the goal of short-duration pain relief that would permit the patient to participate in a rehabilitation program or work. Patients that cannot tolerate NSAIDs, and for whom narcotic pain relievers are not appropriate, may also benefit from the injection.

Prior to the injection, patients are educated about the expected pain relief, as well as possible complications, both systemic (elevated blood glucose) and local (skin depigmentation and atrophy, and the remote risk of tendon rupture). Not more than two injections are administered with a minimum of 1-month interval. A 25 gauge needle is used, and a mixture of 1 ml of 4 mg/ml dexamethasone, 2 ml of 1 % lidocaine, and 2 ml of 0.25 % bupivacaine is prepared. The skin is anesthetized with an ethyl chloride freezing spray (Gebauer, Cleveland, OH), and the needle is aimed at the most tender spot over the common extensor origin. The needle is inserted deep to the subcutaneous tissues (but avoiding placement into the tendon itself), and a wide area is injected with several needle passes, making sure the material is not injected in the superficial subcutaneous tissues or the tendon origin.

After the injection, patients are instructed to apply ice for comfort, and to resume normal use of their extremity, while avoiding activities that exacerbate their symptoms. Importantly, they are prescribed an active rehabilitation program emphasizing stretching exercises, which is scheduled to begin within a week of the injection. Once pain is under control, progressive resistive exercises are initiated.

Summary

Corticosteroid injections have a long and safe record of use in LE, and are frequently requested by patients. Moreover, it is not uncommon for a patient to present to the hand surgeon's office having already received one or multiple corticosteroid injections from their primary care provider. However, the current literature presents a strong argument against corticosteroid injections for LE. While there is a proven short-term benefit, there appears to be a detrimental effect with longer follow up. Thus, the routine use of corticosteroid injections should be discouraged, and the injections should be limited only to those cases when short-term pain relief is desirable in order to increase patient compliance with a long-term rehabilitation process.

References

1. Nichols AW. Complications associated with the use of corticosteroids in the treatment of athletic injuries. Clin J Sport Med. 2005;15(5):370–5.
2. Baily RA, Brock BH. Hydrocortisone in tennis elbow: a controlled series. Proc R Soc Med. 1957;50(6):389–90.
3. MacMahon PJ, Eustace SJ, Kavanagh EC. Injectable corticosteroid and local anesthetic preparations: a review for radiologists. Radiology. 2009;252(3):647–61.
4. Torp-Pedersen TE, et al. Effect of glucocorticosteroid injections in tennis elbow verified on colour Doppler ultrasonography: evidence of inflammation. Br J Sports Med. 2008;42(12):978–82.
5. Krogh TP, et al. Treatment of lateral epicondylitis with platelet-rich plasma, glucocorticoid, or saline: a randomized, double-blind, placebo-controlled trial. Am J Sports Med. 2013;41(3):625–35.
6. Khan KM, et al. Histopathology of common tendinopathies. Update and implications for clinical management. Sports Med. 1999;27(6):393–408.
7. Alfredson H, Lorentzon R. Chronic tendon pain: no signs of chemical inflammation but high concentrations of the neurotransmitter glutamate. Implications for treatment? Curr Drug Targets. 2002;3(1):43–54.
8. Barr S, Cerisola FL, Blanchard V. Effectiveness of corticosteroid injections compared with physiotherapeutic interventions for lateral epicondylitis: a systematic review. Physiotherapy. 2009;95(4):251–65.
9. Carayannopoulos A, et al. Prolotherapy versus corticosteroid injections for the treatment of lateral epicondylosis: a randomized controlled trial. PM R. 2011;3(8):706–15.
10. Nirschl RP, Pettrone FA. Tennis elbow. The surgical treatment of lateral epicondylitis. J Bone Joint Surg Am. 1979;61(6A):832–9.
11. Ashe MC, McCauley T, Khan KM. Tendinopathies in the upper extremity: a paradigm shift. J Hand Ther. 2004;17(3):329–34.
12. Kraushaar BS, Nirschl RP. Tendinosis of the elbow (tennis elbow). Clinical features and findings of histological, immunohistochemical, and electron microscopy studies. J Bone Joint Surg Am. 1999;81(2):259–78.
13. Ljung BO, Forsgren S, Friden J. Substance P and calcitonin gene-related peptide expression at the extensor carpi radialis brevis muscle origin: implications for the etiology of tennis elbow. J Orthop Res. 1999;17(4):554–9.
14. Stefanou A, et al. A randomized study comparing corticosteroid injection to corticosteroid iontophoresis for lateral epicondylitis. J Hand Surg Am. 2012;37(1):104–9.
15. Coombes BK, et al. Effect of corticosteroid injection, physiotherapy, or both on clinical outcomes in patients with unilateral lateral epicondylalgia: a randomized controlled trial. JAMA. 2013;309(5):461–9.
16. Coombes BK, et al. Optimising corticosteroid injection for lateral epicondylalgia with the addition of physiotherapy: a protocol for a randomised control trial with placebo comparison. BMC Musculoskelet Disord. 2009;10:76.
17. Kazemi M, et al. Autologous blood versus corticosteroid local injection in the short-term treatment of lateral elbow tendinopathy: a randomized clinical trial of efficacy. Am J Phys Med Rehabil. 2010;89(8):660–7.
18. Lindenhovius A, et al. Injection of dexamethasone versus placebo for lateral elbow pain: a prospective, double-blind, randomized clinical trial. J Hand Surg Am. 2008;33(6):909–19.
19. Okcu G, et al. Evaluation of injection techniques in the treatment of lateral epicondylitis: a prospective randomized clinical trial. Acta Orthop Traumatol Turc. 2012;46(1):26–9.
20. Tonks JH, Pai SK, Murali SR. Steroid injection therapy is the best conservative treatment for lateral epicondylitis: a prospective randomised controlled trial. Int J Clin Pract. 2007;61(2):240–6.

21. Price R, et al. Local injection treatment of tennis elbow-hydrocortisone, triamcinolone and lignocaine compared. Br J Rheumatol. 1991;30(1):39–44.
22. Newcomer KL, et al. Corticosteroid injection in early treatment of lateral epicondylitis. Clin J Sport Med. 2001;11(4):214–22.
23. Goodman LS, et al. Goodman & Gilman's the pharmacological basis of therapeutics. 10. Aufl. New York: McGraw-Hill; 2001. S. 2148.
24. Scott A, Khan KM. Corticosteroids: short-term gain for long-term pain? Lancet. 2010;376(9754):1714–5.
25. Lin YC, et al. Comparison between botulinum toxin and corticosteroid injection in the treatment of acute and subacute tennis elbow: a prospective, randomized, double-blind, active drug-controlled pilot study. Am J Phys Med Rehabil. 2010;89(8):653–9.
26. Bellapianta J, et al. Randomized prospective evaluation of injection techniques for the treatment of lateral epicondylitis. Orthopedics. 2011;34(11):e708–12.
27. Dogramaci Y, et al. Treatment of lateral epicondilitis using three different local injection modalities: a randomized prospective clinical trial. Arch Orthop Trauma Surg. 2009;129(10):1409–14.
28. Altay T, Gunal I, Ozturk H. Local injection treatment for lateral epicondylitis. Clin Orthop Relat Res. 2002;398:127–30.
29. Bisset L, et al. Mobilisation with movement and exercise, corticosteroid injection, or wait and see for tennis elbow: randomised trial. BMJ. 2006;333(7575):939.
30. Smidt N, et al. Corticosteroid injections, physiotherapy, or a wait-and-see policy for lateral epicondylitis: a randomised controlled trial. Lancet. 2002;359(9307):657–62.
31. Bisset L, et al. Conservative treatments for tennis elbow do subgroups of patients respond differently? Rheumatology (Oxford). 2007;46(10):1601–5.
32. Wolf JM, et al. Comparison of autologous blood, corticosteroid, and saline injection in the treatment of lateral epicondylitis: a prospective, randomized, controlled multicenter study. J Hand Surg Am. 2011;36(8):1269–72.
33. Gunduz R, et al. Physical therapy, corticosteroid injection, and extracorporeal shock wave treatment in lateral epicondylitis. Clinical and ultrasonographical comparison. Clin Rheumatol. 2012;31(5):807–12.
34. Gosens T, et al. Ongoing positive effect of platelet-rich plasma versus corticosteroid injection in lateral epicondylitis: a double-blind randomized controlled trial with 2-year follow-up. Am J Sports Med. 2011;39(6):1200–8.
35. Krogh TP, et al. Comparative effectiveness of injection therapies in lateral epicondylitis: a systematic review and network meta-analysis of randomized controlled trials. Am J Sports Med. 2012.
36. Coombes BK, Bisset L, Vicenzino B. Efficacy and safety of corticosteroid injections and other injections for management of tendinopathy: a systematic review of randomised controlled trials. Lancet. 2010;376(9754):1751–67.
37. Korthals-de Bos IB, et al. Cost effectiveness of interventions for lateral epicondylitis: results from a randomised controlled trial in primary care. Pharmacoeconomics. 2004;22(3):185–95.
38. Smith AG, et al. Common extensor tendon rupture following corticosteroid injection for lateral tendinosis of the elbow. Br J Sports Med. 1999;33(6):423–4 (discussion 424–5).
39. Beyzadeoglu T, Bekler H, Gokce A. Skin and subcutanous fat atrophy after corticosteroid injection for medial epicondylitis. Orthopedics. 2011;34(8):570.

Chapter 8
Tennis Elbow: Blood and Platelet-Rich Plasma (PRP) Injections

Christopher Judson and Jennifer Moriatis Wolf

Introduction

Lateral elbow epicondylitis, or tennis elbow, is a common musculoskeletal condition affecting 1–3 % of the adult population [1, 2]. The ailment affects men and women equally and presentation most often occurs between ages 35 and 50 [3]. Pain in the lateral elbow and weakened grip, especially with wrist extension, are the most common complaints. Symptoms tend to present between 6 months and 2 years [2, 4].

Lateral epicondylitis was originally described as an inflammatory condition, but no inflammatory cells have been demonstrated in pathologic specimens [3, 5, 6]. Alfredson et al. found normal levels of the inflammatory marker PGE-2 in postoperative tissue specimens from patients with lateral epicondylitis [6]. Instead, the pathologic findings have been described as angiofibroblastic tendinosis. Therefore, lateral epicondylitis is likely better characterized as a tendinopathy. The origin of the extensor carpi radialis brevis, or less commonly the extensor digitorum communis, are most commonly affected [5]. The extensor muscle origin at the lateral humeral epicondyle is thought to be at risk for multiple reasons. It may be susceptible to microtrauma from overuse and eccentric loading, and it may have impaired healing due to an inadequate vascular supply. Two relatively hypovascular zones in the common extensor origin have been described, one at the origin of the lateral epicondyle and the other 2–3 cm distal along the tendinous insertion [7].

C. Judson (✉) · J. M. Wolf
Department of Orthopaedic Surgery, University of Connecticut Health Center, 263 Farmington Avenue, MARB4-ORTHO, Farmington, CT 06030, USA
e-mail: cjudson@resident.uchc.edu

J. M. Wolf
e-mail: jmwolf@uchc.edu

J. M. Wolf (ed.), *Tennis Elbow,* DOI 10.1007/978-1-4899-7534-8_8

Treatment

Despite the multiple treatment methods that have been described, there is no unanimously supported algorithm for the treatment of lateral epicondylitis. An observational approach is the most conservative, and many patients will report improvement of symptoms by 1 year after initial onset [8]. However, the choice to passively allow the disease to run its course can be unacceptable for many patients, as it can entail decreased functional ability and consistent pain. Patients who are unable to work can face economic hardships. Symptomatic treatment consists of activity modification and nonsteroidal anti-inflammatory medications. Other conservative treatment modalities include various types of physiotherapy, including exercises, bracing, and ultrasound.

For the cohort of patients who do not respond to these treatments, injections have been utilized prior to any surgical treatment. Historical injections included lidocaine, alcohol, and carbolic acid [3]. Currently, the combination of corticosteroids with a local anesthetic is most widely used. However, in recent literature a number of alternative injections have been described in randomized controlled trials. These include autologous blood, platelet-rich plasma (PRP), botulinum toxin, hyaluronic acid, polidocanol, glycosaminoglycan, and prolotherapy. Beyond injections, approximately 4–11 % of patients with refractory cases will progress to requiring operative intervention [4].

Blood-Based Injections

There has been increasing interest in orthopedics in the use of autologous and platelet-rich preparations to stimulate bone, tendon, muscle, and cartilage healing. These preparations have been applied for chronic tendinopathies, acute muscle and ligamentous injuries, and intraoperative augmentation [9]. Growth factors such as transforming growth factor-beta, fibroblast growth factor-2, platelet-derived growth factors, insulin-like growth factor-1, epidermal growth factor, and vascular endothelial growth factor can be found in the alpha granules of platelets [9]. These growth factors have a number of functions, including cellular proliferation, cell migration, collagen synthesis, and angiogenesis [10]. Blood-based preparations also have a number of proteins, such as cell-adhesion molecules, that may participate in promoting inflammatory cell migration to the site of injury. Delivery of these bioactive factors has been achieved in various injection forms, including whole autologous blood, leukocyte-depleted moderate-yield PRP, and leukocyte-rich high-yield PRP [11].

In chronic tendinopathies, such as lateral epicondylitis, it has been theorized that relative hypovascularity of the tendon combined with repetitive overuse can lead to tendinopathy. Autologous blood preparations can ideally bring the body's own growth factors to the hypovascular site of injury. This could result in increased healing potential by the body's own means. Based on this hypothesis, preparations of autologous blood or PRP have been used in the treatment of lateral epicondylitis, Achilles tendinopathy, patellar tendinopathy, and plantar fasciitis. In the following

sections, the evidence for use of autologous blood and PRP injections in patients with lateral epicondylitis is reviewed.

Autologous Whole Blood

Autologous blood injection for the treatment of lateral epicondylitis was first described by Edwards and Calandruccio [12]. The authors noted that techniques such as forceful closed manipulation, traumatic injection, and percutaneous release resulted in improved outcomes for patients, and theorized that this was due to bleeding at the extensor origin following the trauma. This bleeding would then stimulate an inflammatory cascade to begin a healing response for the tendinopathy. They proposed that autologous blood injection, specifically composed of 2–3 ml of autologous blood combined with lidocaine, would deliver the cellular and humoral mediators to the elbow for a similar healing process.

In a case series of 28 patients with lateral epicondylitis symptoms present for 6 or more months who had failed conservative therapy, Edwards and Calandruccio found that after receiving one to three autologous blood injections, pain scores and Nirschl stages decreased at an average follow up of 9.5 months [12]. Overall, they found 79% relief of pain following autologous blood injections.

Preparation of autologous blood is relatively standard among various studies. A volume of 2–3 ml of blood is typically collected. Some studies advocate injecting a local anesthetic such as lidocaine or 2 ml bupivacaine a few minutes prior to blood injection to allow the anesthetic time to take effect. Others support combination of autologous blood with 1 ml local anesthesia in the same preparation in order to only perform one injection (Fig. 8.1). A single-shot or peppering injection technique can be used (Fig. 8.2).

There have been a number of randomized controlled trials evaluating autologous blood injections for lateral epicondylitis, although only one with comparison to a

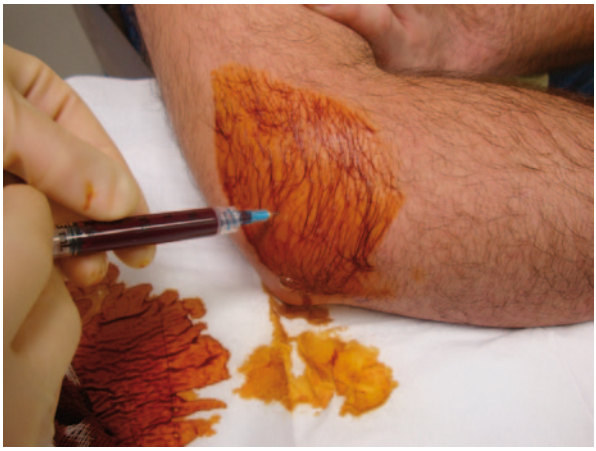

Fig. 8.1 Combination injection: autologous blood and 1 ml local anesthesia in one injection

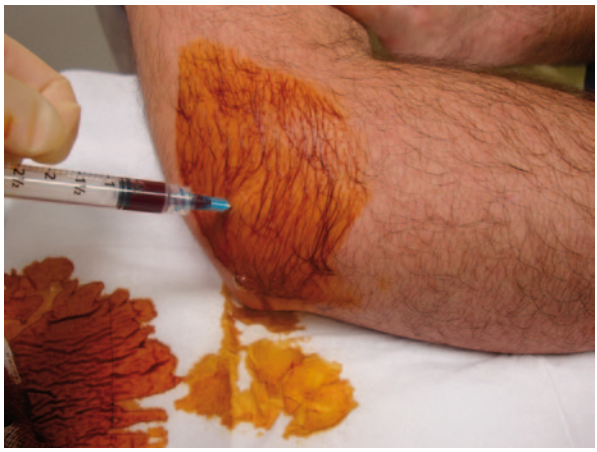

Fig. 8.2 Single-shot or peppering technique

placebo injection. Wolf et al. performed a randomized controlled trial (RCT) of 28 patients comparing autologous blood, corticosteroid, and a saline injection [13]. The study was double-blinded and patients were evaluated at 2 weeks, 2 months, and 6 months after injection with Visual Analog Scale (VAS), Disabilities of the Arm, Shoulder, and Hand (DASH), and the patient-related forearm evaluation. Although all of these outcomes demonstrated improvement from baseline in each group, there were no significant differences in any of the groups. However, the authors point out that the small number of patients in the study may limit their power to detect a difference between groups.

In 2010, Ozturan compared autologous blood injection to both corticosteroid injection and extracorporeal shock wave therapy in a three-armed randomized trial of 60 patients [14]. Although corticosteroid treatment showed the best outcomes at 4 weeks, success rates at 1 year were greatest for the autologous blood (83 %) and extracorporeal shock wave therapy (90 %) compared to only 50 % for corticosteroids. This study concluded that while corticosteroid injections provided better short-term relief of symptoms, autologous blood injections showed significantly better long-term results with decreased recurrence.

Kazemi directly compared autologous blood to corticosteroid injections in a short-term RCT of 60 patients [15]. As opposed to Ozturan et al.'s study, the authors found improved outcomes measures in the short-term for autologous blood. At 4 weeks, autologous blood was significantly more effective at decreasing pain scores at rest and with grip, as well as increasing QuickDASH scores ($p > 0.001$, $p = 0.002$, $p = 0.004$). These results persisted at 8 weeks ($p < 0.001$ for all measures).

Dojode performed a randomized study with 60 patients comparing autologous blood with local corticosteroid injection in a labor-intensive population [16] with 6 month follow up. Patients receiving corticosteroid injections had significantly decreased pain and Nirschl stage at 1 week ($p < 0.001$, both) and 4 weeks ($p = 0.002$, $p = 0.018$). However, outcomes were reversed as time went on. At 12 weeks and 6 months, patients who had received autologous blood had significantly lower pain and Nirschl stage scores ($p = 0.013$, $p = 0.018$ at 12 weeks, $p = 0.006$, $p = 0.006$ at

6 months, respectively). At the 6 month time follow up, 90% of patients who had received autologous blood injection reported complete relief of pain, compared to 47% of patients receiving steroid injection. This study concluded that autologous blood injections provide improved long-term relief of symptoms compared to corticosteroid injections.

There have been few side effects demonstrated from autologous blood injections. Most commonly authors cite the pain after injection as the most difficult side effect for patients. Ozturan describes 89% of patients having cessation of pain within 2 days, and the remaining 11% of patients had pain from 4 to 6 days[14]. In addition, 21% had elbow erythema, 16% had swelling, and 21% had nausea. Wolf et al. and Kazemi et al. described no side effects [13, 15]. Dojode reported 60% of patients having pain after the injection that resolved within a few days after injection [16].

In summary, autologous blood injections offer numerous factors to stimulate a healing cascade in the degenerative tendinous origin. Studies have shown beneficial effects for patients receiving these injections in the short- and long-term, predominantly compared to steroid injections. However, in the only placebo-controlled study, no significant benefit was observed for autologous blood injection. Additionally, one study showed no difference between autologous blood injections and extracorporeal shock wave therapy. Further investigation comparing autologous blood injections to placebo injections or conservative treatment with larger patient groups will shed more light on their efficacy.

Platelet Rich Plasma (PRP)

Autologous PRP is a concentrated source of platelets and platelet-derived growth factors that has been used for numerous musculoskeletal diagnoses. PRP is theorized to enhance the healing of wounds, bone, and tendons through release of specific growth factors upon platelet activation [17]. PRP has the theoretical advantage of increased concentration of platelets and therefore platelet-derived growth factors [17].

PRP is prepared by drawing 20–60 cc of blood from the patient. An FDA-approved blood separation device is used to centrifuge the blood for 15 min to isolate PRP [17]. This produces 3–6 mL of PRP (Fig. 8.3), which can be combined with or given after injection of 1–2 mL of local anesthetic (Fig. 8.4). Carofino et al. reported that lidocaine can cause inhibitory effects on tenocyte proliferation after exposure to PRP in vitro [18]. However, as the most common side effect from this injection is pain, it is standard to inject at least a small amount of local anesthetic into the skin with or prior to the injection.

Mishra et al. were the first to study the efficacy of PRP for lateral epicondylitis treatment [19]. In an unblinded prospective study, the authors treated 20 patients with chronic lateral epicondylitis using PRP in 15 and control bupivacaine in 5 [19]. At 8 weeks, patients who received PRP injections had significantly better VAS scores than the bupivacaine group. At final follow up of 1–3 years, 93% had reduction in VAS pain scores.

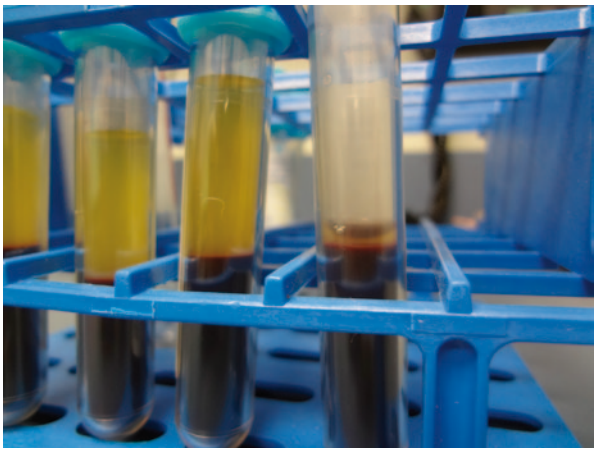

Fig. 8.3 Platelet-rich plasma (*PRP*). 20–60 cc of blood will, after 15 min centrifuge, produce 3–6 mL PRP

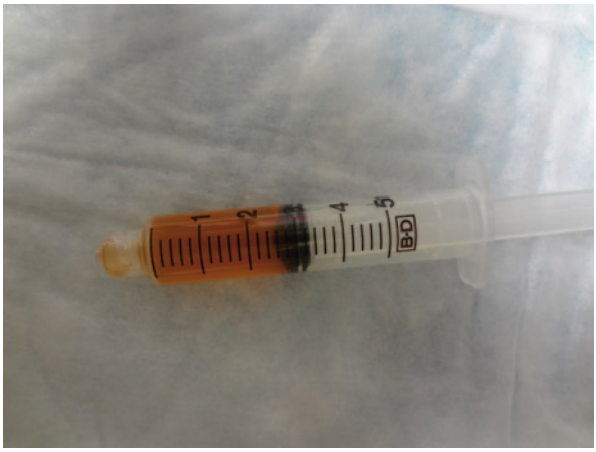

Fig. 8.4 Platelet-rich plasma (*PRP*) can be administered after local anesthetic, or be combined with it

There have been a number of randomized controlled trials evaluating PRP in the treatment of tennis elbow. Peerbooms et al. compared PRP with corticosteroid injection in a double-blind randomized trial of 100 patients [20]. Successful treatment was defined as >25% reduction in VAS score with no reintervention. The authors found that at the early 4-week time point, patients in the corticosteroid group showed slightly more improvement. However, at 26 and 52 weeks, VAS and DASH scores were significantly better for the PRP group ($p<0.001$ and $p=0.005$), with resolution in 73% of the PRP group vs. 49% the corticosteroid group. At 2 years, 81% of PRP patients reported successful outcomes compared to 40% of the corticosteroid group [21].

Krogh et al. compared PRP to corticosteroid and placebo injections with 60 patients in a short-term, randomized, double-blind trial [22]. Similar to the results of Peerbooms et al., improved pain relief was demonstrated at 1 month in the corticosteroid group compared to PRP and placebo. However, at 3 months follow up, there were no significant differences between the three groups using the patient-related tennis elbow evaluation (PRTEE).

Stenhouse et al. performed a randomized trial comparing 2 ml PRP injection with dry needling in 28 patients with refractory tennis elbow, with a mean duration of symptoms of 19 months [23]. The authors found that there was a trend towards greater clinical improvement, as measured by reduction in VAS scores, at 2 and 6 months for the PRP group compared to dry needling, but the differences were not significant. However, the small cohort sizes may have impacted power to determine a difference.

Mishra et al. recently reported the largest randomized controlled study to date, in which 230 patients were blinded and randomized to either needling the extensor origin with either PRP or nothing, after injection of lidocaine in both groups [24]. At 12 weeks with 83% follow up, the groups were not significantly different with regards to improvement in pain scores. However, for the 119 patients who had data available at 24 weeks, those receiving PRP had a 71% improvement in their pain scores compared to 56% for the control group ($p=0.027$). The percentage with remaining significant elbow tenderness was 29% for the PRP group vs. 54% for the control group ($p<0.001$).

The safety of PRP is similar to autologous blood, with minimal concern for immunogenic reactions. A number of patients report some magnitude of postinjection pain that can last up to 3–4 weeks [20]. Thanasas et al. found that patients who received PRP had more postinjection pain as compared to autologous blood injections [25]. Mishra et al. found no difference in the number of adverse events between the PRP and control needling groups, both causing pain in just under 20% of patients [24]. However, 2/116 patients did report severe pain that lasted 2–4 days.

It is important to note that the components of PRP can differ considerably depending on preparation methods. Mazzocca et al. demonstrated significantly different platelet and white-blood cell concentrations among different single-spin and double-spin separation techniques [26]. The literature on PRP in lateral epicondylitis includes different preparation methods that may lead to variable concentrations of platelets and growth factors, and therefore variable results. Additionally, Mazzocca et al. determined that each individual had varying concentrations of platelets and growth factors following different blood draws. These results suggest that differing concentrations of platelets and growth factors may contribute to the variable results seen among patients and in the literature.

PRP injection has demonstrated benefits in a difficult cohort of patients with chronic lateral epicondylitis who have failed other therapies. Research thus far has not supported any superiority for PRP over corticosteroids or placebo in the short-term, however, its superiority to corticosteroids in long-term (>3 months) follow up was demonstrated in two large double-blinded RCT with 2 years follow up [21, 24]. As compared to placebo injections, PRP has shown some long-term superiority in one study [24], but no significant differences in two smaller RCT's [22, 23].

Literature Comparisons of Autologous Blood and PRP Injections

Creaney et al. and Thanasas et al. both compared PRP with autologous blood injections in RCTs of 150 and 28 patients, respectively, who had failed first-line therapy for lateral epicondylitis [11, 25]. Creaney et al. defined success as a 25-point

reduction in the PRTEE [11]. In their trial, all patients were given two injections under ultrasound guidance. They found 66% success for the PRP group and 72% success for the autologous blood group, which was not significantly different. Twice as many patients in the autologous blood group (20% vs. 10%) sought eventual surgery. The study achieved 90% power to detect a difference of 10 points on the PRTEE scale but was limited by lacking a control group.

Thanasas et al. randomized patients to one injection of autologous blood or PRP in their single-blind study. They found their PRP group to have significantly better pain improvement than autologous blood at 6 weeks ($p<0.05$), but that the differences were not significant beyond this time point [25]. There were no significant differences in Liverpool elbow scores at any time points. A higher proportion of patients in the PRP group (64% vs. 29%) reported postinjection pain that gradually decreased. The authors theorized that this may result from the higher white blood cell concentration in PRP.

These studies suggest no definitive long-term difference in outcomes between PRP and autologous blood injections. Creaney et al. hypothesized that the reason for no difference in outcomes between PRP and autologous blood injections may be due to saturation of the beneficial capabilities of the growth-factors [11]. For instance, if the maximum collagen-producing capability has been reached with the platelets and growth factors in autologous blood, the higher concentration of these components in PRP may be unnecessary. Thanasas et al. described better short-term (6-week) pain scores for the PRP group with the caveat of more immediate postinjection pain, however, both injections show similar benefits in the long-term [25]. Therefore, with the current body of evidence, it is difficult to justify the additional expense of preparing PRP compared to autologous blood injections for lateral epicondylitis.

Conclusion

In reviewing the evidence for both of these treatments, the high-quality literature has shown mixed results. Both PRP and autologous blood injections have been compared with corticosteroid injection, which had long been considered the standard injection therapy for lateral epicondylitis. The majority of studies have found that although corticosteroids may provide better temporary relief of symptoms in the first month, both PRP and autologous blood demonstrate improved outcomes from 6 months to 1 year. Therefore, current evidence supports that once injection therapy is considered, autologous preparations should be considered over corticosteroids.

PRP and autologous blood injections have not been shown to have significantly different effects in comparative trials. In RCT's comparing autologous blood injection or PRP to placebo, no significant differences were appreciated in the majority of randomized trials [13, 14, 22, 23]. The largest randomized study of 230 patients demonstrated a benefit for PRP over placebo at 24 weeks, however, it was biased by a 48% loss-to follow up by that time point. Krogh et al. performed a systematic review and meta-analysis of 17 trials with 1381 patients comparing injection therapies in lateral epicondylitis, although only five of these trials looked at autologous blood

Table 8.1 Summary of Level I and II evidence

Study	Subject	Number of participants	Findings	Level of evidence
Wolf et al. [13]	ABI vs. Corticosteroid vs. Saline	28	No significant differences	Level II
Ozturan et al. [14]	ABI vs. Corticosteroid vs. Extracorporeal Shock Wave Therapy	60	Corticosteroids had better outcomes at 4 weeks, but ABI showed improved outcomes at 1 year	Level I
Kazemi et al. [15]	ABI vs. Corticosteroid	60	ABI with better outcomes at 4 and 8 weeks	Level I
Dojode et al. [16]	ABI vs. Corticosteroid	60	ABI provides better outcomes at 3 and 6 months, while results are better for corticosteroids at 1 and 4 weeks	Level I
Peerbooms et al. [20]	PRP vs. Corticosteroid	100	PRP better outcomes at 6 and 12 months, steroids better at 4 weeks	Level I
Gosens et al.* [21]	PRP vs. Corticosteroid	100	PRP better outcomes at 2 years	Level I
Krogh et al. [22]	PRP vs. Corticosteroid vs. Saline	60	Corticosteroids better outcomes at 1 month, no difference at 3 months	Level I
Stenhouse et al. [23]	PRP vs. Dry needling	28	No significant differences at 2 or 6 months	Level II
Mishra et al. [24]	PRP vs. Dry needling	230	No significant differences at 12 weeks, PRP with better outcomes at 24 weeks	Level I
Creaney et al. [11]	ABI vs. PRP	150	No significant differences up to 6 months	Level I
Thanasas et al. [25]	PRP vs. ABI	28	PRP better outcomes at 6 weeks, but no significant differences at 3 or 6 months	Level I

ABI Autologous blood injection, *PRP* Platelet-rich Plasma injection
*[21] was a follow-up study of [20]

or PRP injections [27]. Autologous blood and PRP were both shown to be superior to placebo with effect sizes of 1.43 (2.15–0.71) and 1.13 (1.77–0.49), respectively.

It is possible that the smaller randomized trials have lacked power to determine an advantage for autologous injections, or even that placebo injections that cause microtrauma at the site of injury may have a clinical benefit as opposed to conservative treatment. Nonetheless, at best there is a limited amount of evidence supporting the beneficial effect of these injections over placebo. Future large, randomized trials will be of importance to determine if these injections prove beneficial and cost-effective compared to conservative therapies (Table 8.1).

