Review Article

Distal Triceps Rupture

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Abstract

Distal triceps rupture is an uncommon injury. It is most often associated with anabolic steroid use, weight lifting, and laceration. Other local and systemic risk factors include local steroid injection, olecranon bursitis, and hyperparathyroidism. Distal triceps rupture is usually caused by a fall on an outstretched hand or a direct blow. Eccentric loading of a contracting triceps has been implicated, particularly in professional athletes. Initial diagnosis may be difficult because a palpable defect is not always present. Pain and swelling may limit the ability to evaluate strength and elbow range of motion. Although plain radiographs are helpful in ruling out other elbow pathology, MRI is used to confirm the diagnosis, classify the injury, and guide management. Incomplete tears with active elbow extension against resistance are managed nonsurgically. Surgical repair is indicated in active persons with complete tears and for incomplete tears with concomitant loss of strength. Good to excellent results have been reported with surgical repair, and very good results have been achieved even for chronic tears.

Distal rupture of the triceps tendon is rare and is among the least common of reported tendon injuries. In a review of 1,014 tendon ruptures, 0.8% constituted triceps tendon injuries.[1] In a 6-year span, only 21 cases of triceps rupture were recorded in the National Football League.[2] There is a male predominance of roughly 2 to 1 in all age groups.

Most reports have associated triceps tendon rupture with anabolic steroid use and weight lifting.[3] Professional football players may be at a higher risk for rupture than the general population, possibly because of the training regimen, the potential for steroid use, and the violent nature of the sport itself.[2] Other factors that may play a role in distal triceps rupture include local steroid injection,[2-4] metabolic bone diseases (ie, hyperparathyroidism), renal osteodystrophy, olecranon bursitis,[5] hypocalcemic tetany, Marfan syndrome, osteogenesis imperfecta, rheumatoid arthritis, and type I diabetes.[6] Persons with chronic renal disease generally possess elevated levels of parathyroid hormone, which stimulates osteoclastic activity and bone resorption. Such circumstances may ultimately predispose to tendon rupture. Adolescent persons with incompletely fused or recently fused physes are also susceptible to triceps tendon rupture.[7]

Anatomy

The triceps brachii is a pennate muscle with three heads: lateral, long, and medial. The triceps extends the entire length of the posterior humerus and is the only muscle located in the posterior compartment of the arm. The triceps is innervated by the radial nerve (C6-C8). The lateral head originates from the posterior humerus between the teres mi-

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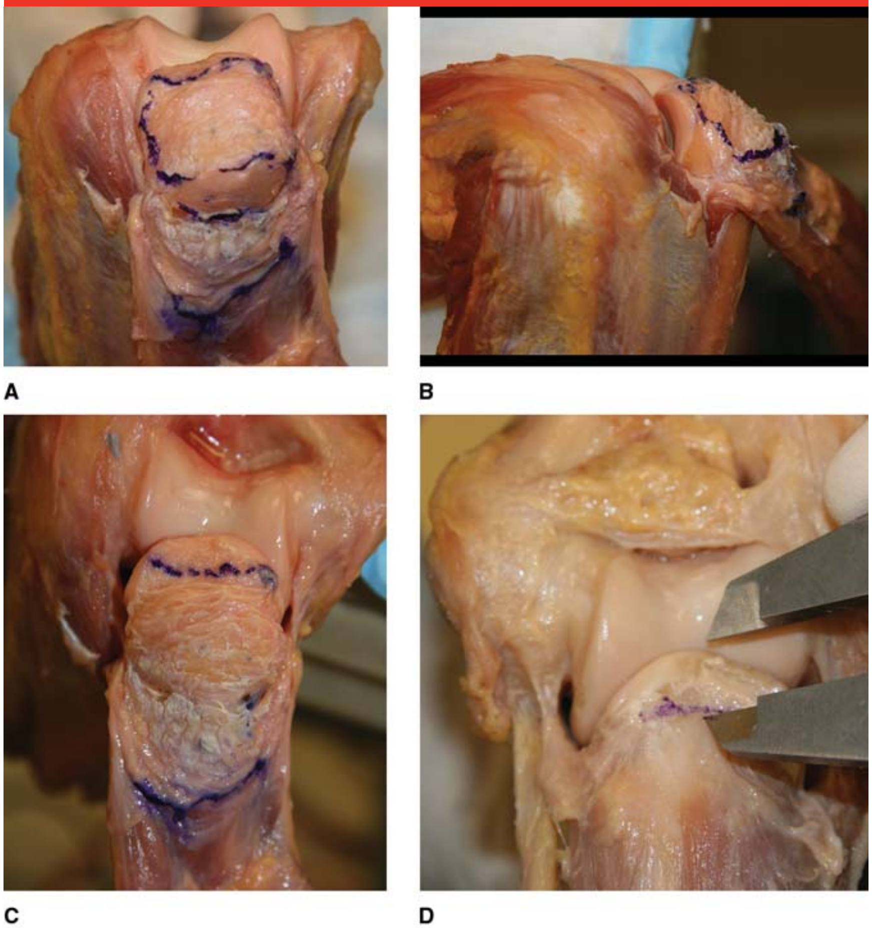
Figure 1



