

SPORTS INJURY BULLETIN

prevention • treatment • rehabilitation

From the editor

Tendon injuries make up around half of a therapist's caseload – and yet there is still disagreement on how best to treat them. Trevor Langford takes a look at the different approaches to dealing with high hamstring problems: is a conservative plan best, or is injection therapy the way forward?

A lesser proportion of sports injuries affect the wrist or hand, but these comparatively smaller problems can have hugely deleterious effects, particularly for athletes whose sports involve loading on the arms. Andrew Hamilton explores the problems and potential treatments.

Elsewhere this issue, Chris Mallac has the second part of his feature on the rehabilitation of the acromioclavicular joint; and also assesses the characteristics of the frequently injured supraspinatus muscle.

And Alicia Filley looks at the incapacitating lower leg pain caused by chronic exertional compartment syndrome of the deep posterior compartment. See you next time.

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Shoulders

Muscle series: supraspinatus

Chris Mallac takes a look at a frequently injured shoulder muscle...

The supraspinatus muscle is one of the four rotator cuff muscles of the shoulder. It originates from the central part of the supraspinatus fossa of the scapula and then courses laterally to cross the joint capsule of the shoulder and attaches onto it; it then passes under the acromion and the coracoacromial ligament and fans out horizontally and inserts to the superior and medial facets of the greater tuberosity of the humerus. The superficial part of the muscle is covered by the fascia supraspinata. Superiorly it contacts the deltoid and trapezius muscles, whereas inferiorly the deep fascia attaches to the joint capsule.

Functionally, the supraspinatus has a role in both initiating active shoulder abduction and providing abduction torque (particularly in the first 30 degrees of shoulder abduction) along with the more powerful deltoid, and it also plays a role in depressing the humeral head and centralising the humeral head in the glenoid during abduction/flexion movements. It has been shown in biomechanical studies that during active shoulder abduction, the force vector created by the supraspinatus (which acts as a humeral head compressor and depressor) and the anterior/middle deltoids (humeral head elevators) creates a

force couple whereby the humeral head remains centred in glenoid fossa. In the instance of a weakened supraspinatus, greater deltoid activity ensues and this will result in a superior shear of the humeral head in relation to the glenoid^(2,3). This may then create a mechanical impingement between the humeral head and the overlying superior glenoid rim, acromion process and coracoacromial arch.

Finally, the supraspinatus may also contribute to external rotation and internal rotation torque production; however, this is varied upon the starting abduction and flexion/extension angles of the shoulder. For the most part, it contributes little towards pure rotation torque at the glenohumeral joint.

Incidence of Injury

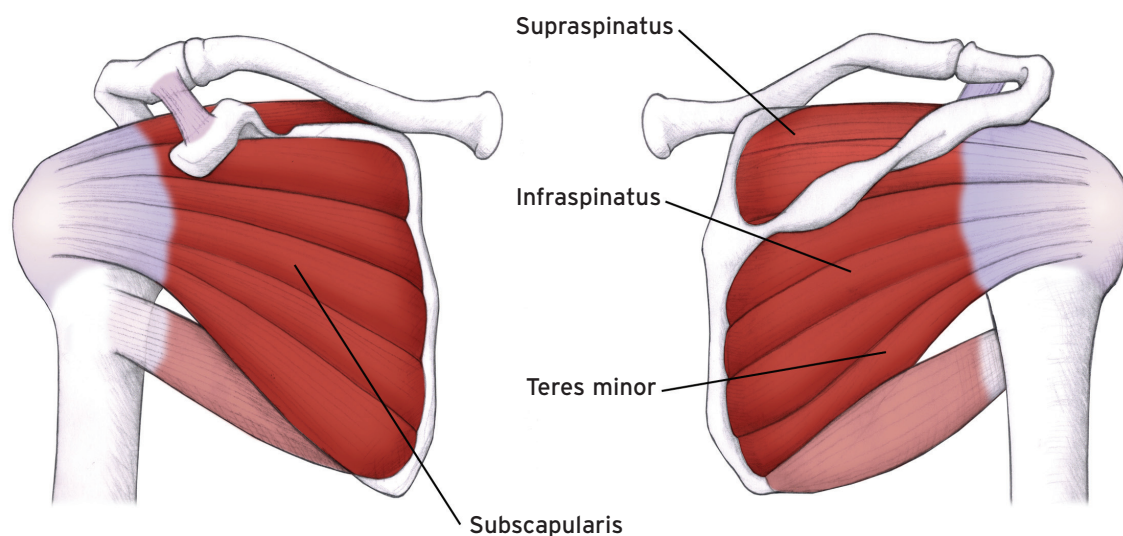
Supraspinatus injury has a high prevalence in the population. It is particularly common in the older population which indicates that progressive degeneration is a factor in developing a partial or full thickness tear in the supraspinatus tendon. Research has postulated that in individuals under the age of 40 the incidence is a small 4%; however, in those ages 60 and above the incidence rapidly rises to 54%⁽⁴⁾.

Even in patients without shoulder pain, the incidence of rotator cuff injury is relatively high. Tempelhof et al (1999) found that even in asymptomatic individuals, ultrasound finding showed that in the 50-59 age group 13% had tears, in the 60-69 age group 20%, in the 70-79 age group 31% and in the older 80+ age group 51% of asymptomatic shoulders had tears in the rotator cuff⁽⁵⁾.

Yamamoto et al (2010)⁽⁶⁾ supports this age-related predisposition by suggesting that as well as arm dominance and previous shoulder trauma, age was another risk factor in developing a rotator cuff tear. What they found was that the incidence of rotator cuff tears increased linearly with age (0% in the 20s group, 2.5% in the 30s group all the way up to 50% in the 80s group). Interestingly, 17% of subjects with rotator cuff tears complained of no symptoms in their shoulders. In essence, many older individuals may have a true partial/full thickness tear of the supraspinatus and be completely unaware the injury exists⁽⁶⁾.

Countless research shows that in the absence of a supraspinatus (due to a full thickness tear of the supraspinatus tendon), the humeral head will migrate superiorly

Cross-section of the shoulder



and abut the acromion, highlighting the importance of the supraspinatus in centering the humeral head in the glenoid and preventing a superior migration of the humeral head^(7,8,9,10). Repeated superior migration of the humeral head will create an impingement situation and repeated episodes of impingement will lead to breakdown and damage to the subacromial bursa, arthritic changes in and around the glenoid and humeral head and acromioclavicular joint changes in the inferior aspect of the joint.

In the younger athletic population, true partial/full thickness tears are rare unless it is associated with a more serious shoulder trauma such as a dislocation or if the arm is forcefully adducted whilst in an abducted position, a situation that may occur in contact sports such as rugby and NFL. More commonly, injury to the supraspinatus involves early stage tendinopathy that may result in an inflamed supraspinatus tendon due to mechanical overload. Finally, active trigger points can develop in the supraspinatus that may create radiating pain across the deltoid and upper arm.

Mechanism of injury

Interestingly in the older population, injuries to the supraspinatus may result from innocuous activities such as lifting a heavy bag or holding a dog on a lead and the dog suddenly stops or runs, resulting in a yank on the arm. In the younger population, direct injury to the supraspinatus may arise due to a trauma

episode such as fall onto an outstretched hand, dislocating a shoulder, sustaining a strong adduction force on a flexed/abducted shoulder or falling off a bike and keeping hold of the handles. It may come through heavy overload movements such as Olympic-type lifting, powerlifting and repetitive trauma from repetitive exercise such as swimming, tennis and other overhead sports.

The supraspinatus is a tendon that is highly subjected to mechanical forces over an extended period of time, highlighting the incidence of tears in the older population. Neer (1983)⁽¹¹⁾ was the first to describe the three stages of rotator cuff disease, in particular the changes seen in the supraspinatus. However, he did not elucidate if the cause was mechanical impingement or age-related intrinsic tendon degeneration or a combination of both. Stage I occurs in patients < 25 years with oedema and haemorrhage of the tendon and bursa. Stage II involved inflammation and fibrosis of the rotator cuff in patients aged between 25 and 40 years of age. Stage III involves tearing of the rotator cuff, either partial or full-thickness, and occurs in patients > 40 years of age⁽¹¹⁾.

It is possible that some small supraspinatus tears may heal or become smaller; however, Yamanaka and Matsumoto (1994) showed that about 53% develop further and 28% may progress to full thickness tears. It is believed that full thickness tears do not heal due to poor vascularisation within the tendon⁽¹²⁾.

Presenting signs and symptoms

Subjective

The patient with a low grade supraspinatus injury or the more serious partial thickness tears will complain of antero-lateral shoulder pain that is made worse by any activity where the arm is lifted up to 90 degrees of flexion or abduction. Sleeping on the shoulder can be painful. Holding weights and lifting simple things such as hanging towels on a clothes line may prove to be quite painful and functionally impossible to perform. However, as highlighted above, many incidences of supraspinatus pathology may in fact be painless and asymptomatic.

Objective

Palpation

The patient will usually be tender to palpate in and around the humeral head under the acromion process.

Active Movements

Full flexion and abduction movements will most likely be painful and difficult to fully execute depending on severity of the injury. Stage I-type injuries will have an arc of pain throughout abduction whereby the pain will be present from 80-160 degrees of abduction. In more serious partial thickness and full thickness tears, full abduction may be impossible to perform due to an inability of the muscle to initiate abduction, or the quality of the movement may be quite poor whereby the patient elevates or 'hitches' the scapula to initiate abduction.

The classic test for supraspinatus/rotator cuff impingement is the Neer test first described in 1972. In this test the patient is asked to flex the shoulder past 90 degrees whilst the scapular is held stable by the examiner. Typically the pain is felt at around 120 degrees of shoulder flexion⁽¹³⁾. Jia et al (2011) looked at the internal structure of the shoulder using an arthroscope and found that in most cases at 120 degrees of flexion, the rotator cuff (supraspinatus included) contacted the superior glenoid rim. These findings correlated with the position patients felt the pain when assessed clinically. Therefore injuries to the supraspinatus may present as pain in the anterior/lateral shoulder when the arm is flexed to 120 degrees⁽¹⁴⁾.