References

1. Allander E. Prevalence, incidence, and remission rates of some common rheumatic diseases or syndromes. Scand J Rheumatol. 1974;3(3):145–53.
2. Verhaar JA. Tennis elbow. Anatomical, epidemiological and therapeutic aspects. Int Orthop. 1994;18(5):263–7.
3. Faro F, Wolf JM. Lateral epicondylitis: review and current concepts. J Hand Surg Am. 2007;32(8):1271–9.
4. Calfee RP, et al. Management of lateral epicondylitis: current concepts. J Am Acad Orthop Surg. 2008;16(1):19–29.
5. Nirschl RP, Ashman ES. Elbow tendinopathy: tennis elbow. Clin Sports Med. 2003;22(4): 813–36.
6. Alfredson H, et al. In vivo investigation of ECRB tendons with microdialysis technique–no signs of inflammation but high amounts of glutamate in tennis elbow. Acta Orthop Scand. 2000;71(5):475–9.
7. Bales CP, et al. Microvascular supply of the lateral epicondyle and common extensor origin. J Shoulder Elbow Surg. 2007;16(4):497–501.
8. Smidt N, et al. Corticosteroid injections, physiotherapy, or a wait-and-see policy for lateral epicondylitis: a randomised controlled trial. Lancet. 2002;359(9307):657–62.
9. Foster TE, et al. Platelet-rich plasma: from basic science to clinical applications. Am J Sports Med. 2009;37(11):2259–72.
10. Kampa RJ, Connell DA. Treatment of tendinopathy: is there a role for autologous whole blood and platelet rich plasma injection? Int J Clin Pract. 2010;64(13):1813–23.
11. Creaney L, et al. Growth factor-based therapies provide additional benefit beyond physical therapy in resistant elbow tendinopathy: a prospective, single-blind, randomised trial of autologous blood injections versus platelet-rich plasma injections. Br J Sports Med. 2011;45(12):966–71.
12. Edwards SG, Calandruccio JH. Autologous blood injections for refractory lateral epicondylitis. J Hand Surg Am. 2003;28(2):272–8.
13. Wolf JM, et al. Comparison of autologous blood, corticosteroid, and saline injection in the treatment of lateral epicondylitis: a prospective, randomized, controlled multicenter study. J Hand Surg Am. 2011;36(8):1269–72.
14. Ozturan KE, et al. Autologous blood and corticosteroid injection and extracoporeal shock wave therapy in the treatment of lateral epicondylitis. Orthopedics. 2010;33(2):84–91.
15. Kazemi M, et al. Autologous blood versus corticosteroid local injection in the short-term treatment of lateral elbow tendinopathy: a randomized clinical trial of efficacy. Am J Phys Med Rehabil. 2010;89(8):660–7.
16. Dojode CM. A randomised control trial to evaluate the efficacy of autologous blood injection versus local corticosteroid injection for treatment of lateral epicondylitis. Bone Joint Res. 2012;1(8):192–7.
17. Sampson S, Gerhardt M, Mandelbaum B. Platelet rich plasma injection grafts for musculoskeletal injuries: a review. Curr Rev Musculoskelet Med. 2008;1(3/4):165–74.
18. Carofino B, et al. Corticosteroids and local anesthetics decrease positive effects of platelet-rich plasma: an in vitro study on human tendon cells. Arthroscopy. 2012;28(5):711–9.
19. Mishra A, Pavelko T. Treatment of chronic elbow tendinosis with buffered platelet-rich plasma. Am J Sports Med. 2006;34(11):1774–8.
20. Peerbooms JC, et al. Positive effect of an autologous platelet concentrate in lateral epicondylitis in a double-blind randomized controlled trial: platelet-rich plasma versus corticosteroid injection with a 1-year follow-up. Am J Sports Med. 2010;38(2):255–62.
21. Gosens T, et al. Ongoing positive effect of platelet-rich plasma versus corticosteroid injection in lateral epicondylitis: a double-blind randomized controlled trial with 2-year follow-up. Am J Sports Med. 2011;39(6):1200–8.

22. Krogh TP, et al. Treatment of lateral epicondylitis with platelet-rich plasma, glucocorticoid, or saline: a randomized, double-blind, placebo-controlled trial. Am J Sports Med. 2013;41(3):623–35.
23. Stenhouse G, Sookur P, Watson M. Do blood growth factors offer additional benefit in refractory lateral epicondylitis? A prospective, randomized pilot trial of dry needling as a standalone procedure versus dry needling and autologous conditioned plasma. Skeletal Radiol. 2013;42(11)1515–20.
24. Mishra AK, et al. Platelet-rich plasma significantly improves clinical outcomes in patients with chronic tennis elbow: a double-blind, prospective, multicenter, controlled trial of 230 patients. Am J Sports Med. 2014;42(2):463–71.
25. Thanasas C, et al. Platelet-rich plasma versus autologous whole blood for the treatment of chronic lateral elbow epicondylitis: a randomized controlled clinical trial. Am J Sports Med. 2011;39(10):2130–4.
26. Mazzocca AD, et al. Platelet-rich plasma differs according to preparation method and human variability. J Bone Joint Surg Am. 2012;94(4):308–16.
27. Krogh TP, et al. Comparative effectiveness of injection therapies in lateral epicondylitis: a systematic review and network meta-analysis of randomized controlled trials. Am J Sports Med. 2013;41(6):1435–46.

Chapter 9
Alternative Treatments for Tennis Elbow: Acupuncture, Prolotherapy, and Shock Wave Lithotripsy

Carlton M. Clinkscales

Introduction

Lateral epicondylitis is primarily a nonoperative problem [1]. While surgery for lateral epicondylitis is usually not indicated, as there are certain circumstances outlined elsewhere in this text where surgery may be the appropriate or preferred treatment for this condition. However, most cases of lateral epicondylitis can be managed successfully nonoperatively until they resolve in 80–95 % of cases.

Lateral epicondylitis is a painful condition afflicting the lateral epicondyle at the insertion of the extensor carpi radialis brevis tendon (ECRB) [2]. It is an enthesopathy, a degenerative condition, rather than inflammatory condition. Typically, it affects healthy, vigorous, active males and females usually in middle age. It can be traumatic, associated with activity, or even spontaneous. In fact, one of my primary observations (having had it myself) is that the pain is quite dramatic, extreme, and piercing. Furthermore, I have discovered both in my own case and in the case of almost all of my patients that there is significant frustration that others cannot understand or appreciate just how bad the pain is. It is natural therefore to form two conclusions: (1) "If the pain is bad, the problem must be bad," and (2) "If there is a problem, it must be fixed and right away." Most surgeons who have tried to fix it have discovered that postoperative healing can be prolonged and patients' satisfaction mixed. It is challenging to operate on pain.

It is therefore easy to understand that lateral epicondylitis is a degenerative condition that needs to be managed until it runs its natural course. A good analogy might be the child with flu symptoms whose family insists on antibiotics only to claim that, when the flu resolves, it proves that the antibiotics were curative. Yet we know that the natural course of the disease was to inevitably resolve. Who are we treating?

C. M. Clinkscales (✉)
Department of Orthopaedics, Littleton Hospital, 7935 Endora Circle, Centennial, CO 80122, USA
e-mail: doctorcmc@msn.com

J. M. Wolf (ed.), *Tennis Elbow,* DOI 10.1007/978-1-4899-7534-8_9

The goals of treatment therefore should be pain management (maintaining, and even maximizing function during resolution of symptoms) and prevention of its recurrence. Patient education is tantamount. The first thing to tell the patient is that the magnitude of the pain does not correlate to the magnitude of the problem. Second, the patient must know that in the majority of cases, their problem will resolve successfully. Third, the patient must learn his or her role in recovery, maintenance, and prevention.

Alternative Treatments for Tennis Elbow

This chapter will present the potential benefits of several noninvasive treatments, specifically, acupuncture, prolotherapy, and extracorporeal shock wave therapy (ESWT). They are all controversial, but potentially helpful and therefore possibly valuable.

Acupuncture

Acupuncture is a viable modality for the treatment of epicondylitis. Acupuncture is an Eastern tradition, and practitioners are required to complete 3–4 years of formal training. Acupuncturists are licensed by state, with varying scopes of practice, and considerable experience is required. The practice of acupuncture depends on traditional needle placement (Fig. 9.1) based on established meridians, vectors, and locations. It often relies on symmetry and specific trigger points.

Acupuncture purportedly stimulates the nervous system releasing neurochemicals that lead to biochemical changes, thus promoting physical and emotional wellbeing. Specific acupuncture point stimulation has been shown to affect areas of the brain, which reduce sensitivity to pain and stress and or promote relaxation as well

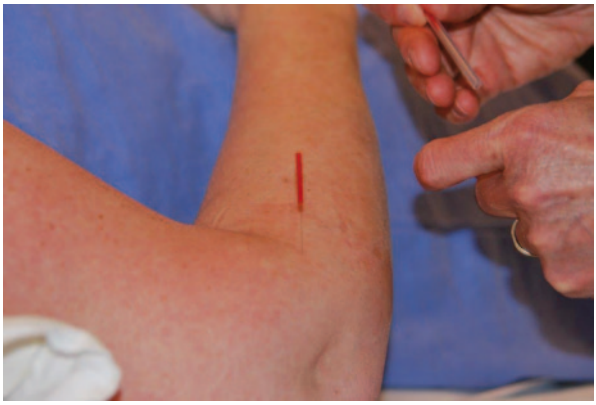

Fig. 9.1 Acupuncture involves traditional needle placement based on established meridians, vectors, and locations. Symmetry and trigger points are frequently determinative

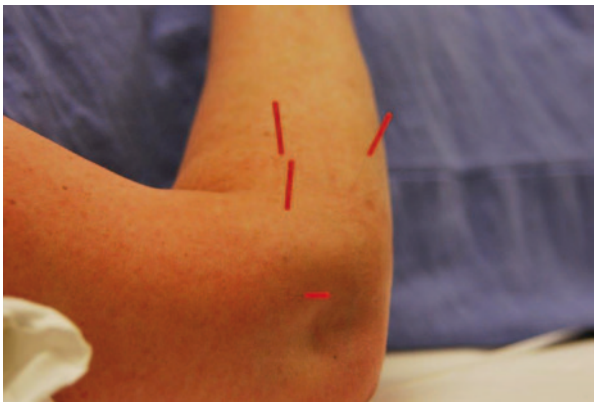

Fig. 9.2 Acupuncture should be distinguished from the technique of dry needling, or insertion of needles into an affected area

as decreasing anxiety, according to the British Acupuncture Council [3]. The general theory of acupuncture is based on the flow of qi; abnormal flow supposedly causes disease. Acupuncture describes a family of procedures intending to rebalance the flow of qi through channels known as meridians. This is accomplished by stimulating specific areas on or under the skin (so called acu points). Currently, acupuncturists use metal needles manipulated manually or with electrical stimulation [4].

Acupuncture for lateral epicondylitis often involves needles placed on the opposite extremity, even if asymptomatic. Acupuncture may address epicondylitis differently depending on whether it is chronic or acute. There are trigger points in the auricular area which may be included. One session weekly for 4 weeks followed by every 2 or 3 weeks, then monthly is recommended [5]. It is important to make sure that there are no underlying intrinsic problems about the elbow joint itself based on examination, X-rays, or other imaging studies such as MRI. Proximal or distal issues should also be ruled out.

Acupuncture (Fig. 9.2) should be distinguished from the technique of dry needling, or insertion of needles into an affected area. This is thought to stimulate blood flow on a microscopic level and potentially lead to a healing process [6]. Stenhouse et al. noted improvement with dry needling alone in a randomized trial, with no additional benefit of autologous conditioned plasma [7].

Fink et al. performed a randomized control trial evaluating acupuncture in the treatment of chronic lateral epicondylitis. A group of 23 patients treated with acupuncture were compared to 22 patients who received ‘sham’ acupuncture. There were two treatments per week for a total for ten treatments, and outcome was determined by strength, pain, and disabilities of the arm, shoulder, and hand (DASH) score. The group that received true acupuncture showed significant improvement compared to the sham group at 2 weeks, although both groups noted pain improvement. At 2 and 12 months posttreatment, there were no significant differences between groups. The authors concluded that using accurate acupuncture points had an important effect on short-term outcomes in tennis elbow [8].

Buchbinder et al. presented a literature review of acupuncture for lateral elbow pain in 2008. They searched multiple databases and found four small randomized controlled trials for analysis. One study showed that needle acupuncture alleviated pain longer than placebo. A second study showed improvement in the short-term but no differences in the long-term (defined as 3 or 12 months). A study of laser acupuncture vs. placebo demonstrated no differences. A fourth study from China showed no difference between vitamin B12 injection and acupuncture vs. vitamin B12 injection alone. The authors concluded that there is insufficient evidence to either support or refute the use of acupuncture (either needle or laser) in the treatment of lateral epicondylagia.

Vickers and Linde published a meta-analysis of 29 trials examining individual treatment data of acupuncture for chronic pain. Eighteen studies compared acupuncture to no acupuncture control and 20 studies compared acupuncture to sham acupuncture control. They noted that acupuncture had improved outcomes compared with sham acupuncture and no acupuncture as control. They noted reduction of pain after treatment in 30% for no acupuncture controls, 42.5% for sham acupuncture and 50% for acupuncture treatment [9].

Currently both civilian and military institutions are integrating acupuncture into the care they provide. Translational research is also elucidating effects of interventions like meditation and acupuncture on the ventral mechanism of pain perception and processing, regulation of emotion and attention, and placebo responses. Although not yet fully understood, these effects point toward scientifically plausible mechanisms—often unrelated to the traditional mechanistic explanations—by which these interventions might exert benefit [10].

Prolotherapy

Prolotherapy is also known as “proliferation therapy,” “regenerative injection therapy,” [11], or “proliferative injection therapy”. It involves injecting an otherwise nonpharmacological and nonactive irritant solution into the body, generally in the region of tendons or ligaments. It is thought to strengthen weakened connective tissue and alleviate musculoskeletal pain. Theoretically, prolotherapy stimulates a local inflammatory process which facilitates tissue repair of tendons, ligaments, or soft tissue. Possibly, this occurs through the release of local growth factors. A more precise mechanism of action has not been identified. A small volume of irritant solution is injected directly into the painful area. There are several commonly used prolotherapy solutions, which may actually act differently. For example, dextrose may cause osmotic rupture of local cells, phenol-glycerine-glucose may cause local cellular irritation, and sodium morrhuate may result in chemotactic attraction of inflammatory mediators [12].

Prolotherapy was originally described in the 1930s and has become increasingly popular with practitioners using a variety of injection protocols, some of which were formalized in the 1950s by George Hackett [13]. A systematic review including a broad search of human studies assessing prolotherapy revealed 34 case reports

and series and two nonrandomized controlled trials [14]. These showed efficacy in many musculoskeletal conditions, but the randomized controlled studies showed conflicting outcomes. These studies did not specifically look at lateral epicondylitis. The authors concluded that further investigation was needed with better-structured studies.

Rabago et al. performed systematic review and noted that the evidence suggested that polidocanol, prolotherapy, autologous blood, and platelet-rich plasma all showed promise in targeting the neovascularity critical to healing tendonopathies. The studies that support this claim included his own work, including randomized pilot studies [15, 16]. A randomized controlled trial of prolotherapy vs. cortisone injection showed no statistical differences between the two groups [17].

Most insurers, including Medicare, do not cover prolotherapy. Websites for Aetna, Blue Cross Blue Shield, Cigna, and United Healthcare state that prolotherapy is not covered for any diagnosis. Medicare reviews took place in September 1992 and then again in September 1999 after increased demand arose. In 1992 reviewers determined that practitioners had not provided "any scientific evidence on which to base a [different] coverage decision," but expressed willingness to reconsider if presented with results of "further studies on the benefits of prolotherapy" (Health Care Finance Administration). According to its website, Colorado's worker's compensation insurer, Pinnacol Assurance does not recommend prolotherapy for any diagnosis, specifically upper-extremity injuries (http://www.sos.state.co.us).

Electro Shock Wave Therapy (ESWT)

ESWT has been studied and published in more traditional orthopaedic journals than either acupuncture or prolotherapy. Initial anecdotal reports of various techniques using ESWT showed promising results [18]. It is thought that ESWT exerts direct pressure or causes cavitation of bone [19]. In a rabbit model ESWT showed increased bone formation and bone mineral density, and improved collagen alignment compared with controls [20]. ESWT has been postulated to improve the biologic environment of the tendon-bone interface via upregulation of TGF-β, VEGF, and BMP [21].

Rompe et al. compared two low-energy doses of ESWT in a prospective study in patients with lateral epicondylitis [22]. One hundred patients who had symptoms for more than 12 months were randomized into two groups: one received a total of 3000 pulses of 0.08 J/mm^2 whereas the other group received 30 pulses. Patients were evaluated at 3, 6, and 24 weeks. Based on significant alleviation of pain and improvement of function after treatment, 48% achieved acceptable results initially and 42% at final review in group 1 compared with 6% initially and 24% at final review in group 2, indicating efficacy at the higher dose.

A multicenter, retrospective study, involving 65 patients showed that the efficacy and safety of ESWT were excellent or good in 74% of patients 6 months after treatment.

ESWT enjoyed some commercial success, but remained controversial. In 2002, Haake et al. presented a randomized controlled multicenter trial of ESWT in the treatment of lateral epicondylitis [23]. Under local anesthesia, shockwave therapy at 2000 pulses or placebo therapy was used to treat patients who were blinded to the type of treatment. The primary outcome measure was the Roles and Maudsley patient-rated pain score and whether additional treatment was required 12 weeks after the intervention. The authors noted no differences between groups, although both improved over time.

Crowther et al. published a prospective randomized study comparing ESWT to injection of steroid for the treatment of tennis elbow [24]. They compared a single dose of 20% of triamcinolone with lidocaine to a second group receiving 2000 shockwaves in three sessions at weekly intervals. They found that after 3 months, 84% of the steroid group had successful treatment, defined as pain relief, whereas only 60% of the shockwave treatment had achieved lower-pain scores. The authors concluded that steroid injection was more effective and less expensive than ESWT in the treatment of tennis elbow.

Several other articles confirmed that ESWT was not superior to placebo [25, 26]. Speed et al. found no difference in improvement between ESWT and sham treatment. The authors concluded that "there appears to be a significant placebo effect of moderate dose ESWT in subjects with lateral epicondylitis but there is no evidence of added benefit of treatment when compared to sham therapy" [27]. Finally, Staples et al. found little evidence to support the use of ESWT for the treatment of lateral epicondylitis in a double-blind, randomized, placebo-controlled trial [28].

In summary, while anecdotal reports are favorable, better-structured studies of ESWT suggest that treatment is not better than placebo, and at least one study refutes its effectiveness entirely. Again, based on the data [29], insurers typically do not cover the treatment modality.

Conclusions

Lateral epicondylitis is generally a nonoperative problem with many treatment modalities available. While the science may not show efficacy, these three modalities have been shown to be safe, and in some studies, effective. Personally, I have had good success with acupuncture referrals. I have encounted anecdotal observations of success with prolotherapy. I do not use shockwave therapy (EWST) due to high cost and limited availability, and unproven efficacy. Nonoperative treatment for lateral epicondylitis should emphasize patient education, self-management, and reasonable expectations for time to resolution. In addition activity modification should be encouraged to decreased repetitive strain and pain. Modalities should focus on pain control and pain management, tolerability, and cost. We should not employ trickery, but if "entertaining the patient" means making them feel better, then Voltaire is right!

References

1. Szabo RM. Steroid injection for lateral epicondylitis. J Hand Surg Am. 2009;34(2):326–30.
2. Faro F, Wolf JM. Lateral epicondylitis: review and current concepts. J Hand Surg Am. 2007;32(8):1271–9.
3. Macpherson H, Scullion A, Thomas KJ, Walters S. Patient reports of adverse events associated with acupuncture treatment: a prospective national survey. Qual Saf Health Care. 2004;13(5):349–55.
4. Wootton J. National Institutes of Health Consensus Development statement on acupuncture. J Altern Complement Med. 1997;3(4):419–20.
5. Trinh KV, Phillips SD, Ho E, Damsma K. Acupuncture for the alleviation of lateral epicondyle pain: a systematic review. Rheumatology (Oxford). 2004;43(9):1085–90.
6. Nagraba L, Tuchalska J, Mitek T, Stolarczyk A, Deszczynski J. Dry needling as a method of tendinopathy treatment. Ortop Traumatol Rehabil. 2013;15(2):109–16.
7. Stenhouse G, Sookur P, Watson M. Do blood growth factors offer additional benefit in refractory lateral epicondylitis? A prospective, randomized pilot trial of dry needling as a standalone procedure versus dry needling and autologous conditioned plasma. Skeletal Radiol. 2013;42(11):1515–20.
8. Fink M, Wolkenstein E, Luennemann M, Gutenbrunner C, Gehrke A, Karst M. Chronic epicondylitis: effects of real and sham acupuncture treatment: a randomised controlled patient-and examiner-blinded long-term trial. Forsch Komplementarmed Klass Naturheilkd. 2002;9(4):210–5.
9. Vickers AJ, Linde K. Acupuncture for chronic pain. JAMA. 2014;311(9):955–6.
10. Loitman JE. Pain management: beyond pharmacology to acupuncture and hypnosis. JAMA. 2000;283(1):118–9.
11. Distel LM, Best TM. Prolotherapy: a clinical review of its role in treating chronic musculoskeletal pain. PM R. 2011;3(6 Suppl 1):S78–S81.
12. Rabago D, Slattengren A, Zgierska A. Prolotherapy in primary care practice. Prim Care. 2010;37(1):65–80.
13. Hackett GS. Prolotherapy in whiplash and low back pain. Postgrad Med. 1960;27:214–9.
14. Rabago D, Yelland M, Patterson J, Zgierska A. Prolotherapy for chronic musculoskeletal pain. Am Fam Physician. 2011;84(11):1208–10.
15. Rabago D, Best TM, Beamsley M, Patterson J. A systematic review of prolotherapy for chronic musculoskeletal pain. Clin J Sport Med. 2005;15(5):376–80.
16. Scarpone M, Rabago DP, Zgierska A, Arbogast G, Snell E. The efficacy of prolotherapy for lateral epicondylosis: a pilot study. Clin J Sport Med. 2008;18(3):248–54.
17. Carayannopoulos A, Borg-Stein J, Sokolof J, Meleger A, Rosenberg D. Prolotherapy versus corticosteroid injections for the treatment of lateral epicondylosis: a randomized controlled trial. PM R. 2011;3(8):706–15.
18. Hammer DS, Rupp S, Ensslin S, Kohn D, Seil R. Extracorporal shock wave therapy in patients with tennis elbow and painful heel. Arch Orthop Trauma Surg. 2000;120(5/6):304–7.
19. Wang L, Qin L, Lu HB, Cheung WH, Yang H, Wong WN, Chan KM, Leung KS. Extracorporeal shock wave therapy in treatment of delayed bone-tendon healing. Am J Sports Med. 2008;36(2):340–7.
20. Qin L, Wang L, Wong MW, Wen C, Wang G, Zhang G, Chan KM, Cheung WH, Leung KS. Osteogenesis induced by extracorporeal shockwave in treatment of delayed osteotendinous junction healing. J Orthop Res. 2010;28(1):70–6.
21. Atesok K, Fu FH, Wolf MR, Ochi M, Jazrawi LM, Doral MN, Lubowitz JH, Rodeo SA. Augmentation of tendon-to-bone healing. J Bone Joint Surg Am. 2014;96(6):513–21.
22. Rompe JD, Hopf C, Kullmer K, Heine J, Burger R, Nafe B. Low-energy extracorporal shock wave therapy for persistent tennis elbow. Int Orthop. 1996;20(1):23–7.
23. Haake M, Konig IR, Decker T, Riedel C, Buch M, Muller HH, Extracorporeal shock wave therapy clinical trial Group. Extracorporeal shock wave therapy in the treatment of lateral epicondylitis: a randomized multicenter trial. J Bone Joint Surg Am. 2002;84-A(11):1982–91.

24. Crowther MA, Bannister GC, Huma H, Rooker GD. A prospective, randomised study to compare extracorporeal shock-wave therapy and injection of steroid for the treatment of tennis elbow. J Bone Joint Surg Br. 2002;84(5):678–9.
25. Spacca G, Necozione S, Cacchio A. Radial shock wave therapy for lateral epicondylitis: a prospective randomised controlled single-blind study. Eura Medicophys. 2005;41(1):17–25.
26. Chung B, Wiley JP, Rose MS. Long-term effectiveness of extracorporeal shockwave therapy in the treatment of previously untreated lateral epicondylitis. Clin J Sport Med. 2005;15(5):305–12.
27. Speed CA, Nichols D, Richards C, Humphreys H, Wies JT, Burnet S, Hazleman BL. Extracorporeal shock wave therapy for lateral epicondylitis—a double blind randomised controlled trial. J Orthop Res. 2002;20(5):895–8.
28. Staples MP, Forbes A, Ptasznik R, Gordon J, Buchbinder R. A randomized controlled trial of extracorporeal shock wave therapy for lateral epicondylitis (tennis elbow). J Rheumatol. 2008;35(10):2038–46.
29. Buchbinder R, Green SE, Youd JM, Assendelft WJ, Barnsley L, Smidt N. Systematic review of the efficacy and safety of shock wave therapy for lateral elbow pain. J Rheumatol. 2006;33(7):1351–63.

Chapter 10
Orthotic Use in the Management of Epicondylitis: What is the Evidence?

Roger A. Daley, David W. Meister and Barbara L. Haines

Introduction

Management of lateral epicondylitis remains a controversial topic. Use of immobilization for treatment and symptom control is not a new concept. Morris described the primary etiology and symptoms of "tennis elbow" in 1882. He also recognized the importance of immobilizing the arm [1]. Splinting remains a key component of most treatment protocols [2–4]. Literature on the efficacy of orthotic use alone is difficult to analyze because studies rarely investigate an isolated treatment modality but rather the efficacy of a comprehensive treatment program [5–12]. With multiple treatment variables to consider, a precise determination of a particular modality is difficult. Therefore, current recommendations for splint use are predominantly based on traditional beliefs and anecdotal experience.

Much of the literature on epicondylitis treatment fails scientific scrutiny. Many of the articles are based on opinions and lack clear scientific methodology. Labelle et al. performed a systematic review to assess the scientific evidence for methods of treatment for lateral epicondylitis [8]. The authors concluded that there was insufficient evidence to support any of the current conservative treatment options, secondary to lack of scientific validity. Randomized controlled studies are rare and multiple variables between studies make comparisons difficult. There are a wide variety of treatment options to alleviate symptoms associated with lateral epicondylitis.

R. A. Daley (✉)
Department of Orthopaedic Surgery, Medical College of Wisconsin—Froedtert Memorial Lutheran Hospital, Milwaukee, WI, USA
e-mail: rdaley@mcw.edu

D. W. Meister
Department of Orthopaedic Surgery, Medical College of Wisconsin, Milwaukee, WI, USA
e-mail: dmeister@mcw.edu

B. L. Haines
Department of Rehab: Hand/UE, Froedtert/Medical College of Wisconsin, Milwaukee, WI, USA
e-mail: Barbara.haines@froedtert.com

J. M. Wolf (ed.), *Tennis Elbow,* DOI 10.1007/978-1-4899-7534-8_10

The aim of this chapter is to review the evidence of bracing options and to provide clinical recommendations based on the available literature.

Objectives of Orthotic Wear

Patients will primarily complain of pain at the lateral elbow that radiates down the forearm as well as weakened grip and difficulty in lifting objects. Strength may be limited because of pain, although some believe that muscle dysfunction may be an independent symptom and not necessarily secondary to pain [2, 3, 13–16]. On examination, patients will have tenderness at the lateral epicondyle and distally in the dorsal forearm and wrist. They will have pain with resisted wrist extension as well as passive wrist flexion. Pain relief and restoration of muscle conditioning are the primary objectives of treatment. Some form of immobilization or splinting is used as an adjunct to various muscle conditioning protocols recommended for treatment.

Several objectives regarding orthotics exist for the treatment of lateral epicondylitis. Theoretically, splinting allows the involved muscles to rest, and counterforce bracing decreases stress on the pathologic tendon. Another favorable feature not readily investigated is the use of the orthotic device as a reminder to both the patient and others (i.e., the employer) to avoid activities that aggravate the condition.

Protective orthotics theoretically provide rest for the wrist extensors, particularly the extensor carpi radialis brevis (ECRB), extensor digitorum communis (EDC), and extensor carpi ulnaris (ECU) during use of the extremity. It is thought that immobilization of the wrist in extension will decrease muscle activity and thereby limit the excursion of the muscles and decrease tension on a diseased tendon [2, 3]. Splinting can be achieved by wrist immobilization, elbow immobilization, or a combination of both (Figs. 10.1 and 10.2). This may also aid in the healing of microscopic tears in the extensor origin since a splint places the muscle in a shortened position.

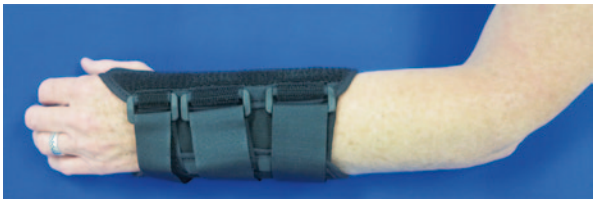

Fig. 10.1 *Wrist Brace*. The theoretical basis for the protective wrist orthotic is to provide rest for the wrist extensors, particularly the extensor carpi radialis brevis (ECRB), extensor digitorum communis (EDC), and extensor carpi ulnaris (ECU) during use of the extremity. The wrist should be held in an extended position (neutral extension or 15° extension)

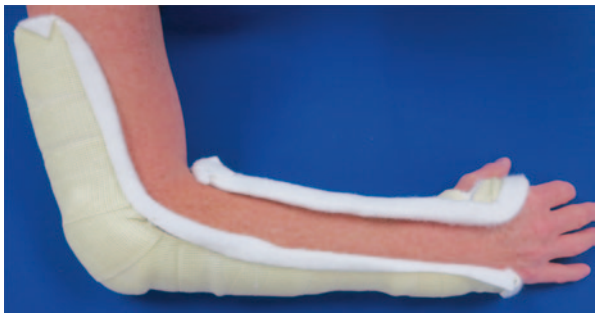

Fig. 10.2 *Long arm splint*. Immobilization of both the elbow and wrist decreases muscle activity across both joints acted on by the wrist extensors, thereby limiting excursion of the muscles and decrease tension on the diseased tendon origin

Evidence to Support Orthotic Use

Counterforce strap bracing refers to a nonelastic strap placed around the proximal forearm (Fig. 10.3), with the intended therapeutic effect of reducing stress on the lateral epicondyle by decreasing force transmission across the extensor muscle tendon unit. Meyer et al. performed a combined cadaveric and clinical study showing a 13–15% force reduction of the ECRB origin. Snyder-Mackler and Epler demonstrated a statistically significant decrease in ECRB and EDC muscle force recruitment with the counterforce strap, when compared to no strap, as measured by electromyography [17]. By inhibiting muscle expansion, the strap decreases the magnitude of muscle contraction, thereby reducing the tension at the musculotendinous junction proximal to the band [18]. Furthermore, the direct compression provided by the strap creates a secondary origin of the extensor tendons, which increases surface area and decreases stress and microtrauma experience by the true origin at the lateral epicondyle.