A, AP photograph of a cadaveric elbow. The triceps footprint on the olecranon is outlined with marker. In an anatomic study, the footprint was found to measure 466 mm^2.[8] **B,** Lateral view of the triceps footprint. Note the distance of the proximal portion of the footprint to the articular surface. The footprint wraps around the tip of the olecranon distally. **C,** AP view of the triceps tendon as it is released from its footprint. The large area of attachment on the tendon itself is clearly visualized (marked in purple). **D,** The distance from the tip of the olecranon to the proximal portion of the footprint is measured with a digital caliper. In this specimen, that measurement is 12 mm.

nor insertion and the superior aspect of the spiral groove, the lateral border of the humerus, and the lateral intermuscular septum. The long head originates at the infraglenoid tuberosity, where the scapula blends with the shoulder capsule. The medial head originates on the posterior humerus distal to the spiral groove, medial humerus, and medial intermuscular septum.

Figure 2

The lateral triceps expansion is marked with a caliper. The olecranon and the triceps tendon proper are dotted with marker.

The triceps insertion is not a focal point on the olecranon. Rather, the distal triceps inserts over a wide area or footprint (Figure 1, A through C). In a cadaver study, the footprint was found to start 12 mm distal to the tip of the olecranon (Figure 1, D) and to blend with the posterior capsule; the footprint measured 466 mm^2.[8] The width of the distal triceps tendon insertion ranges from 1.9 to 4.2 cm[9] and consists of the triceps tendon proper (ie, the confluence of tendon from all three heads inserting on the olecranon) and the lateral triceps expansion (Figure 2).

The insertion of the medial aspect of the triceps expansion is located on the posterior crest of the ulna, adjacent to the medial head. Hypertrophy of this muscle can cause ulnar nerve impingement, a condition that

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is occasionally seen in weight lifters. Laterally, the triceps expansion has a wide insertion on the fascia of the extensor carpi ulnaris muscle and the deep fascia of the anconeus muscle.[10] Because of its wide lateral insertion, the triceps expansion often requires concomitant repair in cases of triceps rupture. The triceps expansion also inserts on the antebrachial fascia of the forearm distally.

The distal portion of the medial head has been shown to have a distinct muscle belly and a tendon that is separate from and deep to the common triceps tendon. However, histologic analysis has revealed that the distal expansion of the medial head becomes confluent with the common triceps tendon as they insert onto the olecranon.[10] This distinct insertion may provide the rationale for a distinct medial (proximal) repair to achieve anatomic restoration of the medial insertion, along with primary repair of the lateral head insertion.

The main function of the triceps is to extend the forearm at the ulnohumeral joint. However, because of its origin at the infraglenoid tuberosity, it is believed that the long head of the triceps may also aid in arm adduction and extension. The overall muscle-tendon length of the triceps critically affects its motor function. Biomechanical studies show that as little as 2 cm of shortening between origin and insertion causes a 40% loss of extension strength.[11]

Presentation and Physical Examination

The most common mechanism of injury is a sudden eccentric load applied to a contracting triceps muscle, such as during weight lifting or from a fall onto an outstretched hand. Lacerations and open injuries can also cause distal triceps rupture. A direct blow may result in such injury, but this finding is less common. Higher-energy mechanisms, such as a motor vehicle accident or a fall from a height, may also cause concomitant ulnar nerve injury and compartment syndrome; these possible causative factors must not be overlooked.[12,13]

Regardless of mechanism of injury, triceps tendon ruptures are usually seen at the osseous insertion.[12] However, there have been reports of rupture within the muscle belly[14] and tears at the myotendinous junction.[6,15] Fatigue failure of the muscle has been suggested as a mechanism for intramuscular tears.[14]

Patients present with pain and swelling over the posterior aspect of the elbow. Physical examination reveals tenderness to palpation, swelling, and ecchymosis. A palpable defect proximal to the olecranon can also be apparent and may confirm the clinical diagnosis (Figure 3). In the acute stage, a defect may not be palpable secondary to swelling, body habitus,[16] or the degree of rupture.