Neer Impingement Test

Resisted muscle tests

Patients with low grade supraspinatus pathology will often test normal with the majority of resisted rotation movements and abduction movements. The more serious tendon lesions such as partial/full tears will usually present as weakness in external rotation and abduction (with or without pain). Specifically, muscle testing for the supraspinatus can be achieved using an 'empty can' or 'full can' test.

Researchers have been studying the 'best' position for testing and retraining the supraspinatus since Jobe and Moynes (1982) first offered the 'empty can' movement as being an effective supraspinatus strengthening exercise⁽¹⁵⁾. In this movement, the arm is abducted in the scaption plane (30° anterior to frontal)



Empty can test



Full can test

with the arm internally rotated – similar to pouring fluid out of a can. The examiner can then push downwards on the hands to add extra resistance. This position will usually be felt as being weak and painful in the presence of a supraspinatus lesion.

However, numerous studies since have shown that the 'empty can' position is not necessarily the best position for testing and exercising for isolated supraspinatus without excessive deltoid activation. The scientific basis for this disagreement has stemmed from the numerous EMG research studies in the last decade that have measured supraspinatus activity relative to other shoulder muscles such as the deltoids. For example, Blackburn et al (1990) suggests the opposite movement for testing/strengthening supraspinatus – the 'full can' movement. This is identical to the 'empty can'; however, the arm is kept in external rotation rather than internal rotation. This elucidates the same level of supraspinatus activity, without the superior shear effect of the deltoid⁽¹⁶⁾.

Exercises and testing positions that produce higher levels of deltoid activity in relation to supraspinatus activity may be counter-productive in patients with shoulder pain, weakness of the rotator cuff, and inefficient dynamic stabilization. Therefore, the empty can position may also give false positives as the source of pain may be direct impingement of the subacromial structures due to the superior migration of the humeral head due to deltoid contraction.

From an anatomical and biomechanical standpoint, the full can exercise also may

be the most beneficial position to both test and exercise and provoke the least amount of pain because of the least amount of humeral head superior migration and increased moment arm of the supraspinatus muscle in this position compared with the empty can position.

Rehabilitation of supraspinatus injuries

Early stage supraspinatus injuries and small partial thickness tears may do well when managed conservatively. Larger partial thickness tears and full thickness tears often will need to be surgically managed to obtain a favourable outcome. For the purposes of this paper, the discussion will focus only on the conservatively managed supraspinatus injury.

It appears that the best exercises for the supraspinatus would elicit the greatest amount of supraspinatus activity while minimizing the surrounding muscular activity, particularly the deltoid. Boettcher et al (2009)⁽¹⁷⁾ studied this exact phenomenon when they assessed the EMG activity (15 subjects) of a number of shoulder muscles including supraspinatus, infraspinatus and deltoids whilst performing the following movements: full can movements, empty can movement, prone elevation, external rotation in 0 degrees abduction and prone external rotation positions. All the exercises were performed with the scapular in a retracted position. All the exercises performed were held isometrically for five seconds, with a one-second build-up phase, a three-second hold and a one-second release phase.

They sought to not only look at which exercises best activated supraspinatus but also which ones recruited deltoids the least. What they found was that all five chosen exercises activated the supraspinatus to a significant degree, and in particular there was no difference between 'full can' and 'empty can' movements. They also found that prone elevation activated muscles in this order of magnitude: posterior deltoid, supraspinatus, anterior deltoid. The rotation at 0 degrees and the prone external rotation activated infraspinatus the most, but supraspinatus activity still outperformed all the deltoids; and more importantly, these two exercises also recruited far less deltoid than the 'full can' and 'empty can' movements. They argued that the best exercise for supraspinatus was the exercises that incorporate external

rotation and not the 'empty can' and 'full can' movements. The researchers argue that exercises need to be chosen that limit deltoids as it has a superior migratory effect on the humeral head, potentially causing impingement of the supraspinatus against the acromion and coracoacromial space.

Reinold et al (2007)⁽¹⁸⁾ conducted a similar study on comparing the 'empty can', 'full can' and 'prone full can' movements and measuring EMG activity in the middle deltoid, posterior deltoid and supraspinatus muscles. What they found was that although all three exercises produced similar amounts of supraspinatus activity, the full can exercise produced significantly less activity of the deltoid muscles and may be considered to be the optimal position to recruit the supraspinatus muscle for rehabilitation and testing. The empty can exercise may be a good exercise to recruit the middle deltoid muscle, and the prone full can exercise may be a good exercise to recruit the posterior deltoid muscle. These findings were supported by Lee et al (2014)⁽¹⁹⁾ who showed using PET/CT imaging that the 'full can' position was more effective as an exercise for supraspinatus without overriding deltoid activity. Finally, Escamilla et al (2009)⁽²⁰⁾ suggests that scapular protraction and scapular anterior tilt, both of which decrease subacromial space width and increase impingement risk, are greater when performing scaption movements with internal rotation ('empty can') compared with scaption with external rotation ('full can').

Therefore it appears that the 'safest' exercises to recruit supraspinatus and also minimise the deltoid are the 'full can position' and the 'prone external rotation' exercise. These two exercises can form the basis of most supraspinatus re-strengthening programs. These exercises are described below. Furthermore, effective supraspinatus function will only be evident with a strong and functioning scapula base. Direct exercises to both strengthen and retrain the serratus anterior and lower trapezius will also be required to allow optimal scapular positioning through shoulder range.

The exercises

1. Full can exercise

Stand holding either small dumb-bells (2.5kg women, up to 5kg for men) or rubber tubing. Start with the hands by the side and the thumbs turned outwards. Slowly raise the arm into abduction whilst

keeping the angle of the arm around 30 degrees to the frontal plane. Initially if the shoulder is painful the range can be limited to 30 degrees; however, as strength and pain improve they may move the arm into further positions of abduction. Work on three sets of 15-20 repetitions.

2. Prone external rotation exercise

Lie face down and start with the arm at 90 degrees abduction and the arm hanging down. Gently retract and depress the scapula. Slowly raise the arm into external rotation whilst attempting to maintain the scapula position. Perform three sets of 15-20 repetitions.

3. Scapular wall slides

As per the image below, start with the forearms in contact with the wall. Gently slide the forearms up the wall above the head, slowly externally rotating the arms/forearms on the way up. This will create scapula upward rotation and protraction, a great exercise to activate the serratus anterior, a necessary muscle in the control

of scapula movement and hence rotator cuff function in overhead sports.

4. Lower trapezius setting

The easiest way to perform this and to teach this as an exercise is to do this drill on a lat pulldown machine. Use only light resistance such as 3-4 plates on the pulldown machine. Seated with the hands gripping the lat pulldown bar, slowly draw the scapula down and in (retract and depress). Hold for a quick 1-2 seconds and then repeat for two sets of 15 reps.



Lower trap setting (start)



Lower trap setting (finish)



Scapular wall slides (start)



Scapular wall slides (finish)

Conclusion

Research shows that the supraspinatus plays an important role in the shoulder as it centres the humeral head into the glenoid during functional movements of the arm/shoulder. Dysfunction in this muscle may lead to excessive upward shearing/gliding of the humeral head that may be a precursor to the more serious shoulder impingements and shoulder instabilities. Injuries to the supraspinatus are common and the most likely cause of rotator cuff dysfunction

particularly in the older athlete. This article has provided a framework for assessment and rehabilitation of dysfunction in the supraspinatus muscle.

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Hamstrings

Management of high hamstring tendinopathies – a conservative versus injection therapy approach

Trevor Langford reviews the possible ways to treat these hamstring problems – and the evidence on offer

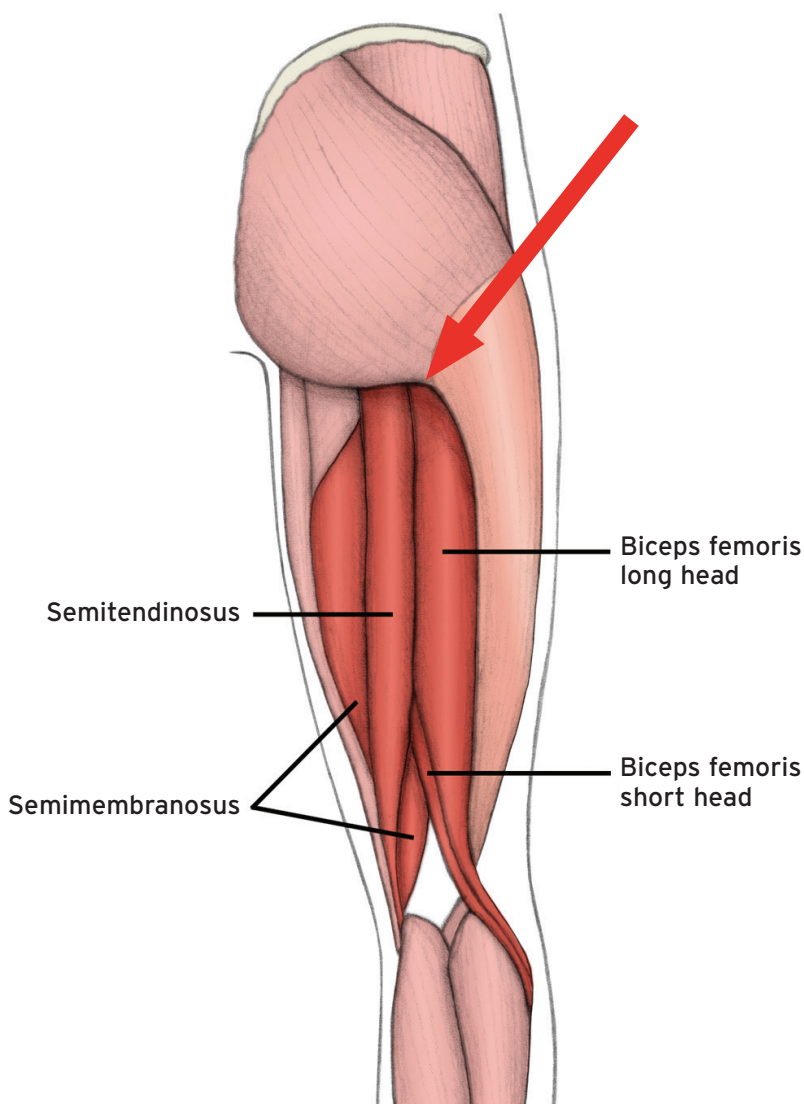
Tendon injuries account for 30-50% of all injuries reported to physiotherapy clinics and with 30% of all running injuries relating to tendon overuse⁽¹⁾. Tendinopathies are complex in nature to manage and may result in a long lay-off from sport and long-term damage if inappropriately treated. High hamstring tendinopathies (HHT) are less commonly diagnosed than other tendons of the lower limb and may present as either deep buttock pain and/or as posterior thigh pain⁽²⁾.