Struijs et al. performed a clinical trial randomizing 180 patients to a forearm band-type splint, physical therapy, or a combination of these and showed no significant differences at 26 and 52 weeks with regard to pain, disability, and satisfaction

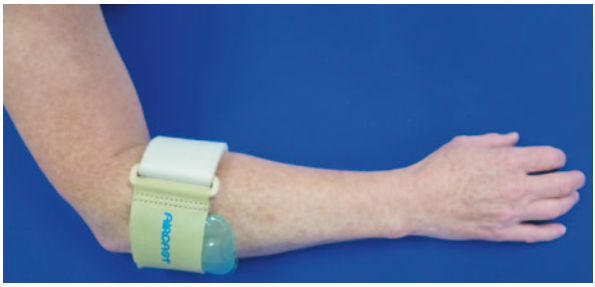

Fig. 10.3 Counter-force strap brace. Counterforce strap bracing refers to a nonelastic strap placed around the proximal forearm, with the intended therapeutic effect of reducing stress on the lateral epicondyle by decreasing force transmission across the extensor muscle tendon unit. Several varieties of this brace are available from different companies, with similar effects (pictured is one from Aircast, DJO Global, Vista, California)

[19]. Success rates at 52 weeks ranged from 85 to 89% within the three treatment groups. The same authors performed a meta-analysis that included all randomized clinical trials describing individuals with diagnosed lateral epicondylitis and comparing the use of an orthotic device as a treatment strategy [15, 20]. Only five studies met their inclusion criteria; overall, there were few outcome measures, large heterogeneity, and limited long-term results. None of the included studies investigated an orthotic as an isolated treatment modality. They stated no definitive conclusions could be drawn concerning the effectiveness of orthotic devices and that more well-designed randomized clinical trials of sufficient power are warranted [3, 15, 16, 20].

Altan and Kanat performed a short-term study of counterforce bracing versus a resting wrist splint and showed significant improvement in all parameters including pain at rest, pain with movement, and hand grip strength in the sixth week for both groups [21]. Comparison of the two groups showed significantly better improvement in resting pain with the wrist splint; otherwise other parameters were the same.

Van De Streek et al. from the Netherlands conducted a study comparing the effect of a forearm-based hand splint compared with an elbow band (counterforce brace) as a treatment for lateral epicondylitis. In this study they explored a new fabricated hand splint (thought to give more rest to the extensors of the wrist versus a cock-up splint) to an elbow band [22]. This was a randomized clinical trial with 43 patients. They were instructed to wear the braces for as much as possible for 6 weeks, with no other interventions. The outcome measures included maximal grip strength and patient-rated forearm evaluation questionnaire (PRFE). This study shows that the hand splint is no more effective than the elbow band as a treatment for lateral epicondylitis.

Garg et al. performed a randomized controlled trial (level of evidence II) investigating the clinical outcomes of a wrist extension splint with that of a counterforce forearm strap [6]. Among the 42 patients (44 elbows) investigated, they found that both modalities improved the Mayo elbow performance (MEP) and American Shoulder and Elbow Society (ASES) elbow assessment scores in the sixth week. The overall function was similar between the two groups. There was no significant difference measured between the braces with the ASES ($p=0.60$) nor MEP ($p=0.63$) scores. However, within the ASES derived score, pain relief was significantly better with the extension splint group ($p=0.027$). No other variables were statistically significantly different. They concluded that the greater degree of pain relief with the wrist extension splint may be due to improved immobilization of the wrist extensor muscles in a resting position.

Derebery et al. reviewed the potential disadvantages of bracing in lateral epicondylitis, particularly in cases involving workers' compensation [5]. They found that patients treated with splints had higher rates of limited duty ($p<0.001$), more medical visits and charges ($p<0.001$), higher total charges (medical and PT, $p<0.001$), and longer treatment durations ($p<.01$) than patients without splints. They concluded that splinting patients with epicondylitis may not optimize outcomes, including rates of limited duty, treatment duration, and medical costs. This article was unique in that it illustrates the variable of worker's compensation and potential negative impact on clinical outcomes.

Luginbuhl et al. performed a randomized study comparing the effect of the forearm-support band versus strengthening exercises for the treatment of lateral epicondylitis [9]. Twenty-nine patients with thirty tennis elbows were randomized into three groups of treatment: (I) forearm-support band, (II) strengthening exercises, and (III) both methods. Patients were evaluated at various time points over 1 year. At the latest follow up, there was a significant improvement of the symptoms compared to before treatment ($p < 0.0001$), considering all patients independently of the methods of treatment. However, no differences in the scores were found between the three groups of treatment ($p = 0.27$), indicating that no beneficial influence was found either for the strengthening exercises or for the forearm-support band. Improvement seems to occur with time, independent of the method of treatment used.

Discussion

A variety of splint types have been proposed to treat lateral epicondylitis. The significance of orthotic use is debatable, with no proven benefit of one orthotic treatment over another. There is evidence to suggest that immobilization with orthotics may be symptomatically beneficial in the short term. The choice of orthotic may be left to personal preference. The evidence that force reduction at the extensor origin occurs as demonstrated by biomechanical and electrodiagnostic findings may support the functional benefit of the counterforce brace. However, in the long-term, the use of orthotics may be no better than the natural course of the disease, left untreated. Despite the evidence presented here, a multimodal approach for lateral epicondylitis management remains the preferred treatment. This approach often includes orthotic wear, counter force brace, non-steroidal medication, activity modification, and therapy. Patients often expect some type of intervention to help with the management of their pain, and the use of orthotics provides a reasonable first line treatment option, with little downside and few side effects.

References

1. Morris H. The rider's sprain. Lancet. 1882;2:133–4.
2. Calfee RP, Patel A, DaSilva MF, Akelman E. Management of lateral epicondylitis: current concepts. J Am Acad Orthop Surg. 2008;16(1):19–29.
3. Struijs PA, Smidt N, Arola H, Dijk vC, Buchbinder R, Assendelft WJ. Orthotic devices for the treatment of tennis elbow. Cochrane Database Syst Rev. 2002;1:CD001821.
4. Tosti R, Jennings J, Sewards JM. Lateral epicondylitis of the elbow. Am J Med. 2013;126(4):357, e351–6.
5. Derebery VJ, Devenport JN, Giang GM, Fogarty WT. The effects of splinting on outcomes for epicondylitis. Arch Phys Med Rehabil. 2005;86(6):1081–8.
6. Garg R, Adamson GJ, Dawson PA, Shankwiler JA, Pink MM. A prospective randomized study comparing a forearm strap brace versus a wrist splint for the treatment of lateral epicondylitis. J Shoulder Elbow Surg. 2010;19(4):508–12.

7. Labelle H, Guibert R. Efficacy of diclofenac in lateral epicondylitis of the elbow also treated with immobilization. The University of Montreal Orthopaedic Research Group. Arch Fam Med. 1997;6(3):257–62.
8. Labelle H, Guibert R, Joncas J, Newman N, Fallaha M, Rivard CH. Lack of scientific evidence for the treatment of lateral epicondylitis of the elbow. An attempted meta-analysis. J Bone Joint Surg Br. 1992;74(5):646–51.
9. Luginbuhl R, Brunner F, Schneeberger AG. No effect of forearm band and extensor strengthening exercises for the treatment of tennis elbow: a prospective randomised study. Chir Organi Mov. 2008;91(1):35–40.
10. Mahaffey P. Tennis elbow. There is no proved treatment. BMJ. 2009;339:b5325.
11. Mahaffey PJ. Tennis elbow myths. BMJ. 2011;342:d3837.
12. Wolf JM. Do we need to treat tennis elbow? Orthopedics. 2012;35(11):921–2.
13. Bisset L, Paungmali A, Vicenzino B, Beller E. A systematic review and meta-analysis of clinical trials on physical interventions for lateral epicondylalgia. Br J Sports Med. 2005;39(7):411–422 (discussion 411–422).
14. Faro F, Wolf JM. Lateral epicondylitis: review and current concepts. J Hand Surg Am. 2007;32(8):1271–9.
15. Smidt N, Assendelft WJ, Arola H, Malmivaara A, Greens S, Buchbinder R, van der Windt DA, Bouter LM. Effectiveness of physiotherapy for lateral epicondylitis: a systematic review. Ann Med. 2003;35(1):51–62.
16. Struijs PA, Smidt N, Arola H, van Dijk CN, Buchbinder R, Assendelft WJ. Orthotic devices for tennis elbow. Cochrane Database Syst Rev. 2001;2:CD001821.
17. Snyder-Mackler L, Epler M. Effect of standard and Aircast tennis elbow bands on integrated electromyography of forearm extensor musculature proximal to the bands. Am J Sports Med. 1989;17(2):278–81.
18. Meyer NJ, Walter F, Haines B, Orton D, Daley RA. Modeled evidence of force reduction at the extensor carpi radialis brevis origin with the forearm support band. J Hand Surg Am. 2003;28(2):279–87.
19. Struijs PA, Kerkhoffs GM, Assendelft WJ, Van Dijk CN. Conservative treatment of lateral epicondylitis: brace versus physical therapy or a combination of both-a randomized clinical trial. Am J Sports Med. 2004;32(2):462–9.
20. Struijs PA, Smidt N, Arola H, van Dijk CN, Buchbinder R, Assendelft WJ. Orthotic devices for tennis elbow: a systematic review. Br J Gen Pract. 2001;51(472):924–9.
21. Altan L, Kanat E. Conservative treatment of lateral epicondylitis: comparison of two different orthotic devices. Clin Rheumatol. 2008;27(8):1015–9.
22. Van De Streek MD Van Der Schans CP De Greef MH Postema K. The effect of a forearm/hand splint compared with an elbow band as a treatment for lateral epicondylitis. Prosthet Orthot Int. 2004;28(2):183–9.

Chapter 11
Mini-open Surgery for Lateral and Medial Epicondylitis (Tendinosis)

Robert P. Nirschl

The term tendinosis is preferred to epicondylitis as this is more descriptive of the true pathology [1]. Epicondylitis (tendinosis) occurs at least five times more commonly on the lateral than on the medial aspect of the joint. The selection factors to determine the candidates for surgery are similar for each process, yet there are some distinct features with regard to the surgical technique [2]. Thus, medial epicondylitis is discussed separately in this chapter.

Indications/Contraindications

Pain is the major indication for surgery of lateral elbow tendinosis. There are three broad indications and a fourth feature to consider.

1) Pain is of significant intensity as to limit function; and interferes with daily activity or occupation; (Nirschl Pain Phases 5, 6, or 7) [3] (Table 11.1).
2) Localization is precisely at the attachment area of the extensor carpi radialis brevis (ECRB) or the extensor digitorum communis (EDC) at and just distal to the lateral epicondyle.
3) A legitimate period of nonoperative management has been attempted; this typically includes at least 6 months of activity modification, counterforce forearm band, anti-inflammatory agents, and a quality rehabilitation program; [4]
4) Failure of cortisone injections is no longer considered an absolute necessity prior to offering surgical intervention. However, if injections have been used and the patient is no longer benefiting or has not benefited from them, then the patient is a candidate for a surgical procedure. The technique of injection (e.g. properly placed under the ECRB tendon) is important regarding these considerations [5].

R. P. Nirschl (✉)
Nirschl Orthopaedic Center, Arlington, Virginia
e-mail: nirschlorthopaedics@comcast.net

J. M. Wolf (ed.), *Tennis Elbow,* DOI 10.1007/978-1-4899-7534-8_11

Table 11.1 Tendinosis phases of pain from [3]

Phase I	Mild pain after exercise activity, resolves within 24 h
Phase II	Pain after exercise activity, exceeds 48 h, resolves with warm-up
Phase III	Pain with exercise activity that does not alter activity
Phase IV	Pain with exercise activity that alters activity
Phase V	Pain caused by heavy activities of daily living
Phase VI	Intermittent pain at rest that does not disturb sleep Pain caused by light activities of daily living
Phase VII	Constant rest pain (dull aching) and pain that disturbs sleep

The contraindications to surgical intervention include an inadequate nonoperative program [4]; and patients who have demonstrated lack of compliance with the recommendations, particularly that of activity modification. Individuals on worker's compensation disability should be assessed on several occasions to assure that the above indications have been met.

Preoperative Planning

Physical findings include local tenderness to palpation over the tendon origin at the epicondyle. Provocative tests of pain with resisted wrist extension for lateral involvement are invariably positive especially with the elbow in full extension. In some cases the symptoms may be aggravated by performing the test with the elbow in 90° flexion (a sign indicating substantial tendinosis) [1, 4]. If forearm pain is a component (which is unusual), examine for posterior interosseous nerve irritation (an independent malady) [6]. The most sensitive test is pain on resistive supination [7].

The most commonly involved tissue, the pathological process, and the principles of the surgical intervention should be reviewed prior to undertaking the surgical procedure [8].

The hallmarks of good surgical concept and technique include precise identification of the pathologic tissue, resection of all involved pathology, maintenance of normal tissue attachments, protection of normal tissue, enhancement of vascular supply, firm repair of the operative site, and quality postoperative rehabilitation.

Kraushaar and Nirschl have now defined the histopathology in precise detail utilizing the methods of electron microscopy and histoimmunochemistry [1]. The ECRB and the anterior edge of the EDC are the tissues most commonly involved laterally (100% and ~35%, respectively) [9–12]. Histologically, the pathological tissue is devoid of inflammatory cells, but has a characteristic pattern of immature fibroblasts and vascular elements (Fig. 11.1a, b) [1, 13]. Recent electron microscopic evidence reveals lack of extracellular cross-linkage (Fig. 11.2a, b, c) [11]. The recommended surgical technique specifically focuses on demonstrated pathoanatomy. It should be noted that although the classical case, in our experience, is angiofibroblastic tendinosis in the ECRB; the anteromedial edge of the EDC origin has concomitant pathology in 35% of cases and when present, should be addressed

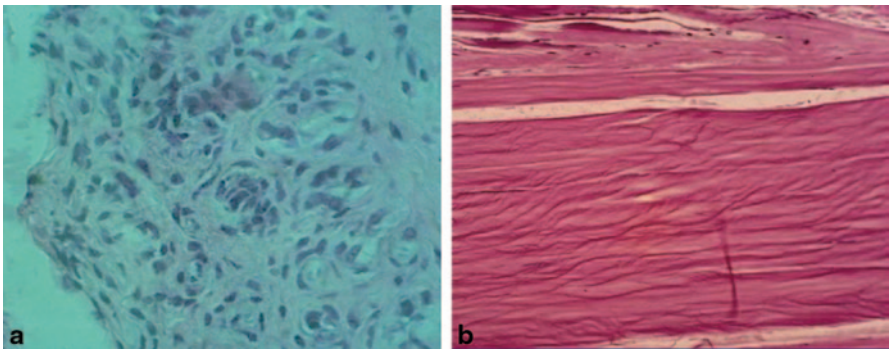

Fig. 11.1 **a** Histology of lateral extensor tendinopathy. Note the predominance of giant cells and monocytes, and the absence of neutrophils. **b** Cross-section of extensor carpi radialis brevis, with abnormal tissue pictured above, compared to normal tissue

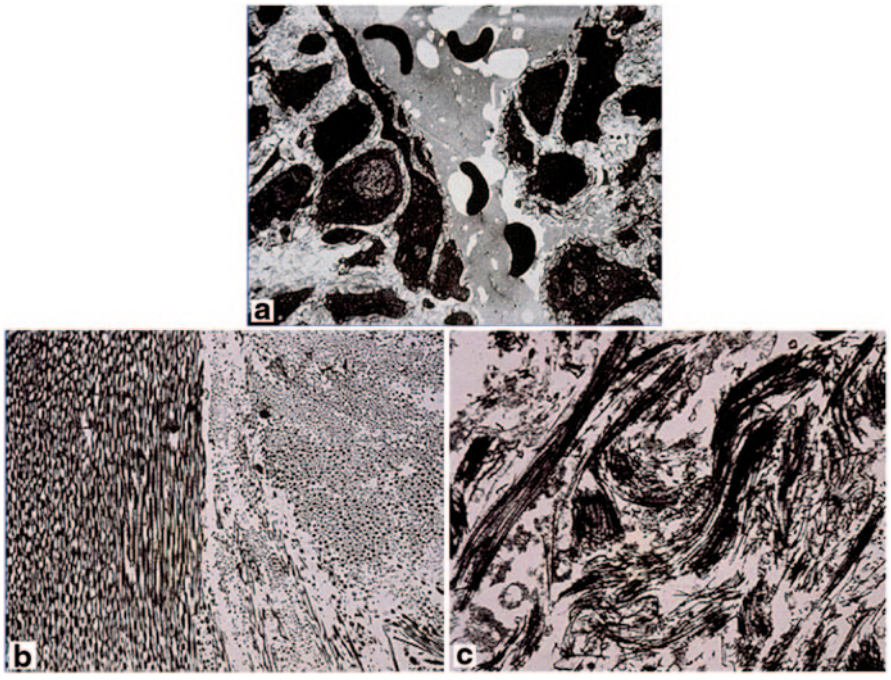

Fig. 11.2 **a–c** Transmission electron microscopy of the extensor tendon origin showing discontinuity of fibers and lack of cross-linkage seen in normal tendon

(Fig. 11.3). Additional pathologies include bony exostosis of the lateral epicondyle in 20% of cases, and anterolateral compartment intraarticular pathology such as synovitis, plica, and chondromalacia (5%) [14].

Intraarticular changes as noted above have also been observed with the advent of elbow arthroscopy as a therapeutic tool. Today we specifically assess by clinical

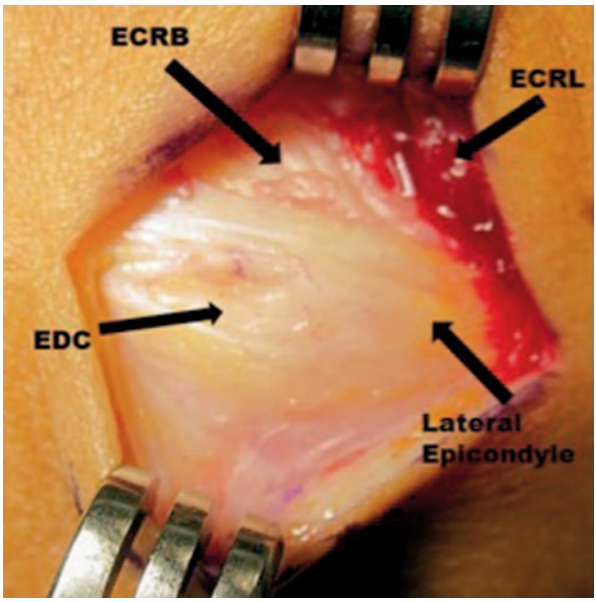

Fig. 11.3 Pathology is often seen both at the ECRB and EDC origin

exam and imaging studies, the possibility of any symptom producing intraarticular elements (5% in our experience) and proceed accordingly with possible arthroscopy or limited arthrotomy in such cases [3, 15, 16].

Surgery for Lateral Tendinosis

The described technique and illustrations apply for the large majority of cases. It should be noted, however, that individual variations can and do occur. In these instances, the pathological variations should be addressed as presented.

The data on arthroscopic debridement are incomplete at this time to draw a conclusion or to recommend this treatment with the exception of clearly identified symptoms producing intraarticular issues [1]. For the typical extraarticular symptomatology, there is no advantage of arthroscopy over our described mini-open technique [16]. Disadvantages of arthroscopy include increased risk to nerves and joint surfaces plus increased costs of OR time and instrumentation. More importantly, the excision of pain producing tendinosis tissue may be incomplete [17].

Technique

After anesthesia (general or arm block) is induced, a tourniquet is applied and the arm is draped free and placed on an arm board.

The incision extends proximal and just anteromedial to the lateral epicondyle for 2 to 3 cm down to the level just proximal to the joint (i.e., 1 cm distal to the epicondyle)

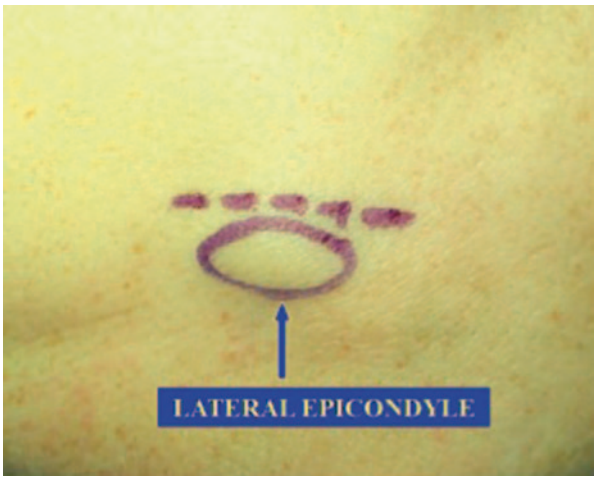

Fig. 11.4 The standard skin incision is approximately 4 cm extending 1–2 cm proximal and just anterior to the lateral epicondyle distally 1–2 cm to the level of the elbow joint and carried further distally 1 cm if the joint is explored. The *circled area* identifies the lateral epicondyle

(Fig. 11.4). It is important to place the incision accurately so as not to compromise the identification of the extensor carpi radialis longus (ECRL)-EDC interface at the deeper level. The subcutaneous tissue and superficial fascia are incised and retracted, locating the interface between the ECRL and the firm anterior edge of the extensor aponeurosis of the EDC. A palpable crevice is present at this interface as the fascia over the ECRL is thin and the anterior edge of the aponeurosis is firm and thick. A splitting incision 1 to 2 mm in depth is made between the ECRL and the extensor aponeurosis in the identified interface extending from 1 cm proximal to the lateral epicondyle distally to the level just proximal to the joint line. The ECRL is undermined with sharp dissection and retracted anteromedially approximately 1.5 cm. This retraction brings the ECRB, under the ECRL, into direct view (Fig. 11.5) [3, 18].

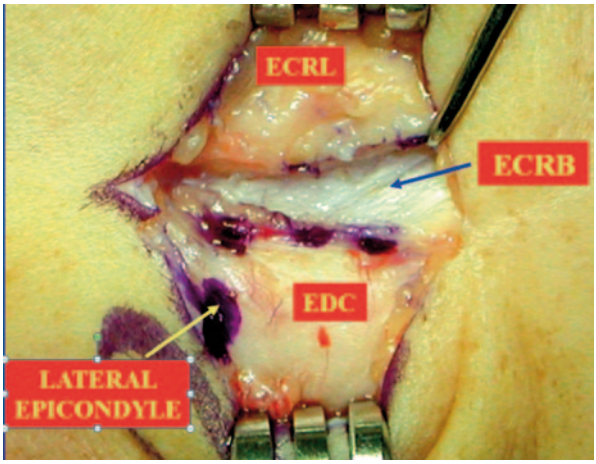

Fig. 11.5 An incision in the extensor longus aponeurosis interface with anteromedial retraction of extensor longus exposes the patholgoical origin of the extensor brevis. A key technical point is not to incise too deeply but more medially as the extensor longus is only 2–3 mm in depth at this level

Technical note: A common error in the incision at the ECRL interface is to penetrate too deeply by vertical dissection. As noted, the extensor longus is only 1 to 2 mm thick at this region. Once the 1 to 2 mm depth is reached, the dissection is primarily horizontal progressing medially. This technical subtlety is important to avoid iatrogenic distortion as well as confusing the identification of the ECRB tendon. Such iatrogenic distortion can easily complicate the identification of the pathological tendinosis tissue in the ECRB origin.

With proper case selection and appropriate exposure, the entire origin of the ECRB is easily identified. The gross appearance of the pathological tendinosis change is most often a dull-grayish tissue, which is typically edematous and friable, and, on occasion, ruptured (Figs. 11.6, 11.7, 11.8a). Normal tendon tissue in contrast is shiny, firm, and has a slightly yellowish-white hue. The pathological tissue often encompasses the entire origin of the ECRB, and in our series, the anterior 10% edge of the extensor aponeurosis is abnormal in approximately 35% of cases [6, 16] (Fig. 11.7).

Excision of all pathological tissue at the ECRB origin is performed en bloc. This tissue block is somewhat triangular in shape with the base distal. The typical size of the tissue excised is 2 × 1 cm (Fig. 11.8b). It should be noted that in this dissection, the brevis origin is released from the lateral epicondyle and the anterior edge of the extensor aponeurosis. If the anterior aponeurosis has pathological alteration, the pathological tissue is also removed (but not normal tendon). Release of the normal EDC aponeurosis from the epicondyle is unnecessary, potentially harmful, and should be avoided.

Pathological tissue in the EDC and ECRB is easily identified by its visual appearance and confirmed by the "Nirschl scratch test" (Fig. 11.9) [3, 16]. This makes use of the friability of pathological tissue, which easily peels off by utilizing a

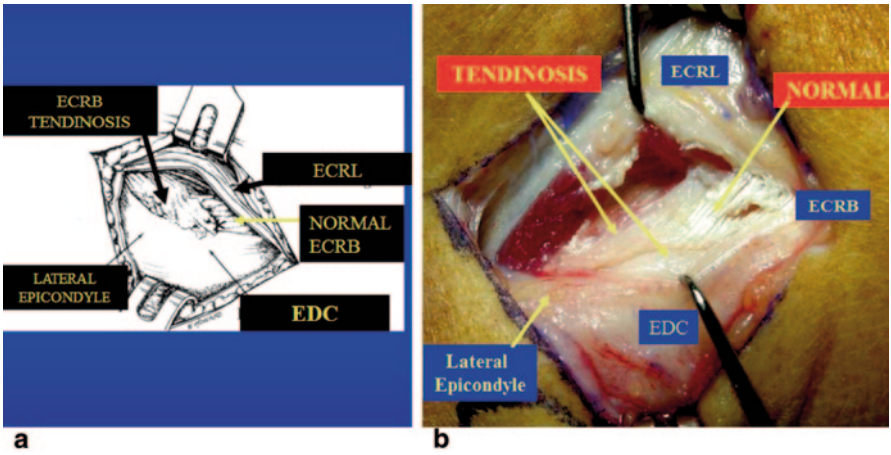

Fig. 11.6 Exposure for resection of pathological tissue. In this rendering, 100% of the origin is involved and a partial rupture is depicted. In no circumstance is the extensor aponeurosis totally released from the epicondyle

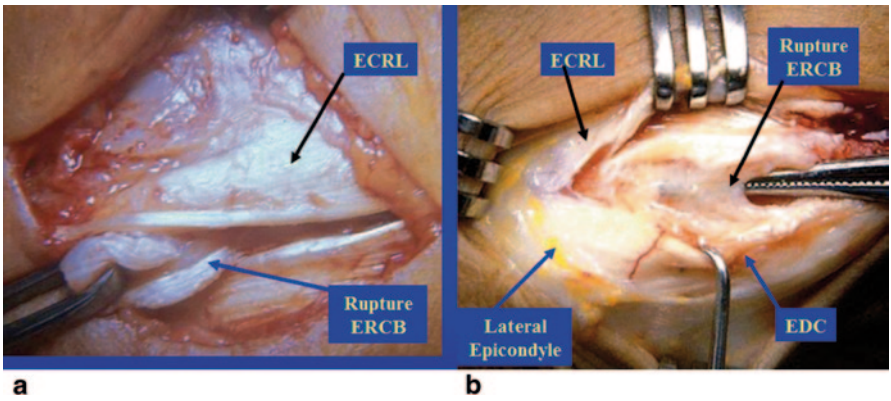

Fig. 11.7 Surgical photograph of resection of pathological extensor brevis origin shows major tendinosis with an underside rupture. Note a small strip of normal tendon at the edge of the extensor longus muscle. The remaining pathological alteration has a dull-grayish edematous gross appearance typical of angiofibroblastic tendinosis

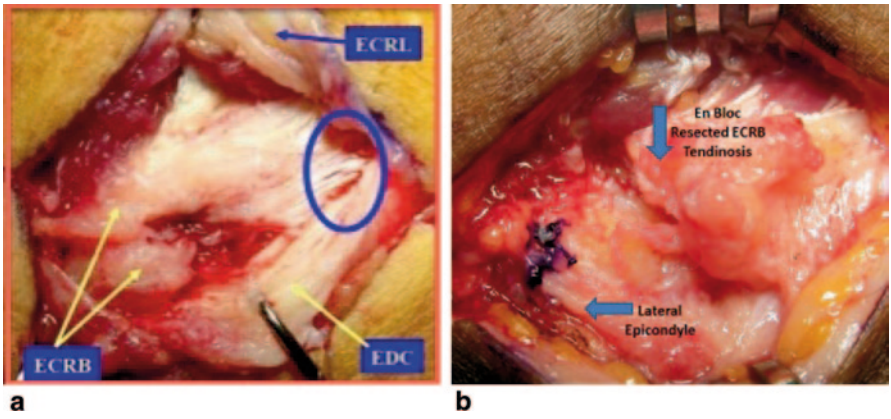

Fig. 11.8 **a** Degenerated extensor brevis origin. **b** Resection specimen of ECRB origin

vigorous scratching motion with the scalpel. When healthy tissue is reached, it no longer peels off with the scratching motion. This technique is especially helpful when removing the pathological changes in the anterior edge of the aponeurosis. The scratching technique should be vigorous to remove all pathological tissue.

In the 20% of cases that present with an exostosis or prominence of the lateral epicondyle, the proximal anteromedial edge of the EDC aponeurosis is temporarily peeled off the epicondyle for adequate exposure and the exostosis removed by rongeur and smoothed by a rasp. When this does occur, the exostosis usually occupies about 15% of the anteromedial edge of the epicondyle (not the entire epicondyle). Thus the majority of the epicondyle and aponeurosis attachment is left undisturbed. We believe that it is unnecessary and contraindicated to do further epicondylar resection.

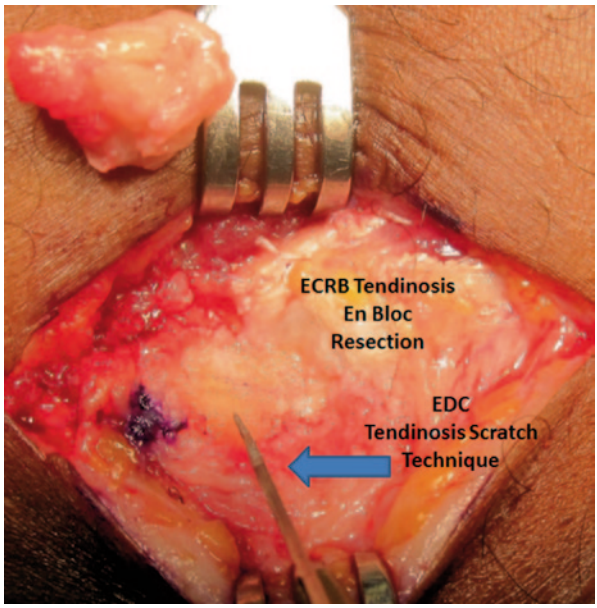

Fig. 11.9 Demonstration of "scratch test." A scalpel edge is used to scrape off degenerated friable tissue

Once the pathological tissue is adequately resected, a defect is present in the area of the ECRB tendon origin. The more distal aspect of the extensor brevis is still attached to the orbicular ligament, distal anterior aponeurosis, and underside of the ECRL. The ECRB, therefore, does not retract distally to any appreciable degree, thereby maintaining an essentially normal working length of the entire extensor muscle-tendon unit (i.e., from elbow to wrist). It is therefore not necessary to reattach the remaining brevis with sutures or a bone anchor. The goal of the operation is resection of all pathological tissue, not tendon release (e.g. the common expression of describing the operation as a tendon release operation is erroneous). It is to be emphasized that all normal tendon attachments are not disturbed and not released.

In the 5 % of cases whose preoperative evaluation indicates intraarticular abnormality, a small synovial opening may be made at this time to inspect the anterolateral joint compartment. This can be easily accomplished by extending the incision distally 5–10 mm [1]. Unless the patient presents with clear intraarticular signs and symptoms preoperatively, it is rare to find intraarticular changes, and the arthrotomy incision is therefore, in the majority of cases, unnecessary and not recommended.

To enhance vascular supply, one small hole is drilled through the cortical bone in the area of ECRB resection (not the epicondyle) (Fig. 11.10). This technique is theorized to encourage rapid replacement of this ECRB resection tissue void with healthy fibrotendinous tissue.