The inability to actively extend against resistance is a sign of complete rupture. However, the converse is not always true—that is, not all complete tears result in the inability to actively extend against resistance. This finding is likely secondary to an intact lateral expansion or a compensating anconeus muscle. Thus, the diagnosis of a triceps tear can be confounding. In fact, one study showed that almost 50% of acute triceps ruptures were initially misdiagnosed.[12] In the setting of chronic tears, the most common complaints are of pain and weakness with extension.

A modified Thompson squeeze test, similar in execution to that performed on the Achilles tendon, has been reported as a potential clinical diagnostic tool.[17] During this passive extension test, the patient lies prone with the elbow at the edge of the examination table. The forearm is allowed to hang down over the edge of the table so that the elbow is flexed 90° in a relaxed position. The examiner firmly squeezes the triceps muscle. The patient with an incomplete triceps rupture will be able to extend the elbow against gravity.[18] The patient with complete disruption of the triceps proper and lateral expansion will not be able to extend the elbow against gravity.

Figure 3

Clinical photograph of a palpable defect proximal to the olecranon in a patient with distal triceps rupture. The abrasion just distal to the olecranon indicates the area of impact during injury.

Fundamental differences between the Thompson squeeze test used on the Achilles tendon and the modification for evaluating the triceps could affect the utility of this evaluation tool. The long lever arm of the forearm distal to a triceps tear makes distal resistance much higher than that seen with the short lever arm of the foot distal to an Achilles tear. The bulk of the triceps is relatively small compared with that of the gastrocnemius-soleus complex, which makes it more difficult to tightly squeeze the triceps muscle.

It may be challenging to accurately see and/or quantify the amount of residual motion. The amount of forearm motion required to declare the modified Thompson squeeze test to be positive or negative is not known. The presence of some extension on

Figure 4

Lateral radiograph of the elbow in a patient with a distal triceps rupture. A flake of bone has avulsed off the olecranon (arrow).

Figure 5

T1-weighted sagittal magnetic resonance image of partial triceps tendon rupture. The arrow points to partial attachment of the tendon. (Reproduced with permission from Mair SD, Isbell WM, Gill TJ, Schlegel TF, Hawkins RJ: Triceps tendon ruptures in professional football players. *Am J Sports Med* 2004:32:431-434.)

Figure 6

Sagittal fat-suppressed T2-weighted spin-echo magnetic resonance image of the elbow demonstrating a large fluid-filled gap (arrowhead) between the completely torn and retracted distal triceps tendon (arrow) and the olecranon process. (Reproduced with permission from Kijowski R, Tuite M, Sanford M: Magnetic resonance imaging of the elbow: Part II. Abnormalities of the ligaments, tendons, and nerves. *Skeletal Radiol* 2005:34:1-18.)

this test does not rule out the possibility of complete rupture of the tendon proper. It may be that the triceps expansion is still intact and is responding to the passive extension squeeze test.

Diagnostic Imaging

AP and lateral radiographs of the involved elbow should be obtained at presentation. The lateral radiograph may show the presence of flecks of avulsed osseous material from the olecranon (ie, flake sign), which is almost always pathognomonic of a triceps tendon rupture[19] (Figure 4). Plain radiographs of the ipsilateral wrist joint should be strongly considered when rupture is clinically suspected.

CT can be helpful in excluding associated osseous injuries, such as radial head fracture and distal radius fracture, both of which have been reported to occur in association with triceps tendon rupture.[15,20] MRI and ultrasonography are useful in difficult cases in which it is hard to distinguish whether an injury is partial or complete.[21-23] Sagittal MRI accurately demonstrates the integrity of the triceps tendon. Partial rupture is characterized by a small fluid-filled defect within the distal triceps tendon, with edema also affecting the surrounding subcutaneous tissues. The defect appears as a bright area on T2-weighted images (Figure 5). In one study, 8 of 10 injuries classified as partial triceps ruptures predominantly involved the medial side of the tendon insertion.[2] Complete rupture of the triceps tendon is characterized by a large fluid-filled gap (ie, paratricipital edema) between the distal end of the triceps tendon and the olecranon process with[23] (Figure 6).

Classification

No formal classification system for distal triceps rupture has been established. We describe these injuries based on results of imaging studies (ie, ultrasonography, MRI), as well as on intraoperative findings. Anatomic factors important to the description include the degree of the tear (ie, complete, partial, intact) and the location of the tear (ie, muscle belly, musculotendinous junction, tendinous insertion, avulsion off bone). It is also important to note the integrity of the lateral expansion (intact versus torn).

