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Failed surgical treatment for lateral epicondylitis: literature review and treatment considerations for successful outcomes

Zachary C. Hanson, MD[a,*], William P. Stults, MD[a], Gary M. Lourie, MD[a,b]

[a]Dept. of Orthopedic Surgery, Wellstar Atlanta Medical Center, Atlanta, GA, USA
[b]The Hand & Upper Extremity Center of Georgia, Atlanta, GA, USA

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Background: Lateral epicondylitis is a common cause of elbow pain in the general population. It is recognized as a degenerative tendinopathy of the common extensor origin believed to be multifactorial, involving elements of repetitive microtrauma associated with certain physiologic and anatomic risk factors.
Methods: Initial treatment typically involves a combination of conservative treatment measures, with up to 90% success at 12-18 months. Surgical treatment is reserved for recalcitrant disease; traditionally involving open surgical débridement of the common extensor origin with reported success rates greater than 90%.
Results: Failure of surgical treatment can be multifactorial and present a challenge in determining the optimum management. Residual symptoms may be due to an incorrect initial diagnosis, inadequate surgical débridement, new pathology as a complication of the initial surgery and/or other patient-related and physician- related factors. Even more of a challenge is the possibility that etiology can be due to a combination of listed factors.
Discussion: In this review, we review the classification scheme for evaluating failed surgical treatment of LE first proposed by Morrey and expand on this classification system based on the senior author's experience. We present the senior author's preferred systematic approach to evaluation and management of these patients, as well as a salvage surgery technique used by the senior author to address the most common etiologies of surgical failure in these patients.

Lateral epicondylitis (LE) affects 3% of adults over the age of 40 and up to 7% of manual laborers, with approximately 1 million people newly diagnosed in the United States each year.[17,53,62,64] Patients are typically treated with some combination of conservative treatment measures such as nonsteroidal anti-inflammatory medications, rest, bracing, physical therapy and/or home stretching programs, ultrasound, ionophoresis and/or injections (eg, corticosteroids or platelet-rich plasma), all of which have been shown to have variable success. A consistent finding among prospective studies of LE is that 80%-90% of patients improve within 12-18 months from the time they're enrolled in the study, regardless of treatment,[26] suggesting that even for cases which persist a year or longer, symptoms may still resolve without surgery.

An estimated 4%-11% of patients with LE eventually undergo surgical intervention.[26] Nirschl[43] described a surgical technique in 1979 for open débridement of the pathologic tissue overlying the lateral epicondyle; since that time, a number of modified-open and arthroscopic surgical techniques have been described, all of which have been reported with relatively good success.[47,51,60] Despite the success rates of surgery, treatment failure and surgical complications remain problematic. Postoperative symptoms may be caused by inadequate surgical treatment, incorrect initial diagnosis, failure to address concomitant pathology or a surgical complication or even a combination of factors; distinguishing the cause of failed surgical treatment is challenging but critical to provide appropriate treatment.

The purpose of this paper is to review the etiologies of failed surgical treatment of LE and expand on the classification of surgical failure types first proposed by Morrey.[40] We present the senior author's preferred systematic approach for evaluation and management of these patients, along with a salvage surgery technique used by the senior author to address the most common etiologies of surgical failure in these patients.

Etiology, pathoanatomy and ntural history

The common extensor origin is formed by the confluence of the extensor carpi radialis brevis (ECRB), extensor digitorum communis (EDC), extensor digiti minimi and extensor carpi ulnaris tendons,

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*Corresponding author: Zachary C. Hanson, MD, Dept. of Orthopaedic Surgery, 4th Fl. GME, 303 Parkway Drive NE, Atlanta, GA 30312, USA.
E-mail address: zchg47@gmail.com (Z.C. Hanson).

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originating from the anterior face of the lateral epicondyle of the distal humerus. While their individual anatomic footprints have been described in cadaveric studies, the EDC and ECRB do not become clinically distinct musculotendinous units until several centimeters distal to their origin. In contrast, the brachioradialis (BR) and extensor carpi radialis longus (ECRL) originate further proximally along the supracondylar ridge and are therefore distinguishable by the presence of muscle belly. LE is thought to involve primarily the superior and deep fibers of the ECRB tendon[13]; involvement of the EDC is thought to occur in approximately one-third of cases and less commonly, the ECRL or extensor carpi ulnaris may be involved as well.[29,42] Recent anatomic studies have shown that the ECRB is uniquely located among the tendons of the common extensor origin, laying slightly medial and superior to the outer edge of the capitellum as it passes directly over the radiocapitellar joint line with robust attachments to the joint capsule.[8] During elbow motion there is considerable contact between the undersurface of the ECRB with the capitellum as it is compressed by the ECRL against the underlying bone, which is thought to lead to abrasion and micro-tearing of the tendon origin during elbow motion.[12]