The interface between the posterior edge of the ECRL and the remaining anterior edge of the extensor aponeurosis is now firmly closed (Fig. 11.11). Current choice is an absorbable number 1 polydioxanone suture (PDS). It is unnecessary to suture

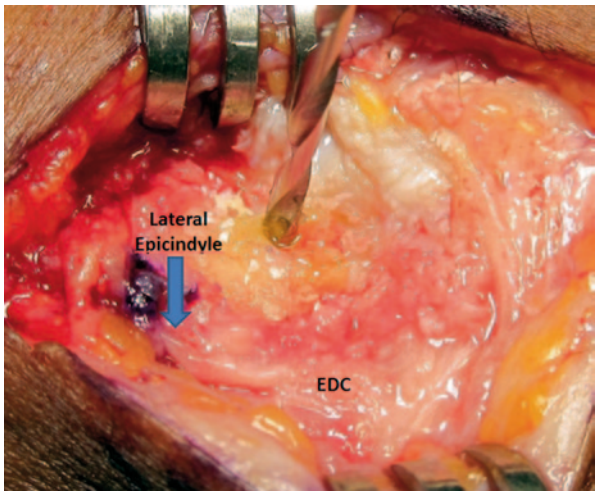

Fig. 11.10 The cortical region distal to epicindyle is drilled to increase blood supply

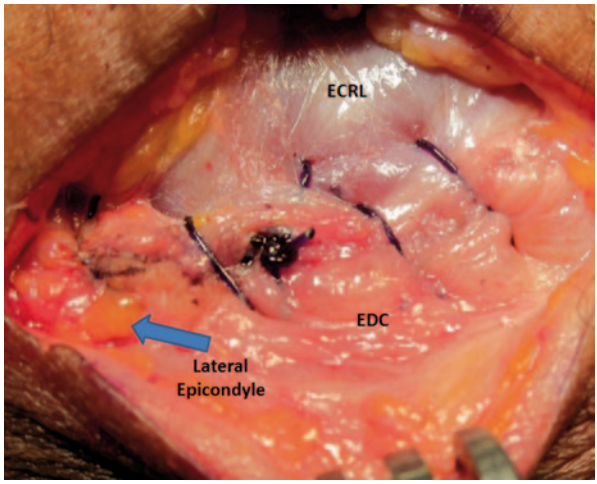

Fig. 11.11 Repair of tendon interface. In all cases the interface between the extensor longus and the extensor aponeurosis is firmly closed. It is theorized that blood clot transformed to biologically healthy fibrous tissue (painless) replaces the proximal defect of the resected area further reinforcing the security of the ultimate brevis origin

the distal ECRB, since a firm attachment is retained to the orbicular ligament, distal aponeurosis, and underside of ECRL distally. The anterior medial edge of the extensor aponeurosis is therefore firmly repaired to the ECRL. In thin patients, place the knots deeply or use a polyglactin (Vicryl) suture. Since the proximal attachment of the EDC is largely undisturbed, rapid mobilization postoperatively is possible and encouraged. The subcutaneous layer is closed in routine fashion by the subcuticular skin technique with absorbable suture. The author's preference is 2–0 or 3–0 poliglycaprone (Monocryl) supported by adhesive skin strips.

New Technique & Technology

Recent ultrasound treatment technology utilizing the above principles of tendinosis pathology identification and excision is on the horizon. The concept is to emulsify tendinosis tissue by a specifically designed high frequency ultrasound probe. The first published article of 20 cases, although preliminary, appears promising. Further investigation and long-term follow-up are indicated [19].

Postoperative Management

The arm is placed in an elbow immobilizer with four Velcro straps (Fig. 11.12). The joint is then immobilized for 2 days at 90° flexion, the forearm is in neutral, and the wrist and hand are free. Motion exercises are usually started within 48 h postoperatively.

Intermittent immobilizer protection is usually maintained intermittently for another 3–4 days, at which time normal activities of daily living are resumed. Counterforce support (forearm band) [20] providing protective function is utilized until full forearm strength returns (usually 3–6 months). The brace is used at times of rehabilitation exercise and more vigorous forearm activities such as heavier household activities. A gradual return to sports often is initiated at 4–6 weeks with brace protection. Participation in more intensive sports, particularly competitive athletics such as tennis [21], usually takes 3–5 months.

Fig. 11.12 Light elbow immobilizer with Velcro straps provides comfortable support in the immediate postoperative period. Motion exercises are usually started 48 h postoperative but intermittent immobilizer protection is usually maintained for 6 to 7 days (Courtesy of Medical Sports Inc.)

Results

We have shown that with the described lateral side surgery, 97% of patients can expect improvement and 93% of patients can expect full return of all prior activities [21]. In 3% no improvement is observed. Thus, less than 3% of patients are considered failures. Success with other techniques has been reported, with 85 to 90% response rates [9, 10, 12, 15].

Complications and Surgical Failure

The most frequent complication after surgery for lateral tennis elbow is residual pain. This is not common with the technique described above. When pain after surgery is present, a logical analysis is conducted and the following determinations must be considered [22]:

1) Has there been sufficient time and/or proper rehabilitation to allow adequate healing?
2) Did the proper diagnosis exist prior to the surgical intervention?
3) Did something occur at the time of surgery to cause iatrogenic symptoms?
4) Was the true pathoanatomy adequately addressed? This is the most common cause of failure (e.g. inadequate surgical excision of pain-producing tendinosis tissue). In this case, a second surgical procedure should be considered (Fig. 11.13) [22, 23]. The success of a second procedure which utilizes our described technique is 83% [23]. Worker's compensation may affect an individual's motivation and should be considered during the rehabilitation phase.

Complications by other techniques can ensue from an aggressive release of the EDC tendon from the lateral epicondyle, which can result in a release of the collateral ligament with resulting joint instability. Occasionally, instability is manifested as residual pain and not as laxity. This is diagnosed by stress view radiographs and occasionally by an arthrogram. The treatment in this circumstance is collateral ligament repair or reconstruction.

Medial Epicondylitis/Tendinosis

Introduction

As with the lateral elbow we prefer the term tendinosis to epicondylitis as the problem is in the common flexor tendons, not the epicondyle. The histopathology also has no inflammatory cells [1, 24]. Medial elbow tendinosis is less common than lateral elbow tendinosis by a factor of one to five [2].

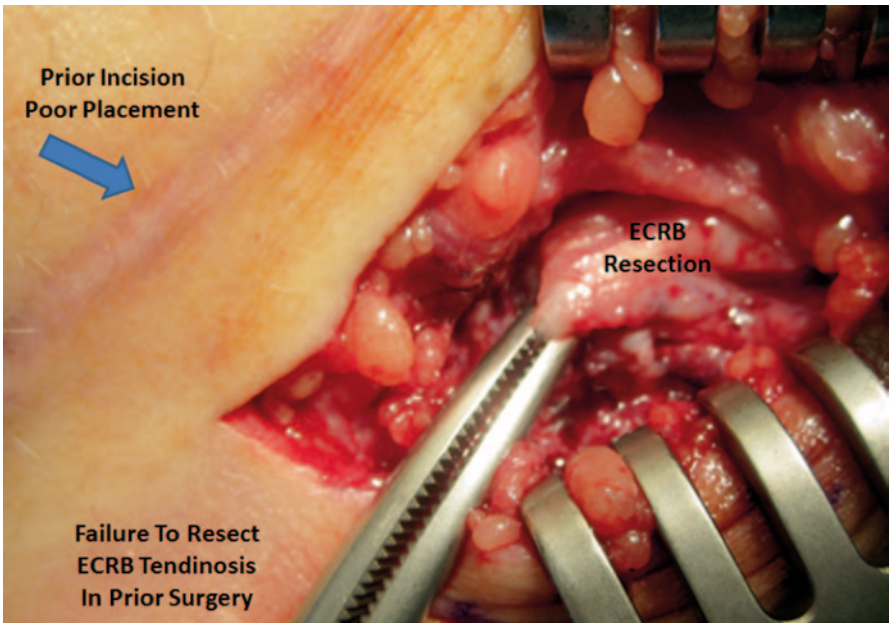

Fig. 11.13 Salvage surgery photos for initial surgery which failed to identify and resect ECRB tendinosis. Misplaced initial incision directly over lateral epicondyle (*arrow*). It is critical to place the incision accurately so as not to compromise the identification of the ECRL-EDC interface. Exposure and removal of abnormal ECRB which was not addressed at initial operation. The most common cause of failed surgery

Indications

As with lateral epicondylitis, the indication for surgery is pain that limits daily activity and/or interrupts sleep (Table 11.1). The duration of nonoperative management is usually 6–9 months, and ideally at least 12 months. All conservative measures should have been tried. At least one cortisone injection is helpful to isolate the lesion location while offering temporary pain control. Limited temporary pain control of this therapy strengthens the indications for surgery. Total failure (e.g. no pain control) raises concern about the etiology of symptoms (emotional factors or secondary gain motivation) or possibly inadequate injection technique.

Contraindications

The most significant contraindication is a history and physical that does not accurately coincide with expectations of medial epicondylitis. Poor motivation, worker's compensation and unrealistic expectations are issues of concern and to be considered before surgical intervention is carried out. Individuals who are

improving or who have had symptoms for less than 6 months are generally not considered candidates for surgery.

Presentation and Classification

Medial epicondylitis is a consequence of acute or chronic loads applied to the flexor-pronator mass of the forearm as a result of activity related to the medial elbow and proximal forearm [24]. It is approximately one-fifth as common as lateral epicondylitis and has a similar demographic profile. The concomitant presence of ulnar neuropathy at the elbow is seen in 30–50% of patients and may be the primary management concern [18, 25–28]. Physical examination reveals common flexor origin and direct epicondylar tenderness and indirect pain with resisted pronation and wrist flexion. Ulnar nerve examination may demonstrate a positive Tinel's sign, elbow flexion test or nerve compression test. Valgus stress examination is essential to assess ulnar collateral ligament sprain or medial instability either as an associated concern or as the primary process. Subluxation of the medial head of the triceps and medial antebrachial cutaneous neuropathy should be ruled out as well [29].

Plain radiographs are helpful to evaluate additional diagnoses, most commonly degenerative arthritis (which may require diagnostic xylocaine injection of the elbow to differentiate an intraarticular vs. an extraarticular source of symptoms). Valgus stress radiographs should be obtained if indicated. Magnetic resonance imaging can be helpful if symptoms suggest additional abnormalities, but is usually not required as this is primarily a clinical diagnosis.

Medial epicondylitis is classified with a combined epicondylitis and ulnar neuropathy classification system [25]. To simplify the original classification, Type I is an isolated medial epicondylitis and Type II is medial epicondylitis with an associated ulnar neuropathy. This may be further classified as (a) minimal or (b) moderate ulnar nerve severity.

The initial management of Type I medial epicondylitis is similar to lateral epicondylitis including corticosteroid injection, counterforce bracing, wrist splinting and a conditioning program [3, 30]. Injections should be placed at the proximal anterior aspect of the common flexor origin just distal to the epicondyle with the elbow in extension to avoid the ulnar nerve [16] and the anterior oblique ligament. Instances of Type I and Type II medial epicondylitis that fail to respond to nonoperative management are indications for surgical intervention.

Preoperative Planning

The surgical procedure of choice relates to the classification of medial epicondylitis. Operative management of Type I medial epicondylitis involves medial common flexor origin debridement alone [3, 18, 24, 26]. In the past, percutaneous release was reported but is currently not recommended [31]. The Type II medial epicondylitis

may require ulnar nerve decompression including cubital tunnel release [2, 3, 16, 26]. Ulnar nerve transfer is indicated for symptoms caused by nerve tension (eg. skeletal or dynamic valgus instability) or a completely dislocating nerve, both uncommon [3, 32]. On occasion a subluxing medial head of the triceps may occur and should not be confused with a dislocating nerve [29]. Occasionally, a small epicondylar exostosis may be removed, if present. Medial epicondylectomy should be avoided, as anterior epicondylar removal (for medial epicondylitis) and posterior epicondylar removal (for the ulnar nerve) may result in compromise of the anterior oblique ligament origin. Tendinosis usually involves the flexor carpi radialis and the medial side of the pronator teres. It is best to excise this tissue longitudinally in elliptical fashion thereby preserving all normal tendon attachments [3, 18, 24, 26, 32].

Valgus instability, if present, may be operatively treated at the same setting with anterior oblique ligament reconstruction in association with a longitudinal split in the common flexor origin. Exposure to the area of ligament repair or reconstruction by this exposure is less punishing and the ulnar nerve does not require transfer in most instances. Ulnar nerve complications and delayed rehabilitation secondary to prior techniques of total release of the flexor pronator mass and submuscular nerve transfer are thereby largely eliminated.

The common flexor origin (CFO) is the primary anatomic focus in medial epicondylitis. It lies immediately anterior and superficial to the anterior oblique ligament with, in most cases, no identifiable interval between these two structures. The CFO serves as the tendon origin for the flexor pronator mass musculature including the flexor carpi ulnaris, flexor carpi radialis, the pronator teres, the palmaris longus, and the deeper positioned flexor digitorum sublimus. At the level of the medial epicondyle, the tendon is fully conjoint. The common flexor origin tendon unit can extend distally up to 4–5 cm into the forearm but the usual is 3–4 cm. Gross pathologic involvement of the tendon is usually seen within the proximal 2–3 cm of the tendon, the level where it is fully conjoint. It is at this level that the surgical debridement in medial epicondylitis is conducted.

Surgery

Type I Medial Epicondylitis—Isolated Medial Epicondylar Debridement

Prior to anesthesia, it is important to clearly reidentify the area of tenderness as this will identify the area of pathology. After induction of a general anaesthetic, the arm is prepped and draped in the usual fashion. A longitudinal skin incision is created starting 1 cm posterior to the proximal margin of the medial epicondyle and extending distally for 3–4 cm [3, 18, 32]. Posterior skin incision placement avoids and therefore protects the anterior branches of the medial antebrachial cutaneous nerve (Fig. 11.14). The skin and subcutaneous tissue are easily retracted upward

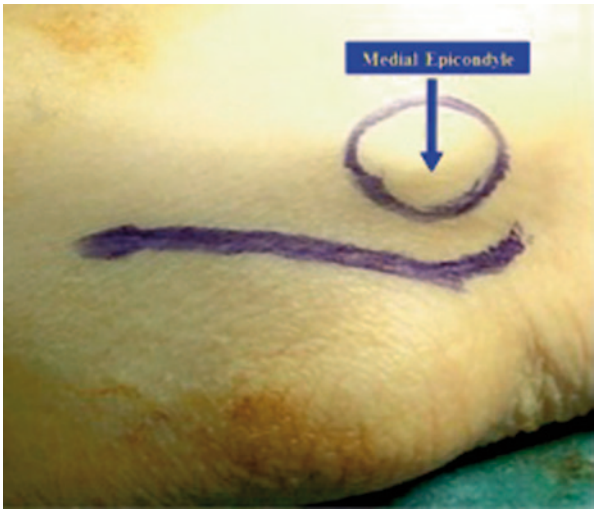

Fig. 11.14 Skin incision—placement of incision posterior to the medial epicondyle decreases the potential of injury to the branches of the medial antebrachial cutaneous nerves

and anterolaterally exposing the proximal margin of the common flexor origin at the superior aspect of the medial epicondyle extending distally to a level approximately 3 cm distal to the inferior aspect of the medial epicondyle. The superficial fascia of the flexor pronator mass is longitudinally incised starting at the proximal edge of tendinosis tissue, usually flexor carpi radialis and medial side of the pronator teres [18, 32, 33]. Elliptical resection or scratch technique resection of all pathological tissue is undertaken (Fig. 11.15).

Large lesions are immediately evident at this site. If a degenerative nidus is present, it is usually seen proximally close to the tip of the medial epicondyle. The anterior oblique ligament can be seen and palpated with a Freer elevator immediately posterior to the common flexor tendon. The interval between these two structures can be developed by passing the Freer elevator down along the posterior margin of the medial conjoint tendon, but this is usually unnecessary as the depth of tendon resection rarely reaches the ligament level. The volume of tendinosis pathology can vary. Larger lesions in the flexor carpi radialis and medial side of the pronator teres can extend 2–3 cm distally from the tip of the medial epicondyle. Elliptical resection is undertaken leaving the normal flexor ulnaris and lateral side of the pronator teres insertion intact (Fig. 11.16) [18, 32]. The width of this resection may reach 1 cm but is usually 3–6 mm (Fig. 11.16). Smaller lesions usually start at the tip of the medial epicondyle extending distally 4–5 mm and are best removed by the Nirschl scratch technique (e.g. vigorously scratching the tissue with a scalpel). The anterior oblique ligament can be inspected, but is usually grossly normal in most instances except in chronic valgus instability situations (this is usually anticipated preoperatively by history and physical examination). At this time, a small drill hole is made in the cortical bone 3–4 mm distal to the epicondyle (not in the epicondyle) to enhance vascular supply to this area [18].

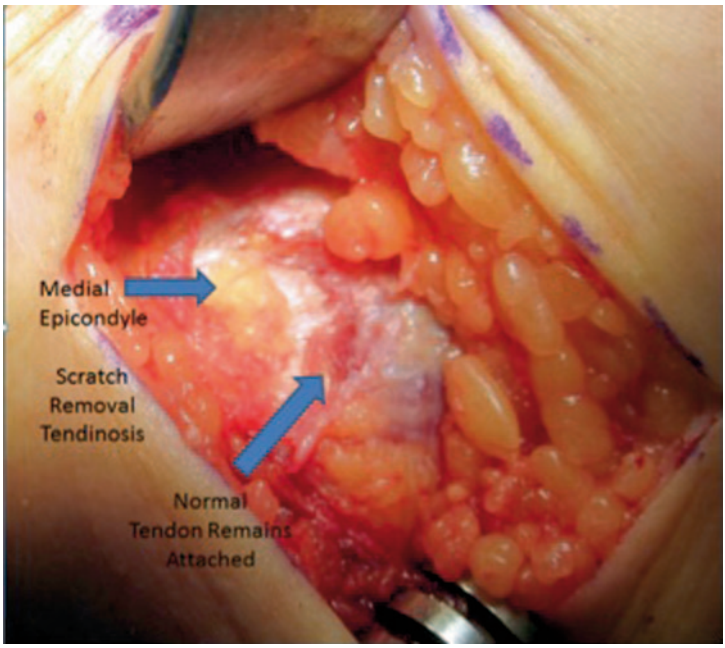

Fig. 11.15 Resection of tendinosis tissue with elliptical longitudinal incision [34]

If a bony exostosis is present (not usual) at the anterior aspect of the epicondyle, it can be "freshened up" with a rongeur. Do not alter the body of the epicondyle as increased postoperative pain is the result as well as the potential of harm to the oblique ligament. The resected elliptical tendon defect is firmly closed with absorbable sutures [16, 18, 33]. (author preference 2.0 PDS). The skin and subcutaneous closure is in routine fashion (author preference 2.0 Monocryl).

A small soft dressing is applied with the elbow in 90° of flexion followed by an elbow immobilizer with the wrist and hand free and the forearm in neutral position [16, 18, 33].

Type II Medial Epicondylitis—Medial Tendon Debridement with Cubital Tunnel Release or Nerve Transposition

In cases with associated ulnar neuropathy (II) the most common site of compression impingement is the Zone 3 of Nirschl (Fig. 11.17) [3, 14, 16]. Cubital tunnel release may be performed in this setting, or in Type I cases where larger tendon resection and repair tightens the area resulting in the potential for increased cubital tunnel compression. To clarify, cubital tunnel release is indicated when the environs of the ulnar nerve at the cubital tunnel are subject to compression (not nerve tension) and are pristine, i.e., no prior trauma, good nerve gliding without scarring, and no

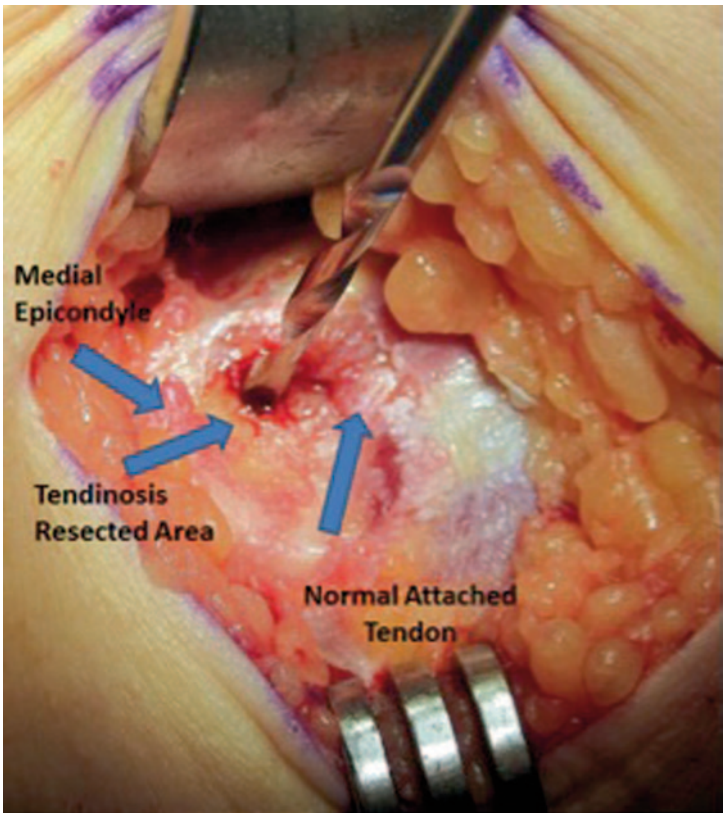

Fig. 11.16 Resection of extensive tendinosis, with cortical drilling to enhance vascular access to resected area. Do not alter or drill epicondyle

complete dislocation with elbow flexion either prior to or following cubital tunnel release. It is recommended in Zone 3 and, if needed, Zone 2 to release the Zones on the posterior side of the nerve [35]. The posterior side release leaves more anterior upper tunnel tissue thereby enhancing nerve stability (i.e**.,** to maintain the nerve in the cubital tunnel). If upward subluxation or perching of the nerve to the upper outer edge of the epicondyle occurs without full dislocation, it often does not require transfer [24]. It is important to always check the opposite elbow preoperatively for signs of asymptomatic congenital subluxation, which may be present and if so reinforces the judgment not to transfer the nerve. In Type II cases, the procedure of debriding the medial conjoint tendon is identical to the above description, but is performed only after the nerve status has been assessed. In most instances with or without subluxation it is unnecessary to transfer the nerve [28, 36]. Indications for transfer include tension on the nerve via skeletal valgus (e.g., prior fracture) or valgus ligamentous instability, scar environment, or complete nerve dislocation and instability [14, 16, 24].

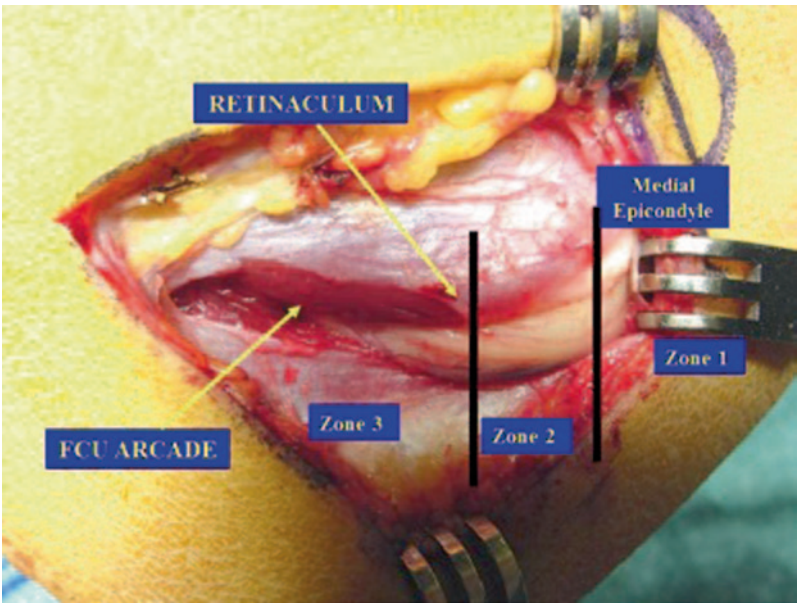

Fig. 11.17 Anatomy of Nirschl ulnar nerve zones—the common flexor tendon in this photo is normal. The decompression of the ulnar nerve is at 2 areas of zone 3 of the cubital tunnel (The retinaculum at the entry of zone 3 and release of the flexor ulnaris muscle at the exit of zone 3)

For nerve decompression, the previously described skin incision posterior and distal to the medial epicondyle is utilized (approximately 2 in in length). The posterior margin of the medial epicondyle is identified and the flexor carpi ulnaris fascia exposed. The fascia overlying the nerve is opened by scalpel and tenotomy scissors at the posterior/inferior corner of the medial epicondyle. The fascia is then incised distally for 1–2 cm exposing the two heads of the flexor carpi ulnaris muscle. As noted, keep the incisional releases on the posterior side of the nerve [35]. The two FCU heads form a V distally. At this point, flex the elbow to 135° to assess nerve stability. To complete the decompression, a split is made at the apex of the V with dissection proceeding distally 1–2 cm, splitting the two heads of the flexor carpi ulnaris, which lies directly over the ulnar nerve. The operative release thus takes place in Nirschl Zone 3, occasionally extending into Zone 2 [3, 14, 16, 24].

The ulnar nerve is then examined without disturbing its bed. The elbow is again flexed to 135° to assess for dislocation. The cubital tunnel is also assessed for other abnormalities (e.g., scar adhesions, or bony exostoses). Nerve transfer may be indicated in the circumstances of hostile scar environment or complete nerve dislocation (uncommon) [14]. In the usual case (e.g., no nerve transfer), the elliptical defect in the common flexor origin is firmly closed (author preference is 2–0 PDS). The cubital tunnel is left open.

The need for ulnar nerve transposition, whether subcutaneous or submuscular, is uncommon. The indications for a transfer include hostile scar environment, nerve

tension secondary to valgus and complete nerve dislocation. The highlights of subcutaneous transfer techniques will be discussed, but we recommend other resources for the techniques in detail. Subcutaneous transfer (preferred by the authors) is very technique-specific**.** The nerve needs to be placed approximately 3/4 inch distal lateral from the epicondyle for best protection. The brachial fascia overlying the nerve posterior to the intermuscular septum, proximal to the medial epicondyle is identified. The fascia is incised proximally (Zone 1) releasing the arcade of Struthers in the process. With the elbow in relative extension to relax the nerve, the fascia is incised distally, ultimately extending into the fascia of the flexor carpi ulnaris (Zone 2). Distal release of the flexor ulnaris arcade (Zone 3) should be generous to ensure adequate relaxed nerve relocation. Proximally, the distal attachment of the medial intermuscular septum to the epicondyle is released. Once the nerve is free and relaxed, transfer can occur. As noted, do not place the nerve over the epicondyle but 3/4 inch anterolateral (Fig. 24). Care is also taken to preserve the vascular arterial and venous elements which often lie just posterior to the nerve. If possible, it is recommended to transfer these vascular elements with the nerve. Submuscular transposition is an alternate consideration, but requires more tissue dissection and is not the author's preference.

Skin closure is completed with or without transfer in usual manner and the arm is placed into an elbow immobilizer, well padded at the posterior elbow to prevent any splint irritation.

Postoperative Management

Postoperative procedures for Type I or II medial elbow surgery are similar to the lateral elbow protocol. The arm is placed in an elbow immobilizer at 90° of flexion, the forearm is in neutral, and the wrist and hand are free. After 2 days, gentle active assisted motion exercises are usually started (tendon and/or tendon with nerve decompression surgery). Intermittent immobilizer protection is usually continued for 6 to 7 days at which time normal activities of daily living are resumed [3, 18]. Counterforce support (not too tight especially if the nerve is decompressed) providing protective function is utilized until full forearm strength returns (usually 3–6 months). The brace is used at times of rehabilitation exercise and more vigorous forearm activities such as heavier household activities. A gradual return to sports often is initiated at 6 to 8 weeks with counter-force brace protection (Fig. 11.13). Full sports participation (such as play to win in tennis) may take 4–6 months (e.g., somewhat longer than lateral elbow rehabilitation).

For the uncommon Type II procedure with nerve transfer, active assistive motion is delayed for 1 week with use of the elbow immobilizer. If a concomitant lateral tennis elbow tendinosis debridement has been performed, a wrist splint may be considered for lateral elbow comfort (not usually needed). For Type I or II surgery strengthening is usually started at 3 weeks. The flexor pronator rehab program is continued until symmetric pain-free strength is gained. A counterforce brace is

continued until full strength is restored. Full activity status for physically demanding activities such as sports is reserved until the same point in time. This takes a minimum of 3–6 months on the average.

Results

There is an emerging body of evidence that has clarified the expectations of surgery for medial epicondylitis. Four recent reports all reveal a greater than 90% satisfaction with this surgery [12, 24, 26, 28]. These results can occur with associated Zone 3 ulnar nerve decompression. However, both Gabel and Morrey [26] and Kurvers and Verhaar [27] emphasize a poorer prognosis associated with more complex ulnar nerve involvement. Final outcome is usually realized in 6 months in most, but may take over a year to fully recover in some. Results of concomitant medial and lateral surgery with 10 year follow-up has been reported by Schipper and Nirschl with a satisfaction success of 95% [2].

Complications and Surgical Failure

The most frequent complication after surgery for medial elbow tendinosis is residual pain. This is not common in the Mayo, Jobe, or Nirschl experience and occurs in less than 10% of patients. However, Kurvers et al. report a significantly worse prognosis with medial than lateral epicondylitis [27].

Incisional complications are not common if the described technique of a skin incision placed posterior to the medial epicondyle and elliptical excisions of tendinosis tissue is utilized. If prior reported techniques of incisions anterior to the epicondyle and transverse resection (e.g., release) of the flexor origin are utilized, complications related to medial antebrachial cutaneous nerves as well as the flexor pronator can occur. Medial antebrachial cutaneous (MABC) neuropathy may result from avulsion, traction or transection of these nerve branches. If this is recognized intraoperatively, the nerve should be mobilized proximally and transposed into the brachialis muscle belly. If the neuropathy is identified postoperatively, a desensitization program as well as neurogenic pain medication, such as nortriptyline or gabapentin may be helpful. A corticosteroid injection at the point of maximum Tinel's sign may be useful if the neuropathic pain persists. Sympathetic mediated pain may result from MABC injury, but is not synonymous. If other hallmarks of a sympathetically-mediated pain process are identified, pain management consultation may be indicated. As noted, a posteriorly placed incision, which avoids the MABC nerve branches, will likely avoid this complication.

Transient exacerbation of the ulnar neuropathy symptoms is not uncommon especially if a transposition has been performed. Objective incomplete loss of function of the ulnar nerve is uncommon, but when it occurs usually resolves spontaneously.

Complete loss of ulnar nerve function is quite rare and may indicate compression from a hematoma, fibrous band, or acute angulation. Complete loss of function requires early re-exploration, but may be associated with no objective level of compression, indicating a possible intraneural vascular event. Recovery is usually seen even in these circumstances, but is typically incomplete.

Medial collateral ligament injury can occur with transverse release of the common flexor origin or epicondylectomy (not recommended) and may result in medial instability of the elbow. If this occurs, anterior oblique ligament reconstruction may prove to be necessary to alleviate symptoms [37].

Persistent postoperative medial epicondylitis symptoms may indicate a prolonged recovery rather than a failure of the procedure. Symptoms that reoccur or continue after 6 months should be managed in a manner similar to the preoperative program. If symptoms are present after a transposition procedure it is most likely nerve induced rather than tendon or scar induced. The therapeutic modalities of cold and heat, and ultrasound may be helpful. If injections are used after a transposition procedure, they should be placed to avoid intraneural injection of the ulnar nerve [36]. This requires a clear understanding of the prior operative technique and nerve position. Significant epicondylar symptoms that persist beyond 18–24 months may require revision, but this is the case in less than 2 to 3% of medial epicondylitis cases. Persistent ulnar nerve symptoms are more common, but still rarely require revision. Ulnar nerve symptoms after decompression usually respond to physiotherapy. Symptoms after nerve transfer usually occur with excess scar and kinking with submuscular transfer or the nerve placed too close to the epicondyle in subcutaneous transfer. As noted, the described techniques of posteriorly placed incisions, elliptical resection of medial elbow tendinosis tissue, decompression of the cubital tunnel at Nirschl Zone 3 and on occasion Zone 2, and subcutaneous transfer (not commonly needed) of the ulnar nerve likely avoid the above noted complications.

