Tendinopathy in the tennis player is common and can either affect playing ability or even prevent a player from being on court. If the tissue pathology is addressed properly, traditional treatments of training load modifications and eccentric strength training that involve a functional component can be quite effective. However, secondary management with more innovative techniques may be necessary in recalcitrant cases. Surgery is not necessary in the majority of cases of chronic tendinopathy. Tennis-specific return may need to involve stroke modifications, bracing and possible volume adjustment. Long-term success with chronic tendinopathy in tennis can still be encouraging if a structured approach is utilised.

GENERAL CONCEPTS OF TENDINOPATHY

Tendinopathy generally refers to an abnormal tendon. This term is much more reflective of the actual pathophysiology of this condition, rather than the more common term of ‘tendinitis’ because there is rarely a pathological finding of ‘-itis’ or inflammation. Nirschl described ‘angiofibroblastic tendinosis’ with few inflammatory cell on histological evaluation of elbow tendinopathy. Tendinosis implies cumulative load to the tendon resulting in some degenerative changes. Australia’s Professor Jill Cook further describes the process of tendinopathy to be in three stages (Figure 1):

1. Reactive
2. Dysrepair,
3. Degenerative, where there is often a combination of all of these stages.

Ultimately, the stage of tendinopathy helps determine the treatment plan. Reactive tendinopathy is increased load from accumulation of eccentric loading or training without degenerative changes, resulting in a painful tendon. In this stage, it is most important to evaluate training load and modify or reduce the eccentric component of this load where possible. Other modifications such as patellar tendon straps or heel lifts for Achilles tendinopathy may help to unload the tendon.

Dysrepair may include stages prior to development of tendinopathy. Evaluating the rehabilitation that would contribute to tendon healing rather than worsening tendon pathology is the main goal during these earlier stages. For example, it may be better to apply loads that reduce overall pain in the earlier phases (such as isometric load or resistance training in the mid-range that is done isometrically).

Degenerative tendinopathy involves the terminal stages of tendon pathology. Degenerative tendinopathy responds best to eccentric strengthening as has been shown in multiple studies. The exception may be when there is reactive on top of degenerative tendinopathy and this type of irritable tendon would need load reduction and modifications prior to initiating eccentric strength training.

EPIDEMIOLOGY

As tendinopathy is degenerative and cumulative, it rarely occurs in players younger than 18 years old. Adult recreational players are most at risk for development of tendinopathy based on age, playing volume and in some cases, technique. A cross-sectional self-report survey of 529 USTA recreational adult league players of an average age of 46.9 years old found that there was an injury incidence of 3.0 injuries/1000 hours of play, while there was a prevalence of 53 injuries/100 players. The elbow was the most common site of injury (20% of all injuries), while the shoulder was
Injuries to the upper extremities are the second most common with 15% of all injuries. Other studies have found a 50% lifetime incidence of elbow pain in adult tennis players over 30 years old. While true 'tennis elbow' is a relatively common condition in the adult tennis player, another study which followed 700 elite players followed at the French Open over 3 years, including the seniors, had only one reported case of tennis elbow. This suggests that proper technique may be protective of this condition despite advancing age.

Rates of shoulder injury have generally been reported between 4 and 17% of players (including rotator cuff tendinopathy as well as all other tennis-specific shoulder conditions). Reports of shoulder pain may be as high as 24% in high level competitive players aged 12 to 19, and 50% in middle aged players. Other common areas for tendinopathy include the Achilles and patellar tendons. The rate of other tendinopathies in tennis players has not been as well documented as in the elbow or shoulder.

**COMMON AREAS OF TENDINOPATHY IN TENNIS**

**Elbow**

Anatomy of elbow tendinopathy

Lateral epicondylitis (or lateral elbow tendinosis) typically involves injury to the extensor carpi radialis brevis muscle but may also involve the extensor digitorum communis muscle. Medial epicondylitis (or medial elbow tendinosis) involves injury to the pronator teres and flexor carpi radialis muscles at the origin on the medial epicondyle.

**Treatment**

The treatment of these conditions most frequently depends on the stage of the tendinopathy (Figure 2).