The purpose of this review is to provide an insight into the conservative management for HHT and when to explore injection therapy. High hamstring tendinopathies may also be referred to as proximal hamstring tendinopathy (PHT) or hamstring origin tendinopathy (HOT). The research pertaining to conservative treatment for HHT is limited⁽²⁾ and therefore some of the research provided in this review is related to tendinopathies of other major tendons of the lower limb.

Anatomy of the hamstring muscle

The hamstring muscle comprises of three separate muscles; i) semitendinosus, ii) semimembranosus and iii) biceps femoris (long head) all originate at the ischial tuberosity of the pelvis. The short head of the biceps femoris, in contrast, originates at the posterior aspect of the femur on a ridge known as the linear aspera. The hamstring muscles course down the posterior thigh with the biceps femoris (long and short heads) attaching laterally to the head of the fibula whereas the semitendinosus and semimembranosus

Box 1: The hamstring muscles with the arrow indicating the primary location for pain in HHT



attach medially to the tibia. The hamstring muscles are innervated by the tibial branch of the sciatic nerve and facilitate hip extension and knee flexion⁽³⁾.

Pathophysiology

The term tendinosis is used to describe the process of degeneration in tendinopathy, although many still use the age old term of tendinitis. The 'itis' implies an inflammatory response is present but intratendinous degeneration is defined as being hypoxic and calcific from factors such as reduced blood flow, aging and microtrauma rather than presenting as an inflammatory response⁽⁴⁾. A hypothesis explaining the pain experienced in a tendinopathy is from the ingrowth of the vessels and nerves termed as 'neovascularisation' which causes a pain response from the swelling of an irritable tendon⁽⁵⁾.

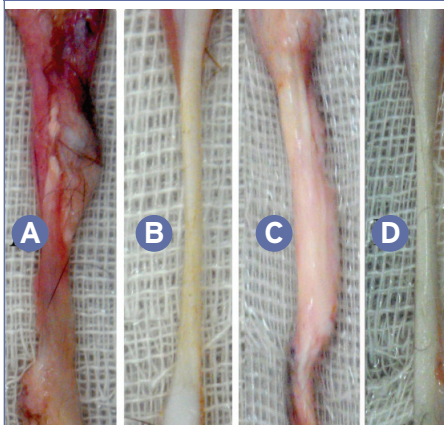
The pathological part of a tendon appears as grey and of dull appearance as it loses its glistening white appearance as seen in Box 2⁽⁶⁾. The illustration of an injured tendon in Box 2 was not a tendinopathy and was a tendon removal coupled with surgical repair of the superficial digital flexor tendon in rabbits. The authors,⁽⁶⁾ however, drew comparisons to the healing stages in tendinopathies in humans at the similar timelines of 28 and 84 days (four and 12 weeks). The picture illustrated in section C of box 2 shows the biomechanical properties of the immature collagen fibres and significantly lesser to the normal tendon in sections B and D.

Clinical assessment

The proximal hamstring tendon is placed under greatest load when the knee is extended and the hip is fully flexed. Running, or running-related sports, predispose an individual to tendinopathy due to the extended duration the hamstring tendon is held in this eccentrically loaded position⁽⁷⁾. Additional factors exposing an individual to HHT may include low hamstring to quadriceps muscle ratio, inadequate warm-up protocols, previous hamstring injury and reduced hamstring flexibility⁽²⁾. Other factors may include pelvic dysfunction and decreased activity of the core stability muscles and therefore a broad assessment should be undertaken with the onset of deep buttock pain⁽³⁾.

Symptoms of HHT may include pain during repetitive eccentric loading, acceleration during running and in extreme cases sitting on hard surfaces. The taking the shoe off test, resisted by the

Box 2: Tendons



- A** Injured tendon of a rabbit of 28 DPI (days post-injury) showing swelling.
- B** Normal tendon of a 28 DPI rabbit having shiny and normal appearance.
- C** Injured tendon of 84 DPI group.
- D** Normal tendon of a rabbit of the 84 DPI group⁽⁶⁾

uninvolved foot, has been shown to have a high sensitivity and specificity of 100% for hamstring-related injuries and should be considered for use with a suspected HHT⁽⁸⁾. Typically pain is felt on active and passive stretching of the hamstring muscles at the origin attachment with pain also experienced on palpation of the ischial tuberosity⁽²⁾. In addition, there is minimal decrease in strength of the hamstring muscles with knee flexion or hip extension in isolation and no neurological deficit is present unless further lumbosacral spine pathology is involved⁽²⁾. Pain is often observed when the knee is extended with the hip fully flexed placing the greatest load on the hamstring muscles such as the terminal of stage of the swing phase in gait.

Box 3: Eccentric loading guidelines used for tendinopathies⁽¹⁾

- One REP MAX to determine load should be avoided to prevent further damage.
- Load should be determined by number of repetitions rather than weight.
- 3-4 sets of 6-12 repetitions to start initially.
- 4 sessions per week as opposed to daily.
- Lift with two legs and lower with involved leg.

Conservative treatment

An eccentric contraction, which refers to increased tension of a muscle-tendon unit during lengthening, has been widely used as a treatment tool for tendon pathologies⁽⁷⁾. Tendons require seven and half times less oxygen than skeletal muscle does and during the eccentric phase oxygen consumption rarely rises to more than twice its resting value⁽⁹⁾. Research compiled during the 1970s indicated that faster concentric contractions demand a greater oxygen supply and as a result an increase in heat production and cellular metabolism occur⁽¹⁰⁾. Consequently a greater amount of waste products are transported to the active site potentially causing a chemical reaction of nerve endings and an increase in pain⁽¹⁰⁾. Therefore the justification for eccentric exercise in the management of tendinopathies is provided as opposed to concentric contractions.

More recent research has suggested that eccentric exercise promotes collagen fibre cross-linkage within the tendon which facilitates remodelling of the injured tissue⁽⁷⁾. This was supported by Langaard and colleagues who found that 12 weeks of eccentric loading of the Achilles tendon increased the collagen synthesis rate of type one fibres⁽⁵⁾. It has been stated that pain and neovascularisation are reduced following a different 12-week eccentric training programme for an Achilles tendinopathy⁽¹¹⁾. A period of 12 weeks is considered adequate for tendon regeneration to occur, although positive results may be observed sooner.

A case study was published in the Journal of Manual and Manipulative Therapy of a 41-year-old female recreational runner who completed five three-mile runs per week⁽⁷⁾. The patient had an onset of right buttock pain but was able to continue training at a lower intensity for 12 months. The patient then developed left buttock pain with worsening pain on her right side which forced the patient to cease running completely. The patient's objective was to make a full return to running. Pain ranged from 0-6/10 on VAS scale depending on activities. Pain was experienced on brisk walking, getting in and out of cars and sitting on hard surfaces and when taking shoes off ('taking a shoe off test').

An assessment of the patient in the case study⁽⁷⁾ indicated straight leg testing with ankle dorsiflexion and slump tests were negative bilaterally and pain-free lumbar spine movements with over-pressure in all directions were also

Box 4: The rehabilitation programme used in the case study

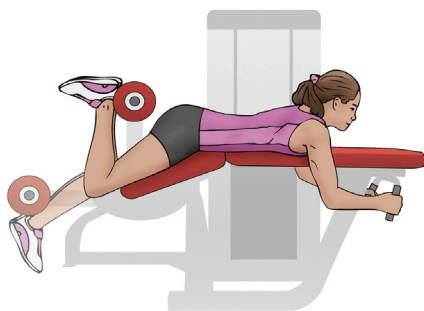


a) Prone hamstring strengthening

Visit 1: ASTYM, a) prone eccentric hamstring curls (3x10 reps, twice per day), b) supine hamstring stretch.



b) Supine hamstring stretch



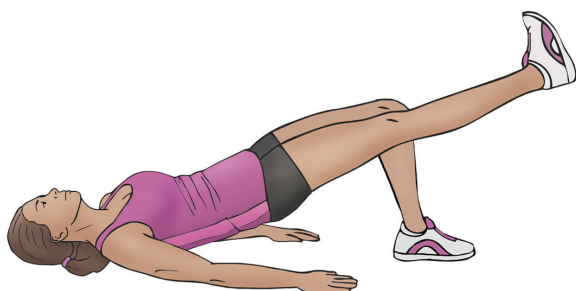
c) Machine hamstring strengthening

Visit 2-4: Warm-up bike, ASTYM, c) Prone eccentric hamstring curls using a machine (lift with two legs, lower with one), resisted hips extension, open chain quad strengthening and contract relax hamstring stretch.



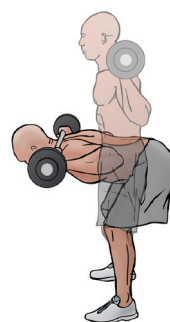
d) Seated hamstring curls

Visit 5-8: Warm up bike, ASTYM, prone eccentric hamstring curls, d) seated hamstring curls from 0-90 degrees,

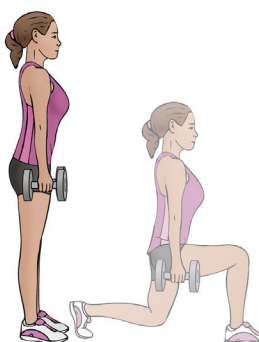


e) Unilateral bridging

Visit 9-12: Warm-up bike, ASTYM, prone eccentric hamstring curls, seated hamstring curls from 0-90 degrees, e) unilateral bridging, f) standing eccentric exercise in the form of good mornings.



f) Good morning



g) Lunges



h) Single-leg squats



i) eccentric Nordic curls

Visit 13-16: As previously completed with the addition of g) lunges, h) single-leg squats and i) Nordic curls.

negative. Pain in buttock with lumbar flexion with her knees fully extended was, however, noted. No pain on palpation of the lumbosacral spine was observed but the patient was tender on the ischial tuberosity and the proximal two inches of the hamstring tendon. Pain was not observed on the hip quadrant test of either hip and no pain was noted on sacroiliac joint testing. Hip flexors, abductors and external rotators were all pain-free and recorded as 5/5 on the Oxford scale. Hip extension was slightly painful on both sides with a reduction in strength 4/5. Knee flexion reproduced greater pain levels on both sides with 4-/5. Pain was reproduced on both sides by passively extending the knee with the hip maintained in 90 degrees of flexion.