The most common issue of persistent tendon pain may reflect failure of identification and complete resection of tendinosis tissue at the index surgery. In this instance, ultrasound, diagnostic imaging, or MRI investigation may be helpful. If this occurs, further treatment effort, either nonsurgical or surgical resection of residual tendinosis tissue may be considered [14, 16].

References

1. Kraushaar BS, Nirschl RP. Tendinosis of the elbow (tennis elbow). Clinical features and findings of histological, immunohistochemical, and electron microscopy studies. J Bone Joint Surg Am. 1999;81(2):259–78.
2. Schipper ON, Dunn JH, Ochiai DH, Donovan JS, Nirschl RP. Nirschl surgical technique for concomitant lateral and medial elbow tendinosis: a retrospective review of 53 elbows with a mean follow-up of 11.7 years. Am J Sports Med. 2011;39(5):972–6.
3. Nirschl RP. Elbow tendinosis/tennis elbow. Clin Sports Med. 1992;11(4):851–70.
4. Nirschl RP, Sobel, J. Conservative treatment of tennis elbow. Phys Sports Med. 1981;9:42–5.

5. Nirschl RP, Rodin DM, Ochiai DH, Maartmann-Moe C, Group D-A-S. Iontophoretic administration of dexamethasone sodium phosphate for acute epicondylitis. A randomized, double-blinded, placebo-controlled study. Am J Sports Med. 2003;31(2):189–95.
6. Roles NC, Maudsley RH. Radial tunnel syndrome: resistant tennis elbow as a nerve entrapment. J Bone Joint Surg Br. 1972;54(3):499–508.
7. Rosen I, Werner CO. Neurophysiological investigation of posterior interosseous nerve entrapment causing lateral elbow pain. Electroencephalogr Clin Neurophysiol. 1980;50(1–2): 125–33.
8. Nirschl RP. Sports and overuse injuries to the elbow. In: Morrey B, editor. Muscle and tendon trauma: medial and lateral tennis elbow. Philadelphia: WB Saunders; 2000.
9. Boyd HB, McLeod AC Jr. Tennis elbow. J Bone Joint Surg Am. 1973;55(6):1183–7.
10. Coonrad RW, Hooper WR. Tennis elbow: its course, natural history, conservative and surgical management. J Bone Joint Surg Am. 1973;55(6):1177–82.
11. Goldie I. Epicondylitis lateralis humeri (epicondylalgia or tennis elbow). A pathogenetical study. Acta Chir Scand Suppl. 1964;57:(SUPPL 339):1+.
12. Nirschl RP, Pettrone FA. Tennis elbow. The surgical treatment of lateral epicondylitis. J Bone Joint Surg Am. 1979;61(6A):832–9.
13. Regan W, Wold LE, Coonrad R, Morrey BF. Microscopic histopathology of chronic refractory lateral epicondylitis. Am J Sports Med. 1992;20(6):746–9.
14. Nirschl RP. Sports and overuse injuries to the elbow. In: Morrey B, editor. Muscle and tendon trauma. Philadelphia: WB Saunders; 2000.
15. Baker CL Jr, Baker CL 3rd. Long-term follow-up of arthroscopic treatment of lateral epicondylitis. Am J Sports Med. 2008;36(2):254–60.
16. Nirschl RP, Ashman ES. Tennis elbow tendinosis (epicondylitis). Instr Course Lect. 2004;53:587–98.
17. Cummins CA. Lateral epicondylitis: in vivo assessment of arthroscopic debridement and correlation with patient outcomes. Am J Sports Med. 2006;34(9):1486–91.
18. Nirschl RP, Ashman ES. Elbow tendinopathy: tennis elbow. Clin Sports Med. 2003;22(4): 813–36.
19. Koh JS, Mohan PC, Howe TS, Lee BP, Chia SL, Yang Z, Morrey BF. Fasciotomy and surgical tenotomy for recalcitrant lateral elbow tendinopathy: early clinical experience with a novel device for minimally invasive percutaneous microresection. Am J Sports Med. 2013;41(3):636–44.
20. Groppel JL, Nirschl RP. A mechanical and electromyographical analysis of the effects of various joint counterforce braces on the tennis player. Am J Sports Med. 1986;14(3):195–200.
21. Dunn JH, Kim JJ, Davis L, Nirschl RP. Ten-to 14-year follow-up of the Nirschl surgical technique for lateral epicondylitis. Am J Sports Med. 2008;36(2):261–6.
22. Morrey BF. Reoperation for failed surgical treatment of refractory lateral epicondylitis. J Shoulder Elbow Surg. 1992;1(1):47–55.
23. Organ SW, Nirschl RP, Kraushaar BS, Guidi EJ. Salvage surgery for lateral tennis elbow. Am J Sports Med. 1997;25(6):746–50.
24. Ollivierre CO, Nirschl RP, Pettrone FA. Resection and repair for medial tennis elbow. A prospective analysis. Am J Sports Med. 1995;23(2):214–21.
25. Gabel GT, Morrey, Bernard F. Medial Epicondylitis. In: Morrey BF, editor. The elbow and its disorders, vol. 1. Philadelphia: Saunders; 2000. p. 537–42.
26. Gabel GT, Morrey BF. Operative treatment of medical epicondylitis. Influence of concomitant ulnar neuropathy at the elbow. J Bone Joint Surg Am. 1995;77(7):1065–9.
27. Kurvers H, Verhaar J. The results of operative treatment of medial epicondylitis. J Bone Joint Surg Am. 1995;77(9):1374–9.
28. Vangsness CT, Jr., Jobe FW. Surgical treatment of medial epicondylitis. Results in 35 elbows. J Bone Joint Surg Br. 1991;73(3):409–11.
29. Spinner RJ, Goldner RD. Snapping of the medial head of the triceps and recurrent dislocation of the ulnar nerve. Anatomical and dynamic factors. J Bone Joint Surg Am. 1998;80(2): 239–47.

30. Nirschl RP, Kraushaar BS. Assessment and treatment guidelines for elbow injuries. Phys Sports Med. 1996;24(5):42–60.
31. Baumgard SH, Schwartz DR. Percutaneous release of the epicondylar muscles for humeral epicondylitis. Am J Sports Med. 1982;10(4):233–6.
32. Nirschl RP. Prevention and treatment of elbow and shoulder injuries in the tennis player. Clin Sports Med. 1988;7(2):289–308.
33. Nirschl RP. Lateral extensor release for tennis elbow. J Bone Joint Surg Am. 1994;76(6):951.
34. Nirschl R. Elbow tendinosis/tennis elbow. In: Renstrom AFH, Leadbetter WB, editors. Clinics in sports medicine, tendinitis II: clinical considerations. Philadelphia: WB Saunders; 1992. p. 865. (11(4)).
35. Hsu PA, Hsu AR, Sutter EG, Levitz SP, Rose DM, Segalman KA, Lee SK. Effect of anterior versus posterior in situ decompression on ulnar nerve subluxation. Am J Orthop. 2013;42(6):262–6.
36. Stahl S, Kaufman T. Ulnar nerve injury at the elbow after steroid injection for medial epicondylitis. J Hand Surg. 1997;22(1):69–70.
37. Jobe FW, Stark H, Lombardo SJ. Reconstruction of the ulnar collateral ligament in athletes. J Bone Joint surg Am. 1986;68(8):1158–63.

Chapter 12
Arthroscopic Treatment of Lateral Epicondylitis

Mark S. Cohen

Introduction

The origin of the extensor carpi radialis brevis (ECRB) has been implicated as the source of pathology in this condition [2, 5, 7–10, 13–16, 18]. Reported histopathologic findings in the affected tendon origin include vascular proliferation and hyaline degeneration, which are consistent with a chronic, degenerative process [10, 14, 18, 20]. Most commonly, surgical treatment is directed at excision of this pathologic tissue through an open approach or more recently arthroscopic methods [1, 3, 5, 6, 11, 15, 17, 19, 21, 22].

This chapter covers the anatomy of the extensor tendon origins at the humeral epicondyle based on anatomic dissections [4]. The location of the ECRB tendon origin is defined relative to intraarticular landmarks. Using this data, a technique for arthroscopic lateral epicondylitis surgery is presented with early clinical results.

Anatomy The ECRL and the ECRB have a unique relationship at the level of the elbow. The ECRL overlies the proximal portion of the ECRB such that the ECRL must be elevated anteriorly in order to visualize the superficial surface of the ECRB. A thin film of areolar connective tissue separates these two structures.

The ECRL origin is entirely muscular along the lateral supracondylar ridge of the humerus (Fig. 12.1). The muscle origin has a triangular configuration with the apex pointing proximally. In contrast, the origin of the ECRB is entirely tendinous. While it blends with the origin of the EDC, it can be separated from the EDC back to the humerus when dissected from a distal to proximal direction and using the tendon undersurface (Fig. 12.1). The anatomic origin of the ECRB is located just beneath the distalmost tip of the lateral supracondylar ridge (Fig. 12.2). The footprint is

M. S. Cohen (✉)
Director, Hand and Elbow Section, Director, Orthopaedic Education, Department of Orthopedic Surgery, Rush University Medical Center, 1611 West Harrison Street, Chicago, IL 60612, USA
e-mail: mcohen3@rush.edu

J. M. Wolf (ed.), *Tennis Elbow,* DOI 10.1007/978-1-4899-7534-8_12

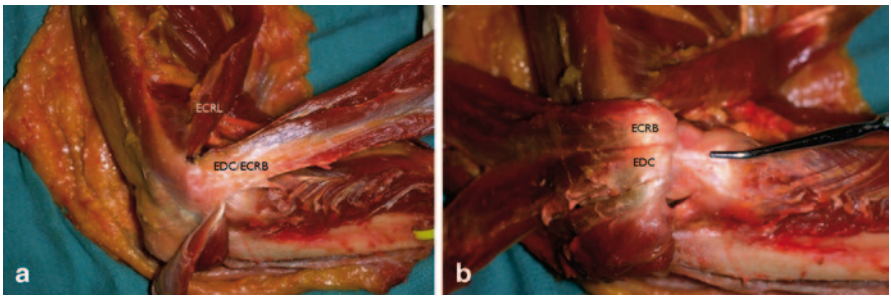

Fig. 12.1 **a** Lateral view of cadaveric specimen. The ECRL has been reflected anteriorly (it has a purely muscular origin) and the extensor carpi ulnaris posteriorly revealing the common extensor tendon origin of the ECRB and EDC. These are indistinguishable when viewed from the outer surface. **b** The muscles and tendons have been reflected proximally. The origins of the ECRB anteriorly and the EDC posteriorly are identifiable on the undersurface of the extensor origin. Note the underlying lateral collateral ligament (probe) (Courtesy of Mark S. Cohen, Chicago, IL with permission.)

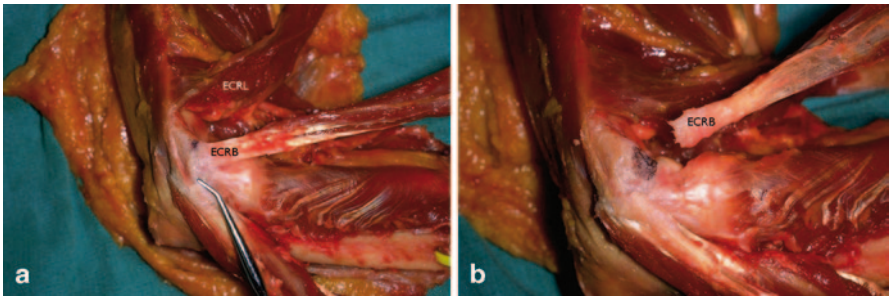

Fig. 12.2 **a** The EDC has been removed allowing better visualization of the bony ECRB origin on the humerus. **b** The ECRB footprint is identified with elevation of the tendon from the humerus (Courtesy of Mark S. Cohen, Chicago, IL with permission.)

diamond-shaped, measuring approximately 13 by 7 mm (Fig. 12.3). At the level of the radiocapitellar joint, the ECRB is closely apposed to the underlying anterior capsule of the elbow joint, but it is easily separable at this level [4]. Using these data, an arthroscopic technique was developed for lateral epicondylitis.

Technique The patient is positioned in the lateral decubitus position with the arm supported and all bony prominences well padded. Regional anesthesia is favored by the authors. Bony landmarks are drawn out including the path of the ulnar nerve. Once the tourniquet is inflated, the elbow is insufflated with an 18-gauge needle introduced through the soft-spot of the elbow.

Next, a standard anteromedial portal is established (Fig. 12.4). This is started several centimeters proximal and anterior to the medial epicondyle and well anterior to the palpable intermuscular septum. Care is taken to slide along the anterior humerus and the joint is entered with a blunt introducer or a switching stick. This

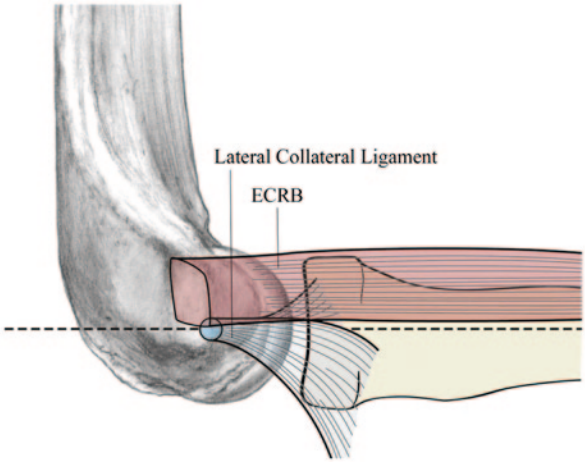

Fig. 12.3 Schematic diagram depicting the relationship between the ECRB origin at the humerus and bony landmarks. Note that the ECRB footprint origin is diamond shaped and located between the midline of the joint and the top of the humeral capitellum beneath the most distal extent of the supracondylar ridge. The tendon does not originate on the epicondyle specifically. Note the relationship between the ECRB origin and the underlying lateral collateral ligament (Courtesy of Mark S. Cohen, Chicago, IL with permission.)

medial portal allows one to view the lateral joint including the radial head, capitellum, and the lateral capsule. It is often helpful at this point to open the inflow to allow distension of the capsule. If visualization is a problem, a retractor can be introduced through a proximal anterolateral portal 2–3 cm proximal and just anterior to the lateral supracondylar ridge. A simple Freer elevator is useful for this purpose. By tensioning the capsule anteriorly, improved visualization of the lateral capsule and soft tissues can be achieved.

A modified anterolateral portal is established using an inside-out technique. This is started 2–3 centimeters above and anterior to the lateral epicondyle (Fig. 12.4). The portal is slightly more proximal than a standard anterolateral portal. This allows instrumentation down to the tendon origin rather than entering the joint through the ECRB tendon itself. If lateral synovitis is present, this can be debrided with a resector.

The capsule is released next. Occasionally in epicondylitis, one can find a disruption of the underlying capsule from the humerus (Fig. 12.5). Most commonly, the capsule is intact although small linear tears can be present (Fig. 12.6). We have found it easier to release the lateral soft tissues in layers using a monopolar thermal device. In this way, the capsule is first incised or released from the humerus. When it retracts distally, one can appreciate the ECRB tendon posteriorly and the ECRL, which is principally muscular, more anterior. As noted above, the ECRB tendon spans from the top of the capitellum to the midline of the radiocapitellar joint.

Once the capsule is adequately resected, the ECRB origin is released from the epicondyle (Figs. 12.4 and 12.6). This is started at the top of the capitellum and

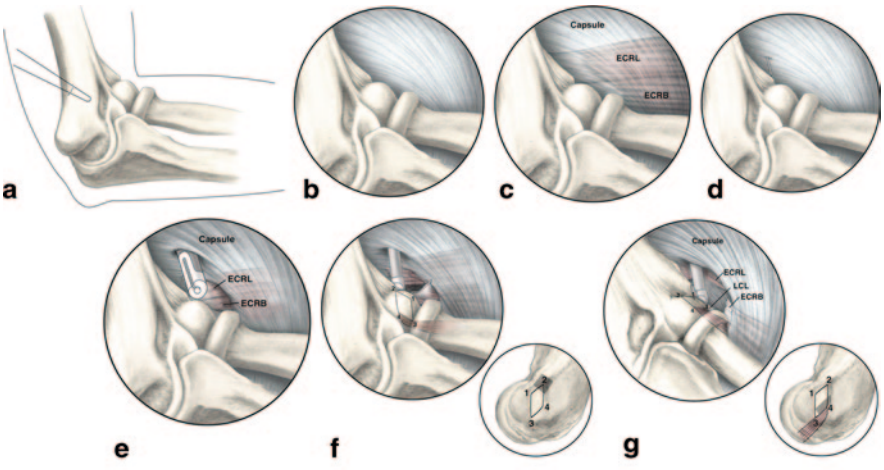

Fig. 12.4 **a** Diagram depicting the medial portal used in visualization for the arthroscopic lateral epicondylar release. **b** Field of view from the medial portal. **c** Diagram depicting the relationship of the extensor tendon origins when viewed intraarticularly. These are located outside (behind) the elbow capsule. **d** Needle used to help establish a modified lateral portal. Note how this has begun slightly proximal and anterior to the proximal margin of the humeral capitellum. **e** Release of the capsule from the lateral humeral margin allowing visualization of the tendinous origins behind. The ECRL is more anteriorly located and is muscular. The ECRB is more posterior. **f** The ECRB is released from the top of the capitellum to the **g** midline of the radiocapitellar joint (Courtesy of Mark S. Cohen, Chicago, IL with permission.)

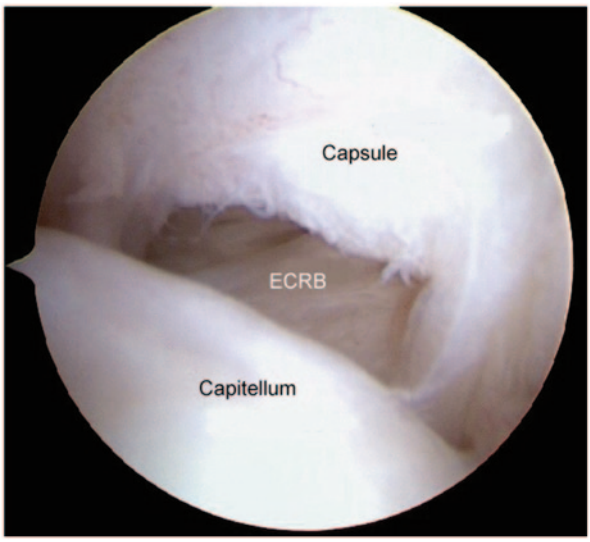

Fig. 12.5 Initial intraoperative view of a patient with recalcitrant lateral epicondylitis. Note the capsular disruption. In some cases, the capsule is noted to have torn away from its humeral origin (Courtesy of Mark S. Cohen, Chicago, IL with permission.)

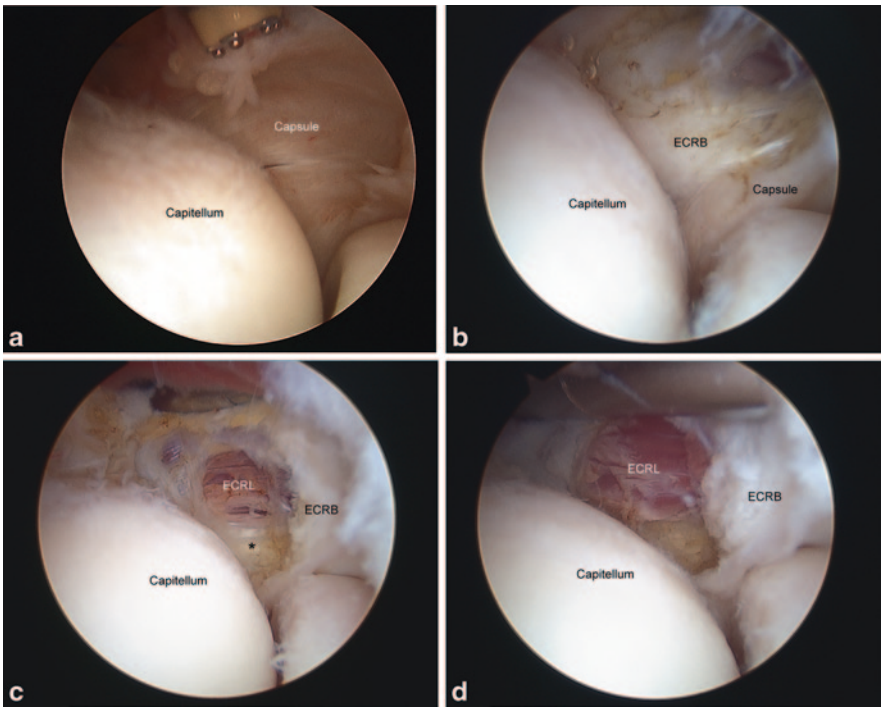

Fig. 12.6 **a** Initial intraoperative view of a patient treated surgically for lateral epicondylitis. The lateral capsule obstructs the view of the extensor tendon origins. Note the small longitudinal rent in the capsule. **b** The capsule has been released revealing the muscular ECRL anteriorly and the tendinous ECRB more posteriorly. Note the capsular layer distally which is deep to the tendon. **c** The ECRB has been released. Behind this, one can see the muscular ECRL anteriorly and the extensor aponeurosis which lies behind the ECRB (asterisk). It is characteristically composed of longitudinally stripped tendinous fibers much less distinct than the ECRB. **d** Final close up view following ECRB release. One can see the thick ECRB origin which has retracted distally following release (Courtesy of Mark S. Cohen, Chicago, IL with permission.)

carried posteriorly. The lateral collateral ligament is not at risk if the release is kept anterior to the midline of the radiocapitellar joint [19]. On average, adequate resection of the ECRB must include approximately 13 mm of tendon origin from anterior to posterior [4]. Care is taken to drive the scope in adequately to view the release down to the midline of the radiocapitellar joint. Typically, the entire ECRB retracts distally away from the humerus.

Care is taken not to release the extensor apponeurosis, which lies behind the ECRB tendon. This can be visualized as a stripped background of transversely (longitudinally) oriented tendon and muscular fibers much less distinct than the ECRB (Fig. 12.6). It is located posterior to the ECRL which again is principally muscular in origin. If the apponeurosis is violated, one will debride into the subcutaneous tissue about the lateral elbow.

Discussion

In recent years, there has been an interest in arthroscopic treatment of lateral epicondylitis [1, 3, 6, 11, 17, 19, 21, 22]. A cadaveric study demonstrated that arthroscopic release of the extensor carpi radialis brevis was a safe, reliable, and reproducible procedure for refractory lateral epicondylitis [11]. However, the results of arthroscopic treatment of this condition have been variable. Tseng reported satisfactory results in 9 of 11 patients [22]. However, he also had a 33 % complication rate. Stapleton and Baker compared five patients treated arthroscopically with ten patients treated by open debridement [21]. They reported similar results and complication rates between the two groups. Later, Baker et al. reported on 39 elbows treated arthroscopically with 37 reporting being "better" or "much better" at follow-up [1]. Peart et al. reported on 33 arthroscopic procedures for lateral epicondylitis with 28 % of patients failing to achieve good or excellent outcomes [17].

The variable results reported using various arthroscopic techniques may be related to increased difficulty in identifying the ECRB origin through the arthroscope [6]. The tendon is extra-articular and capsular release is required to visualize its origin. The tendon footprint is diamond-shaped and located between midline of the radiocapitellar joint and the top of the humeral capitellum averaging 13 by 7 mm (Fig. 12.3). The posterior interosseous nerve should be well medial and distal to the area of dissection. The lateral collateral ligament is not compromised as long as the release does not course posterior to the midline of the radial head [19]. The ligament is not at risk if the release is kept anterior to the midline of the radiocapitellar joint. Care is taken not to release the extensor apponeurosis, which lies superficial to the ECRB tendon.

We reviewed a consecutive series of 36 patients with recalcitrant lateral epicondylitis treated with arthroscopic release using the aforementioned technique [12]. There were 24 men and 12 women with an average age of 42 years at the time of surgery. The cohort had symptoms for an average of 19 months prior to surgical intervention. Intraoperative findings revealed significant lateral intraarticular synovitis in approximately 30 % of patients. Approximately 75 % of cases had an intact elbow capsule or a minor linear capsular tear, while 25 % had a significant proximal capsular disruption. All patients were evaluated by independent examiners for the purposes of this study, at a minimum 2 year follow-up. On average, patients required 4 weeks to return to regular activities and 7 weeks to return to full work duties. No major complications were reported. One patient had a neurapraxia of the superficial radial nerve that resolved by 2 weeks postoperatively. The average functional component of the Mayo Elbow Performance Score at follow-up averaged 11.1 out of 12 (range 5–12). Grip strength averaged 91 % of the opposite, uninvolved side. Subjective pain ratings as measured on a visual analog scale improved 8.1 to 1.5. However, ten patients reported continued pain with strenuous activities and repetitive use of the affected arm. Two patients continued to have significant pain and were considered failures [12]. Thus, the surgical treatment of lateral epicondylitis, whether by open or arthroscopic methods, remains somewhat unpredictable and some patients will have persistent symptoms. This highlights the need for careful patient selection.

In summary, arthroscopic release of the ECRB appears to be an effective option for the surgical treatment of chronic lateral epicondylitis unresponsive to conservative modalities. Knowledge of the anatomy, including the extensor tendon origins as visualized from an intraarticular perspective, is essential for effective surgical release.

References

1. Baker CL, Murphy KP, Gottlob CA, Curd DT. Arthroscopic classification and treatment of lateral epicondylitis: two-year clinical results. J Shoulder Elbow Surg. 2000;9(6):475–82.
2. Bunata RE, Brown DS, Capelo R. Anatomic factors related to the cause of tennis elbow. J Bone Joint Surg Am. 2007;89(9):1955–63.
3. Cohen MS, Romeo AA. Lateral epicondylitis: open and arthroscopic treatment. J Am Soc Surg Hand. 2001;3(1):172–6.
4. Cohen MS, Romeo AA, Hennigan SP, Gordon M. Lateral epicondylitis: anatomic relationships of the extensor tendon origins and *implications* for arthroscopic treatment. J Shoulder Elbow Surg. 2008;17(6):954–60.
5. Coonrad RW, Hooper WR. Tennis elbow: its course, natural history, conservative and surgical management. J Bone and Joint Surg. 1973;55A:1177–82.
6. Cummins CA. Lateral epicondylitis: in vivo assessment of arthroscopic debridment and correlation with patient outcomes. Am J Sports Med. 2006;34:1486–91.
7. Cyriax JH. The pathology and treatment of tennis elbow. J Bone and Joint Surg 1936;18:921–40.
8. Garden RS. Tennis elbow. J Bone and Joint Surg. 1961;43-B:100–6.
9. Gardner RC. Tennis elbow: diagnosis, pathology, and treatment. Clin Orthop. 1970;72:248–51.
10. Kraushaar BS, Nirschl RP. Tendinosis of the elbow (tennis elbow). Clinical features and findings of histological, immunohistochemical, and electron microscopy studies. J Bone and Joint Surg. 1999;81A:259–78.
11. Kuklo TR, Taylor KF, Murphy KP, Islinger RB, Heekin RD, Baker CL Jr. Arthroscopic release for lateral epicondylitis: a cadaveric model. Arthroscopy. 1999;15:259–64.
12. Lattermann C, Romeo AA, Anbari A, Meininger AK, McCarty LP, Cole BJ, Cohen MS. Arthroscopic debridement of the extensor carpi radialis brevis for recalcitrant lateral epicondylitis. J Shoulder Elbow Surg. 2010;19(5):651–56.
13. Morrey BF. Reoperation for failed surgical treatment of refractory lateral epicondylitis. J Shoulder Elbow Surg. 1992;1:47–55.
14. Nirschl RP. Elbow tendinosis/tennis elbow. Clinics in Sports Medicine. 1992;11:851–70.
15. Nirschl RP, Pettrone FA. Tennis elbow: the surgical treatment of lateral epicondylitis. J Bone and Joint Surg. 1979;61A:832–9.
16. Organ SW, Nirschl RP, Kraushaar BS, Guidi EJ. Salvage surgery for lateral tennis elbow. Am J Sports Medicine. 1997;25:746–50.
17. Peart RE, Strickler SS, Schweitzer KM. Lateral epicondylitis: a comparative study of open and arthroscopic lateral release. Am J Orthop. 2004;33:565–7.
18. Regan W, Wold LE, Coonrad R, Morrey BF. Microscopic histopathology of chronic refractory lateral epicondylitis. Am J Sports Medicine. 1992;20:746–9.
19. Smith AM, Castle JA, Ruch DS. Arthroscopic resection of the common extensor origin: anatomic considerations. J Shoulder Elbow Surg. 2003;12(4):375–9.
20. Spencer GE, Herndon CH. Surgical treatment of epicondylitis. J Bone and Joint Surg. 1953;35-A:421–4.
21. Stapleton TR, Baker CL. Arthroscopic treatment of lateral epicondylitis. Arthroscopy. 1996;10:335–6.
22. Tseng V. Arthroscopic lateral release for treatment of tennis elbow. Arthroscopy. 1994;10:335–6.

Chapter 13
Denervation of the Humeral Epicondyles

A. Lee Dellon

Introduction

Painful humeral epicondylar problems are a continuing management problem for those caring for the upper extremity, whether that physician is a specialist in sports medicine, orthopedic surgery, hand surgery, physical medicine and rehabilitation, or pain management. As this textbook demonstrates, a great deal of information is available to support a wide range of nonoperative approaches for acute humeral epicondylar pain and nonoperative and operative approaches for chronic epicondylar pain. A current Internet search on PubMed from the National Institutes of Health will bring up 1498 publications related to the search term "tennis elbow," and approximately the same magnitude of publications are identified searching for "lateral humeral epicondylitis" or "medial humeral epicondylitis." In the last two decades, the "recent" important additions to the treatment options are the use of cold laser [1], shock wave therapy [2], the use of platelet-rich plasma with "growth factors" [3], and the availability of Level I evidence to guide the use of nonoperative modalities [4–7].

Although 90 % of patients with humeral epicondylitis respond to many nonoperative modalities, including placebos, there will ultimately be a population of patients with persistent pain despite the best attempts to manage their condition, and a group of these patients who really need relief in order to work, carry out their activities of daily living, or return to playing tennis. There will also be a group of patients with persistent pain after surgical attempt(s) to relieve their pain. It is the purpose of this chapter to introduce the concept of "denervation" into the armamentarium of those physicians caring for patients with humeral epicondylitis, both lateral and medial.

A. L. Dellon (✉)
Johns Hopkins University, The Exchange Bldg, suite 18, 1122 Kenilworth Dr.,
Towson, MD 21204, USA
e-mail: aldellon@dellon.com

J. M. Wolf (ed.), *Tennis Elbow,* DOI 10.1007/978-1-4899-7534-8_13

Background: Joint Denervation

Denervation was introduced for the hip joint in 1942 by Traviner and Truet, with resection of branches of the obturator nerve [8]. After Wilhelm described the innervation of the wrist joint in 1958 and 1966 [9, 10], Buck-Gramcko reported the experience with total wrist denervation in 1977 [11]. After defining the anatomy of the posterior interosseous nerve in 1979 [12] and the anterior interosseous nerve in 1984, the concept of partial wrist joint denervation was introduced by Dellon and coworkers for the wrist in 1984 and 1985 [13, 14]. After defining the innervation for the knee in 1994 [15], Dellon and coworkers reported partial knee denervation [16] for patients who had a total knee replacement, and described a similar procedure in 1996 [17] for those with residual knee after sports injury or arthroscopy. Similarly, after defining the innervation of the shoulder joint in 1996 [18], Dellon and coworkers reported on the technique and results of partial shoulder denervation in 2004 [19]. The authors also defined the innervation of the sinus tarsi in 2001 [20], with partial ankle denervation described [21]. These concepts have been reviewed in 2009 [22, 23]. While the humeral epicondyles are not joints, their periosteum is innervated and the concept of denervation can be applied to pain arising from the muscle origins of these bony prominences. *Implicit in the concept of partial joint denervation is that the patient must respond to a nerve block with relief of pain and with improved joint function.*

Technique: Denervation of the Lateral Humeral Epicondyle

In 1996, Wilhelm suggested treatment of tennis elbow by denervation, but his approach was quite different from that to be described below [24]. He obtained, "on average 90% success in cases of resistant tennis elbow … denervation is accomplished blindly by disinsertion of certain muscles. The result of this procedure also depends on simultaneous indirect decompression of the posterior interosseous nerve." In some patients he divided a branch from the radial nerve that went through the brachioradialis to the lateral humeral epicondyle.