Short-term solutions for pain control may include non-steroidal anti-inflammatory drugs, counterforce bracing, light eccentric strengthening and activity modifications which may all help to improve symptoms within the first 6 weeks. Counterforce bracing may actually limit forces to the extensor carpi radialis brevis, while addressing the true tendon pathology through isokinetic eccentric strengthening programmes has been shown to be most effective in reducing pain and even the appearance of the tendon at 1 month. A small study demonstrated that a single novel eccentric strengthening exercise using a rubber bar improved all outcome measures compared to standard physical therapy. Shoulder rotation strength is often weak in elbow tendinopathy patients, so a functional component to rehabilitation engaging the entire upper quarter and kinetic chain should be utilised as well. The addition of a steroid injection may be helpful up to 6 weeks, but isolated steroid injections were less effective than physiotherapy and 'wait and see' approach at 52 weeks, with higher rates of recurrence with steroid injection. Injection with fenestrations with limited or no steroid may address the tendon pathology better and result in longer term improvements.

In chronic cases where the symptoms become refractory, other interventional or pro-inflammatory options may need...
Diagnosis of medial epicondylitis; signs include:
- Tender medial epicondyle
- Pain with passive wrist extension
- Pain with resisted wrist flexion

OR

Diagnosis of lateral epicondylitis; signs include:
- Tender lateral epicondyle
- Pain with passive wrist flexion
- Pain with resisted wrist extension

Phase 1: Initial management
(<6 weeks symptom duration)
- Activity modifications
- Counterforce brace
- Topical and oral NSAIDs
- Passive range of motion in terminal extension and flexion with light eccentric strengthening

Improved function and pain:
- +/- brace painful activities
- Continue home exercises
- Injury prevention education
- Expectant management

Continuing or worsening function/pain:
Phase 2 treatment:
- 3-view elbow X-rays
- Formal rehabilitation with eccentric strengthening, +/- iontophoresis
- Activity modification or avoidance (work/sport)
- Counterforce brace, dynamic extension splint or volar splint
- Consider injection (+/- steroid)
- 6-week follow-up

Improved function and pain:
- +/- brace painful activities
- Continue home exercises
- Injury prevention education
- Expectant management

Continuing or worsening function:
Dx: Chronic elbow tendinosis
- Reassess and adjust rehabilitation programme
- Consider different type of brace
- Refer to chronic epicondylitis algorithm

Identify other pathology:
- Treat new diagnosis
- Referral to appropriate specialist

Figure 2: Algorithm for acute epicondylitis (epicondylodesis) (reprinted with permission from www.uptodate.com), NSAIDS=non-steroidal anti-inflammatory drugs.
to be considered (Figure 3). These pro-inflammatory options are emerging rapidly, while the scientific support is still relatively new. These options will be discussed in more detail at the end of this review with regards to all types of tendinopathy.

Nirschl found about 97% improvement with his technique of arthroscopic drilling of the lateral epicondyle and debridement of the ECRB with return to tennis after surgery within 3 months, however this was not a controlled study10. Surgical techniques may also include open repair or percutaneous releases. Most studies show minimal differences in outcomes between the techniques, although most studies evaluate treatment in mainly non-tennis playing populations10.

Tennis-specific recommendations

Equipment: Nirschl criteria were developed to measure appropriate grip size based on the measurement equal to the number of inches from the proximal palmar crease to the tip of the ring finger. Following Nirschl criteria with up to 0.25 inches of difference did not influence forces to the lateral epicondyle, but our tennis-related impression is that assessing grip size may still be a reasonable part of the tennis-specific evaluation10. While limiting vibrations to the elbow is intuitive in limiting progression to injury, this has not been effective. Modifications of vibrations with vibration dampeners are negligible as the weight of strings is about 1/10th the weight of the racquet itself. Similarly, the weight of the racquet, either too heavy or too light, seems to have little effect on development of tendinopathy. A good general rule is to use the heaviest racquet that does not affect swing speed.