The treatment programme of the runner presenting with HHT is highlighted in Box 4. It should be noted that a HHT won't present in exactly the same manner but the case study above does acknowledge other areas to be examining as part of your clinical assessment. This patient was treated with a progression of exercises over the course of 16 treatment sessions with hamstring eccentric exercises, gluteal stability exercises, stretching, and ASTYM. ASTYM is a soft tissue technique proposed to regenerate a response in the soft tissue to promote healing, but published research is yet to be provided on humans. The soft tissue technique is performed by carrying out gliding techniques in the direction of the muscle fibres. The rehabilitation exercises were selected based on the physical capabilities of the patient throughout the rehabilitation and three sets of 10 repetitions were used on each of the exercises.

The outcome of the case published by McCormack⁽⁷⁾ indicated a steady progression throughout with no pain on taking shoes off at visit three. After the eighth treatment the patient was able to walk two and half miles pain-free and after 12 treatments able to jog one mile pain-free. After 16 treatment sessions the patient reported 95% subjective improvement and was able to run for two and a half miles pain-free.

Shockwave therapy

Shockwave therapy has been cited as being an effective tool for managing HHT. A study investigated the effects of hamstring resistance training (not specifically eccentric loading) with anti-inflammatory drugs and shockwave therapy on the tendinopathy of the proximal hamstring in

40 professional athletes⁽¹²⁾. The shockwave applied was 2500 impulses per session without anaesthesia of one session per week for four weeks. The four-week study, which used a randomised controlled research design, found at three months post-study that 17 of the 20 athletes who received shockwave had a reduction in pain of 50%, whereas in the treatment group only two patients received a reduction in their pain by 50%. Although the comparison between the shockwave versus treatment group shouldn't be contrasted against eccentric training, it is substantial evidence for the use of shockwave in treating HHT.

Steroid injection therapy

Research of tendinopathies has indicated that 20% of patients will remain as symptomatic at three to six months after a conservative management programme has been applied⁽¹³⁾. Further interventions should be explored at this point and one of the options is in the form of injection therapy. It should be noted that once injection therapy has been carried out it is key that the conservative management programme be continued for optimal results.

A fluoroscopy-guided peritendinous corticosteroid injection was provided for 18 athletes with HHT diagnosed by MRI⁽²⁾. The follow-up, on average being 21 months, indicated by questionnaire that pain had significantly reduced from 7.22 pre-injection to 3.94 post injection. The results also indicated that athletic participation had significantly increased from 28.76% to 68.82% with 38.8% of patients being completely asymptomatic at a mean follow up of 24.8 months. There was no documentation of a conservative programme and this is the limitation of the research. Had a structured management programme been provided it would be useful to draw on the results obtained and draw comparisons between the groups.

Researchers from the University of Copenhagen studied the effects of corticosteroid injections, eccentric training and heavy slow resistance training on patients with patellar tendinopathy⁽¹⁴⁾. Using a randomized controlled single blind research design, 39 male participants were assigned to one of the three intervention groups. Factors measured were function, symptoms, pain, tendon swelling and vascularisation and tendon mechanical properties prior to the study, at 12 weeks and at six months following the study.

The results of the research yielded that

each intervention significantly improved in pain, function and symptoms during the first 12-week period. Furthermore, pain, function and symptoms continued to improve in the eccentric and heavy slow resistance training groups at the six-month follow-up but had significantly reduced in the corticosteroid injection group. Also at the six-month follow-up the heavy slow resistance training group indicated that they were the most satisfied with the treatment outcome which coincided with increased collagen turnover. Although the corticosteroid injection alone provided positive initial results at 12 weeks the results were not substantive for the long-term clinical effects.

Summary

A rehabilitation programme devised for HHT should be based upon the findings from the initial examination and the load in which the hamstring muscle can withstand. Furthermore, the complexity and load of the rehabilitation exercises should be specific to the patient's pain tolerance. If the tendon isn't responding to conservative treatment then steroid injection is an avenue to explore depending on the patient's age and current symptoms after a period of least 12 weeks. To draw greater comparisons between steroid injection therapy and a conservative management programme, research should endeavour to contrast the effects of both forms of treatment using randomised research design. The current review is unable to provide a firm conclusion at this point as further research is required.

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The lower leg

Getting a leg up

Alicia Filley assesses lower leg pain caused by chronic exertional compartment syndrome of the deep posterior compartment...

Lower leg pain is a common complaint among runners. Pain deep within the calf that starts after 20 to 30 minutes of exercise and resolves with rest is likely caused by chronic exertional compartment syndrome (CECS) of the deep posterior compartment. The pain may be described as a burning, aching, bursting, or tightness along the posterior medial border of the tibia. Pain or numbness may extend to the medial aspect of the foot.

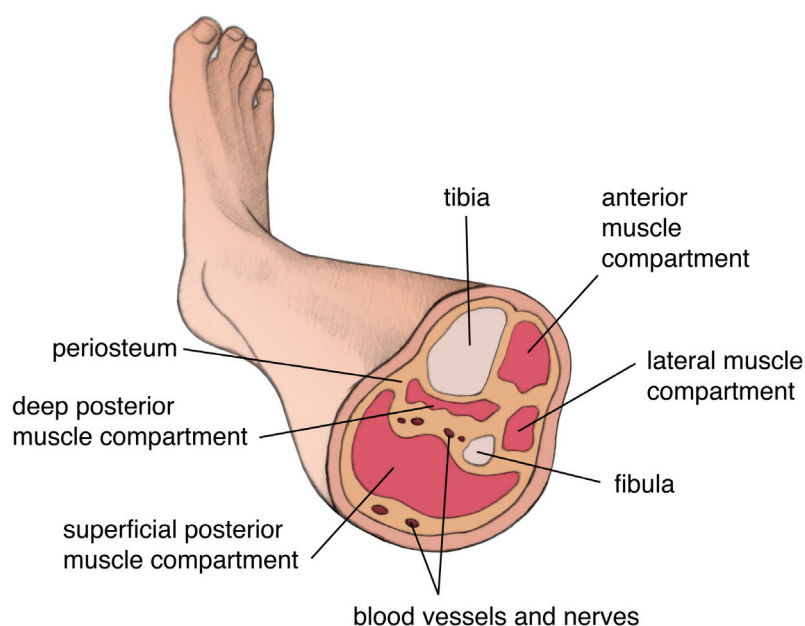
The pain consistently manifests itself shortly after starting an activity and continues to worsen until the athlete is forced to stop the activity. With chronic deep posterior compartment syndrome (CDPCS), the muscles of the calf may feel swollen or tense upon physical exam, especially immediately after exercise. Pain may be present upon aggressive palpation or passive ankle dorsiflexion. Pain, numbness along the posterior-medial aspect of the calf, and weakness in toe flexion, ankle inversion and plantar flexion, may continue for some time after exercise, but usually resolve with a day's rest, only to re-appear when the athlete returns to training. The pain occurs bilaterally in 80-95% of athletes with CECS, and involves the deep posterior compartment in 32%-60% of all cases of CECS⁽¹⁾.

Anatomy

There are four fascial compartments within the lower leg: anterior, lateral, superficial posterior, and deep posterior (see Figure 1). A compartment consists of a fascial sheath and the contents within – muscles, nerves, and blood vessels. Some consider the tibialis posterior to be a compartment unto itself because of its own fascial covering, but for practical purposes, it is included in the deep posterior compartment. Chronic exertional compartment syndrome most often affects the anterior compartment, followed by the deep posterior compartment in frequency⁽¹⁾.

Within the deep posterior compartment lie the tibialis posterior, flexor digitorum longus, flexor hallucis longus, and popliteus muscles. The posterior tibial nerve, artery, and vein also course through the deep posterior compartment. The muscles of the

Figure 1: Muscular compartments of the lower leg



The deep posterior compartment of the lower leg is situated deep within the calf, beneath the gastrocnemius and soleus muscles and nearly sandwiched between the tibia and fibula. In effect, this compartment is 'land-locked' within the anatomy of the leg.

posterior compartment assist with inversion and plantar flexion of the foot and the posterior tibial nerve (L5-S1) innervates them.

Under pressure

Compartment syndrome occurs when the pressure of the fluid inside the compartment becomes so great that it restricts blood flow into the muscles within the compartment. Acute compartment syndrome, caused by extreme swelling after a traumatic event, is a medical emergency and requires immediate treatment with a fasciotomy before the lack of blood flow results in tissue death. Chronic exertional compartment syndrome also occurs due to the build-up of pressure within the compartment, but differs from the acute process in that the increased pressure occurs with exercise, and resolves when the activity is stopped.

During strenuous exercise, muscle volume normally increases up to 20%⁽²⁾. If

the muscle is hypertrophied or the fascia is less compliant, there is less room within the compartment for expansion and the pressure within the compartment increases. It is assumed that, as in acute compartment syndrome, the flow of oxygenated blood into the muscles is impeded if the pressure within the muscle is greater than the fluid pressure within the vessels themselves. To measure this pressure, clinicians use a needle catheter inserted into the compartment. The pressure is generally measured at rest, one minute after exercise, and five minutes post-exercise.

The only way to accurately diagnose CECS is by measuring the pressure within the compartment. This is done using a needle catheter inserted along the under surface of the medial tibial border. Measurements of more than 15 mmHg at rest, 30 mmHg one minute after exercise, or 20 mmHg at five minutes post-exercise signify CECS⁽²⁾.