The course of the posterior interosseous nerve (PIN) through the “radial tunnel” is bordered laterally by the BR, ECRL, and ECRB muscles; compression of the PIN within the radial tunnel can present similar to LE, coexisting in 5% or more of LE cases, and is often implicated in patients with refractory LE.[65]

LE is thought to represent a multifactorial condition associated with elements of repetitive microtrauma in patients with certain physiologic risks. It is recognized as a degenerative tendinopathy/tendinosis of the common extensor origin, rather than an inflammatory tendonitis as its name suggests.[24] Repetitive microtrauma of the common extensor tendons in the setting of its inherent avascularity leads to an impaired healing response and subsequent angiofibroblastic dysplasia, an umbrella term used by Nirschl to describe the characteristic histology fibroblastic hypertrophy, disorganized collagen deposition, fibrovascular hyperplasia and the *absence* of inflammatory cells.[11,24,42,43]

Anatomic dissections have demonstrated one to two posterior branches of the posterior cutaneous nerve of the forearm (PCNF) innervate the periosteum of the lateral humeral epicondyle which serves as the origin of the to the common extensor muscles. Repetitive muscle tearing has been suggested to cause local neurotrauma and lead to painful microneuroma formation which may contribute to pain with the condition.[18,50,66] Proponents of local nerve denervation for recalcitrant LE cite this hypothesis as a possible reason for the effectiveness of the procedure.[9,66]

Recent literature has suggested additional etiologies which may contribute to this condition and should be considered as well. Arrigoni et al propose symptomatic minor instability of the lateral elbow (SMILE) as a potential cause of recalcitrant LE.[2] The authors suggest that incompetence/elongation of the lateral ulnar collateral ligament (LUCL) in patients with subtle elbow instability leads to increased strain on the ECRB, which acts as an extra-articular secondary stabilizer against varus-pronation stress at the elbow. Surgical treatment of LE in these patients should include LUCL-capsular plication for improved lateral elbow stability.[2]

Calcium deposition is often stated to be associated with enthesopathy of the ECRB origin in LE. However, the prevalence of these calcifications has been shown to increase with age while the incidence of LE peaks in the 6th decade indicating that this finding may be incidental.[61]

Despite a number of studies have attempted to identify the etiologies and risks associated with this condition, though conflicting data with respect to certain factors has made this challenging. A recent meta-analysis by Sayampanathan et al determined female sex and patients with a positive smoking history to have 1.29 and 1.49-times higher odds of sustaining LE, respectively.[55] Chronic hyperglycemia has also been implicated as patients with HbA1c > 6 were found to have 3.3-times higher risk.[45] Obesity, occupation, alcohol status, and other metabolic factors have not been consistently associated.[45]

Classifying failed surgical treatment

Indications for surgical treatment of lateral epicondylitis include failed conservative treatment for 6-12 months, with an estimated 4%-11% of patients with LE eventually requiring surgery.[26] While various techniques have been described, surgical treatment typically involves local excision of the degenerative tendinosis of the common extensor origin with débridement of the surrounding tendon bed with or without repair of the extensor tendon origin.[29] Results of surgical treatment are generally very good and reliable, with success rates of >90% reported in the literature.[6,22,23,43]

Despite the high success rates of surgery, failed surgical treatment can occur in 3%-10% of cases. Though this 10% can seem a small insignificant number, for those physicians given the task in correcting the cause, the goal can be difficult as the etiology so elusive. This is confounded by the fact that even successful surgery may still require several months of postoperative rest and rehabilitation before symptoms are relieved. Patients must be made aware of this preoperatively lest they attempt to return to aggressive activity too quickly and perceive the surgical treatment as a failure.[39] In compliant patients with an absence of secondary gain (eg, worker’s compensation), symptoms persisting 6-9 months postoperatively is unusual and is indicative of treatment failure.[38,39]

The first critical step is to assess the characteristics of the patient’s symptoms as compared to preoperatively; this determination allows the patient to be classified into one of several types of treatment failure as first described by Morrey and modified here based on the senior author’s clinical experience with these patients.[40] Patients with type 1 treatment failure refers to patients with symptoms that are unchanged after surgery, while type 2 failure refers to patients who develop new symptoms postoperatively. Type 3 failure refers to patients with a mixed complex with elements of both old and new symptoms. Distinguishing the treatment failure type is an important step in determining how to proceed in the evaluation and management of these patients.

Type 1 failure (symptoms unchanged)

Type 1A: incorrect initial diagnosis

Type 1A treatment failure is characterized by patients who have an incorrect initial diagnosis and therefore confirming the diagnosis of lateral epicondylitis following failed surgical treatment requires ruling out any pathology which might produce similar symptoms (Table I).

Radial tunnel syndrome caused by compression of the PIN in the proximal forearm is often difficult to distinguish from LE.[1,4,30,40,43,46,52,57] Further, these two entities coexist in as many as 5% of lateral epicondylitis, or even more frequently (as many as 44% of cases) in some tertiary care settings, as has been the case with this senior author; complicating accurate diagnosis and treatment.[65] The use of a counterforce brace can create its own potential confounding source of pain through compression of the radial nerve in the radial tunnel, causing further confusion which must be clinically distinguished as a separate entity.[18] Other neurogenic causes which may be confused for LE include cervical

Table I
Differential diagnoses for lateral elbow pain, type 1A treatment failure.