Several nonanatomic factors must be considered when managing triceps ruptures. These include the functional and medical status of the patient as well as the chronicity and atrophy of the muscle-tendon unit. Based on these factors, the surgeon can determine the most appropriate treatment to administer.

Management

Management of triceps tears is generally guided by tear location and by the functional extension strength of the extremity. The amount of extension strength is important because, in triceps injuries, complete anatomic ruptures do not necessarily cause full loss of function.[24] The intact lateral triceps expansion or anconeus may compensate adequately for the loss of triceps function. The patient with complete anatomic rupture but with some function remaining should be identified and treated according to goals set specifically for that patient.

Treatment decisions must be individualized based on the patient's medical and functional status. For example, a partial tear at the tendinous insertion may be managed nonsurgically in a debilitated, elderly patient, whereas the same tear may be managed surgically in a highly functioning athlete.[24] Healthy older persons require normal triceps function for activities of daily living, such as getting out of a chair.

In general, any tear <50% can be treated nonsurgically with satisfactory results.[7] Tears >50% are treated nonsurgically in the sedentary person; however, in the active person, surgical intervention may be appropriate.[2,24] Complete tears are generally managed surgically.[12,16,19]

Nonsurgical

Partial tears at the muscle belly, musculotendinous junction, and tendon insertion with insignificant loss of extension strength can be managed nonsurgically.[25] Nonsurgical management consists of splint immobilization for approximately 4 weeks at 30° of flexion.[7,16,18,25]

Muscle belly tears tend to heal with scar tissue rather than with newly regenerated muscle. Outcomes are relatively similar with these tears regardless of treatment. Thus, even complete tears within the muscle belly can be managed nonsurgically. Nonetheless, surgical intervention of muscle belly tears with direct suturing has been reported[26] and may be an option for the young, highly functioning athlete who desires to return to sports. No objective results for surgical intervention of muscle belly tears have been reported other than the few case reports demonstrating return to previous level of activity.

Good results have been reported with nonsurgical management, with published studies indicating a return to preinjury level of function.[2,18,27] One report of bilateral partial rupture at the tendinous insertion in a weight lifter showed quantitatively normal function at 41 weeks, but fatigue with combined lifting at 55 weeks.[25] A report of a patient who was treated nonsurgically for an intramuscular tear of the triceps demonstrated no acute muscle weakness (performing one push-up).[14] However, fatigue was present after endurance exercises, especially after periods of relative inactivity. This finding was still present 9 years after injury.

Mair et al[2] reported that of 10 professional football players with partial tears, 6 healed with nonsurgical treatment and experienced no residual pain or weakness. Three players were treated with bracing for the remainder of the season, after which they received surgical treatment to correct residual pain and weakness. One player sustained a complete rupture on return to play despite bracing. The mixed results of this study demonstrate that treatment should be individually tailored to the needs of each patient and should not be determined by the type of tear only.

Surgical

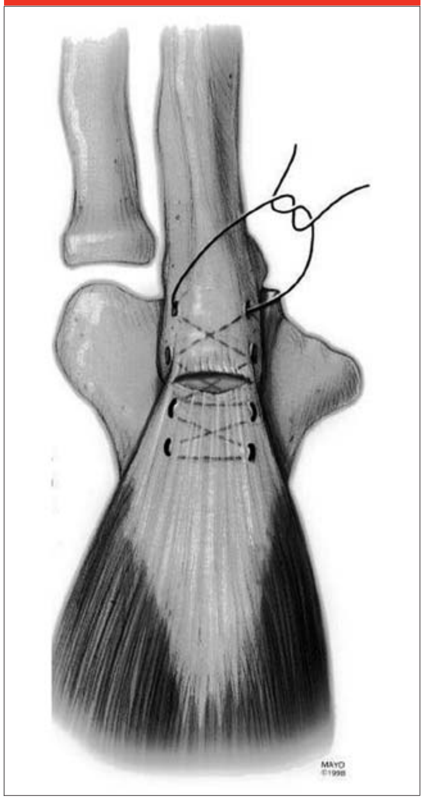
Early primary repair is appropriate for acute, complete triceps tear at the tendinous insertion with significant loss of triceps strength.[24] Surgery is typically performed in patients with such injury who have symmetric or reduced, although still functional, strength. However, medical status and activity level need to be considered. When possible, repair should be performed within 2 weeks, although primary repair has been described as late as 8 months after injury.[28] Surgical treatment is also appropriate for partial tears at the myotendinous junction or tendon insertion in patients who are highly active or who have failed nonsurgical treatment.