Low flow

Theoretically, the pain with CECS is caused by the resulting ischemia, or cell death, within the muscles and nerves when blood flow is compromised. Studies attempting to confirm this theory fail to demonstrate the ischemic changes consistent with such levels of pain, except under extreme tissue pressure ($\geq 160\text{mmHg}$)⁽²⁾. Calling the ischemia theory into question, researchers in Victoria, Australia examined 34 patients under thallium-201 single-photon emission tomography⁽³⁾. This imaging measured the perfusion within the muscles of the offending compartments. Twenty-five of the patients in the study had CECS confirmed by elevated compartment pressure, while nine had normal pressure but positive leg pain and served as controls. The researchers found no significant difference between the perfusion of those with CECS and those without, suggesting there is another aetiology for the pain experienced.

Alternative theory

Normal fascial tissue is a thick connective tissue with little elasticity. Some theorise that in individuals with CECS, the fascia surrounding the compartment is less compliant than in others, due to repetitive loading on the fascia and its attachments to the bone. To discover what type of histological changes may occur within the fascia in these individuals, researchers at the University of Melbourne explored the cellular nature of the fascia in individuals with CDPCS⁽¹⁾. In this case-controlled study, 10 males and 9 females with CDPCS underwent fasciotomy after conservative measures (not defined within the study) failed to improve the condition. Tissue samples were taken from the fascia of the deep posterior compartment and compared with control tissue samples taken from eleven autopsy subjects.

Interestingly, while the tissue samples of each of the subjects with CDPCS differed from one another, they did not differ significantly from the controls in measures of fibrocytic activity, chronic inflammatory cells, or vascular proliferation as assumed. However, the subjects differed significantly from controls in the degree of collagen organisation. The researchers were surprised to find that the collagen in the fascia of the subjects showed more regular organisation than that of the controls. While the exercise history of the cadaver subjects was not available, it was assumed that their collagen would be more

organised. Instead, the alignment in the cadaver fascia was quite irregular.

Researchers hypothesised that the regularity found in the collagen arrangement in subjects with CDPCS was due to the chronic remodelling process experienced as a result of continuous strain on the fascia. This study was small, measured only a few variables, and the controls were not equally matched for activity with the subjects. However, noting changes within the fascia warrants further exploration to determine if the pain from CDPCS occurs due to the stimulation of the pain receptors within the fascia or the periosteum where it attaches to the bone, rather than ischemia.

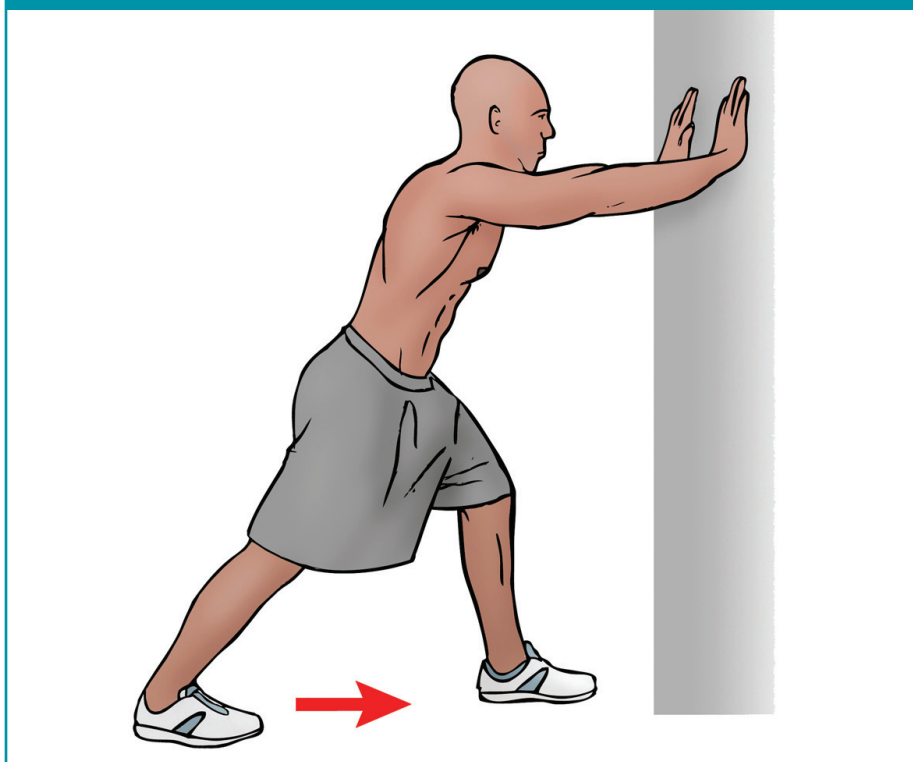
Treatment options

It's possible, then, that repeated strain and remodelling of the fascia, as hypothesised by the researchers at the University of Melbourne, decrease the pliability of the fascia⁽¹⁾. Therefore, stopping the repeated strain and increasing fascial compliance would seem to be the targets for treatment. Biomechanical analysis may reveal long-standing patterns of movement that have added stress to the calf over a prolonged period of time.

Exploring this premise, researchers in West Point, NY, evaluated the running technique of ten armed servicemen with a diagnosis of CECS of the anterior compartment⁽⁴⁾. Each of these patients was awaiting fasciotomy for the treatment of the CECS. Each underwent a six-week program of running re-training to learn a forefoot-strike running technique. The program consisted of running analysis, drills, exercises, and feedback. After six weeks, post-run anterior compartment pressures and reported pain scores decreased significantly, while running distances increased significantly in all subjects. Positive results continued for one year after intervention in all ten subjects.

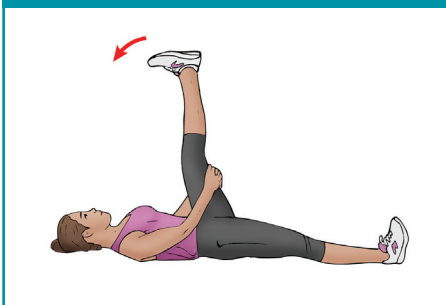
This study was limited in size, analysed patients with anterior compartment CECS, not CDPCS, and did not include subject controls with whom to compare results. However, all subjects avoided surgery with a six-week intervention program of three 45-minute sessions per week. This speaks dramatically to the need for biomechanical analysis of all athletes with CDPCS. If the cause of strain can be eliminated, the hypothesis is that the fascia can heal and function normally again.

Figure 2: Stretch for the deep posterior compartment



Have the athlete perform this calf stretch for limited range of motion in the ankle or tightness in the calf. Leaning against a wall, lunge forward with the unaffected leg until a deep stretch is felt in the calf. Hold for up to a minute, performing several reps once to twice a day.

Figure 3: Neural glide for the tibial nerve



To mobilise the tibial nerve within the deep posterior compartment, perform the above exercise from five to ten reps daily. Supporting the back of the leg as needed, flex and extend the knee, pointing the toes in plantar flexion, especially at the top of the arc of movement.

The biomechanical analysis should include an evaluation of strength, range of motion, skeletal alignment, and equipment, including shoes, orthotics, and running surfaces. Fascial compliance and nerve mobility can be evaluated and treated with stretches and exercises (see *Figures 2, 3 and 4*). Training schedules should also be assessed as year-round sport eliminates the opportunity for

off-season cross-training and a break from continuous strain.

Easing the pressure

In acute compartment syndrome, the way to relieve the build-up of pressure and save the tissue within the compartment from ischemia is to release the fascia through a fasciotomy. The assumption for the surgical management of CECS is the same, that releasing the fascia will normalize the perfusion of the compartment and relieve the pain. Current research, showing perfusion deficits do not exist within the compartment, questions the perfusion theory of pain, and thus the benefit of fasciotomy.

A researcher at the University of California reviewed the literature on the effectiveness of fasciotomy for CDPCS⁽⁵⁾. Her review revealed that the often quoted 80% success rate of fasciotomy treatment for CECS only included rates for treatment of the anterior compartment, not the deep posterior compartment. She proposed, therefore, that a difference exists between the success of anterior and deep posterior compartment fasciotomy, as determined by patient satisfaction.

Seven studies met criteria for inclusion in the meta-analysis. The review revealed

that there was a significant difference between the outcome satisfaction of those with anterior compartment fasciotomy (83%) and those with deep posterior compartment fasciotomy (56%). Closer scrutiny revealed studies with patient satisfaction levels at 75%-100% satisfied reported return to activity levels of only 50%-75%⁵! In addition, the incidence of complication from the procedures in the studies reviewed ranged from 4% to 90% of cases.

There are several factors that may contribute to the low satisfaction scores. Since decreased perfusion is likely not the cause of pain in CDPCS, a fasciotomy may not address the problem at all. Accessing the deep posterior compartment is difficult, thus releasing the fascia along the entire compartment is challenging. Surgeons may not truly release the entire compartment in all cases. Rehabilitation standards also differ among practitioners, thus adding incomplete rehabilitation as a possible contributor to poor satisfaction outcomes.

Conclusion

Surveys show that 26%-33% of athletes with exercise induced lower leg pain have CECS in one of the compartments of the lower leg⁽¹⁾. Pain on exertion in the calf of the lower leg is often caused by CDPCS. The prior assumption that the onset of pain after 20-30 minutes of exercise is due to increased pressure and decreased blood flow within the compartment is now under scrutiny. More likely, fascial strain is the cause of pain and the resulting decreased compliance is what causes the increased compartment pressure⁽²⁾. To improve outcomes using conservative treatment, follow a systematic rehabilitation program, with emphasis on biomechanical analysis and correcting the factors that may have instigated the problem in the first place, namely, biomechanical deficits in running technique, fascial and nerve immobility, decreased range of motion, and muscle weakness.