Tennis stroke modifications

The development of lateral or medial elbow tendinosis may occur through one of the following mechanisms10.11 (Table 1):

1. Excess forearm pronation on serves or forehands (either lateral or medial tendinosis) (Figure 4).
2. Increased wrist extension, leading to forearm pronation or lateral tendinosis excess wrist extension at the point of contact or on the follow through on one handed backhands (Figure 4).

Table 1: Stroke modifications for shoulder and elbow pathology in tennis (table adopted with permission from CSMR). SLAP=superior labrum anterior and posterior.

<table>
<thead>
<tr>
<th>Stroke affected</th>
<th>Stroke modification</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Shoulder</strong></td>
<td></td>
</tr>
<tr>
<td>Rotator cuff</td>
<td></td>
</tr>
<tr>
<td>Impingement/tear</td>
<td>Cocking/loading phase of serve</td>
</tr>
<tr>
<td>Internal impingement</td>
<td>Follow through/ deceleration phase</td>
</tr>
<tr>
<td>SLAP/labral tear</td>
<td></td>
</tr>
<tr>
<td>Follow through/ deceleration phase</td>
<td></td>
</tr>
<tr>
<td><strong>Elbow</strong></td>
<td></td>
</tr>
<tr>
<td>Later epicondylitis</td>
<td>One-handed backhand, forearm pronation (forehand, serve, follow through)</td>
</tr>
<tr>
<td>Early eccentric phase of wrist extension or late eccentric follow through on one-handed backhand</td>
<td>Reduce forearm pronation on forehand serve</td>
</tr>
<tr>
<td>Medial epicondylitis</td>
<td>Forehand, extreme topspin</td>
</tr>
</tbody>
</table>

3. Extreme forehand western or semi-western grips that result in extreme supination with ulnar deviation of the wrist with early rapid rotation and acceleration on topspin forehands (medial elbow tendinosis) and slice/spin serves. This may often occur in the more advanced or competitive player. Ilfeld found that stroke modification combined with conservative therapy led to resolution in more than 90% of cases of tennis elbow12. Interestingly, many cases were not just painful on backhands, but also with forearm pronation on forehand and serve follow-through. The two-handed backhand may help limit the end ranges of motion of the wrist, even though measured wrist extensor activity may be the same. Volume of tennis of >2 hours/day has been associated with increased risk of tennis elbow, however there was no increase in risk of injury with up to 6 hours/weekly13. The risk of injury in adult recreational players related to playing tennis between 6 and 10 hours weekly has not been investigated.

**Rotator cuff tendon**

Tendinopathy in the tendon may be similar in appearance to partial thickness tears on the bursal or articular side. It has been postulated that bursal-sided tears and tendinopathy may be related to mechanical trauma such as a downsloping acromion, while articular-sided pathology are felt to be related to age-related degenerative factors14. Rotator cuff tendinopathy is most commonly found in the supraspinatus tendon, while infraspinatus and subscapularis may also be involved. Scapular dyskinesis may contribute to the impingement process and play a role in functional deficits during tennis overhead strokes. The rotator cuff is commonly involved and also noted to have more MRI documented degenerative changes on the dominant side of overhead athletes than the non-dominant side, even when asymptomatic.
Diagnosis of chronic medial epicondylitis (>12 weeks symptoms)
Signs include:
• Tender medial epicondyle
• Pain with passive wrist extension
• Pain with resisted wrist flexion
OR
Diagnosis of chronic lateral epicondylitis (>12 weeks symptoms)
Signs include:
• Tender lateral epicondyle
• Pain with passive wrist flexion
• Pain with resisted wrist extension

Phase 2 treatment: 6-12 weeks
• Continue or initiate eccentric strengthening
• Continue activity avoidance or modification
• Consider or repeat injection (+/- steroid)
• Injection with multiple fenestrations

Improved function and pain:
• +/- brace painful activities
• Continue home exercises
• Injury prevention education
• Expectant management

Continued or worsened function/pain:
• Review negative predictors
• Review secondary gain issues

Identify other pathology:
• Treat new diagnosis
• Referral to appropriate specialist