Surgical repair involves identification of the level of tendinous rupture with primary reattachment of the avulsed triceps tendon to the olecranon. Most of the recently published repair techniques describe use of the Bunnell or Krackow whipstitch technique, which includes placement of nonabsorbable sutures through the tendon. The sutures are then passed through transosseous drill holes in the olecranon and are tied over a bone bridge[12,29-31] (Figure 7). The transosseous drill hole technique can also be used in skeletally immature patients.[32] Other repair techniques include direct tendon repair to a periosteal flap raised from the olecranon. Intraosseous suture anchors may be used to firmly seat the triceps tendon against the olecranon.[15,19]

An alternative technique involves the creation of three parallel drill holes.[33] The drill holes must be angled in an oblique and dorsal fashion from proximal to distal through the olecranon, taking care to avoid the joint surface. Using nonabsorbable sutures, two parallel Krackow-type stitches are placed through the detached tendon, leaving four tails. The two middle tails are passed through the central hole, and the medial and lateral tails are passed through the medial and lateral holes, respectively (Figure 8). The arm is held in approximately 35° to 40° of flexion for tensioning, and the sutures are tied.

A subset of patients presents with a

Figure 7

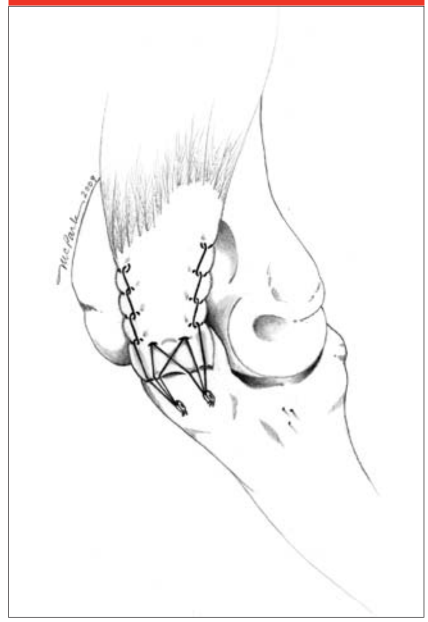


The Bunnell suturing technique for primary repair of the triceps tendon. The sutures are passed through transosseous cruciate drill holes made in the olecranon. (Reproduced with permission from the Mayo Foundation of Medical Education and Research, Rochester, MN.)

Figure 8

Intraoperative photograph demonstrating triceps tendon repair. Krackow stitches were placed through the detached tendon and passed through three parallel drill holes in a proximal-to-distal direction, angling dorsally. (Reproduced with permission from Sierra RJ, Weiss NG, Shrader MW, Steinmann SP: Acute triceps ruptures: Case report and retrospective chart review. *J Shoulder Elbow Surg* 2006:15:130-134.)

Figure 9



Triceps suture bridge construct for distal triceps repair. Four suture anchors form a "bridge" over the entire tendon footprint. (Illustration by Maxwell C. Park, MD.)

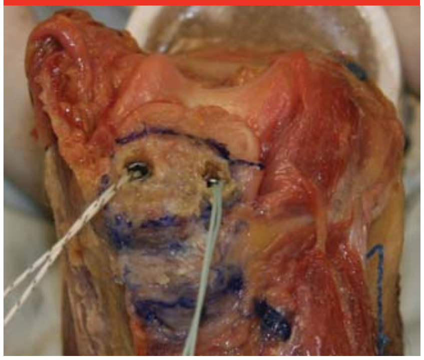
torn triceps in which a portion of the olecranon has avulsed with the attached tendon on the bony fragment. This is more commonly seen in skeletally immature patients whose ossification center of the olecranon has yet to fuse. In these cases, the olecranon fracture can be reduced and fixed either with a screw and washer or with stainless steel wires via a tension-band construct.[17] Smaller fragments can be reduced and secured with suture fixation or can be excised with primary reattachment of the tendon to the olecranon.[34] The use of wire suture (as opposed to braided nonabsorbable suture) has been reported in the literature.[35,36] However, there is a risk of bursitis postoperatively with this construct, and it is mentioned here for historical perspective only.

Triceps rupture at the myotendinous junction can be challenging to manage because of the poor quality of tissue available for primary repair. Wagner and Cooney[6] described the use of a V-Y triceps tendon advancement technique in which strength was augmented by interweaving an autologous plantaris tendon with the remaining proximal and distal triceps tissue. Postoperative care included immobilization for 6 weeks, followed by use of a static elbow brace for protected motion for an additional 6 weeks. Strength training was instituted at 12 weeks postoperatively.