It was while treating patients with "failed tennis elbow surgery" that I realized that pain simulating failed tennis elbow surgery could result from a neuroma of the posterior cutaneous nerve of the forearm. A clinical example of this is given in Fig. 13.1. The patient, who was a basketball coach and avid tennis player, had four surgical attempts to help his lateral humeral epicondylar pain. His evaluation was complex, and included pain from compression of the radial nerve at the elbow from his "counterforce" brace, pain over the epicondyle itself, and pain in the surgery incisions with an area of dysesthesias in the posterior forearm. The approach to identify the cutaneous neuroma revealed branches from the posterior cutaneous nerve to the lateral humeral epicondyle. The treatment of the neuroma, which has

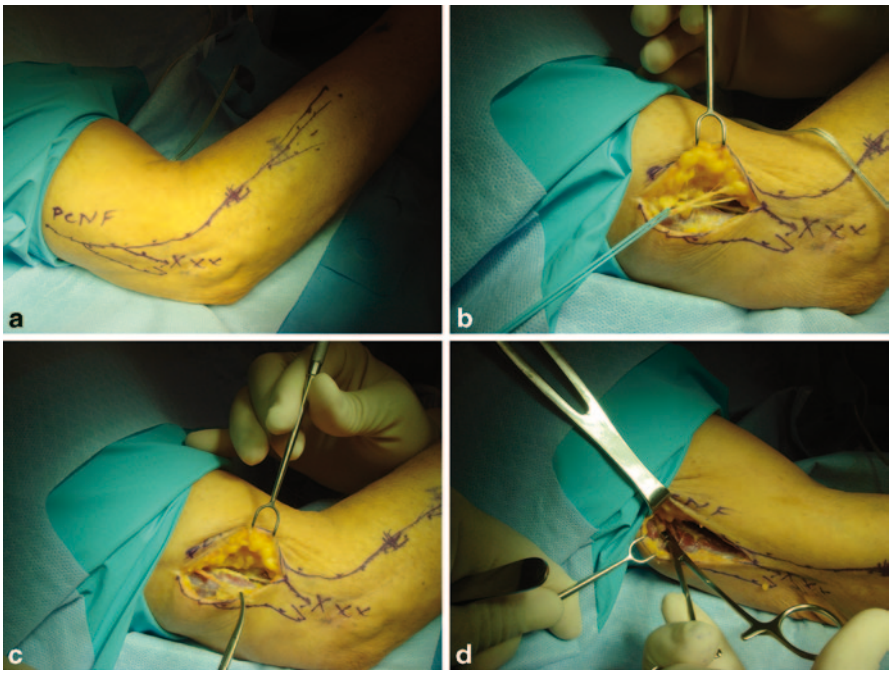

Fig. 13.1 Right forearm and elbow of man who had four previous surgical attempts to treat his "tennis elbow" pain. **a** Outline of the path of the posterior cutaneous nerve of the forearm with *asterisk* over the painful neuroma. The pathways distally into the area of dysesthesias are shown as is the origin and innervation of the branches to the epicondyle. **b** The branches to the dysesthetic skin are shown. **c** The branches to the lateral humeral epicondyle are shown. **d** The proximal end of the entire posterior femoral cutaneous nerve is implanted into the lateral head of the triceps muscle

been reported previously [25] is to resect the posterior cutaneous nerve and implant the proximal end into the lateral head of the triceps. In doing this, it became apparent that there were one or two branches that arose more proximally and innervated the periosteum. These branches were resected along with the main portion of the nerve, and the result was not only relief of the neuroma pain, but also relief of the lateral humeral epicondylar pain. These types of observations led to a series of cadaver dissections that demonstrated (Fig. 13.2) that the posterior cutaneous nerve to the forearm, after arising from the radial nerve at the spiral groove of the humerus, continues distally deep to the deep fascia, at which point it gives off one or two branches, between 3 and 5 cm proximal to the lateral humeral epicondyle, and that these branches continue distally, deep to the fascia, to innervate the periosteum of the lateral humeral epicondyle. The cutaneous portion of the posterior cutaneous nerve then becomes subcutaneous where it travels at this level distal to the elbow to innervate the skin of the posterior and lateral portions of the forearm. The group of patients that provided the insight into this problem had not only denervation, but also a neuroma resection and a neurolysis of the radial nerve, often both at the elbow and also in the forearm (Fig. 13.3).

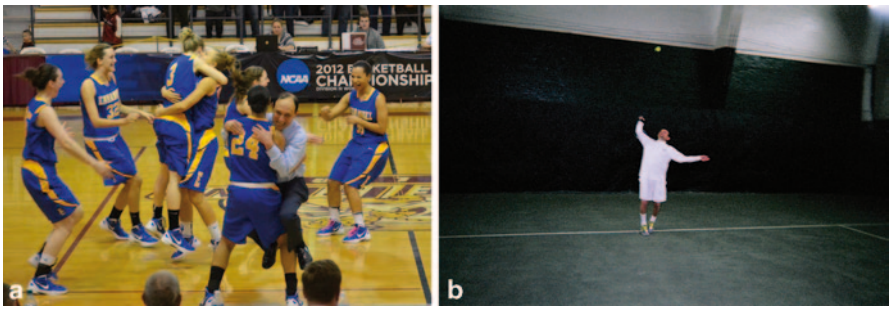

Fig. 13.2 Patient from Fig. 13.1 at a 1-year follow up. **a** Having resumed his coaching activities, he is with his team as they win the championship for their league. **b** Back playing tennis

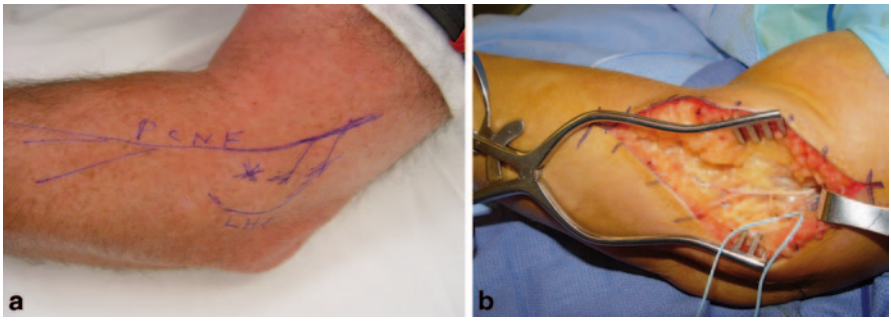

Fig. 13.3 In a cadaver, the anatomy of the posterior cutaneous branch of the radial nerve is outlined in **a** and demonstrated in **b**

The next phase of the investigation involved a collaborative study with the hand surgeons at the Southern Illinois College of Medicine, whose preferred surgical approach was to do a lateral epicondylectomy. In our retrospective study, we studied the patients that had only had a denervation as well as patients treated with epicondylectomy, compared to a group of patients that had both an epicondylectomy and a denervation. While one can argue that epicondylectomy effectively denervates the epicondyle, the proximal end of the nerve is left in close proximity to the bone resection and can regenerate into that scar. The results of that study [26] demonstrated that the denervation alone group, and denervation plus epicondylectomy group had significantly better pain relief, as measured with a visual analog scale (VAS; $p<0.001$), and a significantly shorter recovery time to return to work ($p<0.001$) than did the epicondylectomy alone group (Tables 13.1 and 13.2).

A prospective study was then performed with surgeons from Irvine, CA, with patients who had epicondylar symptoms that had persisted for more than 6 months despite nonoperative measures [27]. These patients underwent a simple denervation of the lateral epicondyle, as demonstrated in Figs. 13.3 and 13.4. Inclusion criteria included a successful preoperative nerve block with 1% xylocaine mixed 1:1 with 0.5% Marcaine. This was performed with the placement of approximately 3–5 cc at

Table 13.1 Demographics from study comparing denervation results to epicondylectomy. [26]

Group	Age	Gender	Number in group
Epicondylectomy	46.8 years	9 male, 8 female	17
Epicondylectomy and denervation	43.1 years	1 male, 6 female	7
Denervation	44.7 years	4 male, 2 female	6

Table 13.2 Results from study comparing return to work in treatment groups. [26]

Group	Average time to return to work	Statistical significance
Epicondylectomy	125 days	
Epicondylectomy and denervation	41 days	$p<0.001$
Denervation	28 days	$p<0.001$

the level of the fascia and more deeply about 3–4 cm proximal to the lateral humeral epicondyle. A visual analog scale preinjection level was compared with the pain 15 min after the injection, with a decrease in level of ≥ 5 being required to consider the block a success. Also, preinjection grip strength with the elbow extended and with the elbow flexed at 90° was compared with the same measurements 15 min after the block.

Denervation Technique

As the technique is done currently, the patient is positioned supine under either local or general anesthesia, and no pneumatic tourniquet is used. The incision site is 2–3 cm proximal to the lateral humeral epicondyle and is longitudinal, being about 4–5 cm in length depending upon the size of the arm (Figs. 13.4 and 13.5). The fat is gently dissected until one or sometimes two branches are identified, usually,

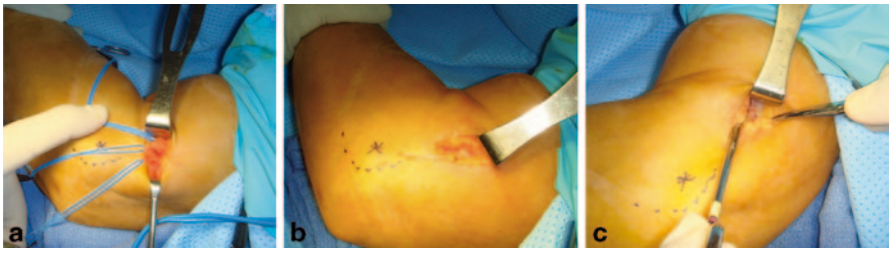

Fig. 13.4 Clinical intraoperative example of denervation of the left lateral humeral epicondyle. **a** Typical incision demonstrates the posterior cutaneous nerve to the forearm in the *top blue* vessel loop, as determined by gently pulling on it and observing the skin move, and two branches to the lateral epicondyle, demonstrated by pulling on the nerves and seeing the skin move directly over the perisoteum. **b** The two branches divided and lying on the skin. **c** These branches have been turned 180° and are implanted loosely into the lateral head of the triceps without a suture

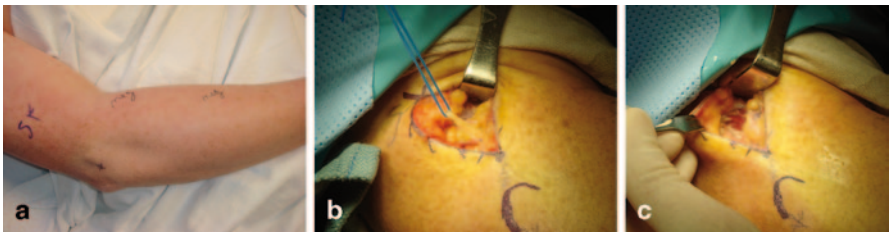

Fig. 13.5 Clinical intraoperative example of denervation of the right lateral humeral epicondyle. **a** Forearm demonstrates with *asterisk* the site of pain, and the "*negative*" indicates no clinical entrapment of the radial nerve at the elbow or forearm. **b** Typical incision demonstrates the posterior cutaneous nerve to the forearm branches to the lateral epicondyle, demonstrated by pulling on the nerves and seeing the skin move directly over the periosteum. **c** These branches have been divided and turned 180° and are implanted loosely into the lateral head of the triceps without a suture

but not always, above the deep fascia. A gentle pull on these nerves will cause the skin at the lateral humeral epicondyle to move. Sometimes, the posterior cutaneous nerve of the forearm is identified first, in which case it can be followed distally or proximally until these branches are identified. The posterior cutaneous nerve of the forearm is then injected with local anesthetic, usually 0.5% Marcaine without epinephrine. Then the branch(es) to the lateral epicondyle is/are cauterized distally, divided, a piece sent to pathology, and the proximal end turned and implanted deep to the fascia into the triceps muscle. The posterior cutaneous nerve itself can usually be preserved. Sometimes an intraneural dissection to obtain length on the divided nerves is necessary to bring them up for implantation into the triceps muscle (Fig. 13.4 and 13.5).

Denervation of the Medial Humeral Epicondyle

During anterior transposition of the ulnar nerve, it has always been the author's practice to excise the medial intermuscular septum, not simply to divide it. Wearing loupes during this surgery, a fascicle within the septum was often observed (Fig. 13.6). In order to understand this, 20 consecutive specimens were evaluated pathologically for the presence of neural tissue. Histology identified a nerve in 15 of the 20 specimens, and in the remaining 5, a nerve was identified with an S-100 stain. This demonstrated that there was a nerve present, and since there was never any clinical deficit associated with this "denervation," it was assumed this was a nerve to the medial humeral epicondyle.

The most common source of complaints of pain in the medial epicondyle were in patients referred to me as recurrent ulnar nerve entrapment. Some of these patients had pain around the elbow, which was not from the usual neuroma of the medial antebrachial cutaneous nerve. The pain could be localized to the juncture of where the medial intermuscular septum joined the medial humeral epicondyle, and it was the

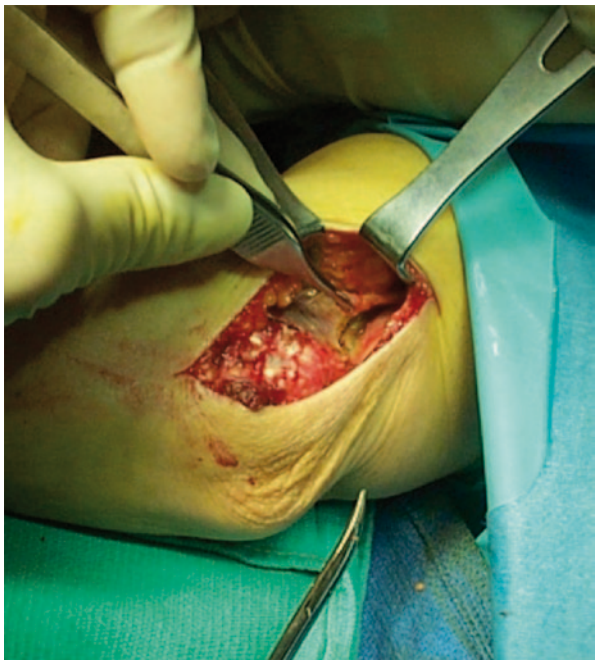

Fig. 13.6 Intraoperative view of resection of the medial intermuscular septum during ulnar nerve anterior transposition. The resected specimen usually contains the nerve to the medial humeral epicondyle, and this resection can be the cause of a painful neuroma

author's impression this represented a true neuroma of the nerve that was located within that septum (Fig. 13.7). At surgery, in addition to resecting a neuroma of the medial antebrachial cutaneous nerve, and doing the neurolysis of the ulnar nerve, the author's practice evolved to resect more proximally the medial intermuscular septum, and send it to the pathologist, where often a true neuroma was identified. This experience has been reported anecdotally [22].

The specific origin of this nerve was identified in a cadaver dissection study involving six fresh-frozen cadavers. The nerve was noted to originate in the axilla, most often as a branch of the radial nerve (Fig. 13.8), and travels either below the intermuscular septum or within it until it reaches the medial humeral epicondyle. There was one instance in which a contribution from the ulnar nerve occurred in the axilla [28].

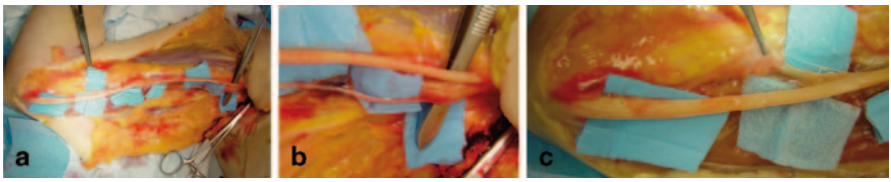

Fig. 13.7 Cadaver dissection of the nerve to the medial humeral epicondyle. **a** Overall, axilla to epicondyle view. **b** Close-up of the origin of the nerve from the radial nerve in the axilla. **c** Close-up of the distal end of the nerve, in the forceps, in relation to the epicondyle and the ulnar nerve (lying on the *blue* paper)

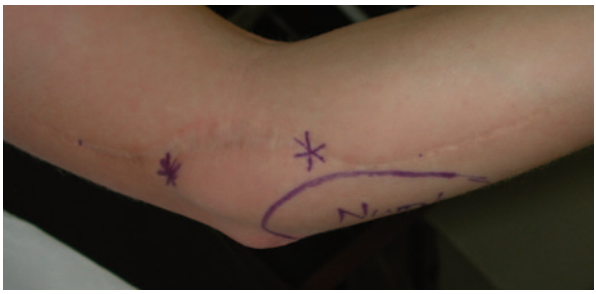

Fig. 13.8 A preoperative view of the medial *left* elbow, with hand to the *right*. The previous ulnar nerve transposition scar is clearly seen. The more *distal* of the two *asterisks* is the painful neuroma of the medial antebrachial cutaneous nerve, with distal radiation. The more *proximal asterisk* is the painful neuroma of the nerve to the medial humeral epicondyle

Technique of Medial Epicondylar Denervation

The technique is done under general anesthesia with the patient supine and without a pneumatic tourniquet (Figs. 13.8 and 13.9). A longitudinal incision is made about 2 cm proximal to the median humeral epicondyle, overlying the medial intermuscular septum. The ulnar nerve is identified and protected. Then, the medial intermuscular septum is carefully inspected to determine if the nerve to the medial humeral epicondyle is just medial to it, or beneath it. When identified, it is gently pulled upon and its connection to the medial humeral epicondyle confirmed. If it is found easily, it is blocked with a local anesthetic, such as 0.5% marcaine, and then cauterized distally, a section excised and submitted to pathology, and the proximal end turned

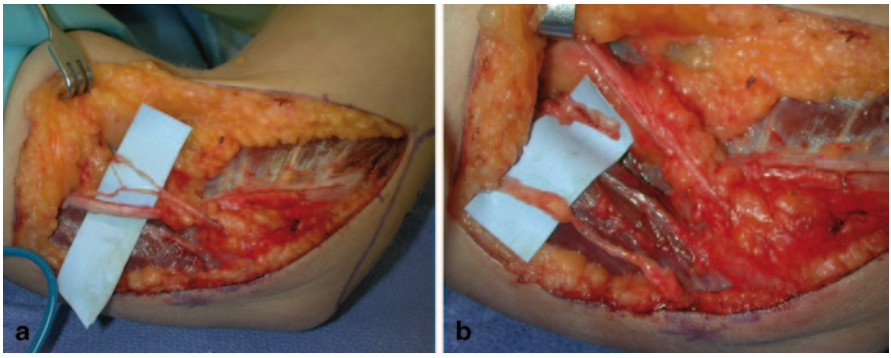

Fig. 13.9 Intraoperative view of a patient similar to the one in Fig. 13.8. **a** The ulnar nerve is noted transposed, and after its neurolysis. The medial antebrachial cutaneous nerve crosses the ulnar nerve to enter scar where it has formed a painful neuroma. **b** After resecting the neuroma of the medial antebrachial cutaneous nerve and implanting it into the medial head of the triceps, the medial brachial cutaneous nerve, posteriorly, was identified and preserved and is note intact on the *blue* paper. Just above this is the divided end of the medial intermuscular septum and the nerve to the medial epicondyle can be seen adherent to it. This nerve will be dissected proximally and implanted into a different tunnel in the medial head of the triceps to prevent recurrent painful neuroma formation

and implanted proximally into the medial head of the triceps. If it is not found in this manner, then a 1 cm section of the medial intermuscular septum is removed to determine if the nerve lies within the septum itself. Once found, it is treated as described above.

Results: Denervation of the Lateral Humeral Epicondyle

In the study by Rose et al., 29 painful tennis elbows in 26 patients were treated with denervation only. At 6 months postoperatively, the grip measurements were repeated and compared with the preoperative measurements (Table 13.3). These demonstrated significant functional improvement in grip strength ($p<0.001$) for both elbow positions. The mean VAS score decreased from 7.86 preoperatively to 1.91 postoperatively ($p<0.001$). Overall, of the 29 elbows, there were 19 excellent, 6 good, 1 fair, and 3 failures. Two of the three failures were "converted" to good or excellent results after neurolysis of the radial nerve at the elbow. The conclusion of this study was that lateral humeral denervation relieved pain and restored function in 86% of the patients [27].

Denervation of the Medial Humeral Epicondyle

This surgery is less common, as this problem presents rarely. A treatment example: an 18-year-old high school star baseball player presented with inability to pitch due to medial epicondylar pain. (Fig. 13.10) Radiographic imaging and orthopedic evaluation did not demonstrate any problem with the medial collateral ligament. The patient underwent a diagnostic block at the medial intermuscular septum just proximal to the epicondyle, staying above or within the septum so as not to block the ulnar nerve. He had no ulnar nerve symptoms and the ulnar nerve was not tender. Following the block, he could demonstrate his throwing motion without pain. At surgery, the nerve to the medial humeral epicondyle was identified, resected, and implanted into the medial head of the triceps (Fig. 13.11). Three weeks after surgery, he was throwing warm-up pitches again (Fig. 13.12) without pain.

These anecdotal findings indicate positive results from denervation, but further evidence is necessary. The next step would be a randomized controlled study of the effect of the block, done using anesthetic in one group and saline in the other group. So far we only have level 5 evidence.

Table 13.3 Results from study comparing return to work in treatment groups. [27]

Elbow position	Preoperative grip (lb.)	Postoperative grip (lb.)	*p* value
Flexion	45.9	62.8	$p<0.001$
Extension	26.0	53.3	$p<0.001$

Fig. 13.10 Medial epicondylitis in a high school baseball pitcher. With his throwing motion being demonstrated with the *right* arm, the *left* index finger points to the site of pain that occurs at this location during the pitch

Rehabilitation after Lateral and Medial Epicondylar Denervation

In contrast to surgery that releases the wrist/finger extensors or flexors, or resects the lateral or medial humeral epicondyle, rehabilitation after denervation is simply a matter of permitting wound healing to take place. Table 13.4 contrasts the postoperative recovery between these two approaches. The critical concepts here are that no tendons or ligaments or major structures are cut during denervation. Wound healing involves only the incision and subcutaneous tissues. Patients know they are better in the first few days after surgery. Often, narcotics are not needed for more than a week. Anti-inflammatory medication is usually sufficient during and after the second postoperative week. Since the nerve is implanted into a muscle proximal to the elbow, full finger, hand, wrist, and elbow range of motion is permitted the day after surgery. The sutures are removed on the 14th day and the patient may then begin to carry a tennis racquet, or a baseball or bat or golf club, as the case may be, in their hand. They can be guided going forward, based upon how long they had been deconditioned prior to the surgery. They should begin hitting the ball with a coach who hits to the same spot for them, to get their form back, before playing competitively again.

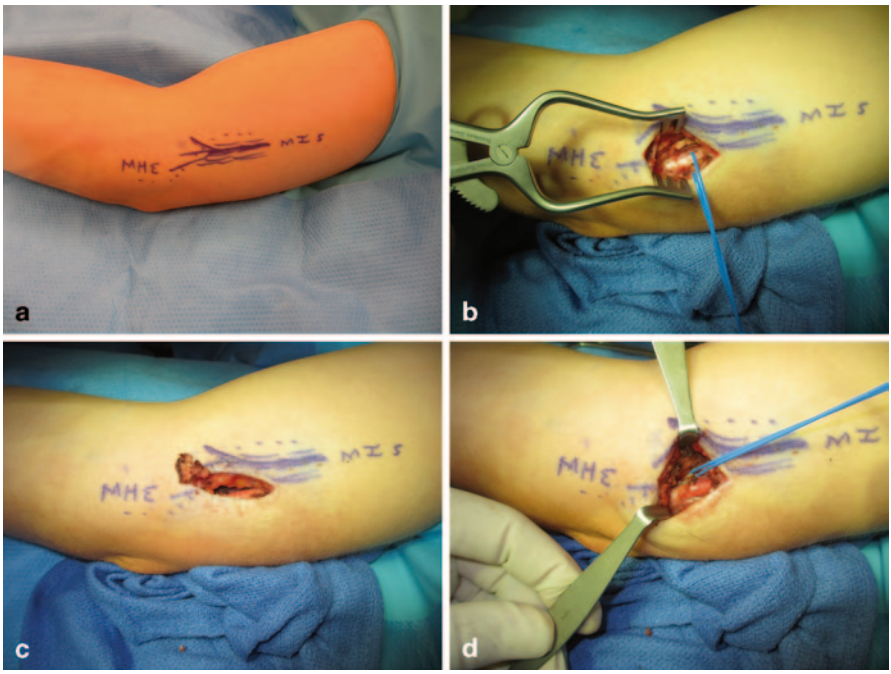

Fig. 13.11 Intraoperative views of the surgery for the person in Fig. 13.10. **a** The *right* arm is noted without a tourniquet. The nerve is drawn overlying or within the medial intermuscular septum (*MIS*) going towards the medial humeral epicondyle (*MHE*). **b** The vessel loop is around the ulnar nerve. The nerve was not identified superficial or within the MIS in this patient. **c** The segment of the MIS adjacent to the MHE has been excised, and is noted on the skin. **d** Just deep to the MIS, the nerve to the MHE was identified and is within the *blue* vessel loop. It was then excised and the proximal end implanted into the medial head of the triceps

Table 13.4 Rehabilitation regimens compared: tendon surgery versus denervation, for lateral humeral epicondylitis [27]

Tendon release surgery	Denervation
Long arm splint for 4 weeks	No splint
Activity restriction for 3 months	Immediate range of motion
Resume tennis or golf at 6 months	Immediate return: activities of daily living
	Resume tennis or golf at 4 weeks

Conclusions

The anatomy for the innervation of the humeral epicondyles has been documented, and surgical approaches developed to permit their denervation. The evidence presented in this chapter makes denervation of the humeral epicondyles available for the treatment of recalcitrant or recurrent humeral epicondylitis in patients who have failed other forms of therapy, who do not wish to go through a long rehabilitation/recovery time, and who have responded to a local anesthetic block with

Fig. 13.12 At the third week after surgery, the pitcher from Figs. 13.10 and 13.11 is shown throwing the ball again. Views of the pitch: front (**a**) and side (**b**)

demonstrated relief of pain and improved function. Most likely, the future will see denervation included with other arms of a prospective randomized study to document effectiveness in a level I study.

References

1. Haker E, Lundeberg T. Is low-energy laser treatment effective in lateral epicondylalgia? J Pain Symptom Manag. 1991;6(4):241–6.
2. Hammer DS, Rupp S, Ensslin S, Kohn D, Seil R. Extracorporeal shock wave therapy in the treatment of tennis elbow and heel pain. Acta Orthop Trauma Surg. 2000;120:104–7.
3. Mishra A, Pavelko T. Treatment of chronic elbow tendinosis with buffered platelet-rich plasma. Am J Sports Med. 2006;34(11):1774–8.
4. Speed CA, Nichols D, Richards C, Humphreys H, Wies JT, Burnet S, Hazleman BL. Extracorporeal shock wave therapy for lateral epicondylitis—a double blind randomised controlled trial. J Orthop Res. 2002;20(5):895–8.
5. Stasinopoulos DI, Johnson MI. Effectiveness of low-level laser therapy for lateral elbow tendinopathy. Photomed Laser Surg. 2005;23(4):425–30.
6. Oken O, Kahraman Y, Ayhan F, Canpolat S, Yorgancioglu ZR, Oken OF. The short-term efficacy of laser, brace, and ultrasound treatment in lateral epicondylitis: a prospective, randomized, controlled trial. J Hand Ther. 2008;21(1):63–7.
7. Wolf JM, Ozer K, Scott F, Gordon MJV, Williams AE. Comparison of autologous blood, corticosteroid, and saline injection in the treatment of lateral epicondylitis: a prospective, randomized, controlled multicenter study. J Hand Surg. 2011;36A:1269–72.

8. Travernier L, Truchet P. La section des branches articulares du nerf Obturator dans le traitement de l'arthritite Chronique de la hanche. Rev Orthop. 1942;28:62–8.
9. Wilhelm A. Zur Innervation der Gelenke der Oberen Extremitaet. Z Anat Entwicklungsgesch.1958;120:331–71.
10. Wilhelm A. Die Gelendendervation und ihre anatomicshecn Grundlagen. Ein neues Behandleungsprinzip in der Handchirurgie. Hefte Unfallheikd. 1966;86:1–109.
11. Buck-Gramcko D. Denervation of the wrist joint. J Hand Surg. 1977;2A:54–61.
12. Dellon AL, Seif SS. Neuroma of the posterior interosseous nerve simulating a recurrent ganglion: case report and anatomical dissection relating the posterior interosseous nerve to the carpus and etiology of dorsal ganglion pain. J Hand Surg. 1978;3:326–32.
13. Dellon AL, Mackinnon SE, Daneshvar A. Terminal branch of anterior interosseous nerve as source of wrist pain. J Hand Surg. 1984;19B:316–22.
14. Dellon AL. Partial dorsal wrist denervation: resection of distal posterior interosseous nerve. J Hand Surg. 1985;10A:527–33.
15. Horner G, Dellon AL. Innervation of the human knee joint and implications for surgery. Clin Orthop Rel Res. 1994;301:221–6.
16. Dellon AL, Mont MA, Hungerford DS. Partial denervation for treatment of persistent neuroma pain after total knee arthroplasty. Clin Orthop Rel Res. 1995;316:145–50.
17. Dellon AL, Mont M, Mullik T, Hungerford D. Partial denervation for persistent neuroma pain around the knee. Clin Orthop Rel Res. 1996;329:216–22.
18. Aszmann OC, Dellon AL, Birely B, McFarland E. Innervation of the human shoulder joint and its implications for surgery. Clin Orthop Rel Res. 1996;330:202–7.
19. Dellon AL. Anterior shoulder denervation. Clin Exper Plast Surg. 2004;36:175–80.
20. Rab M, Ebmer J, Dellon AL. Innervation of the sinus tarsi: implications for treating anterolateral ankle pain. Ann Plast Surg. 2001;47:500–4.
21. Dellon AL. Denervation of the sinus tarsi for chronic post-traumatic lateral ankle pain. Orthopedics 2002;25:849–51.
22. Dellon AL. Partial joint denervation I: wrist, shoulder, elbow. Plast Reconstr Surg. 2009; 123:197–207.
23. Dellon AL. Partial joint denervation II: knee, ankle. Plastic Reconstr Surg. 2009;123:208–17.
24. Wilhelm A. Tennis elbow: treatment of resistant cases by denervation. J Bone Joint Surg. 1996;21B:523–33.
25. Dellon AL, Kim J, Ducic I. Painful neuroma of the posterior cutaneous nerve of the forearm after surgery for lateral humeral epicondylitis. J Hand Surg Am. 2004;29(3):387–90.
26. Berry N, Neumeister MW, Russell RC, Dellon AL. Epicondylectomy versus denervation for lateral humeral epicondylitis. Hand. 2011 Jun;6(2):174–8.
27. Rose NE, Forman SK, Dellon AL. Denervation of the lateral humeral epicondyle for treatment of chronic lateral epicondylitis. J Hand Surg Am. 2013;38(2):344–9.
28. Dellon AL, Ducic I, Dejesus RA. The innervation of the medial humeral epicondyle: implications for medial epicondylar pain. J Hand Surg Br. 2006;31(3):331–3.

Chapter 14
Outcomes of Treatment and Return to Play: The Evidence

Brian A. Tinsley and Augustus D. Mazzocca

The treatment goal of lateral epicondylitis is to reduce pain, thereby allowing the patient to return to full activities [1]. Treatment modalities and duration of rest must balance between patients' rapid return to activity with avoiding treatment side effects and symptom recurrence. There is significant debate regarding the optimal treatment algorithm and which treatment modalities are the most effective. Additionally, there is limited evidence for how soon a patient can return to full activities while avoiding prolonged or recurrent symptoms.