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Figure 4: Mobilisation of the fascia of the deep posterior compartment



To mobilise the fascia of the deep posterior compartment, palpate just posterior to the tibia on the medial side of the leg. In sitting with knee flexed or in $\frac{1}{2}$ kneeling, place a golf ball in this area and one directly opposite on the other side of the leg. Slowly apply pressure medially to the area, while flexing or scrunching the toes and inverting the foot. Hold the pressure while moving the foot repeatedly, and then progress the balls along the tibia holding each spot for several seconds. This stretch can be performed as part of the therapeutic regimen, and may be helpful prior to activity.

Rehabilitation

Rehabilitation masterclass – AC joint reconstruction (part 2)

Chris Mallac takes you through the second part of his Rehabilitation Masterclass for the surgically repaired acromioclavicular joint (ACJ).

Surgery for ACJ injuries

Type III injuries and type II injuries in the high-level throwing athlete is the start of the spectrum for the decision to operatively stabilise the ACJ. This is usually determined on a case-by-case basis, and the criteria for surgery vs conservative management may be based on:

1. Previous injury to the ACJ that has rendered the joint a little degenerative (new on old injury).
2. For those in high-risk sports (contact sport, combat sports, motocross) where the risk for re-injury may be quite high, the initial preference is to treat the ACJ conservatively. If the ACJ is re-injured, this may then push the surgeon to consider a surgical stabilisation.
3. In throwing sports where this is the dominant arm, early surgery may be preferred to avoid any unwanted sensations of ACJ instability or clicking and popping in the ACJ due to the high biomechanical loads imposed on the joint.
4. Arm dominance. Injuries to the ACJ on the dominant side may be a determining factor in early surgery.
5. Degree of instability. Instability in the antero-posterior direction tend to do poorly with conservative management compared with the up-down type instabilities.

The decision to manage Type III injuries surgically versus non-surgically still remains controversial. Some researchers have found that the outcome following surgical versus non-surgical ACJ injuries is quite similar (Calvo et al 2006).

If the decision is to delay surgery on a Type II and III ACJ injury, then the usual time frame is three months of conservative rehabilitation. If the athlete complains of residual pain, sensations of instability or an inability to perform sport at previous levels of function, then surgery is then considered.

The more serious type IV, V and VI will always need surgery.

Types of surgery

There are four basic types of surgical

procedures that have been described for treatment of ACJ injuries. These include:

(1) Primary repair of the AC joint with pins, screws, plates, tension band wiring or rods.

This procedure involves an open repair of the ACJ using a host of fixating options. These may be done with or without CC ligament reconstruction. A comparative study performed by Sugathan and Dodenhoff (2012) found that tension band wiring, although preferable over a Weaver-Dunn procedure (see below) in terms of ACJ strength and functional outcome in acute ACJ injuries, had greater risk of early post-operative complications compared to the Weaver-Dunn procedure and the need for future surgery to remove any metal work in and around the ACJ. They recommended the Weaver-Dunn procedure, particularly in those with failed conservative management.

(2) Distal clavicle excision with soft tissue reconstruction (Weaver-Dunn).

This procedure involves resection of the distal clavicle followed by release of the CC ligament from its attachment on the acromion. The detached end of the ligament is then attached to the distal clavicle to help hold it in a reduced position. Transfer of the conjoined tendon, where the lateral half of the tendon is transferred to the distal clavicle, has recently been described. Transfer of the conjoined tendon has been argued to be superior to the original Weaver-Dunn technique because the functioning CC ligament is left intact.

(3) Anatomic coracoclavicular reconstruction (ACCR).

The ACCR procedure entails a diagnostic shoulder arthroscopy and arthroscopic distal clavicle excision. The AC ligament is detached from its acromial insertion and tied to the distal clavicle through two drill holes. An autograft (donor site being the gracilis or semitendinosus) or an allograft is then looped underneath the coracoid and through two drill holes in the clavicle. The

graft is then tied to itself in a figure-of-eight fashion or fixed to the clavicle with interference screws. Several biomechanical studies have been completed which illustrate that ACCR more closely approximates the stiffness of the CC ligament complex and produces less anterior to posterior translation at the AC joint compared with the Weaver-Dunn procedure.

(4) Arthroscopic suture fixation.

Two types of surgical techniques for restoring the CC ligaments without a graft exist. The first technique involves using two suture anchors through four drill holes in the clavicle for fixation. The suture anchors are fixed in the coracoid and tied over a bone bridge in the clavicle. As part of this procedure the CC ligament is transferred as well. The second type of procedure involves using two tightrope devices to reconstruct the CC ligaments through two single tunnels in the clavicle and coracoid.

Post-operative rehabilitation

Irrespective of the surgical procedure used, the post-operative rehabilitation protocol will be similar for all surgical types. The major point of difference will be that if screw/plate fixation has been used these will usually be removed at around eight weeks post-operatively.

Stage 1: protection and immobilisation (0-6 weeks).

The majority of surgeons would request a conservative six-week period of complete sling immobilisation to allow full tissue healing without any unwanted stretch on the reconstructed ligaments or augmentation devices used in surgery. This differs greatly with other major shoulder surgeries such as shoulder reconstructions and rotator cuff repairs whereby the surgeon encourages pendulum type exercises in these types of shoulder surgeries early in the rehabilitation phase. The concern with sling removal in the early stage is that the weight of the arm and scapular provide a significant traction force to the ACJ, and if

this is allowed to occur in the early stages, then the ACJ may end up with excessive post-operative laxity. To avoid this, most surgeons will advocate no pendulum in the first six weeks and not allow the arm to be unsupported whilst in the upright position.

The goals therefore at this stage are:

1. Allow healing of soft tissues;
2. Decrease pain/inflammation;
3. Early protected range of movement;
4. Retard muscle atrophy in scapular stabilisers.

For the first two weeks, the sling can be removed for hygiene purposes only. At two weeks post-op, the patient may start passive range of movement (therapist-guided) or active assisted (patient-guided) flexion and abduction movements whilst lying in supine. These flexion and abduction movements are slowly progressed to 70° from week two to six as pain allows. Usually internal and external rotation can be pushed to the limits as long as pain allows. Extension movements are avoided in this early stage as this movement produces the greatest amount of stress on the ACJ.

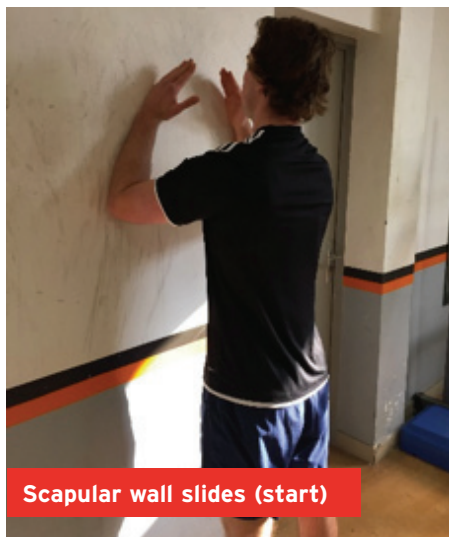
Soft tissue work to the pec major/minor, the latissimus dorsi and subscapularis if the arm can be abducted comfortably away to expose these muscles are usually also started early. Due to the restriction on pendulum exercises in the ACJ-reconstructed shoulders, the arm tends to 'stick' to the side quite easily due to soft tissue contracture and adhesive capsulitis in the shoulder joint. Therefore, if the therapist is able to access the shoulder comfortably, then gentle passive mobilisations of the shoulder joint (physiological as well as accessory) are allowed for the glenohumeral joint.

Gentle scapular setting exercises can be performed in a supported sitting position with the sling in situ. Only allow pain-free ranges of retraction and depression. These can be held as 10-second isometric contractions. This can be enhanced with muscle stimulators placed on the lower trapezius and the stimulator set to an 'atrophy' mode.

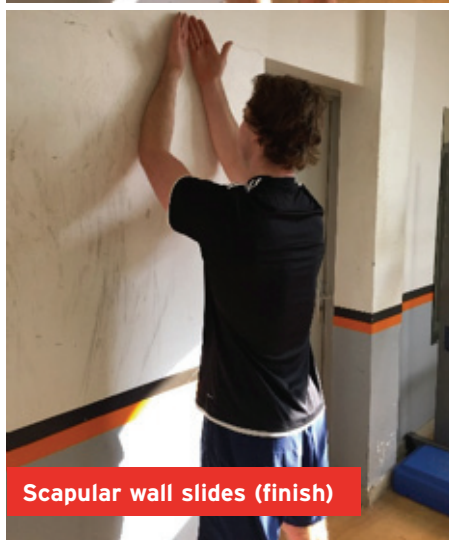
Similarly, muscle stimulators can be used on the deltoids and pec major in an 'atrophy' mode. In supine lie, the patient may start gentle isometric shoulder abduction and rotation exercises at four weeks post operative.

Exit criteria for stage 1

1. Minimal pain and inflammation in the ACJ.



Scapular wall slides (start)



Scapular wall slides (finish)

Stage 2: regain range of movement (7-12 weeks).

The primary goals in this stage are:

1. Gradual increase in range of movement;
2. Gradual increase in isometric strength;
3. Maintain pain-free ACJ and minimal inflammation.

The sling is usually discarded at six weeks post-op. Due to the severe restrictions placed on movement in the first 6 weeks, the usual progression of movement is to allow active assisted flexion and abduction in weeks 7 and 8, and then progress to active only in weeks 9 through to 12. Rotation movements with the arm by the side can be progressed unrestricted early; however, extension is still avoided until 10 weeks post-op. It is expected that the patient will have achieved 90% of range of movement into flexion, abduction and hand behind back by week 12 post-op.

Isometric deltoid, pec major and lat dorsi can be progressed at this stage in neutral and pain-free positions; rotation strength can be worked through range

with therabands. More aggressive prone lying scapular retraction and depression drills can also be progressed early in this stage.

As the patient achieves comfortable ranges of shoulder flexion, gentle wall slide exercise can be started to actively strengthen the serratus anterior. To perform a wall slide exercise (*see image left*) start with the forearms in contact with the wall. Gently slide the forearms up the wall above the head, slowly externally rotating the arms/forearms on the way up. This will create scapula upward rotation and protraction, a great exercise to activate the serratus anterior, a necessary muscle in the control of scapula movement.