Authors' Preferred Technique

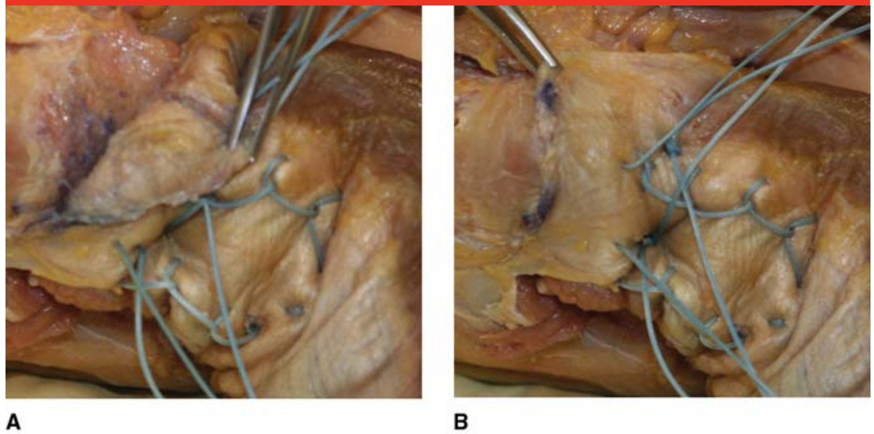
Anatomic (ie, footprint) repair of the rotator cuff, Achilles tendon, and biceps tendon has gained in popularity because it is believed that restoring preinjury anatomy may play a role in postoperative rehabilitation and function. A biomechanical study showed the triceps footprint area to be larger than a point insertion (triceps footprint area, 466 mm^2).[8] In this study, footprint repair yielded the most anatomic restoration of distal triceps ruptures, and significantly less motion occurred at the repair site under cyclic loading ($P < 0.001$). Based on our enhanced understanding of the triceps footprint anatomy and on the biomechanical basis for repair shown in our study, our preference is to perform anatomic triceps tendon footprint repair. We do this using suture anchors to create a suture bridge (Figure 9). This technique

Figure 10



Photograph of a cadaver elbow demonstrating placement of a proximal row of suture anchors in distal triceps repair. The purple markings indicate the triceps footprint on the olecranon. The suture anchors rest just distal to the proximal portion of the footprint.

Figure 11



A, AP photograph of a cadaver elbow demonstrating the distal free triceps tendon (held up and reflected by Adson forceps) after placement of the proximal row of suture anchors into the proximal portion of the triceps tendon. **B,** The distal free triceps tendon is laid down on its native anatomic footprint. Press-fit anchors will be placed distal to the footprint of the laid-down tendon. The Krackow stitches have been partially removed from the triceps tendon for the purpose of this illustration.

restores preinjury anatomy, thereby restoring a wider area of tendon-bone contact.

The patient is placed in the lateral decubitus position. The upper extremity is draped free and is hung over an arm rest so that the elbow may be manipulated comfortably. A tourniquet is not used because of the risk of inhibiting triceps tendon mobilization during repair. A posterolateral approach to the elbow is performed so that wound healing is not hindered by postoperative range of motion (ROM), considering that the tip of the olecranon becomes more prominent with elbow flexion. Dissection is carried proximally to identify the extent of tendon rupture. The exposure should be made distal enough to visualize the entire insertion of the tendon onto the olecranon. The incision should extend approximately 2 cm distal on the shaft of the olecranon. The bony insertion site is excoriated to remove any interposed soft tissue. A locking-type whipstitch (ie, Krackow) is placed on the tendon, with four throws placed on each side.

Two suture anchors are placed on the proximal end of the footprint site, one on the lateral edge and the other on the medial edge (Figure 10). A biomechanical study showed the distance from the olecranon tip to the articular surface to be 12 mm.[8] As a result, the anchors are placed 13 to 15 mm distal to the tip of the olecranon and are directed distal to the articular surface to avoid the risk of joint penetration.