Conservative Management Outcomes

Lateral epicondylitis has been attributed to repetitive microtrauma causing a degenerative tendinosis rather than simply an inflammatory reaction [2]. Although it is typically self-limited, cases of severe or persistent symptoms can be difficult to treat. Since recovery rates of 80 % to over 90 % have been reported with conservative treatment [3, 4], the mainstay of initial management includes rest, activity modification, and nonsteroidal anti-inflammatory medications (NSAIDS) [4–6]. The use of bracing, splinting, physiotherapy, and other conservative modalities has been studied, but no method has proven clearly superior [7–11].

Currently, the literature lacks high quality, large, randomized trials that have consistently shown benefit of injection therapies for management of lateral epicondylitis. While injection therapies have been shown to be safe with few adverse events,

A. D. Mazzocca (✉) · B. A. Tinsley
Department of Orthopaedic Surgery, University of Connecticut Health Center, 263 Farmington Ave, MARB 4th Floor, Farmington, CT 06033, USA
e-mail: mazzocca@uchc.edu

B. A. Tinsley
e-mail: brianatinsley@gmail.com

J. M. Wolf (ed.), *Tennis Elbow,* DOI 10.1007/978-1-4899-7534-8_14

there is mixed evidence demonstrating efficacy over other conservative modalities [10, 11, 13–16].

There are no major studies that evaluate return to activity and the effect of early return on recurrence or persistence of symptoms in conservatively treated patients. Typically with conservative management, patients are instructed to avoid exacerbating activities initially and then resume activities as their symptoms allow. Patient education is an important part of initial management. Based on the literature, 30–50 % of patients will have significant improvement in the 3-week to 6-month period after presentation, and over 90 % of patients may have significant improvement at 1 year regardless of treatment [4, 8, 9, 12].

Surgical Outcomes

In patients with persistent lateral epicondylitis that have failed conservative treatment, surgery may be an option. Since approximately 90 % of patients will improve after one year of conservative treatment, it follows that few will need to proceed to operative intervention. In patients that desire surgery, various techniques for open, percutaneous, and arthroscopic procedures have been described.

The long term outcomes following open surgical treatment have been favorable in patients with lateral epicondylitis who fail conservative management. A retrospective study by Dunn et al [17] reported that 97 % of patients had improvement at the 10–14 year follow-up on 92 cases treated with open extensor origin release, debridement, and repair with lateral epicondyle drilling. These patients were conservatively treated for 2 months to 10 years with a mean of 2.2 years prior to surgery. The postoperative course allowed light activities at 3–5 days, light strengthening at 3 weeks, and gradual return to full activity or sports at 5–6 weeks. At final follow-up, 84 % of patients had little or no pain and 93 % were able to return to their sport after surgery.

Another long term case series retrospectively evaluated 137 patients at a mean of 9.8 years after treatment by a single surgeon with open release, debridement, repair, and lateral epicondyle decortication [18]. Patients had symptoms for 10–60 months and failed conservative treatment prior to surgery. After surgery, they were immobilized in a collar and cuff sling for 10 days followed by gentle range of motion exercises. They were allowed to return to work after 6 weeks and return to sport after 12 weeks. Early results showed that 85.4 % were pain free by 12 weeks. At a mean of 9.8 years, 94.6 % reported good to excellent results with similar outcomes reflected in the Hospital for Special Surgery score and the Mayo elbow performance score.

Reports of outcomes after arthroscopic extensor carpi radialis brevis release have described similar results compared to open surgical release. Several studies with a mean follow-up of 1.8–3.5 years showed improvement in 85–95 % of patients at the final visit [19–21]. Patients returned to work at an average of 2.2–4.8 weeks postoperatively. Long term follow-up after arthroscopic treatment of 30 patients evaluated after 106–130 months found 97 % of patients were “much better” or “better” after

surgery and 87% said they were satisfied [22]. None of those patients had a repeat injection or additional surgery after the initial procedure.

Studies evaluating percutaneous release of the common extensor origin have reported favorable results in patients with recalcitrant lateral epicondylitis. In one study, 26 patients who were symptomatic for a mean of 8.9 months underwent percutaneous release. At 2 months post-procedure, 24 patients had an excellent result, 1 had a good result, and 1 had a poor result [23]. Grundberg and Dobson had similar results reporting excellent or good outcomes in 29 of 32 elbows after percutaneous release [24]. Dunkow et al. [25] performed a prospective randomized trial comparing open and percutaneous releases for lateral epicondylitis. The authors included 45 patients (47 elbows) who failed 12 months of conservative management prior to surgical treatment. At 1 year, the percutaneous group had more patients with an excellent result and fewer unsatisfied patients compared to the open group. Patients who received percutaneous release went back to work significantly faster than patients who underwent open release (2 weeks versus 5 weeks, respectively). Both groups had significant improvement in the Disability of the Arm, Shoulder, and Hand (DASH) scores. The improvement in the DASH score was significantly better in the percutaneous group; however, this may not represent a clinically meaningful difference.

Szabo et al. compared percutaneous, open, and arthroscopic release in 102 patients with recalcitrant lateral epicondylitis after 3–60 months (mean 13.2 months) of conservative treatment [26]. They reported that all groups improved postoperatively and found no differences in postoperative measures of pain, function, and range of motion at 24–108 months of follow-up.

Although the literature reports good outcomes following surgery, the nonoperative course prior to inclusion in these studies is variable. In one study, the patients had 3 months of conservative treatment, while another study included patients with 3–144 months of nonoperative care. The data have shown that many patients will significantly improve after 12 months of conservative treatment, so including patients with only 3 months of symptoms prior to surgery makes the outcome data difficult to interpret. It is unclear how many of these patients would have resolved without operative intervention. In spite of these limitations, the currently available literature, primarily based on retrospective studies, supports operative intervention after failure of exhaustive conservative treatment.

In patients who undergo operative treatment, postoperative return to activity is more gradual compared with conservatively managed patients. Postoperative protocols vary depending on the surgeon and procedure; however, most authors describe an initial period of immobilization followed by range of motion exercises. A gradual return to sport is allowed beginning at 6–12 weeks [17–19, 26]. Studies have not demonstrated a difference in postoperative outcome based on the length of rest prior to return to full activities.

Physiotherapy and Return to Sport

The literature is inconclusive regarding which physiotherapy programs are the most effective in treating lateral epicondylitis and allowing more rapid return to play [26]. In treating the general population, physiotherapy has not proven to be clearly superior to the wait and see approach [27, 28]. In the athlete, however, sport specific rehabilitation protocols have been suggested in order to hasten recovery and reduce the risk of recurrence.

Strength and range of motion differences are common in the dominant versus nondominant arm of elite baseball and tennis players [29, 30]. These muscular adaptations may lead to increased stress in the elbow, forearm, and wrist. In the setting of lateral epicondylitis, improper biomechanics or proximal muscle imbalance may lead to the development or progression of elbow pathology [31, 32]. Therefore, postinjury rehabilitation focusing on developing appropriate endurance and improving sport specific biomechanics may lead to reduced recurrence [31].

While there is limited evidence comparing rehabilitation protocols, several authors suggest a symptom based multiphase program with emphasis on maintaining range of motion followed by progressive strengthening [29, 32]. The first phase includes range of motion exercises which are performed until the patient is able to complete them without significant discomfort (Fig. 14.1a–d). Once this is accomplished, a patient can begin a progressive strengthening program.

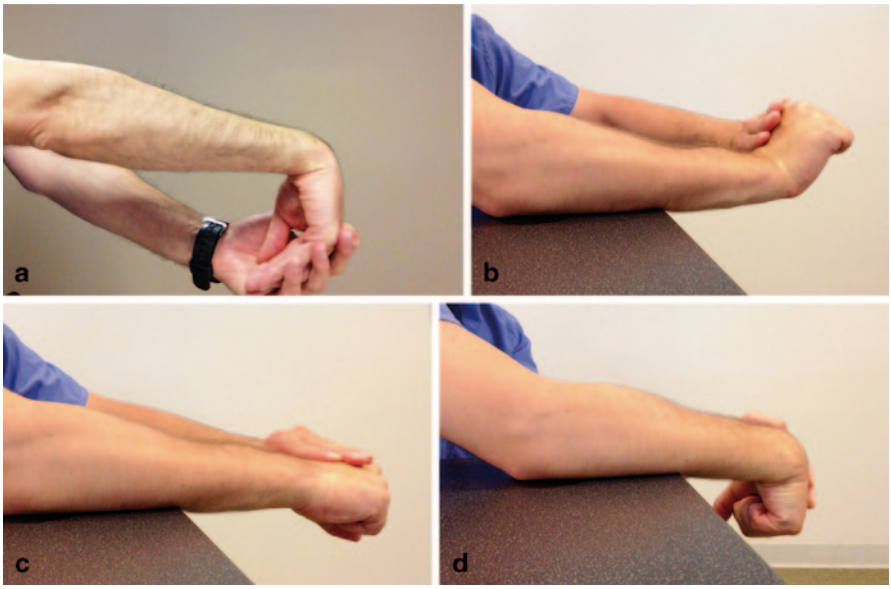

Fig. 14.1 Many physical therapy programs treating lateral epicondylitis include static stretching of the extensor mechanism (**a**) as well as eccentric stretching (**b**–**d**). Eccentric stretching involves contracting the extensor mechanism against resistance as the muscle is lengthened by moving from extension to flexion

Croisier et al. compared age, sex, and activity matched groups who underwent either passive rehabilitation or an active eccentric strengthening program. The passive rehabilitation protocol included use of analgesics, ice, ultrasound, deep friction massage, and stretching. The eccentric program included a progressive eccentric strengthening regimen [33]. They found that the eccentric group had a more rapid reduction in pain, significant improvement in strength and less disability compared to the passive group at 9 weeks postinitiation. Another study comparing stretching only, an eccentric strengthening program, and a concentric strengthening program demonstrated no difference between the groups at 6 weeks; however, a recent systematic review concluded that there is moderate evidence supporting the use of eccentric exercise [28, 34].

Ellenbecker et al. propose additional activity specific strengthening that focuses on the scapula and rotator cuff in addition to the forearm and wrist [32]. In addition to strengthening, proper mechanics should be addressed to reduce stress on the arm and possibly reduce recurrence. Once patients are pain-free and have the affected extremity strength equal to the contralateral side, they may return to sport. A gradual increase in play is recommended until the athlete can resume their preinjury activity level without discomfort.

Conclusions

There is limited evidence for when a patient can return to full activity after conservative or operative treatment. Additionally, there is no evidence examining the effect of early return to activities on the severity or duration of symptoms. The evidence for treatment outcomes favors conservative management.

The authors' preferred treatment begins with a discussion with the patient about the often self-limited nature of the disease and the treatment options. After ruling out other pathology, we typically recommend that patients take a common sense approach to initially managing their symptoms. This includes self-limiting their activities and taking oral NSAIDs as needed. Patients are instructed to return to full activities as they are able. If the pain is severe enough to eliminate a patient from an important event or competition, we would consider 1–2 corticosteroid injections for short term pain relief.

Patients with persistent symptoms after conservative management may undergo surgical treatment. The decision to proceed to surgery is based on symptom persistence despite extensive conservative care, symptom severity, and patient preference. Our approach emphasizes educating patients about the relapsing and remitting nature of their symptoms as well as the possibility for a long duration of symptoms. A thorough discussion about other treatment options including risks, benefits, and the limitations of our evidence is important in order to arrive at a treatment plan that suits each individual.

References

1. Nirschl RP, Pettrone FA. Tennis elbow. The surgical treatment of lateral epicondylitis. J Bone Joint Surg Am. 1979;61(6A):832–9.
2. Kraushaar BS, Nirschl RP. Tendinosis of the elbow (tennis elbow). Clinical features and findings of histological, immunohistochemical, and electron microscopy studies. J Bone Joint Surg Am. 1999;81(2):259–78.
3. Labelle H, Guibert R, Joncas J, Newman N, Fallaha M, Rivard CH. Lack of scientific evidence for the treatment of lateral epicondylitis of the elbow. An attempted meta-analysis. J Bone Joint Surg Br. 1992;74(5):646–51.
4. Smidt N, van der Windt DA, Assendelft WJ, Deville WL, Korthals-de Bos IB, Bouter LM. Corticosteroid injections, physiotherapy, or a wait-and-see policy for lateral epicondylitis: a randomised controlled trial. Lancet 2002;359(9307):657–62.
5. Leach RE, Miller JK. Lateral and medial epicondylitis of the elbow. Clin Sports Med. 1987;6(2):259–72.
6. Vangsness CT Jr, Jobe FW. Surgical treatment of medial epicondylitis. Results in 35 elbows. J Bone Joint Surg Br. 1991;73(3):409–11.
7. Barr S, Cerisola FL, Blanchard V. Effectiveness of corticosteroid injections compared with physiotherapeutic interventions for lateral epicondylitis: a systematic review. Physiotherapy 2009;95(4):251–65.
8. Bisset L, Beller E, Jull G, Brooks P, Darnell R, Vicenzino B. Mobilisation with movement and exercise, corticosteroid injection, or wait and see for tennis elbow: randomised trial. BMJ 2006;333(7575):939.
9. Garg R, Adamson GJ, Dawson PA, Shankwiler JA, Pink MM. A prospective randomized study comparing a forearm strap brace versus a wrist splint for the treatment of lateral epicondylitis. J Shoulder Elbow Surg. 2010;19(4):508–12.
10. Krogh TP, Bartels EM, Ellingsen T, Stengaard-Pedersen K, Buchbinder R, Fredberg U, Bliddal H, Christensen R. Comparative effectiveness of injection therapies in lateral epicondylitis: a systematic review and network meta-analysis of randomized controlled trials. Am J Sports Med. 2013;41(6):1435–46.
11. Wolf JM, Ozer K, Scott F, Gordon MJ, Williams AE. Comparison of autologous blood, corticosteroid, and saline injection in the treatment of lateral epicondylitis: a prospective, randomized, controlled multicenter study. J Hand Surg Am. 2011;36(8):1269–72.
12. Hay EM, Paterson SM, Lewis M, Hosie G, Croft P. Pragmatic randomised controlled trial of local corticosteroid injection and naproxen for treatment of lateral epicondylitis of elbow in primary care. BMJ 1999;319(7215):964–8.
13. Edwards SG, Calandruccio JH. Autologous blood injections for refractory lateral epicondylitis. J Hand Surg. 2003;28(2):272–8.
14. Hayton MJ, Santini AJ, Hughes PJ, Frostick SP, Trail IA, Stanley JK. Botulinum toxin injection in the treatment of tennis elbow. A double-blind, randomized, controlled, pilot study. J Bone Joint Surg Am. 2005;87(3):503–7.
15. Wong SM, Hui AC, Tong PY, Poon DW, Yu E, Wong LK. Treatment of lateral epicondylitis with botulinum toxin: a randomized, double-blind, placebo-controlled trial. Ann Intern Med. 2005;143(11):793–7.
16. Judson CH, Wolf JM. Lateral epicondylitis: review of injection therapies. The Orthop Clin North Am. 2013;44(4):615–23.
17. Dunn JH, Kim JJ, Davis L, Nirschl RP. Ten- to 14-year follow-up of the Nirschl surgical technique for lateral epicondylitis. Am J Sports Med. 2008;36(2):261–6.
18. Coleman B, Quinlan JF, Matheson JA. Surgical treatment for lateral epicondylitis: a long-term follow-up of results. J Shoulder Elbow Surg. 2010;19(3):363–7.
19. Jerosch J, Schunck J. Arthroscopic treatment of lateral epicondylitis: indication, technique and early results. Knee Surg Sports Traumatol Arthrosc. 2006;14(4):379–82.

20. Lattermann C, Romeo AA, Anbari A, Meininger AK, McCarty LP, Cole BJ, Cohen MS. Arthroscopic debridement of the extensor carpi radialis brevis for recalcitrant lateral epicondylitis. J Shoulder Elbow Surg. 2010;19(5):651–6.
21. Baker CL, Jr., Murphy KP, Gottlob CA, Curd DT. Arthroscopic classification and treatment of lateral epicondylitis: two-year clinical results. J Shoulder Elbow Surg. 2000;9(6):475–82.
22. Baker CL, Jr, Baker CL, 3rd. Long-term follow-up of arthroscopic treatment of lateral epicondylitis. Am J Sports Med. 2008;36(2):254–60.
23. Kaleli T, Ozturk C, Temiz A, Tirelioglu O. Surgical treatment of tennis elbow: percutaneous release of the common extensor origin. Acta Orthop Belg. 2004;70(2):131–3.
24. Grundberg AB, Dobson JF. Percutaneous release of the common extensor origin for tennis elbow. Clin Orthop Relat Res. 2000;(376):137–40.
25. Dunkow PD, Jatti M, Muddu BN. A comparison of open and percutaneous techniques in the surgical treatment of tennis elbow. J Bone Joint Surg Br. 2004;86(5):701–4.
26. Szabo SJ, Savoie FH 3rd, Field LD, Ramsey JR, Hosemann CD. Tendinosis of the extensor carpi radialis brevis: an evaluation of three methods of operative treatment. J Shoulder Elbow Surg. 2006;15(6):721–7.
27. Smidt N, Assendelf JJ, Arola H, Malmivaara A, Green S, Buchbinder R, van der Windt D, Bouter LM. Effectiveness of physiotherapy for lateral epicondylitis: a systematic review. Ann Med. 2003;35:51–62.
28. Martinez-Silvestrini JA, Newcomer KL, Gay RE, Schaefer MP, Kortebein P, Arendt KW. Chronic lateral epicondylitis: comparative effectiveness of a home exercise program including stretching alone versus stretching supplemented with eccentric or concentric strengthening. J Hand Ther. 2005;18:411–9.
29. Wilk KE, Arrigo CA, Andrews JR. Rehabilitation of the elbow in the throwing athlete. J Orthop Sports Phys Ther. 1993;17:305–17.
30. Ellenbecker TS, Roetert EP, Riewald S. Isokinetic profile of wrist and forearm strength in elite female junior tennis players. Br J Sports Med. 2006;40:411–4.
31. Ellenbecker TS. Rehabilitation of shoulder and elbow injuries in tennis players. Clin Sports Med. 1995;12(1):87–110.
32. Ellenbecker TS, Nirschl R, Renstrom P. Current concepts in examination and treatment of elbow tendon injury. Sports Health. 2013;5:186–94.
33. Croisier JL et al. An isokinetic eccentric programme for the management of chronic lateral epicondylartendinopathy. Br J Sports Med. 2007;41:269–75.
34. Raman J, MacDermid JC, Grewa R. Effectiveness of different methods of resistance exercise in lateral epicondylosis—a systematic review. J Hand Ther. 2012;25:5–26.

Chapter 15
Tennis Elbow: Complications of Surgical Treatment and Salvage Procedures for Failed Surgery

Jay V. Kalawadia and David M. Kalainov

Abbreviations

ECRB	Extensor carpi radialis brevis
OCD	Osteochondritis dissecans
PIN	Posterior interosseus nerve

Introduction

Lateral epicondylitis (i.e., tennis elbow) is the most common complaint of the elbow seen in adults, affecting approximately 3% of people over the age of 40 [1–8]. While different etiologies can contribute to lateral elbow pain, the diagnosis of lateral epicondylitis denotes pathology within the extensor carpi radialis brevis (ECRB) tendon origin and, in approximately one-third of cases, the anterior margin of the extensor digitorum communis tendon origin as well [9–14]. Histological features reveal predominantly hyaline degeneration and neovascularization, compatible with an aborted effort at healing rather than an inflammatory response [1, 6, 15]. Whereas nonoperative modalities remain the mainstay of treatment, with advances in technology and surgical techniques, the number of surgical interventions is increasing [1]. Unfortunately, and common to all surgical procedures, there are associated complications and modes of failure.

D. M. Kalainov (✉)
Department of Orthopaedic Surgery, Northwestern University,
737 N. Michigan Avenue, Suite 700, Chicago, IL 60611, USA
e-mail: dkalaino@nmff.org

J. V. Kalawadia
Department of Orthopaedic Surgery, Northwestern University,
676 N. Saint Clair, Suite 1350, Chicago, IL 60611, USA
e-mail: Kalawadia@gmail.com

J. M. Wolf (ed.), *Tennis Elbow,* DOI 10.1007/978-1-4899-7534-8_15

Nonoperative Treatment Options

Nonoperative management of tennis elbow is successful in approximately 80–90 % of cases [1, 2, 4, 10, 13, 16–18]. Recognized treatment measures include activity modifications, counterforce strapping, wrist splinting, local steroid injections, and use of topical and oral antiinflammatory medications. Assistance from a physical or occupational therapist for stretching, strengthening, massage, iontophoresis, and phonophoresis may be beneficial in some patients. Injection of botulinum toxin A into the extensor tendon origin has shown promise; however, the administration of this product for tennis elbow is considered an off-label use [19–21]. More recently, the clinical efficacy of platelet-rich plasma injections has been studied but with mixed results [22].

Surgical Treatment Options

Among patients who seek medical treatment for lateral epicondylitis, only 4–11 % will eventually undergo surgery [4, 10, 13, 23]. The operation is directed at removing pathological tissue from the common extensor tendon origin, alone or in conjunction with a partial lateral epicondylectomy, and can be performed in an open or arthroscopic manner [4, 10, 13, 23]. Open debridement procedures may include reattachment of the extensor tendon origin to bone and coverage of a tendinous defect with an anconeus muscle rotational flap [4, 10, 13, 23–25]. Percutaneous release of the damaged tendon(s) has been proposed as a less invasive operative technique. An alternative surgical approach for lateral epicondylitis involves denervation of the lateral epicondyle in place of tendon debridement [24, 26–28] (Fig. 15.1).

Debridement procedures are expected to lead to a satisfactory result in up to 90 % of patients over time [1, 10, 11, 13, 23, 29–36]. In a prospective cohort study of 63 patients undergoing open tennis elbow release surgery, Verhaar et al. [37] found

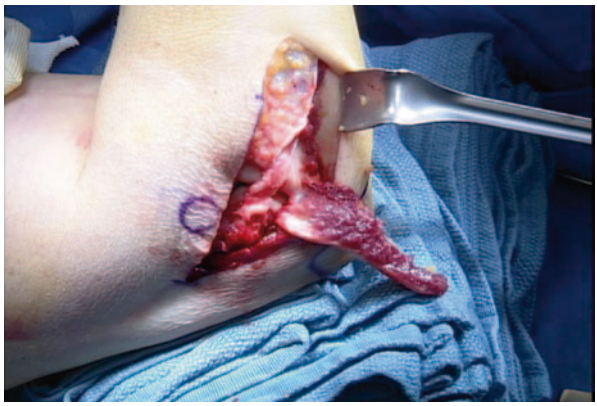

Fig. 15.1 Photograph of a right elbow showing an exposed radiocapitellar joint and a mobilized anconeus muscle flap. The muscle flap is used to cover the soft tissue defect and joint

residual pain over the lateral epicondylar region in 40% of patients at 6 weeks, 24% at 1 year, and 9% at 5 years. Coleman et al. [38] studied 171 elbows at 10 years after open tennis elbow surgery and found good to excellent outcomes in approximately 94% of patients. Using arthroscopic methods to debride the ECRB tendon origin, several authors have reported symptomatic improvement in 93–100% of patients after 2 years [4, 36, 39–41].

There are few studies comparing the various surgical approaches for tennis elbow. Szabo et al. [42] studied arthroscopic, percutaneous, and open techniques and, after 2 years, they found no statistical differences in failure rates between the treatment groups. Conversely, Solheim et al. [43] followed 305 elbows over 3 years and found that arthroscopic release of the ECRB tendon origin resulted in a significantly greater improvement in QuickDASH scores when compared to open release surgery, but with no difference in complications.

Modes of Failure

Morrey proposed three categories of failure following primary surgical treatment of lateral epicondylitis [1]. A growing body of literature supports a fourth category, which includes misguided rehabilitation, patient noncompliance, workers' compensation, and psychological disorders. This fourth category was originally included as a subtype in Morrey's Type 1 failure group.

Type 1 Failure—Inaccurate or Concomitant Diagnosis

Type 1 failure occurs when an inaccurate initial diagnosis is made or a concomitant diagnosis contributing to symptomatology persists [1]. As the underlying source of pain is not sufficiently addressed at the time of surgery, the patient will report lingering elbow pain that is comparable to discomfort experienced before surgery [1, 30]. Causes for Type 1 failure include nerve irritability, a synovial plica, osteoarthritis, osteochondritis dissecans (OCD) of the capitellum, a snapping triceps tendon, lateral ligament insufficiency, an anconeus compartment syndrome, and an osteoid osteoma of the capitellum (Fig. 15.2).

Radial tunnel syndrome, signifying irritability of the posterior interosseus nerve (PIN) in the proximal forearm without a clearly identified impinging structure, is the most common cause of Type 1 failure [1, 9, 13, 30, 39, 40, 44–49]. Werner et al. [46] found that 13% of failures after tennis elbow surgery were due to this condition. Lateral elbow pain from entrapment of the PIN by a ganglion cyst has also been reported [50]. Other potential neurological causes of lateral elbow pain include cervical radiculopathy, brachial plexopathy, and entrapment of the lateral antebrachial cutaneous nerve or the posterior brachial cutaneous nerve [27, 51].

A synovial plica represents a focal thickening of synovial tissue [1, 30, 48, 52]. Patients may describe a snapping sensation or pain in the elbow with inclusion of

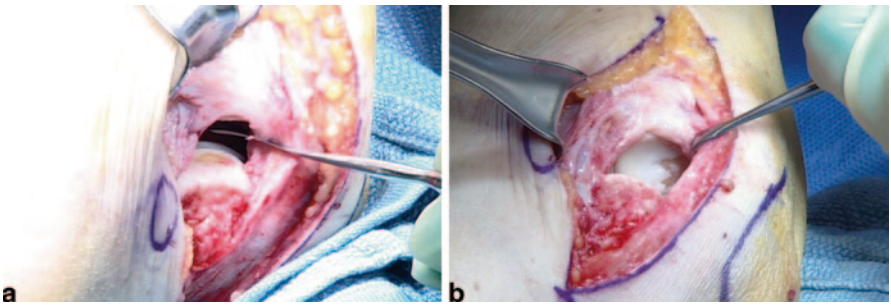

Fig. 15.2 **a**, **b** The photographs show radiocapitellar arthritis that can lead to persistent pain after tendon debridement surgery (Type 1 failure)

the redundant tissue in the radiocapitellar joint [52]. Underlying structural defects in the radiocapitellar joint such as osteoarthritis and OCD of the capitellum can lead to discomfort, clicking, and catching sensations [1, 13, 30, 39, 40, 47, 48, 53]. Sasaki et al. [54] arthroscopically investigated the correlation of radiocapitellar cartilage integrity with lateral epicondylitis and detected cartilage defects in the capitellum and radial head in 65 and 81 % of elbows, respectively. Other authors have found intraarticular pathology in 11–69 % of patients with lateral epicondylitis [4, 13, 40, 42, 55] (Fig. 15.3).

Snapping triceps syndrome involves painful translation of the lateral margin of the triceps tendon over the lateral epicondyle during elbow flexion [56]. Patients with a cubitus valgus deformity of the elbow may be particularly susceptible to this condition. A less common cause of Type 1 failure includes lateral ligament insufficiency with varus posterolateral rotatory instability of the elbow [1, 35]. Painful instability may develop following trauma or insidiously, as has been reported in patients with a preexisting cubitus varus deformity, and after local steroid injections into the common extensor tendon origin [47, 57, 58] (Fig. 15.4).

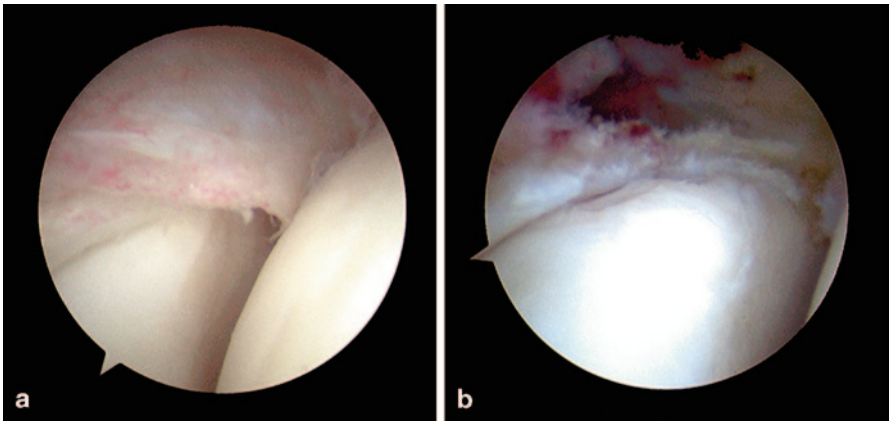

Fig. 15.3 **a**, **b** Arthroscopic views of a synovial plica in the anterolateral aspect of the radiocapitellar joint before and after debridement

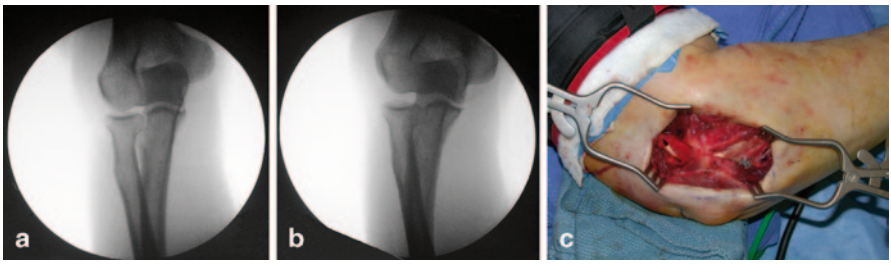

Fig. 15.4 **a**, **b**, **c** Varus posterolateral rotatory instability of the elbow can be a source of persistent pain. The anteroposterior fluoroscopic images show normal radiocapitellar joint alignment without applied stress and widening of the radiocapitellar joint space with varus stressing of the elbow, suggestive of lateral ligament incompetence. The patient eventually required lateral ligament reconstruction using a palmaris longus tendon autograft

Very infrequent causes of lateral elbow pain include a muscle compartment syndrome (e.g., anconeus muscle) or an osteoid osteoma [1, 14, 44, 59]. The possibility of an osteoid osteoma should be considered in a younger patient with atypical pain, especially at night [60].

Type 2 Failure—Inadequate Debridement of Tendinous Tissue

Type 2 failures result from inadequate debridement of pathological tissue from the common extensor tendon origin [1]. Patients will typically describe residual pain at the same site as being less severe or different in character from their pain experienced before surgery [1, 30]. This scenario may require revision debridement to address the problem.

Type 3 Failure—Iatrogenic

Type 3 failures result from the introduction of new pathology following surgical intervention [1]. Patients may describe a myriad of upper extremity symptoms including pain, numbness, weakness, joint laxity, stiffness, swelling, and catching.

Injuries to the radial nerve, the posterior interosseus nerve, and the anterior interosseous nerve have been reported [41, 61]. Formation of a troublesome synovial fistula can result from extensive debridement of the extensor origin and joint capsule [33, 47]. Overly aggressive removal of tissue may also lead to disruption of the lateral collateral ligament complex and varus posterolateral rotatory instability of the elbow (Figs. 15.5 and 15.6).

Less common etiologies of Type 3 failure include infection and osteophyte formation [62]. Growth of an osteophyte at the lateral epicondyle may lead to a snapping sensation with joint motion [62]. Elbow pain, swelling, and stiffness in the presence or absence of systemic symptoms may be indicative of a deep joint infection.