Diagnosis	Pathology	Distinguishing characteristics
Radial tunnel syndrome	PIN compression within radial tunnel	Tenderness over mobile wad, 3-5 cm distal to LE. +Pain with resisted supination, long finger extension (none with resisted wrist extension)
Elbow osteoarthritis	Degenerative changes ± impinging osteophytes of radiocapitellar joint	Older patients; pain over RC joint, crepitus. Pain at ends of motion±fixed flexion deformity (eg, impinging osteophytes)
OCD of capitellum	Commonly adolescent males, overhead throwing activities	Young patient; pain/effusion, mechanical symptoms (eg, clicking)
Cervical radiculopathy	C6 nerve root compression	History of neck pain; +Spurling's maneuver; paresthesias in lateral elbow/dorsal forearm, BR and wrist extension weakness
LABCN entrapment	Compression between biceps and brachialis m. at lateral margin of biceps tendon	Pain at site of entrapment, relief with localized anesthetic injection, +NCV/EMG findings
Snapping triceps syndrome[35]	Painful subluxation of lateral triceps tendon over lateral epicondyle with elbow flexion	Cubitus valgus deformity Painful snapping with elbow flexion past 90 degrees
Synovial plica[36]	Focal thickening of posterolateral synovial fold underlying the joint capsule and lateral epicondyle enthesis	Mechanical symptoms (clicking, snapping, catching) Painful impingement with elbow extension, forearm pronation
Posterolateral rotatory instability	Preexisting LUCL insufficiency	Cubitus varus, history of trauma (eg, dislocation), previous surgery/steroid injections Lateral pivot-shift test, varus instability
Anconeus compartment syndrome	Chronic compartment syndrome with anconeus inflammation/ edema	Swelling, painful bogginess/ bulging over anconeus m. Edema of anconeus m. on MRI; Elevated compartment pressures

RC, radiocapitellar; *PIN*, posterior interosseous nerve; *NCV*, nerve conduction velocity; *EMG*, electromyography; *LABCN*, lateral antebrachial cutaneous nerve; *BR*, brachioradialis; *OCD*, osteochondritis dissecans; *LUCL*, lateral ulnar collateral ligament; *m.*, muscle.

radiculopathy, brachial plexopathy, and entrapment of lateral antebrachial cutaneous nerve (LACN) or posterior brachial cutaneous nerve.[41,50]

LUCL insufficiency also commonly presents as lateral elbow pain and must be distinguished from lateral epicondylitis. Patients with a history of mechanical symptoms such as clicking or catching with elbow extension or a physical examination demonstrating varus laxity or positive pivot-shift, push-up, chair, or tabletop tests should be further evaluated with stress radiographs or fluoroscopy before any intervention at the lateral epicondyle.

Intra-articular pathologies may also produce similar symptoms and should be considered for patients with type 1A surgical failure. Radiocapitellar osteochondral lesions, synovial plica, loose bodies, and radiocapitellar arthritis have been reported to cause similar lateral-based elbow pain.[52,65] Undiagnosed intraarticular osteoid osteomas have been incorrectly diagnosed as lateral epicondylitis as well.[10]

Other, less frequent diagnoses such as snapping triceps syndrome lateral overload from medial collateral ligament instability, and avascular necrosis of the capitellum should be ruled out as these conditions may similarly be confused for LE. Similarly, isolated chronic compartment syndrome of the anconeus muscle can present with persistent laterally based elbow pain; painful swelling, bogginess and bulging overlying the anconeus muscle is suggestive of this diagnosis, which can be confirmed by elevated resting, pre-exercise and postexercise compartment pressures as well as isolated inflammation and edema of the anconeus muscle on magnetic resonance (MR) imaging.[1,14,58]

Type 1B: inadequate surgical treatment

Type 1B treatment failure is due to inadequate or incomplete surgery, most commonly due to inadequate débridement/excision of pathologic tissue.[31,39] These patients will have residual pain in the same location as before surgery, though symptoms may be less severe or slightly different in character.[31,39,40] Thorough evaluation of these patients to confirm a diagnosis of LE should be performed; patients will have maximal tenderness slightly distal and anterior to the lateral epicondyle, overlying the common extensor origin, and pain with resisted wrist and finger extension.