Referral to appropriate specialist:
Phase 3 treatment options

Advanced imaging
• MRI
• Musculoskeletal US

Alternate treatment options
• Nitroglycerin patches
• Buffered PRP injections
• Autologous blood injections
• Acupuncture
• Prolotherapy
• Botulinum toxin A injection
• Sclerosing polidocanol injections
• US-guided tenotomy

Surgical options (>6 months)
• Debridement with +/- arthroscopic drilling
• Open debridement
• Percutaneous tenotomy

Improved function and pain:
• Continue home exercises
• Injury prevention education
• Expectant management

Figure 3: Algorithm for chronic epicondylitis (epicondylosis) (reprinted with permission from www.uptodate.com) MRI=magnetic resonance imaging, PRP=platelet rich plasma, US=ultrasound.
Tennis-specific recommendations

Return to tennis following rotator cuff tendinopathy may involve some important volume and stroke recommendations. Kibler describes incorporating the kinetic chain to utilise optimum (>50%) lower body force generation\(^\text{17}\), while >10° knee bend may limit forces to the shoulder and elbow. These numbers tend to be reversed in the recreational player where upper body forces predominate. Utilising the kinetic chain on a serve may involve a deeper knee bend and more powerful hip rotation, Incorporating scapular retraction into the serve may help reduce rotator cuff impingement symptoms related to tendinopathy. Kovacs describes an eight-stage model (which include preparation, acceleration and follow-through phases) to limit upper extremity problems in the serve which also involves optimum shoulder elevation without excess

Figure 4: (a) Forearm pronation at end of aggressive forehand topspin stroke, (b) reducing forearm pronation on forehand at end of stroke, (c) increased early wrist extension with leading elbow on backhand, (d) proper one-handed backhand with linear shoulder/elbow/arm movements and reduced wrist extension.
Patient presents with non-insertional or insertional Achilles tendinopathy (<6 weeks)

Exclude rupture, other causes evaluate peritendinous structures

Phase 1: Evaluate and treat biomechanical faults (heel lift or orthotics)
Reduce total load in acute (reactive) Achilles tendinopathy

Phase 2: Begin eccentric heel-drop protocol 6-12 weeks (with some pain)

No response

Response, progress to decline, higher load, higher rate, and then functional return to tennis

Secondary options (chronic, degenerative >12 weeks): Continue eccentric strengthening
Consider GTN patch, US-guided PRP or autologous blood, sclerosants, Botulinum A

Consider US-guided tenotomy versus MRI

Surgical evaluation (more likely if insertional)

Figure 5: Achilles tendinopathy algorithm. GTN=glycerol trinitrate, PRP=platelet rich plasma, MRI=magnetic resonance imaging, US=ultrasound.

‘hyperangulation’ and external rotation, in addition to proper lower body force generation.

Achilles tendinopathy
Anatomy of Achilles tendinopathy

Tennis players are required to repetitively perform sudden eccentric contractions of the Achilles tendon with forefoot loading during most strokes, particularly when on the forefoot for volleys, split steps and directional changes on groundstrokes. The Achilles tendon may develop tendinopathy in the non-insertional and the insertional region. Non-insertional tendinopathy of the Achilles tendon may typically occur approximately 2 to 6 cm proximal to the calcaneal insertion and develop thickening, nodularity and disorganised tendon architecture. This is considered a relatively avascular region. Insertional Achilles tendinopathy may be the result of mechanical irritation such as squaring of the posterior superior calcaneus (Haglund’s
deformity) or be mistaken for retrocalcaneal bursitis, retroachilles bursitis or even posterior ankle impingement from such structures as an os trigonum.

**Treatment**

A natural history study of 5-year outcomes of Achilles tendinopathy suggests that the majority of cases have good outcomes with Alfredson’s exercise-based heel-drop eccentric strengthening programme alone. Eccentric strength training has been shown to be effective in the majority of cases on non-insertional Achilles tendinopathy, even through pain (Figure 5). The important component is the heel-drop eccentric portion of the strengthening. This should be progressive, starting with body weight loading then loading with extra weight, such as a backpack and even the speed at which it is done to deliver even higher loads.