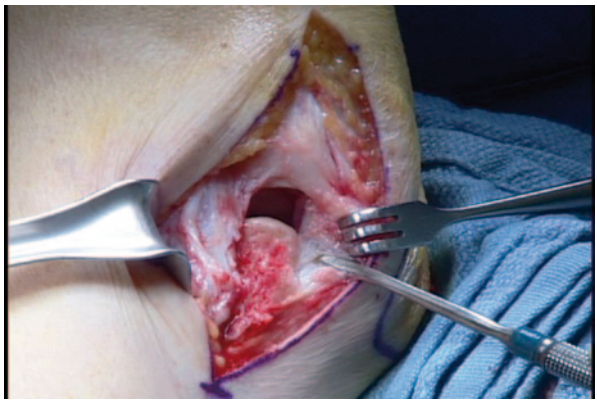

Fig. 15.5 Photograph of a right elbow showing aggressive debridement of the common extensor origin and resultant detachment of the lateral collateral ligament complex origin from bone. The ligament complex requires reattachment or reconstruction to avoid symptomatic varus posterolateral instability of the elbow

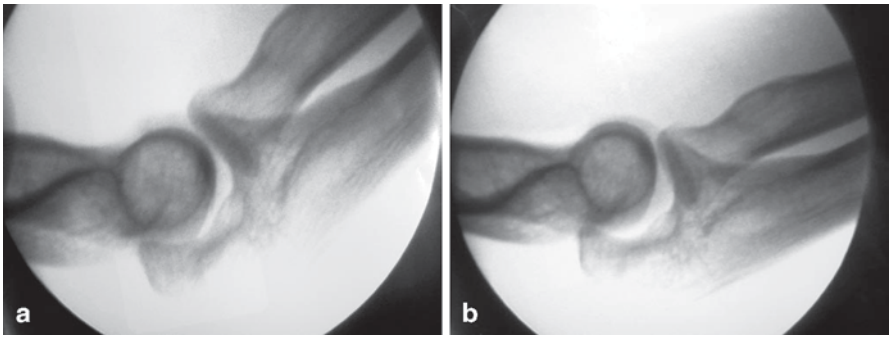

Fig. 15.6 **a**, **b** Varus posterolateral instability of the elbow can result from aggressive debridement of the common extensor origin and detachment of the lateral collateral ligament complex from bone (Type 3 failure). The lateral fluoroscopic images show normal alignment of the radial head and capitellum without stressing the joint and posterior subluxation of the radial head with lateral pivot shift testing

Type 4 Failure—Rehabilitation and Patient-Related Factors

Misguided rehabilitation and patient noncompliance have been conjectured as the most common causes of persistent lateral elbow pain after tennis elbow release surgery [1, 13]. Studies have shown higher levels of pain and longer periods of convalescence in patients receiving workers' compensation benefits when compared to patients not receiving benefits. Das De et al. [63] recently proposed psychological dysfunction as a primary etiology for lateral epicondylitis. In a cross-sectional study of persons with an upper-extremity-specific disability, these authors found that lower preoperative DASH scores correlated significantly with anxiety, depression, and kinesiophobia in patients undergoing treatment for lateral epicondylitis [63].

Patient Evaluation

The patient history is paramount in deciphering the cause of persistent or new symptoms after tennis elbow release surgery. Failure may be related to one or more problems in more than one category. The patient is asked to describe the symptoms and make a comparison to those experienced before surgery [30]. An inquiry is also made into precipitating, aggravating, and relieving factors. The presence of night pain may be indicative of osteoarthritis, OCD of the capitellum, an osteoid osteoma, or a septic joint [1, 30].

Physical Examination

The physical examination for failed tennis elbow surgery includes inspection and palpation of the elbow, and assessments of elbow motion, strength, and stability. An evaluation of the cervical spine and peripheral nervous system are also important for diagnosis. Ideally, the examiner's findings are compared with the findings recorded before the index operation.

A visible elbow deformity such as cubitus varus may be indicative of preexisting varus posterolateral instability [57, 64]. Lateral joint swelling may result from infection or a synovial fistula. A high index of suspicion is necessary to diagnose infection, as ongoing therapy with a nonsteroid antiinflammatory drug, narcotic pain medication, and/or an oral antibiotic may delay diagnosis.

Tenderness over the arcade of Frohse suggests irritability of the posterior interosseus nerve, while pain over the radiocapitellar joint suggests OCD of the capitellum, a synovial plica, or arthritis [55]. Extensor origin tenderness may result from incomplete debridement of the ECRB tendon or formation of a neuroma, osteophyte, or synovial fistula. Lateral elbow pain with resisted forearm supination and wrist extension may also indicate inadequate debridement. Pain with the arm in terminal extension and full supination may represent a plica in the radiocapitellar joint [48].

Decreased range of motion may be seen with arthrofibrosis, arthritis, intraarticular loose bodies, and OCD of the capitellum [55]. Pain throughout an arc of elbow motion is associated with generalized arthritis, whereas pain at the end range of elbow motion is seen in conjunction with impinging osteophytes. Decreased strength and/or a sensory disturbance may provide clues into a neurological deficit.

Elbow stability should be evaluated in all patients. Varus stress and lateral pivot-shift testing may elicit pain and a sensation of giving way in cases of lateral ligamentous insufficiency [64]. Comparing joint laxity between the symptomatic and asymptomatic elbows under fluoroscopic imaging can be particularly useful in equivocal circumstances.

Selective Local Anesthetic Injections

Local anesthetic injections are potentially both diagnostic and therapeutic. Lidocaine, with or without a corticosteroid product, can be injected in the area of maximum pain. Relief of pain after injection over the lateral epicondyle may represent incomplete debridement of the ECRB tendon or neuroma formation of the posterior cutaneous nerve of the forearm.

If an anesthetic injection over the lateral epicondyle does not provide symptom relief, other injection sites may be considered. An injection over the posterior interosseous nerve around the supinator muscle may cause a temporary palsy of the ulnar wrist and digital extensors, as well as provide transient pain relief when radial tunnel syndrome exists [1]. Entrapment of the lateral antebrachial cutaneous nerve can be discerned by an injection at the intersection of the epicondylar humeral line with the lateral biceps tendon [51]. A positive response to a subcutaneous local block of the posterior branches of the posterior cutaneous nerve of the forearm may support denervation of these nerve branches [27].

If an intraarticular source of pain is likely, a lidocaine injection into the posterior elbow compartment through the lateral soft spot may be useful for diagnostic confirmation (the soft spot overlies the junction of the radial head, the capitellum, and the olecranon). Partial or complete relief of pain is expected. Botulinum toxin A has been studied as an alternative means of diagnosing and treating persistent tennis elbow pain; however, administration of botulinum toxin A for this purpose is still considered off-label [20, 21].

Imaging and Electrodiagnostic Testing

Elbow radiographs are typically obtained before advanced imaging studies. Plain radiographs may show pathology contributing to symptoms, including a loose body, elevation of the anterior fat pad indicative of a joint effusion, asymmetric joint widening secondary to instability, an epicondylar osteophyte, and OCD of the capitellum. Stress radiographs or fluoroscopy are useful in diagnosing elbow joint instability. Magnetic resonance imaging is arguably the most useful imaging study to detect ligament damage, a joint effusion, synovitis, a synovial fistula, and osteomyelitis [65]. If a snapping triceps tendon is suspected, dynamic ultrasound may be beneficial for diagnosis as well. Although no longer commonly ordered, a technetium-99 m bone scan may be helpful in the detection of early arthritis or osteomyelitis [1] (Figs 15.7 and 15.8).

In cases of suspected entrapment of the lateral antebrachial cutaneous nerve, brachial plexopathy, or cervical radiculopathy, electrophysiological testing can be helpful for diagnosis [51]. However, nerve irritability is not always discerned by electrodiagnostic studies, as in the case of radial tunnel syndrome [1]. In situations where the etiology of pain remains uncertain, diagnostic elbow arthroscopy may be useful.

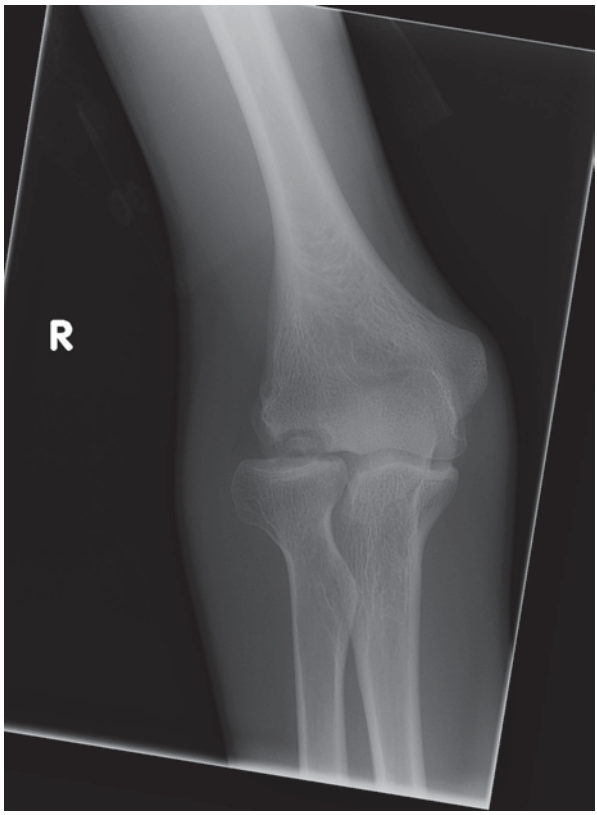

Fig. 15.7 Radiographs assessing the osseous integrity of the elbow should be performed prior to any surgical intervention. The anteroposterior radiograph reveals an osteochondral defect of the capitellum which can result in a Type 1 failure if not recognized

Joint Fluid Analysis and Serology

A crystalline arthropathy or infection of the elbow joint may be detected by synovial fluid analysis. However, microorganism stains and cultures are not universally diagnostic for infection. Adjunct serological studies, including the erythrocyte sedimentation rate, the C-reactive protein level, the white blood cell count, the blood glucose level, and the uric acid level, can be helpful in the diagnosis [66–70]. The clinical relevance of these studies will need to be interpreted in the context of the patient's history, symptoms, and physical examination. Gout can arise in the presence of a normal uric acid level, and septic arthritis can occur without neutrophilia. Recent surgery will often lead to transient elevations in the erythrocyte sedimentation

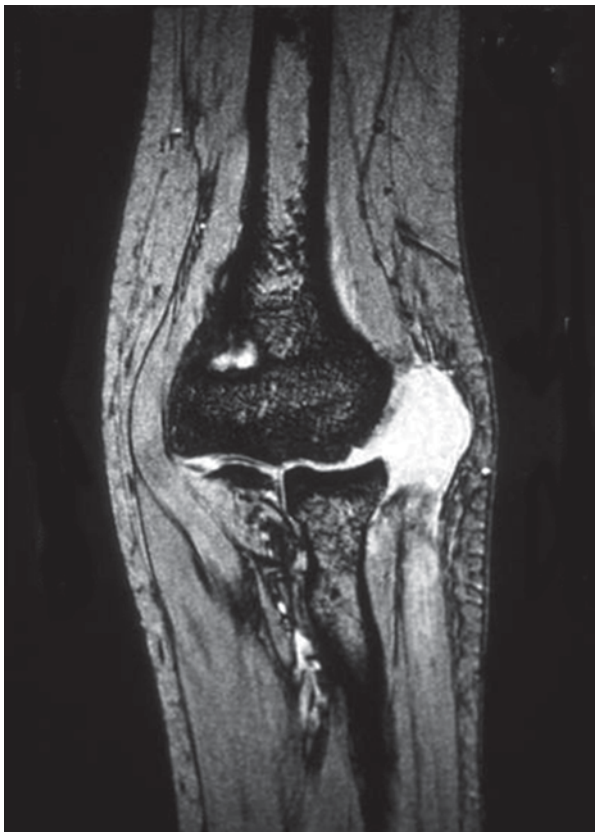

Fig. 15.8 A synovial fistula can form following debridement surgery for lateral epicondylitis. In this T2-weighted MRI image, joint fluid extravasation through a defect in the lateral collateral ligament complex is seen. The patient presented with persistent pain and a sensation of joint laxity that required revision surgery and ligament reconstruction

rate and the C-reactive protein level [71–73]. Elevations in these markers are also seen in association with inflammatory arthritides, inflammatory bowel disease, coronary artery disease, postoperative inflammation, and neoplasms.

Treatment and Outcomes

The success of primary operative treatment for lateral epicondylitis is usually seen within a few months after surgery [30]. Posch et al. [14] found that fewer than 2.3% patients had further symptom improvements after 1 year from surgery. Suboptimal stretching and strengthening efforts after surgery may be the primary reasons for lingering elbow pain [1]. In patients with continued discomfort several weeks after surgery, many will improve with continued therapy measures [35].

Surgical intervention for a suspected surgical failure depends on the root cause(s). In Type 1 failure, a concomitant problem such as a nerve compression syndrome may require nerve decompression or denervation. A tender epicondylar

osteophyte may necessitate excision, symptomatic osteoarthritis of the capitellum may dictate capitellar resection, and a problematic plica may require arthroscopic excision of the redundant synovial tissue for pain relief. Treatment of snapping triceps syndrome entails incision and medial reflection of a portion of the lateral triceps tendon [56].

In Type 2 failure, additional debridement of degenerative tissue from the extensor tendon origin may be indicated. Adjunct denervation of the lateral epicondyle can be considered, in addition to transfer of anconeus muscle tissue to cover any concerning tendinous defect [24–28].

In Type 3 failure with iatrogenic varus posterolateral instability of the elbow, reconstruction of the lateral ligament complex is warranted. In the case of a deep joint infection, surgical debridement and antibiotic treatment are necessary. The initial antibiotic regimen should be directed at common skin flora, taking into consideration the possibility of infection with a methicillin-resistant staphylococcus species.

Physicians need to be cautious in treating patients with a suspected Type 4 failure, assessing the benefits of additional surgery in light of secondary gain and/or contributing psychological dysfunction. Patients with workers' compensation claims or pending litigation should be observed longer than 6–9 months before considering further surgical intervention, unless a clear etiology for recalcitrant elbow pain is identified [1] (Fig. 15.9).

There are relatively few articles pertaining to the treatment of failed tennis elbow surgery. Morrey et al. [30] noted 85% success after reoperation in 13 patients for failed primary surgical treatment of lateral epicondylitis. Surgery was directed at correcting the root causes of persistent pain, including radial tunnel syndrome,

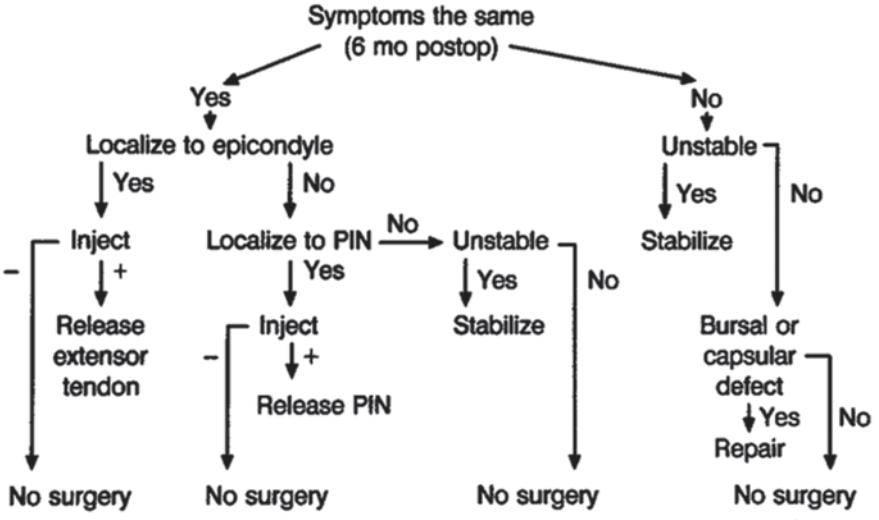

Fig. 15.9 Algorithm proposed by Morrey [30] to diagnose and treat failures following lateral epicondylitis surgery. (Borrowed from [30]. Permission obtained from Elsevier)

incomplete debridement of the ECRB tendon origin, lateral ligament insufficiency, and fistula formation [30].

Nirschl et al. [13] investigated causes for failed lateral epicondylitis surgery in 82 patients and found that the degenerative ECRB tendon origin was not addressed at the index procedure in 77% of cases and was inadequately debrided in 20% of cases. Complete excision of pathologic tissue resulted in a good or excellent outcome in 83% of cases [13]. Organ et al. [35] studied 35 elbows after failed surgical intervention and noted residual tendinosis of the ECRB tendon in 34 cases. The pathological tissue was debrided in all patients, exostoses were removed in 12 elbows, and concurrent radial collateral ligament insufficiency was addressed by ligament reconstruction in one patient. At a mean follow-up of 64 months, 83% of patients achieved a good or excellent outcome.

Dellon et al. [74] reported nine patients who developed a painful incisional neuroma involving the posterior interosseous nerve of the forearm after open tennis elbow release surgery. All patients in their series obtained good to excellent pain relief following neurectomy and resection of scar tissue. The results after treatment of a deep infection are presumably less optimal due to degeneration of articular cartilage and the development of fibrous adhesions [75]. Further outcomes research is necessary to determine expectations and the best means for addressing the various modes of failure.

References

1. Morrey BF. Surgical failures of tennis elbow. In: Morrey BF, Sanchez-Sotelo J, editors. The elbow and its disorders. 4th ed. Philadelphia: Saunders/Elsevier; 2009. p. 650–7.
2. Boyer MI, Hastings H 2nd. Lateral tennis elbow: "Is there any science out there?" J Shoulder Elbow Surg. 1999;8(5):481–91.
3. Allander E. Prevalence, incidence, and remission rates of some common rheumatic diseases or syndromes. Scand J Rheumatol. 1974;3(3):145–53.
4. Calfee RP, Patel A, DaSilva MF, Akelman E. Management of lateral epicondylitis: current concepts. J Am Acad Orthop Surg. 2008;16(1):19–29.
5. Verhaar JA. Tennis elbow. Anatomical, epidemiological and therapeutic aspects. Int Orthop. 1994;18(5):263–7.
6. Faro F, Wolf JM. Lateral epicondylitis: review and current concepts. J Hand Surg Am. 2007;32(8):1271–9.
7. De Smedt T, de Jong A, Van Leemput W, Lieven D, Van Glabbeek F. Lateral epicondylitis in tennis: update on aetiology, biomechanics and treatment. Br J Sports Med. 2007;41(11):816–9.
8. Shiri R, Viikari-Juntura E, Varonen H, Heliovaara M. Prevalence and determinants of lateral and medial epicondylitis: a population study. Am J Epidemiol. 2006;164(11):1065–74.
9. Briggs CA, Elliott BG. Lateral epicondylitis. A review of structures associated with tennis elbow. Anat Clin. 1985;7(3):149–53.
10. Coonrad RW, Hooper WR. Tennis elbow: its course, natural history, conservative and surgical management. J Bone Joint Surg Am. 1973;55(6):1177–82.
11. Nirschl RP. Elbow tendinosis/tennis elbow. Clin Sports Med. 1992;11(4):851–70.
12. Nirschl RP, Ashman ES. Elbow tendinopathy: tennis elbow. Clin Sports Med. 2003;22(4):813–36.

13. Nirschl RP, Pettrone FA. Tennis elbow. The surgical treatment of lateral epicondylitis. J Bone Joint Surg Am. 1979;61(6A):832–9.
14. Posch JN, Goldberg VM, Larrey R. Extensor fasciotomy for tennis elbow: a long-term follow-up study. Clin Orthop Relat Res. 1978;135:179–82.
15. Kraushaar BS, Nirschl RP. Tendinosis of the elbow (tennis elbow). Clinical features and findings of histological, immunohistochemical, and electron microscopy studies. J Bone Joint Surg Am. 1999;81(2):259–78.
16. Binder AI, Hazleman BL. Lateral humeral epicondylitis—a study of natural history and the effect of conservative therapy. Br J Rheumatol. 1983;22(2):73–6.
17. Haahr JP, Andersen JH. Prognostic factors in lateral epicondylitis: a randomized trial with one-year follow-up in 266 new cases treated with minimal occupational intervention or the usual approach in general practice. Rheumatology (Oxford). 2003;42(10):1216–25.
18. Smidt N, van der Windt DA, Assendelft WJ, Deville WL, Korthals-de Bos IB, Bouter LM. Corticosteroid injections, physiotherapy, or a wait-and-see policy for lateral epicondylitis: a randomised controlled trial. Lancet. 2002;359(9307):657–62.
19. De Andres J, Cerda-Olmedo G, Valia JC, Monsalve V, Lopez A, Minguez A. Use of botulinum toxin in the treatment of chronic myofascial pain. Clinical J Pain. 2003;19(4):269–75.
20. Placzek R, Drescher W, Deuretzbacher G, Hempfing A, Meiss AL. Treatment of chronic radial epicondylitis with botulinum toxin A. A double-blind, placebo-controlled, randomized multicenter study. J Bone Joint Surg Am. 2007;89(2):255–60.
21. Wong SM, Hui AC, Tong PY, Poon DW, Yu E, Wong LK. Treatment of lateral epicondylitis with botulinum toxin: a randomized, double-blind, placebo-controlled trial. Ann Inter Med. 2005;143(11):793–7.
22. Thanasas C, Papadimitriou G, Charalambidis C, Paraskevopoulos I, Papanikolaou A. Platelet-rich plasma versus autologous whole blood for the treatment of chronic lateral elbow epicondylitis: a randomized controlled clinical trial. Am J Sports Med. 2011;39(10):2130–4.
23. Boyd HB, McLeod AC Jr. Tennis elbow. J Bone Joint Surg Am. 1973;55(6):1183–7.
24. Almquist EE, Necking L, Bach AW. Epicondylar resection with anconeus muscle transfer for chronic lateral epicondylitis. J Hand Surg Am. 1998;23(4):723–31.
25. Luchetti R, Atzei A, Brunelli F, Fairplay T. Anconeus muscle transposition for chronic lateral epicondylitis, recurrences, and complications. Tech Hand Up Extrem Surg. 2005;9(2):105–12.
26. Berry N, Neumeister MW, Russell RC, Dellon AL. Epicondylectomy versus denervation for lateral humeral epicondylitis. Hand (N Y). 2011;6(2):174–8.
27. Rose NE, Forman SK, Dellon AL. Denervation of the lateral humeral epicondyle for treatment of chronic lateral epicondylitis. J Hand Surg Am. 2013;38(2):344–9.
28. Wilhelm A. Tennis elbow: treatment of resistant cases by denervation. J Hand Surg Br. 1996;21(4):523–33.
29. Bosworth DM. The role of the orbicular ligament in tennis elbow. J Bone Joint Surg Am. 1955;37-A(3):527–33.
30. Morrey BF. Reoperation for failed surgical treatment of refractory lateral epicondylitis. J Shoulder Elbow Surg. 1992;1(1):47–55.
31. Regan W, Wold LE, Coonrad R, Morrey BF. Microscopic histopathology of chronic refractory lateral epicondylitis. Am J Sports Med. 1992;20(6):746–9.
32. van Rossum J, Buruma OJ, Kamphuisen HA, Onvlee GJ. Tennis elbow–a radial tunnel syndrome? J Bone Joint Surg Br. 1978;60-B(2):197–8.
33. Yerger B, Turner T. Percutaneous extensor tenotomy for chronic tennis elbow: an office procedure. Orthopedics. 1985;8(10):1261–3.
34. Wittenberg RH, Schaal S, Muhr G. Surgical treatment of persistent elbow epicondylitis. Clin Orthop Relat Res. 1992;278:73–80.
35. Organ SW, Nirschl RP, Kraushaar BS, Guidi EJ. Salvage surgery for lateral tennis elbow. Am J Sports Med. 1997;25(6):746–50.
36. Mullett H, Sprague M, Brown G, Hausman M. Arthroscopic treatment of lateral epicondylitis: clinical and cadaveric studies. Clinical Orthop Relat Res. 2005;439:123–8.

37. Verhaar J, Walenkamp G, Kester A, van Mameren H, van der Linden T. Lateral extensor release for tennis elbow. A prospective long-term follow-up study. J Bone Joint Surg Am. 1993;75(7):1034–43.
38. Coleman B, Quinlan JF, Matheson JA. Surgical treatment for lateral epicondylitis: a long-term follow-up of results. J Shoulder Elbow Surg. 2010;19(3):363–7.
39. Baker CL Jr., Murphy KP, Gottlob CA, Curd DT. Arthroscopic classification and treatment of lateral epicondylitis: two-year clinical results. J Shoulder Elbow Surg. 2000;9(6):475–82.
40. Owens BD, Murphy KP, Kuklo TR. Arthroscopic release for lateral epicondylitis. Arthroscopy. 2001;17(6):582–7.
41. Lattermann C, Romeo AA, Anbari A, Meininger AK, McCarty LP, Cole BJ, Cohen MS. Arthroscopic debridement of the extensor carpi radialis brevis for recalcitrant lateral epicondylitis. J Shoulder Elbow Surg. 2010;19(5):651–6.
42. Szabo SJ, Savoie FH 3rd, Field LD, Ramsey JR, Hosemann CD. Tendinosis of the extensor carpi radialis brevis: an evaluation of three methods of operative treatment. J Shoulder Elbow Surg. 2006;15(6):721–7.
43. Solheim E, Hegna J, Oyen J. Arthroscopic versus open tennis elbow release: 3- to 6-year results of a case-control series of 305 elbows. Arthroscopy. 2013;29(5):854–9.
44. Abrahamsson SO, Sollerman C, Soderberg T, Lundborg G, Rydholm U, Pettersson H. Lateral elbow pain caused by anconeus compartment syndrome. A case report. Acta Orthop Scand. 1987;58(5):589–91.
45. Stovell PB, Beinfield MS. Treatment of resistant lateral epicondylitis of the elbow by lengthening of the extensor carpi radialis brevis tendon. Surg Gynecol Obstet. 1979;149(4):526–8.
46. Werner CO. Lateral elbow pain and posterior interosseous nerve entrapment. Acta Orthop Scand Suppl. 1979;174:1–62.
47. Kalainov DM, Cohen MS. Posterolateral rotatory instability of the elbow in association with lateral epicondylitis. A report of three cases. J Bone Joint Surg Am. 2005;87(5):1120–5.
48. Ruch DS, Papadonikolakis A, Campolattaro RM. The posterolateral plica: a cause of refractory lateral elbow pain. J Shoulder Elbow Surg. 2006;15(3):367–70.
49. Roles NC, Maudsley RH. Radial tunnel syndrome: resistant tennis elbow as a nerve entrapment. J Bone Joint Surg Br. 1972;54(3):499–508.
50. Hashizume H, Nishida K, Nanba Y, Inoue H, Konishiike T. Intraneural ganglion of the posterior interosseous nerve with lateral elbow pain. J Hand Surg Br. 1995;20(5):649–51.
51. Naam NH, Massoud HA. Painful entrapment of the lateral antebrachial cutaneous nerve at the elbow. J Hand Surg Am. 2004;29(6):1148–53.
52. Tsuji H, Wada T, Oda T, Iba K, Aoki M, Murakami G, Yamashita T. Arthroscopic, macroscopic, and microscopic anatomy of the synovial fold of the elbow joint in correlation with the common extensor origin. Arthroscopy. 2008;24(1):34–8.
53. Morrison DL. Tennis elbow and radial tunnel syndrome: differential diagnosis and treatment. J Am Osteopath Assoc. 1981;80(12):823–6.
54. Sasaki K, Onda K, Ohki G, Sonoda T, Yamashita T, Wada T. Radiocapitellar cartilage injuries associated with tennis elbow syndrome. J Hand Surg Am. 2012;37(4):748–54.
55. Baker CL 3rd, Baker CL Jr., Romeo AA. Osteochondritis dissecans of the capitellum. J Shoulder Elbow Surg. 2010;19(2 Suppl):76–82.
56. Spinner RJ, Goldner RD, Fada RA, Sotereanos DG. Snapping of the triceps tendon over the lateral epicondyle. J Hand Surg Am. 1999;24(2):381–5.
57. O'Driscoll SW, Spinner RJ, McKee MD, Kibler WB, Hastings H 2nd, Morrey BF, Kato H, Takayama S, Imatani J, Toh S, Graham HK. Tardy posterolateral rotatory instability of the elbow due to cubitus varus. J Bone Joint Surg Am. 2001;83-A(9):1358–69.
58. Dzugan SS, Savoie FH 3rd, Field LD, O'Brien MJ, You Z. Acute radial ulno-humeral ligament injury in patients with chronic lateral epicondylitis: an observational report. J Shoulder Elbow Surg. 2012;21(12):1651–5.
59. Steinmann SP, Bishop AT. Chronic anconeus compartment syndrome: a case report. J Hand Surg Am. 2000;25(5):959–61.

60. Spiteri V, Aslam N, Gatt R. Osteoid osteoma of the radial tuberosity presenting as lateral elbow pain. J Shoulder Elbow Surg. 2006;15(6):e5–7.
61. Carofino BC, Bishop AT, Spinner RJ, Shin AY. Nerve injuries resulting from arthroscopic treatment of lateral epicondylitis: report of 2 cases. J Hand Surg Am. 2012;37(6):1208–10.
62. Shapiro GS, Weiland AJ. Reactive bone formation after surgery for lateral epicondylitis. J Shoulder Elbow Surg. 2002;11(4):383–5.
63. Das De S, Vranceanu AM, Ring DC. Contribution of kinesophobia and catastrophic thinking to upper-extremity-specific disability. J Bone Joint Surg Am. 2013;95(1):76–81.
64. McRae R. Clinical orthopaedic examination. Edinburgh: Churchill Livingstone; 2010. viii, p. 323.
65. Karchevsky M, Schweitzer ME, Morrison WB, Parellada JA. MRI findings of septic arthritis and associated osteomyelitis in adults. Am J Roentgenol. 2004;182(1):119–22.
66. Della Valle C, Parvizi J, Bauer TW, Dicesare PE, Evans RP, Segreti J, Spangehl M, Watters WC 3rd, Keith M, Turkelson CM, Wies JL, Sluka P, Hitchcock K. Diagnosis of periprosthetic joint infections of the hip and knee. J Am Acad Orthop Surg. 2010;18(12):760–70.
67. Fink B, Makowiak C, Fuerst M, Berger I, Schafer P, Frommelt L. The value of synovial biopsy, joint aspiration and C-reactive protein in the diagnosis of late peri-prosthetic infection of total knee replacements. J Bone Joint Surg Br. 2008;90(7):874–8.
68. Greidanus NV, Masri BA, Garbuz DS, Wilson SD, McAlinden MG, Xu M, Duncan CP. Use of erythrocyte sedimentation rate and C-reactive protein level to diagnose infection before revision total knee arthroplasty. A prospective evaluation. J Bone Joint Surg Am. 2007;89(7):1409–16.
69. Kamme C, Lindberg L. Aerobic and anaerobic bacteria in deep infections after total hip arthroplasty: differential diagnosis between infectious and non-infectious loosening. Clin Orthop Relat Res. 1981;154:201–7.
70. Schinsky MF, Della Valle CJ, Sporer SM, Paprosky WG. Perioperative testing for joint infection in patients undergoing revision total hip arthroplasty. J Bone Joint Surg Am. 2008;90(9):1869–75.
71. Mathews CJ, Coakley G. Septic arthritis: current diagnostic and therapeutic algorithm. Curr Opin Rheumatol. 2008;20(4):457–62.
72. Coutlakis PJ, Roberts WN, Wise CM. Another look at synovial fluid leukocytosis and infection. J Clin Rheumatol. 2002;8(2):67–71.
73. Weston VC, Jones AC, Bradbury N, Fawthrop F, Doherty M. Clinical features and outcome of septic arthritis in a single UK Health District 1982–1991. Ann Rheum Dis. 1999;58(4):214–9.
74. Dellon AL, Kim J, Ducic I. Painful neuroma of the posterior cutaneous nerve of the forearm after surgery for lateral humeral epicondylitis. J Hand Surg Am. 2004;29(3):387–90.
75. Butters KP, Morrey BF. Septic arthritis. In: Morrey BF, Sanchez-Sotelo J, editors. The elbow and its disorders. 4th ed. Philadelphia: Saunders/Elsevier; 2009. pp. 1056–67.

Index

J. M. Wolf (ed.), *Tennis Elbow,* DOI 10.1007/978-1-4899-7534-8