Types 1C & 1D: patient and physician-related factors

Type 1C surgical failure refers to treatment failures attributed to patient-related factors. Misguided or noncompliance with postoperative rehabilitation represent a common cause of persistent pain after surgical treatment of lateral epicondylitis.[39,42] Patient motivation, job satisfaction and underlying secondary gain should be considered as well. Patients receiving worker's compensation have been shown to have longer duration of pain and recovery following surgery.[34] A study by Das De et al found that among patients being treated for lateral epicondylitis, lower preoperative Disabilities of the Arm, Shoulder and Hand (DASH) scores were associated with significantly higher rates of anxiety, depression and kinesophobia; they proposed psychologic disorders may be a significant contributor to symptoms in patients being treated for lateral epicondylitis.[15] Failure of the physician to adequately establish and maintain a patient-physician relationship can similarly affect outcome success.

As with type 1C failure, physician related factors can play an important role in treatment failure as well. If the physician fails to establish a supportive and caring relationship, regardless of the best surgery, the ultimate result is in jeopardy.

Type 2 failure (new symptoms)

Type 2 treatment failure is characterized by the development of *new* symptoms after surgery, indicating new pathology introduced from iatrogenic injury (Table II). Patients with pain in a new location, change in quality of pain, and/or the development of new symptoms (eg, local swelling or fullness, elbow laxity, mechanical symptoms) are suggestive of type 2 surgical failure.

Excessive débridement at the common extensor origin can easily result in LUCL injury. Posterolateral rotatory instability must always be considered in those having undergone multiple corticosteroid injections or surgery at the lateral elbow. Inadvertent capsular excision with subsequent synovial fistula formation and

Table II
Differential diagnosis for type 2 treatment failure.

Diagnosis	Pathology	Distinguishing characteristics
Posterolateral rotatory instability	Excessive débridement with disruption of LUCL	+Lateral pivot-shift, varus stress tests LUCL disruption on MR arthrogram; +Stress radiographs/ dynamic fluoroscopy
Synovial fistula	Excessive débridement of extensor origin and joint capsule	Painful swelling/bogginess over RC joint; +MR arthrogram
Adventitial bursa formation	Subcutaneous bursa formation over LE due to local inflammation	Painful subcutaneous swelling, bogginess over lateral elbow
Nerve injury	Painful neuroma of SRN or PCNF	Paresthesia/dysesthesia along nerve distribution; +response to local anesthetic injection
Low-grade infection	*Cutibacterium acnes*	Painful swelling of RC joint Infectious laboratory studies (CRP, ESR)

LUCL, lateral ulnar collateral ligament; *MR*, magnetic resonance; *RC*, radiocapitellar; *LE*, lateral epicondyle; *SRN*, superficial radial nerve; *PCNF*, posterior cutaneous nerve of forearm; *CRP*, cardiac reactive protein; *ESR*, erythrocyte sedimentation rate.

adventitial bursal formation are also common following an overly aggressive débridement.[38,39] The formation of heterotopic ossification at the lateral elbow following surgical treatment has also been reported.[21] Low-grade, indolent infections (eg, *Cutibacterium acnes*) can present insidiously after surgery and cause persistent pain in these patients as well.[28]

The PCNF is a cutaneous branch of the radial nerve crossing approximately 1.5 cm anterior to the lateral epicondyle on the fascia of the BR; neuroma following iatrogenic injury may be a cause of postoperative pain typically associated with paresthesia and dysesthesia distal to the incision.[19] Similarly, painful neuroma following injury to the superficial radial nerve (SRN) can occur as well; the superficial nature of the SRN as it branches off the radial nerve proper at the radiocapitellar joint predisposes it to injury (eg, crush injury from aberrant retractor placement, injury during concomitant radial tunnel release).[20] These patients typically have paresthesias and/or dysesthesias distal to the incision. Diagnosis can be confirmed by symptom relief following diagnostic injection in this area will have signs of a nerve injury on exam.

Further studies may be warranted depending on the history and exam of patients with type 2 treatment failure. The presence of instability on exam concerning for LUCL injury can be further evaluated with stress radiographs. An MR arthrogram can be helpful to identify the presence of ligamentous disruption, bursal and/or capsular defects. Diagnostic injections at the lateral epicondyle and arcade of Frohse can be helpful to distinguish lateral epicondylitis from radial tunnel syndrome.

Type 3 failure (mixed symptom complex)

Type 3 surgical treatment failure refers to a combination of types 1 and 2 treatment failure. These patients will have a complex/mixed presentation and, in our experience, are most frequently due to (1) unrecognized coexisting radial tunnel syndrome (type 1A failure), (2) inadequate débridement of ECRB tendinosis in its entirety during the initial procedure (type 1B failure) and (3) iatrogenic soft tissue defects in areas of overaggressive débridement and/or repetitive steroid injection (type 2 failure). Due to the combined symptom complex, identifying each underlying etiology for these patients can be difficult but is critical in successful revision treatment.