For the athlete involved in a running sport, treadmill running with the affected arm holding onto the hand grip is allowed from week 7 onwards. Due to the difficulty with this running technique, running velocities have to be limited to 12-14 km/hour. In weeks 9 and 10, on-field running is allowed with the arm kept locked in by the side to minimise excessive shoulder flexion and extension movements. Full running is allowed in weeks 11 and 12 and high speeds can be slowly progressed. It is difficult to reach top end speeds in this stage due to the aggressive flexion and extension of the shoulder required in the arm drive phase, therefore speeds can be curbed to 80% maximum.

Exit criteria for stage 2

1. Range of movement achieves 90+%.
2. No residual pain in ACJ one hour post-exercises.
3. No night pain in the ACJ.
4. Pain-free running at 80% speed.

Stage 3: strengthening phase (13-16 weeks).

The primary goals in this stage are:

1. Regain full range of movement.
2. Regain 90+% pre-injury pulling strength.
3. Regain 70% pre-injury pushing strength.
4. Improve neuromuscular control.
5. Integrate skill components into rehabilitation.

Range of movement which should be close to 90+% at 12 weeks post-operative is now pushed into end of range positions. This can be done with a lot of athlete directed self-stretching for the global mobilisers such as pectoralis major/minor and latissimus dorsi and local

rotator cuff flexibility in infraspinatus. Furthermore, therapist-directed deep tissue myofascial releases to restricted muscles as well as more aggressive ACJ and glenohumeral joint mobilisations can be used to improve arthrokinematics of the affected joints.

More traditional strength work is now started or progressed if started earlier. As a rule of thumb, regaining gym-based strength in an ACJ is quite similar to regaining strength in a glenohumeral joint. It should progress based on movement directions. The order of movements directions that can be safely progressed, and a new direction added weekly are:

1. Horizontal pulling (for example, seated rows, prone flies, prone pulls, 1 arm rows).
2. Vertical pulling (close grip pulldowns, 1 arm pulldowns, lat pulldowns, chin up variations).
3. Horizontal pushing (push-up variations, bench/dumbbell/cable presses, incline bench).
4. Vertical pushing (dumbbell/barbell shoulder press, lateral/front raises).
5. PNF diagonal patterns (flexion/abduction/external rotation to extension/adduction/internal rotation).

It is expected that by the end of week 16 most of the movement directions have been re-introduced however the strength of the pushing movements will only be around 70% of pre-injury levels. Furthermore, any heavy traction movements to the shoulder such as deadlifts are also avoided at this stage. Lighter deadlifts with the scapular held in retracted positions may be started, however most of the posterior chain strength work will need to be performed away from deadlifts.

Medium to high level proprioceptive work can also be integrated into this stage with exercises such as:

1. Swiss ball arm wrestle.
2. Push-ups on instable surfaces.
3. Bodyblade type shoulder exercises.

For the contact sport athlete involved in hand-ball type sports such as rugby, AFL, basketball, skills can now commence in non-contact situations.

Exit criteria for stage 3

1. Full painless range of movement.
2. Pain-free scarf test.
3. Pulling strength 90% pre-injury.
4. Pushing strength 70% pre-injury.
5. Pain-free running at full speed.

Stage 4: return to sport phase (16-24 weeks).

The primary goals in this stage are:

1. Maintain painless full range of movement.
2. Regain 90+% pre-injury strength.
3. Integrate back into full training/contact training.

This phase is a continuation of phase 3 in that the athlete is still progressing back to full shoulder strength whilst in parallel increasing return to full training. Pushing movements can be really progressed in this stage to regain 90+% of pre-injury strength. The athlete should have full painless range of shoulder flexion, extension, abduction, hand behind back and horizontal flexion (scarf test).

If the athlete is involved in a contact sport such as rugby, American Football, AFL, MMA/wrestling then the decision to start controlled contact is also a decision based on certain criteria. Prior to starting full contact, the athlete should be able to perform:

1. Pain-free clap push-up;
2. Pain-free bench dip.

These two movements impose a high tensile and compressive force on the ACJ therefore they are good screening movements to ascertain if the ACJ has fully recovered from injury and surgery.

Exit criteria for stage 4

1. Full painless range of movement.
2. Pain-free scarf test/clap push-up/bench dip
3. Pulling strength close to 100% pre-injury.
4. Pushing strength 90%+ pre-injury.
5. Completed full contact training.

Return to contact training

Staging an ACJ-injured athlete back to a full competitive training situations requires a stepwise progression of drills and skills that resemble the demands of the competition whilst still allowing appropriate protection of the shoulder/ACJ at critical stages of recovery. A logical way to prepare the athlete to develop match readiness is to modify the training environment from safe and controlled situations initially to more advanced game-specific events as they progress. For example, starting in kneeling positions and then progressing to standing, walking and running positions allows the athlete to confidently practise contact components without fear of further ACJ injury.

Oveleaf is an example of how an ACJ-injured athlete would progress contact situations for a combative sport such as rugby.

Conclusion

Returning an athlete back from a surgically reconstructed ACJ is similar in content and time frame to other shoulder surgeries except for a few key differences. Firstly, the initial six-week protection stage is far more important to adhere to in the ACJ-repaired athlete as early movement out of the sling may lead to traction on the joint that may render the ACJ hypermobile in the early post-operative stage. Furthermore, the progression of functional range of movement is also different to other shoulder surgery in that rotation movements are allowed early; however, extension is avoided for the first 10 weeks. Following these slight differences, the remainder of the rehabilitation process is quite similar in content to other shoulder surgeries in the development of range of movement, strength and also return to sport guidelines especially contact in training.

The later stages of rehabilitation will be highly dependent on the sport chosen. For the throwing athlete, appropriate interval throwing has to be woven into the last stages of rehabilitation, similarly with the pitching, tennis, golf and swimming. The contact sports athlete has a host of other complicating integrations that are not an issue with non-contact athletes.

Most of the ACJ-repaired athletes can return to full sport participation within six months of surgery depending on the sport played. Some non-contact sports may be back competing at 14-16 weeks post-operative. Power athletes may take much longer and sometimes up to nine months post-operatively.

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Stage	Intensity	Mode	Aims	Content
1	Low	Kneel	Simple contact/collision in knee-protected positions	1. Falling Mechanics 2. Wrestling Mechanics 3. Impact Absorption 4. Forward Hits 5. Fending
2	Low	Stand	Simple contact/collision in static stance	1. Falling Mechanics 2. Wrestling Mechanics 3. Impact Absorption 4. Forward Hits 5. Fending
3	Low	Walk	Simple contact/collision in safe and controlled walking situations	1. Falling Mechanics 2. Wrestling Mechanics 3. Impact Absorption 4. Forward Hits 5. Fending 6. Hit and spinning
4	Medium	Walk-Jog	Progressions to game simulation in walking	1. Down + Ups 2. Specific Wrestling 3. Being Tackled/Hit in Diff Situations (High-Low) 4. Double Combined Efforts 5. Footwork (Attack + Defence)
5	Medium	Jog	Increase impact forces	1. Down + Ups 2. Specific Wrestling 3. Being Tackled/Hit diff situations 4. Double Combined Efforts 5. Footwork
6	Medium	Run	Increase impact forces	1. Down + Ups 2. Specific Wrestling 3. Being Tackled/Hit in Diff Situations 4. Double Combined Efforts
7	High	Run	Match situations	Combination of different areas of contact and running WITH CONDITIONING COMPONENT
8	High	Sprint		Position-Specific WITH CONDITIONING COMPONENT
9	High	Maximum		Position-Specific WITH CONDITIONING COMPONENT

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Wrist injuries

Ulnar impaction syndrome

Studies suggest that between 3 and 9% of all sports injuries involve the wrist and/or hand.

Andrew Hamilton looks at ulnar impaction, one of the more common injuries to affect this region, especially among older athletes...

Ulnar impaction syndrome (UIS – sometimes called ulnocarpal abutment) is a condition in which the ulna of the forearm is too long relative to the radius, resulting in excessive loading on the ulnar side of the wrist. In most cases, this condition is congenital and present from birth, but sometimes ulnar impaction syndrome can be secondary to shortening of the radius after a fracture. Regardless of the origin, however, most patients only become symptomatic in later life, when accumulated and degenerative wear and tear takes its toll on the ligaments and cartilage, causing ulnar-sided wrist pain. For athletes whose sports involve loading of the upper limbs, this can be a particular problem.

Wrist stability

To fully appreciate how ulnar impaction can result in ulnar-sided wrist pain, it helps to understand the structure and role of the triangular fibrocartilage complex (TFCC) and loading across the ulnocarpal joint (*see Figure 1*). Ulnar-sided wrist stability is enhanced via the TFCC, an arrangement of ligaments and fibrocartilage originating from the sigmoid notch on ulnar border of the radius and inserting into the base of the ulnar styloid and fovea of the ulnar head.

Studies have shown that there is a direct relationship between increasing ulnar length (relative to radius length) and increased force transmission across the TFCC. In a neutral wrist, the ulnocarpal joint takes around 18% of the total load applied to the wrist (with the radiocarpal joint taking the other 82% or so). However, a positive variance of 2mm will increase the ulnocarpal load to approximately 40%, while an increased dorsal tilt due to previous injury of the radius can further increase the ulnar load to 65% of total load transferred^(1,2). In addition, thinning of the articular disc (which is common with increased ulnar length) also increases the risk of TFCC wear and perforation⁽³⁾.

While it's most commonly associated with congenital or acquired positive ulnar variance, UIS can also occur in ulnar

neutral or even negative ulnar variance wrists^(4,5). Athletes performing power and/or gripping tasks associated with axial loading and rotation are particularly at risk of ulnar impaction syndrome because of the 'dynamic ulnar variance' that occurs during tasks requiring maximal grip and pronation⁽⁶⁾. More generally, athletic events that place repetitive compression and rotation demands on the upper limbs increase the risk of ulnar impaction via traumatic development.

Although symptoms of UIS rarely present in younger athletes, the risk for these symptoms in later life may be increased by events during these formative years. One reason for this is that distal radius fractures are the most frequently occurring fracture in children under the age of sixteen. Research shows that when significant radial shortening (5mm or more) occurs as a result of such fractures, there's a greatly increased risk of long-term functional impairment⁽⁷⁾. Moreover, even in the absence of distal radius fractures, we know that submitting an immature wrist to prolonged compression and repetitive micro-trauma has can lead to a premature arrest of radial growth plate and subsequent ulnar

overgrowth^(8,9), which of course greatly increases the risk of UIS in later years.