Outcomes for insertional Achilles tendinopathy are less reliable as there may be other mechanical components involved such as a Haglund’s deformity. The heel drop eccentric strengthening programme should be used, but modified to the heel drop only to neutral in cases of insertional Achilles tendinopathy. Pro-inflammatory options have been described and will be outlined at the end of this review. Surgery should only be considered as a last resort in non-traumatic cases of Achilles tendinopathy.

Tennis-specific modifications

Heel lifts may unload the Achilles tendon during tennis. Reduction of off-court eccentric training and limiting forefoot loading and repetitive plantar flexion in training and competition is also recommended. Also, consider playing on surfaces with low friction that allow sliding (e.g. clay vs hard court) and to make sure the outsole of the shoe matches the court surface.

**Patellar tendinopathy**

Anatomy of patellar tendinopathy

Repetitive knee eccentric loading occurs where a flexed knee is extended in most strokes of competitive tennis players, particularly when utilising lower body force generation. Most pathology of patellar tendinopathy develops in the proximal patellar tendon, while mid-substance and distal tendinopathy may still occur.

**Treatment**

Eccentric strength training of the patellar tendon has been effective in treating chronic patellar tendinopathy (Figure 6). This treatment has been shown to be equivalent to patellar tenotomy in one randomised study. This effect is not seen when done in season, presumably due to excess load to the tendon. To augment results, a decline squat eccentric strengthening programme should be done at 25° and may have weights added as well to increase rehabilitative load. While numerous anecdotal cases exist of chronic patellar tendinopathy in elite tennis players, there is little tennis-specific data of this condition. Quadriceps tendinopathy may occur much less commonly and can be treated with similar eccentric strengthening as for patellar tendinopathy.

**Tennis-specific recommendations**

Reduce load with training (i.e. plyometrics, post-exercise eccentric training, in-season eccentrics, including uphill or downhill training). Patellar tendon straps may reduce forces to the tendon during play. Reduction of high load and rapid directional changes during training or competition such as limiting serve and volley is recommended. A player may consider slicing balls (under-spin) more often to reduce lower body load for lower balls rather than needing lower body force generation for heavy spin. Knee pain ratings have also been found to be better on clay courts than hard courts.

**OTHER TENDINOPATHIES**

Less common tendinopathies may occur in tennis players in other areas of the body around the lateral hip, ankle and the knee. Gluteus medius tendinopathy may often be labelled as other conditions such as greater trochanter syndrome or trochanteric bursitis. Many coaches may not be aware of this improved understanding
so health professionals may need to provide education via available channels. Traditionally, hip abduction strengthening combined with manual treatments to restore normal gluteus medius tendon pathology and symmetric pelvic stability are typical first line treatments. Procedures may also include injection of glucocorticoid, but probably done with fenestrations and may even include other pro-inflammatory or regenerative injections. Tennis players may get asymmetric pain patterns in the pelvis related to training and style of play.

Ankle tendinopathy may occur in the posterior tibialis tendon or peroneal tendons, in addition to Achilles tendinopathy. Non-achilles ankle tendons function to stabilise the ankle and the posterior tibialis tendon may play a role in forming the arch of the foot. Addressing foot alignment with orthotics, ankle bracing and combining this with proper eccentric strength training may help with treatment of these conditions. Occasionally, longitudinal tears and/or progression of the posterior tibialis tendon may require surgical debridement or larger reconstructive procedures.

SECONDARY OPTIONS FOR TENDINOPATHY

When traditional methods for treating tendinopathy fail, particularly appropriate mechanical loading with eccentric

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**Figure 6:** Patellar tendinopathy algorithm. GTN=glycerol trinitrate, PRP=platelet rich plasma, MRI=magnetic resonance imaging, US=ultrasound.
strengthening and volume reduction over extended periods, then it may be worthwhile to consider other non-surgical approaches. To induce or stimulate the ‘healing process’ of the tendon, pro-inflammatory (or regenerative) measures may be considered. There has been a large growth in the use of these agents, while there are still limited data to support their widespread use. Data are emerging in specific locations with specific types of procedures.