Evaluation

Careful history and examination are critical in the evaluation of patients who have failed surgical treatment, including patient motives (eg, secondary gain). Time is an important consideration when evaluating these patients; it should be determined whether a sufficient length of time has elapsed since surgery (ie, greater than 6-9 months) and if the patient has been compliant with the postoperative rehabilitation regimen. Assessment of aggravating and relieving factors may be helpful as well; night pain, for example, may be suggestive of osteoarthritis, osteochondritis dissecans (OCD) lesions of the capitellum or septic arthritis.[39]

Physical examination should be performed keeping failure types in mind and should include inspection, palpation, evaluation of elbow motion, strength, stability, and overall alignment. Swelling or fullness over the lateral elbow may be suggestive of an underlying infection or a synovial fistula. Thorough examination for any concomitant signs of infection should be performed as postoperative pain medication and/or oral antibiotics can mask this presentation. Precise localization of the patient's pain on exam is important to confirm a diagnosis of LE. For patients with type 1 failure, tenderness should be localized over the common extensor origin just anterior to the lateral epicondyle; patients with PIN irritation due to radial tunnel syndrome will have neuropathic pain in the proximal-lateral forearm over the arcade of Frohse, 3-5 cm distal to the lateral epicondyle. Patients will classically not have pain with resisted wrist extension but will have pain reproduced with resisted supination and long finger extension.

Diagnostic nerve blocks

Selective injection of local anesthetic can be useful both diagnostically and therapeutically in the initial evaluation of these patients, particularly for patients with type 1 or 2 failure. Because the pain in LE is thought to be related to repetitive neuro-microtrauma, resolution following injection over the lateral epicondyle is useful to confirm this as the diagnosis. The associated nerve endings are terminal branches of the posterior branch of the PCNF and can be blocked with 1-2 mL of local anesthetic injected 2-3 cm proximal to the lateral epicondyle.[18,66] Similarly, pain relief following injection in other locations can suggest alterative diagnoses such as painful postoperative neuroma (eg, posterior cutaneous nerve of forearm, superficial radial sensory nerve), or an unrelated diagnosis if there is a lack of improvement any improvement following nerve block (type 1 failure).

For the latter, other injection sites may be considered to identify alternative diagnoses. An injection over the PIN can be performed near the proximal supinator muscle, with a PIN palsy and resolution of pain after injection confirming a diagnosis of radial tunnel syndrome; care must be taken to avoid diffusion of local anesthetic to the lateral epicondyle as this can confuse the clinical picture.[41] Similarly, if dynamic compression/entrapment of the LACN is suspected, selective injection can be performed by near the intersection of a line along epicondylar axis and the lateral edge of the biceps tendon.[7,19] A positive response to subcutaneous nerve block of the posterior cutaneous nerve branches of the forearm can also support local denervation if revision surgery for LE is required.[50] Intraarticular injections with 4-5 mL of lidocaine through the posterolateral soft spot of the elbow can be helpful to identify intraarticular pathology as the source of pain.

Pain localized to the radiocapitellar joint is more suggestive of intraarticular pathology such as OCD lesions of the capitellum, osteoarthritis, and synovial plica. These diagnoses will be associated with decreased motion and/or mechanical symptoms. Pain throughout range of motion is consistent with generalized osteoarthritis while pain at terminal motion (eg, painful clicking at terminal elbow extension and forearm supination) is more consistent with impingement from osteophytes or a hypertrophic synovial plica.[5]

Posterolateral instability of the elbow should be ruled out in patients with lateral elbow pain. Instability may be associated with patients suffering from LE due to excessive corticosteroid injections or iatrogenic injury to the LUCL, or type 3 treatment failure. The presence of preexisting cubitus varus, elbow trauma or previous elbow surgery should be assessed. Lateral elbow stability is evaluated on exam with varus stress and lateral pivot shift testing, with pain, laxity and/or apprehension indicating a positive test. In cases where the physical exam findings are equivocal, stress radiographs or fluoroscopy of the elbow can be helpful to identify laxity.

Arthroscopic treatment has been proposed for revision surgery, with the proposed benefit of the ability to assess for concomitant pathology. Certainly, patients with imaging or physical exam findings suggestive of intraarticular pathology warrant an examination of the joint, and arthroscopy can provide a clear advantage in such cases. Recent studies have proposed that subclinical evidence of symptomatic minor instability at the lateral elbow can only be seen during arthroscopy.[3,25] Arrigoni et al demonstrated that 48% of patients in their cohort with recalcitrant lateral epicondylitis but no clinical signs of instability displayed intraarticular findings consistent with lateral ligamentous patholaxity.[2] However, multiple studies which have compared arthroscopy to open treatment without examination of the joint demonstrated similar outcomes.[33,35,48,60] As such, patients are known to improve regardless of whether these common intraarticular findings are addressed. This seems to indicate that intraarticular signs without associated clinical findings are simply incidental and unrelated to the patient's pain. Arthroscopic treatment is discussed further in the "Treatment" section, below.

Imaging and diagnostic studies

Plain film radiographs should be obtained for initial imaging evaluation of these patients and may be useful to reveal OCD lesions of the capitellum, loose bodies, arthritic change or a fat pad sign suggestive of an effusion. In cases of long-standing LE, calcifications may be visible over the ECRB tendon insertion may be present.