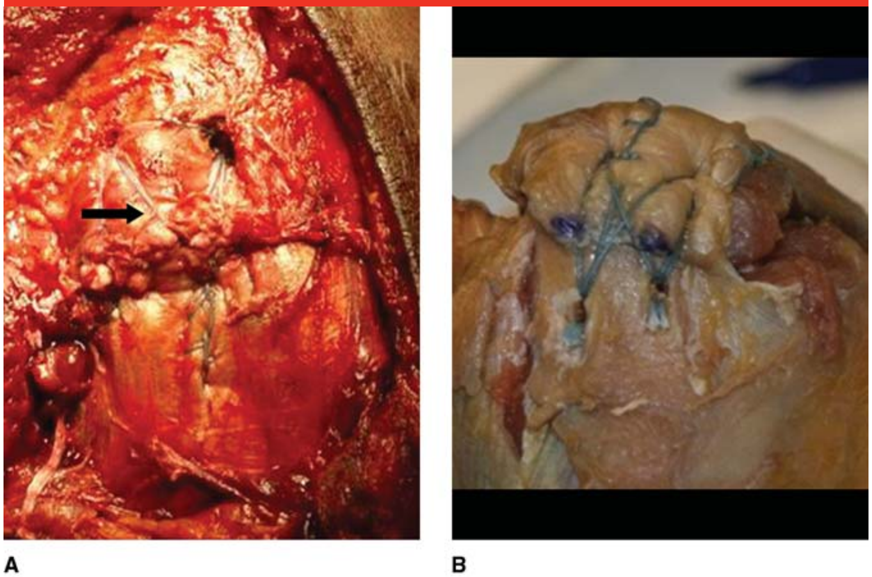
A mattress suture from each anchor is placed in the distal tendon 2 cm proximal to the tendon edge. This secures the proximal tendon down to the bone and restores the distinct medial insertion (medial-side anchor) and lateral insertion (lateral-side anchor). The sutures from these anchors should not be cut because they will be used later to form the bridge. There should be free tendon still remaining distal to the anchors (Figure 11).

The distal footprint is estimated, and two pilot holes are created to accommodate the next two anchors. The holes should be placed just distal to the anatomic footprint. Care should be taken not to drill directly toward the joint surface but rather to drill obliquely, aiming distally on the ulnar shaft.

Three suture tails (one suture tail from each anchor and one suture limb of the free Krackow suture) are then threaded into the anchor eyelet of a 3.5-mm knotless press-fit anchor. Tension is held on the sutures to reduce the triceps tendon to its desired position on the footprint. With this tension applied, the anchor is advanced with a mallet into the pilot hole until it sits flush. The sutures can then be cut flush to the level of the press-fit anchor. The remaining three sutures are threaded into the eyelet of a second knotless press-fit anchor; this anchor is driven into the other pilot hole, and the suture ends are cut flush (Figure 12). Repair of the triceps expansion is then performed using side-to-side sutures.

Postoperatively, the patient is not splinted. Rather, a sling is used for comfort. The patient is allowed to discontinue use of the sling when she or he feels comfortable. Active mo-

Figure 12



A, Intraoperative photograph of repaired triceps tendon rupture with a suture bridge construct. Note the crossing sutures that help to restore the footprint area of the triceps attachment (arrow). **B,** Photograph of a cadaver specimen demonstrating the crossing sutures used to restore the wide footprint attachment of the triceps insertion.

tion exercises, including supination and pronation, are begun immediately, and the patient is allowed to hang the arm against gravity as tolerated. Weight bearing restrictions are set at ≤5 lbs during this phase of recovery. Strengthening is begun 4 weeks postoperatively. Although no data in the literature support one protocol over another, we allow patients to begin earlier ROM given the biomechanical data for the anatomic repair.[8]

Postoperative Care

Most postoperative protocols involve immobilization of the elbow in 30° to 45° of flexion for 2 weeks until the wound heals. Therapy, consisting of passive extension and guided active flexion, is begun following immobilization. At 4 weeks postoperatively, active ROM is initiated and efforts are refocused on regaining full elbow motion. Weight lifting should be avoided for at least 4 to 6 months postoperatively.[12]

Chronic, Revision, and Difficult Acute Tears

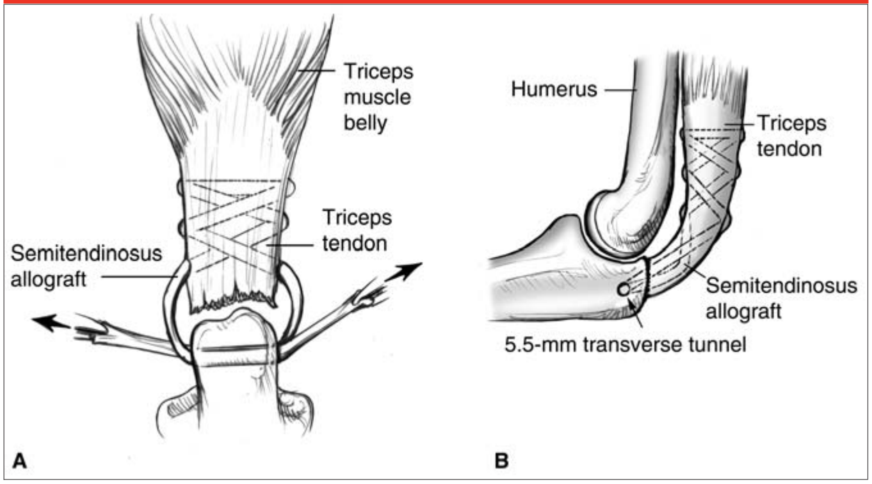
Chronic ruptures (injuries >6 weeks old) are usually the result of either a delay in diagnosis or a delay between the time of injury and when the patient sought treatment. Multiple procedures have been described to augment the management of these more challenging triceps injuries (ie, chronic, revision, acute tear with poor tissue quality). The use of anconeus or Achilles rotation flaps, plantaris or hamstring tendon augmentation, and ligament augmentation devices have all been reported with varying degrees of success.[6,37-40] Factors that affect surgical technique include degree of chronicity and retraction, quality of the tendon tissue, and the presence of medical comorbidities.