The more common methods have included topical glycerin nitrate patches, platelet rich plasma (PRP)/autologous blood injections, ultrasound-guided percutaneous tenotomy, extracorporeal shock-wave therapy, acupuncture and other types of injections. Aggressive, augmented soft tissue mobilisation using tools during rehabilitation has also been utilised with widespread anecdotal successes, but without controlled studies demonstrating its efficacy.

These types of options should be considered secondary management when traditional therapies fail and the patient also would not like to consider surgical options. Surgical treatment typically may be considered in chronic tendinopathy in severe cases where appropriate primary and secondary management has failed for >6 months and the patient is not willing to continue to try non-operative treatments.

**Topical glyceryl trinitrate**

The use of daily topical nitroglycerin patches as a non-invasive means to improve collagen synthesis to a relatively avascular tendinopathy has been shown to be effective in chronic lateral elbow tendinopathy over a 6-month period. This can be applied in other settings of tendinopathy in patients who have no hypotension, use of other nitrates or other contraindications. One of the frequent side-effects that may limit usefulness is migraine-like headaches but this can be limited with a ‘start low, go slow’ dosing schedule.

**Ultrasound-guided percutaneous tenotomy**

Non-surgical tenotomies to address chronic tendinopathies have been studied in the elbow, Achilles and patellar tendon, but have been applied in other regions. McShane et al demonstrated that fenestrations of the tendon, breaking up of calcifications and potentially abrading the bone showed improvement in severe cases of chronic lateral elbow pain/tendinopathy in series of patients who were considering surgery in this non-randomised trial. These improvements continued even with long-term follow-up of up to 28 months with avoidance of surgery. Maffulli et al found long-term improvement with multiple longitudinal percutaneous tenotomy in mid-portion chronic Achilles tendinopathy when there was no involved paratendinopathy. Similar findings of improvement with percutaneous tenotomy in chronic patellar tendinopathy have also been reported. None of these studies had control groups, i.e. the quality of evidence is ‘low’.

**PRP injections**

PRP injections have been used in a variety of settings to introduce high volumes of growth factors (platelets) to promote healing when using centrifuged blood of the patient. PRP injections have more recently come into favour as secondary management for chronic tendinopathy. A small study on chronic epicondylitis suggests that 80% of patients with buffered PRP injections showed improvement, however the number of patients in the control group was small. Additionally, PRP injections have been evaluated in patellar tendinopathy and actually found to accelerate healing compared to eccentric strength training alone, however the treatment effect dissipated with time. PRP has not been shown to be effective in rotator cuff tendinopathy.

**Autologous blood injections**

These injections involve simply taking autologous blood without isolating the platelet component and injecting into chronic tendinopathy. Some feel that it is more likely that multiple treatments may be necessary compared to PRP. However, it appears that there may be a similar treatment effect with this in refractory tendinopathy, but the effect dissipates by 6 months. It is unclear if the effect is from the autologous blood or the dry needling (or tendon fenestrations).

**Extracorporeal shock wave therapy**

This is an aggressive way to treat chronic tendinopathy. There are studies in chronic elbow tendinosis and other tendinopathies, but this isolated treatment can be quite painful and data is not convincing for its regular use.

**Other injections**

Other experimental treatments such as prolotherapy (injection of irritant), sclerosing polidocanol injections and Botulinum A injections have evidence in small supportive studies in chronic tendinopathy (typically of the elbow). There does appear to be a potential risk of transient weakness after injection with Botulinum A injections.

**CONCLUSIONS**

Tendinopathy in tennis players is generally limited to adult players where age and excess playing volume, combined with sub-optimal technique may contribute to degenerative tendinopathy. The majority of tendinopathies in tennis players are to the elbow, rotator cuff, Achilles tendon, and patellar tendon where appropriate primary management, volume reduction to reduce load, and alterations in technique where appropriate may improve symptoms. Depending on the stage of tendinopathy, a properly supervised program of eccentric strengthening rehabilitation is generally effective. Secondary management techniques and procedures have limited early and anecdotal data and should be reserved for recalcitrant cases where the player is not interested in surgery. Long term successful comprehensive programs are possible, and the need for surgical procedures is uncommon. Returning the tennis player successfully to the court may need medical and tennis specific recommendations.

**References**

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