Stress radiographs can be useful to evaluate instability if LUCL insufficiency is suspected. Advanced imaging with MR arthrogram is perhaps the most useful imaging modality to detect any effusion, ligamentous injury, capsular defect, synovitis or synovial fistula or other soft tissue pathology.[32] MR imaging evaluation is not routinely utilized for diagnosing lateral epicondylitis, but may show increased signal intensity over the ECRB and/or common extensor origin consistent with edema and tendinosis; findings, however, are inconsistent and have been shown to persist postoperatively without reliably correlating to clinical severity.[54]

Ultrasonography (US) can be a useful tool for evaluating these patients and can be effective in identifying structural changes to the ECRB tendon (eg, thickening, degeneration, tearing), bony irregularities and/or calcium deposits associated with lateral epicondylitis. Doppler US is effective for detecting neovascularization, and is highly sensitive for diagnosing LE[63]; the absence of greyscale US changes and neovascularization on doppler US effectively rules out a diagnosis of LE, and alternative diagnoses should be considered.[63]

Electrodiagnostic testing can be helpful if certain neurologic etiologies (eg, brachial plexopathy, cervical radiculopathy, lateral antebrachial cutaneous nerve entrapment) are suspected, though these tests are not always able to detect the presence of nerve irritability, such as radial tunnel syndrome.[39,41]

Treatment

Timing of any subsequent surgical intervention is an important consideration. Resolution of symptoms following successful primary surgical intervention for lateral epicondylitis is expected within 3-4 months of surgery.[39] Although lingering symptoms can be expected to improve for some patients, symptoms are unlikely to improve if present after 1 year postoperatively.[49]

For patients with type 1A failure, optimal treatment requires accurate identification of the underlying pathology. Surgical intervention may be appropriate for certain pathologies, such as compression neuropathies including radial tunnel syndrome, and entrapment of LACN or posterior brachial cutaneous nerve.[41,50] Concern for symptomatic intraarticular pathology (eg, OCD lesions, painful osteophytes, symptomatic synovial plicae, loose bodies) or instability (eg, medial collateral ligament instability, posterolateral rotatory instability) warrants further arthroscopic evaluation and management. Chronic exertional compartment syndrome of anconeus or the extensors may require fasciotomy.[27]

Type 1B failure is most frequently thought to occur after incomplete resection of the pathologic tissue. Making this diagnosis requires a sufficient amount of time has passed (ie, 6-9 months since surgery) and alternate or coexisting pathologies have been ruled out. A positive response to a local anesthetic injection over the lateral epicondyle indicates the patient may be a good candidate for revision surgery. An open procedure with a more thorough débridement of the common extensor origin is recommended in the revision setting often combined with an anconeus muscle flap to promote healing and address frequent steroid-induced soft tissue defect.[16,37,44,59]

Patients with types 1C and 1D failure should be approached cautiously. Inadequate postoperative rehabilitation can be responsible for lingering elbow pain following surgery, and often these patients can continually improve with several weeks of stretching and strengthening exercises. In patients with underlying psychiatric diagnoses contributing to their physical symptoms, a multidisciplinary approach should be considered rather than revision surgery. Similarly, in patients with secondary underlying motives or worker's compensation cases, revision surgery is unlikely to yield much benefit in comparison to the risks of additional surgery.

For patients with type 2 failure, or new symptoms postoperatively, the pathology may be addressed as soon as it is identified. Posterolateral rotatory instability of the elbow due to iatrogenic LUCL injury should be surgically reconstructed. Synovial fistula and adventitial bursal formation typically require revision surgical débridement with possible closure of the capsular defect or anconeus muscle flap coverage; postoperative infections are treated with revision débridement as well an appropriate postoperative antibiotic regimen with empiric coverage of common skin flora as well as methicillin-resistant staphylococcus species. Postoperative painful neuroma of the SRN of PCNF can be addressed with neurectomy and excision of scar tissue.[19] Heterotopic bone formation may treated with resection if symptomatic or functionally limiting.[21]