Hamstring allograft reconstruction has been used to augment repairs in these difficult cases.[41] The allograft semitendinosus tendon is woven in a Bunnell fashion through the remaining proximal triceps tendon. A 5.5-mm transosseous tunnel is then drilled through the proximal olecranon, 1 cm distal from its tip and centered between the articular surface and the posterior cortex. The two free ends of the hamstring tendon are then passed through the transosseous tunnel in a retrograde fashion, and the elbow is placed in full extension as the tendon stump is reduced to the olecranon (Figure 13). The use of an interference screw, rather than suturing the tendon to itself, improves the fixation strength and resistance to displacement. Improved fixation strength at the repair site may allow for earlier initiation of active motion and may decrease potential elbow stiffness or joint contracture.

Results and Complications

Results following primary repair of acute triceps ruptures are very good. Most published reports do not provide detailed quantitative data or subjective outcomes based on standardized measures such as the Disabilities of the Arm, Shoulder and Hand score and the Medical Outcomes Study 36-Item Short Form score. Instead, most studies offer a retrospective review of cases performed and state that the patients clinically demonstrated a certain ROM and strength with return to preinjury levels of function.[2,3,13,15-17,20,24,29] van Riet et al[12] used the transosseous cruciate technique for primary repair of 13 acute tears at the tendinous insertion. Surgery was performed an average of 63 days after injury. At 1-year follow-up, the authors demonstrated peak strength of 92% (range, 75% to 106%) and 8° of loss of extension compared with the uninjured side.

Figure 13



AP (**A**) and lateral (**B**) views of the hamstring allograft technique used to augment distal triceps repair. The allograft semitendinosus tendon is woven through the remaining proximal triceps tendon edge. The transverse tunnel is drilled through the olecranon 1 cm from the tip. The tunnel is centered between the articular surface and the posterior cortex.

Quantitative data and subjective outcome measures are lacking in reports of chronic or difficult ruptures. However, van Riet et al[12] quantified the outcome of nine chronic ruptures managed with triceps reconstruction. The time from injury to treatment averaged 163 days (range, 28 to 393). These patients demonstrated an average peak strength of 66% (range, 35% to 100%) and average loss of extension of 13° compared with the uninjured arm. Despite the wide range in peak strength, this report demonstrates that reconstructions of chronic tears are inferior to primary repairs of acute tears. In contrast, augmentation of primary repairs with hamstring autograft have demonstrated good outcomes. Case reports indicate that patients have returned to manual labor as well as competitive weight lifting with 5/5 manual strength testing in the clinic.[6,40]

Potential postoperative complications include olecranon bursitis secondary to wire suturing, flexion contractures ranging from 5° to 20°, irritation from underlying internal fixation, and rerupture.[31] Rerupture is rare and usually results from a traumatic fall after complete recovery from the first rupture.[12] Results of repairs and reconstructions following three cases of rerupture in the study by van Riet et al[12] were found to be functionally equivalent to results with primary repair of first-time ruptures.

Summary

Acute triceps ruptures are uncommon, occurring mainly in athletes, weight lifters (especially those who use anabolic steroids), and persons who sustain elbow trauma. Acute triceps rupture is a clinical diagnosis, although MRI may aid in confirmation and surgical planning. Anatomic classification of these ruptures, in conjunction with functional and medical status, is helpful in determining the proper treatment protocol. Surgical repair offers predictable return of function with a small risk of loss of elbow motion. Even surgical treatment of chronic tears has demonstrated improved elbow extension and strength as well as patient satisfaction. We favor anatomic footprint repair of the triceps to provide optimal tendon-to-bone healing and, ultimately, optimal functional outcome.

References

Evidence-based Medicine: Levels of evidence are listed in the table of contents. The references in this article are level IV case series or case report studies.

Citation numbers printed in **bold type** indicate references published within the past 5 years.

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