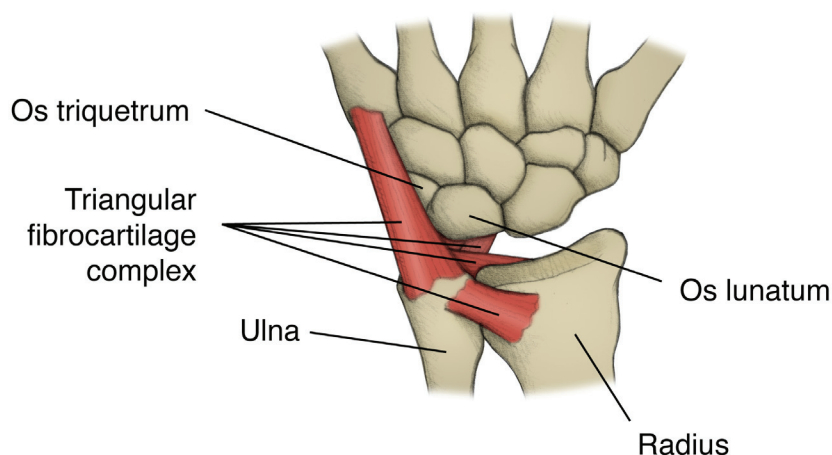
Symptoms of ulnar impaction syndrome

The development of UIS leads to the progressive degeneration and increased abutment of the distal ulna or TFCC against the ulnar carpus. Although any athlete can suffer from this condition, gymnasts, boxers, racquet and stick sport athletes are particularly at risk, with symptoms of pain particularly occurring during wrist rotation. It's important to understand, however, that the development of this condition is not always linear; the load-bearing demand placed on the TFCC means that there's an increased susceptibility towards an acute traumatic injury, as well as the secondary degenerative concerns implicated with ulnar impaction⁽¹⁰⁾.

Common symptoms of ulnar impaction syndrome include the following:

- Pain (especially during rotation), aggravated with activity and (generally) relieved with rest;
- Painful clicking or locking during pronation and supination;
- Occasional edema;
- Decreased wrist range of motion;

Figure 1: diagrammatic representation of the ulnocarpal joint and triangular fibrocartilage complex (TFCC)



- Decreased forearm rotation;
- Tenderness to palpation dorsally, just distal to the ulnar head and just volar to the ulnar styloid process;
- All of the above tend to be aggravated by forceful grip, forearm pronation, and ulnar deviation.

What tends to distinguish chronic ulnar impaction syndrome from an acute TFCC injury (which may itself be made more likely by ulnar impaction) is the insidious, progressive nature of the pain, which gradually limits range of motion, grip strength, and performance. In 1981, Palmer and Werner introduced (a now widely used) classification system to help clinicians determine whether a TFCC injury is primarily progressive and degenerative or acute in nature (or indeed both). This is shown in Box 1.

Diagnosis

When attempting to make a diagnosis of UIS, a comprehensive wrist examination is needed, together with a detailed patient history (for example, has the patient suffered a radius fracture in the past?). Unfortunately, however, there's no single clinical test that can fully diagnose UIS, not least because many tests performed in the clinic are inconclusive as to whether TFCC-related pain is acute or degenerative in nature (*see Box 1*). For this reason, diagnostic imaging (*eg* MRI) should be performed to support the findings from the clinical exam. Having said this, the clinician can gain valuable supporting evidence from a thorough examination that includes the following:

On palpation, is there:

- Tenderness dorsally just distal to ulnar head?
- Tenderness just volar to the ulnar styloid process?
- Positive ulnar variance, while static or dynamic?

Do range of motion tests show:

- Painful passive ulnar deviation and forceful pronation?
- Decreased flexion, extension, radial & ulnar deviation?

Does a strength measurement reveal:

- Decreased grip strength compared to the unaffected wrist when using dynamometer?

Is an ulnocarpal stress test positive? (*see Box 2*)

Box 1: Palmer and Werner Classification of traumatic and degenerative conditions of the TFCC⁽¹¹⁾

Class I: Traumatic

- A Central perforation
- B Medial avulsion (ulnar attachment) with or without distal ulnar fracture
- C Distal avulsion (carpal attachment)
- D Lateral avulsion (radial attachment) with or without sigmoid notch fracture

Class II: Degenerative (Ulnocarpal Impaction Syndrome)

- A TFCC wear
- B TFCC wear + lunate and/or ulnar chondromalacia
- C TFCC perforation + lunate and/or ulnar chondromalacia
- D TFCC perforation + lunate and/or ulnar chondromalacia + L-T* ligament perforation
- E TFCC perforation + lunate and/or ulnar chondromalacia + L-T* ligament perforation + ulnocarpal arthritis

NB* L-T = lunotriquetral ligament

Box 2: Diagnostic testing for ulnar impaction syndrome

Although additional imaging is often required to confirm a diagnosis of UIS, the ulnocarpal stress test and the gripping rotary impaction test (GRIT) can provide clinicians with useful supporting evidence⁽¹²⁾.

Ulnocarpal stress test – The clinician places the patient's wrist in maximum ulnar deviation and then applies an axial load to wrist followed by a passive rotation of the forearm through supination and pronation. A positive test will reproduce the patient's pain. However, while this test is sensitive for ulnar impaction syndrome, other pathologies, such as LTIL injury, TFCC injury or isolated arthritis can also produce a (false) positive result.

GRIT – The patient's arm is placed by their side with the elbow flexed at 90 degrees. Using a grip dynamometer, the clinician measures grip strength in three positions: neutral, full supination and full pronation. The GRIT ratio is then calculated as follows: supinated grip strength ÷ pronated grip strength. A GRIT strength ratio greater than 1.0 indicates ulnar impaction syndrome.

Is the Gripping Rotary Impaction Test (GRIT) positive? (*see Box 2*)

The ulnocarpal stress test described in Box 2 was originally introduced by Nakamura and his colleagues⁽¹³⁾. In the authors' original study, 33 of 45 patients (73%) with positive ulnocarpal stress test results demonstrated positive ulnar variance of 1mm or more on the affected wrist. In the 33 patients who had a positive ulnar variance, 19 (58%) were confirmed as suffering from class II TFCC lesions resulting from ulnocarpal impaction. The majority of these patients suffered a spontaneous onset of pain, and were diagnosed with class IIB lesions involving TFCC wear with lunate and/or ulnar chondromalacia (*see Box 1*). More generally, a detailed history of spontaneous ulnar sided wrist pain combined with provocative testing should prompt the astute clinician to seek further evaluation using imaging.

Treatment options

When treating athletes with UIS, earlier is better; studies show that early diagnosis and intervention may significantly reduce the risk of long-term disability and injury progression^(14,15). Conservative treatment should be attempted before surgery and can include immobilisation or limiting aggravating movements such as pronation, gripping and ulnar deviation for 6-12 weeks. Following immobilisation/restriction, other conservative treatment options include, non-steroidal anti-inflammatories (NSAIDs) and corticosteroid injections.

However, while conservative treatments such as anti-inflammatories combined with immobilisation or restricting range of movement may be reasonably effective for the general population, they are often insufficient for athletes because they don't address the fundamental biomechanical factors that predispose the athlete to UIS. Therefore, when conservative management fails to produce a significant improvement, evidence suggests that surgery is indicated^(16,17).

When surgery is required, many athletes opt to postpone surgery until the close of the season, allowing recovery from surgery to take place during the off-season. In terms of surgical options, this is best determined by the surgeon after thorough radiologic screening. These options may include:

- **Ulnar shortening osteotomy** – the ulna is shortened by 2-3mm of shaft and fixated

with a tubular or standard compression plate. This option is indicated when there is ulnar wrist pain worsened by rotation and ulnar deviation, a positive ulnocarpal stress test, and positive ulnar variance with or without cystic changes.

● **Arthroscopic wafer procedure** – this procedure uses arthroscopy to debride the central triangular fibrocartilage complex tear, along with debridement of the distal pole of the ulna causing the impaction. The debridement of the ulna is performed to the degree at which the patient is ulnar neutral or slightly ulnar negative. The benefit of this procedure is that open surgery is not required, and recovery is faster. However, if the ulnar variance is greater than +4mm, this option is not suitable.

Following surgery, athletes can expect to undergo at least 3-4 months or recovery/rehab. Depending on the surgical procedure and individual response, this period will typically consist of:

● **Weeks 1 and 2** – control pain and swelling using ice/anti-inflammatory medication. Wearing a sugar-tong splint or long-arm cast to protect the surgical site while maintaining as much range of movement (ROM) in unaffected joints.

● **Weeks 3 to 6** – Protect surgical site and continue to maintain ROM in unaffected joints. Switch to a removable elbow-

hinged splint or wrist cock-up brace. Try to increase elbow & wrist ROM. Attend to scar management.

● **Weeks 7 and 8** – Switch to removable splint worn at night. Progress to full ROM exercise for elbow & wrist and introduce isometric elbow flexion/extension and supination/pronation.

● **Weeks 9 onwards** – continue with stretching and mobilisation. Introduce low-load resistance exercises to develop strength.

Athletes should expect a return to full activity around 3-4 months after an arthroscopic wafer procedure, and around six months after an ulnar shortening osteotomy.

Summary

Although ulnar impaction syndrome is often congenital in its aetiology, advancing years and athletic activity can combine to produce progressively more debilitating symptoms in athletes. For the clinician, early diagnosis is desirable but not always straightforward; even if range of movement tests strongly suggest UIS, imaging will be required to determine whether TFCC-related pain is acute or degenerative in nature, which in turn affects treatment options. Unless the UIS is mild or the athlete is able to

significantly modify his/her activity, it may be that conservative treatment is ineffective and surgery is required. The surgical options will vary according to the individual but in many cases, the longer-term outcomes are favourable despite the requirement for extended recovery and rehab after surgery.

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