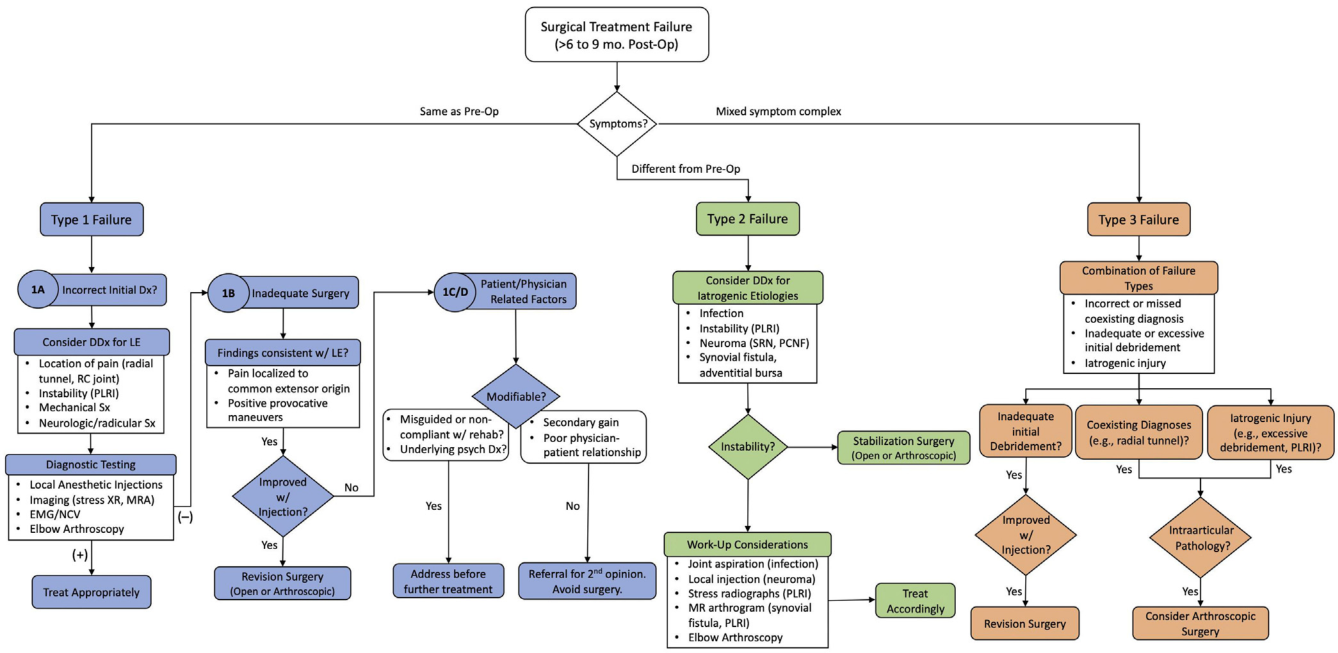

Figure 1 A general method for approaching patients following failed surgical treatment of lateral epicondylitis, with some important considerations for evaluation, diagnosis and treatment of these patients. Thorough knowledge of the differential for lateral epicondylitis and possible iatrogenic pathologies introduced from surgery is important to determine the type of failure, making an accurate diagnosis and providing appropriate treatment. *RC*, radiocapitellar; *EMG*, electromyographyl; *NCV*, nerve conduction velocity; *LE*, lateral epicondylitis; *SRN*, superficial radial nerve; *PCNF*, posterior cutaneous nerve of forearm; *MR*, magnetic resonance.

Arthroscopic approach

An arthroscopic approach to treatment has been described and is typically performed utilizing a proximal anteromedial portal, proximal anterolateral portal and direct anterolateral portal. Diagnostic arthroscopy and synovectomy is performed using a small (3 mm) shaver along the anterolateral capsule and lateral gutter; impinging radiocapitellar plica, capsular tearing and/or chondral pathology can be addressed at this point as well.[46] Concomitant elbow laxity can be addressed via capsular plication, performed with a suture anchor over the anterolateral aspect of the capitellum has been described for treatment of symptomatic minor instability of the elbow.[2]

Extra-articular access to the common extensor origin is obtained via blunt dissection with the shaver to develop the plane between the anterolateral capsule and overlying extensor tendons; an anterolateral capsulotomy can then be performed for a window through which to address any common extensor tendon pathology.[46] Anatomic insertional release procedure has been described for the treatment of recalcitrant lateral epicondylitis as well, and involves the identification and anatomic release of the bony insertions of ECRB and EDC tendons.[46] Excision of calcification, small osteophytes, and tendon insertional drilling have been described as well. Postoperatively patients are placed in a bulky soft dressing with a sling, transitioned to a portal dressing on postoperative day 3 or 4, and cleared for immediate full with restrictions on heavy lifting for 12 weeks.[46]

Expected outcomes

Outcomes reported in the literature following revision surgery for lateral epicondylitis are generally very good, with greater than 80% success rates. Inadequate or incomplete excision of tendinosis is common, reported in as many as 97% of cases. Revision surgery including a more thorough resection of pathologic tissue and repair of extensor aponeurosis has been reported with success rates of 83%.[44] For revision cases in which a wide zone of tendinosis requires excision, anconeus muscle flap transposition has been reported with favorable outcomes, with patient satisfaction rates of 95% in one series.[36,52,56]

Author's preferred approach

Our approach for evaluating and treating these patients is summarized in Fig. 1. In practice, we've found failed surgery is most commonly represented by type 3 failure, with multiple elements contributing to continued symptoms. These patients will have a complex/mixed presentation and, in our experience, are most frequently due to (1) unrecognized coexisting radial tunnel syndrome (type 1 failure), (2) inadequate débridement of ECRB tendinosis in its entirety during the initial procedure (type 2 failure) and (3) iatrogenic soft tissue defects in areas of overaggressive débridement and/or repetitive steroid injection (type 3 failure).

For patients with a symptom complex consistent with a combination of multiple failure types, and who meet the criteria for consideration of revision surgery, we recommend a surgical technique to address the most common underlying pathology in these patients (Fig. 2). Intraoperative fluoroscopy is utilized to evaluate stability prior to any intervention at the lateral epicondyle. Any evidence of posterolateral rotatory instability is addressed with LUCL reconstruction. We do not recommend routine arthroscopic evaluation of the joint unless there is evidence of intraarticular pathology on physical examination or advanced imaging. Revision débridement with exploration of the entire extensor origin and excision of all pathologic tissue is performed in conjunction with PIN decompression and vascularized anconeus muscle rotational flap coverage to address any tendinous defects.

An open technique should be utilized to better assess the extent of pathologic tissue and allows visualization of the entire course of the PIN as it traverses the radial tunnel, ensuring adequate decompression. In these revision cases, prior surgical and corticosteroid treatments combined with a thorough repeat débridement will frequently result in a large poorly vascularized defect at the

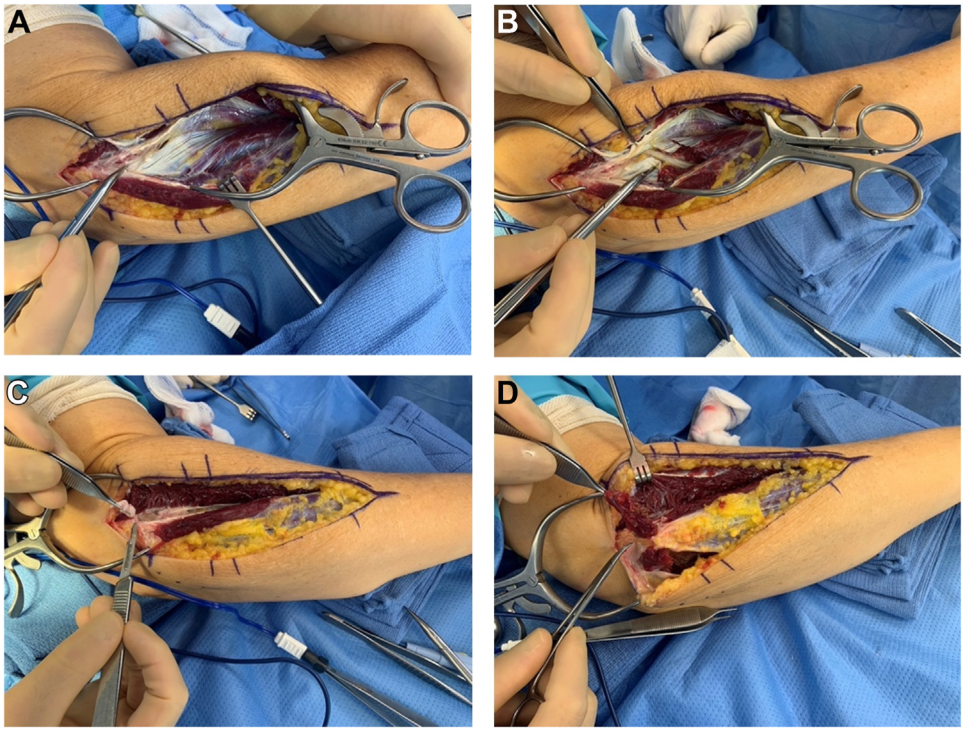

Figure 2 Clinical photos demonstrating a proposed salvage treatment technique after failed primary surgery for lateral epicondylitis. (**A**) The fascial septum between the ECRB and EDC is divided to expose the underlying supinator muscle. (**B**) The PIN is decompressed and visualized in its entirety as it passed through the supinator muscle and radial tunnel. (**C**) All residual pathologic tissue is identified over the common extensor origin and sharply excised. (**D**) The inferior border of the anconeus muscle is subperiosteally elevated off the ulna and rotated into the tissue defect created at the site of pathologic tissue débridement. *ECRB*, extensor carpi radialis brevis; *EDC*, extensor digitorum communis; *PIN*, posterior interosseous nerve.

lateral epicondyle. A vascularized anconeus muscle flap provides a soft tissue cushion and promotes healing through increased blood supply to the area. This operation has been met with good success in treating these challenging presentations.[59]

Conclusion

Patients who have undergone surgical treatment for lateral epicondylitis are expected to have improvement and near complete symptom resolution by 6-9 months postoperatively. Surgical treatment failure requires a thorough evaluation to determine the failure type(s) to appropriately guide further workup and optimize the treatment plan for each patient. Any revision surgery should be planned to address all coexisting pathologies. A surgical technique involving revision ECRB débridement with concomitant PIN/radial tunnel release and anconeus rotational flap coverage of soft tissue defects is an effective means of addressing the most common etiologies for treatment failure in our experience.

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