

Pain in the Achilles Region

CHAPTER

32

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Although Achilles—the legendary warrior and hero of Homer’s *The Iliad*—died as a result of an arrow that pierced the midportion of his tendon, today’s patient with a painful presentation in this region usually has a good long-term prognosis.¹ Nevertheless, runners have a 15 times greater risk of Achilles tendon rupture, and 30 times greater risk of tendinopathy than do sedentary controls.² This chapter highlights clinically useful evidence-based treatments for pain in the Achilles region.^{3,4}

Functional anatomy

The key structures that contribute to pain in the Achilles region (posterior heel and proximal towards the calf) are illustrated in Figure 32.1. The Achilles tendon, the thickest and strongest tendon in the human body,⁵ is the combined tendon of the gastrocnemius and soleus muscles. The tendon has no synovial sheath but is surrounded by a paratenon (also known as peritendon), which is continuous with the perimysium of the muscle and the periosteum of the calcaneus. Pain in the main body of the tendon (2–3 cm [1–1.5 in.] above the insertion, which we refer to as the ‘midportion’) appears to respond much better to treatment than pain at the insertion itself. The bursae are important potential sources of pain, as is the superolateral tubercle of the calcaneus, which, when excessively large, is called a ‘Haglund’s deformity’. A large and rather square-shaped calcaneus appears to predispose to bursitis. The posterior process of the talus approximates the Achilles insertion and, thus, can contribute to pain in this region. Blood vessels enter the Achilles tendon from the deep surface (Fig. 32.1c); this is where abnormal vessels are ablated using sclerotherapy (see p. 602).

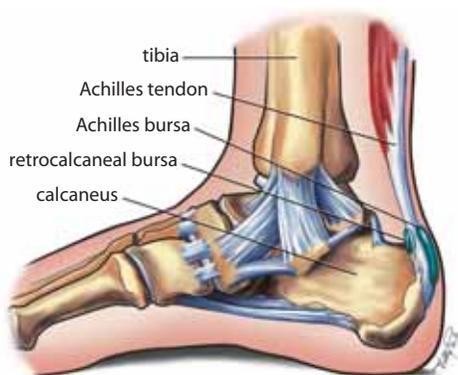
Clinical perspective

Acute tendon rupture is most common among men aged 30–50 years (mean age, 40 years); it causes sudden severe disability. Most textbooks suggest that rupture limits active plantarflexion of the affected leg—but beware, the patient can often plantarflex using an intact plantaris and the long toe flexors.

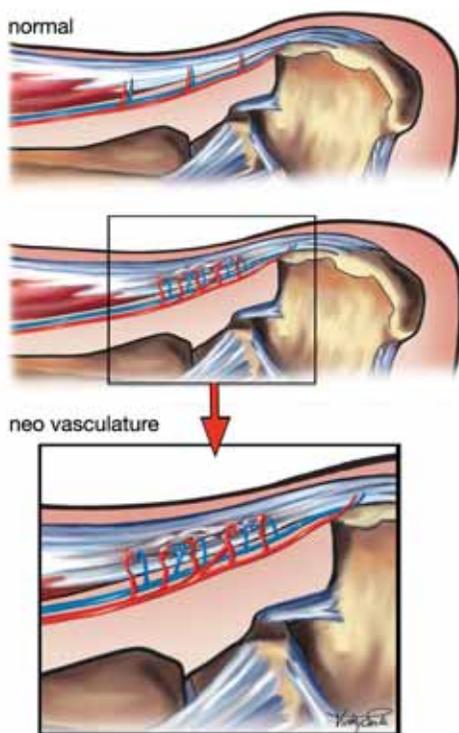


Figure 32.1 The Achilles region

(a) Surface anatomy



(b) Anatomy



(c) Stylized and magnified depiction to illustrate abnormal vasculature in Achilles tendinosis



They key diagnostic test is the ‘calf squeeze test’ (also called Simmond’s or Thompson’s test) (see Fig. 32.2h).

Overuse Achilles tendon injuries—tendinopathy—may arise with increased training volume or intensity but may also arise insidiously. Because the prognosis for midportion Achilles tendinopathy is much better than for insertional tendinopathy, these conditions

should be distinguished clinically. The condition that was previously called ‘Achilles tendinitis’ is not truly an inflammatory condition and, thus, should be referred to as ‘Achilles tendinopathy.’⁶ Chapter 2 details the pathology that underlies the common tendinopathies.

The main differential diagnoses of gradual onset pain in the Achilles region arise from the neighboring anatomy (Table 32.1). There are two bursae in this region: the retrocalcaneal bursa, which lies between the posterior aspect of the calcaneus and the insertion of the Achilles tendon, and the Achilles bursa, which lies between the insertion of the Achilles tendon and the skin (Fig. 32.1b). The posterior process of the talus or a discrete anatomical variant, the os trigonum, can each be involved in posterior impingement syndrome (Chapter 34). This is most commonly seen in ballet dancers but occurs occasionally in sprinters and in football players. Other, much less common differential diagnoses include dislocation of the peroneal tendons, an accessory soleus muscle, irritation or neuroma of the sural nerve, and systemic inflammatory disease. These pathologies cause pain in and also *around* the Achilles tendon; true tendon pain is almost always confined to the tendon itself.

In adolescents, it is important to consider the diagnosis of Sever’s lesion, a traction apophysitis at the insertion of the Achilles tendon into the calcaneus (Chapter 40). Referred pain is a very rare cause of pain in the Achilles region.

History

The athlete with overuse tendinopathy notices a gradual development of symptoms and typically complains of pain and morning stiffness after increasing activity level. Pain diminishes with walking about or applying heat (e.g. a hot shower). In most cases, pain diminishes during training, only to recur several hours afterwards.

The onset of pain is usually more sudden in a partial tear of the Achilles tendon. In this uncommon condition, pain may be more disabling in the short term. As the histological abnormality in a partial tear and in overuse tendinopathy are identical (see below), we do not emphasize the distinction other than to suggest that time to recovery may be longer in cases of partial tear. A history of a sudden, severe pain in the Achilles region with marked disability suggests a complete rupture. The patient often reports hearing a ‘shot’.

Examination

Palpate the painful area for tenderness, thickening and crepitus (a ‘crackling’ feeling that arises because

Table 32.1 Causes of pain in the Achilles region

Common	Less common	Not to be missed
Midportion Achilles tendinopathy (this includes tendinosis, paratendinitis and partial tears)	Achilles bursitis	Achilles tendon rupture
Posterior impingement syndrome	Accessory soleus muscle	Achilles tendinopathy due to the inflammatory arthropathies (Chapter 50)
Insertional Achilles tendinopathy including retrocalcaneal bursitis and Haglund's disease	Referred pain	
Sever's lesion (adolescents)	Neural structures	
	Lumbar spine	

of the hydrophilic [water-attracting] excess matrix proteins found in the peritendon). Also, seek possible predisposing factors, such as unilateral calf tightness, joint stiffness at the ankle or subtalar joints, and abnormal lower limb biomechanics. If the Achilles tendon seems to be the cause of pain, and the examiner is confident that the tendon is intact, the examination should aim to provoke tendon pain during tendon-loading activity. In most patients, simple single-leg heel-raises will be sufficient to cause pain. In more active individuals, however, it may be necessary to

ask the patient to hop on the spot, or hop forward, to further load the tendon and reproduce pain. In some athletes repeated loading (i.e. multiple hops, jumps) tests may be necessary to evaluate the tendon fully. These functional tests provide a baseline against which treatment response can be compared. Another method of monitoring the clinical progress of Achilles tendinopathy is to use the VISA questionnaire⁷ (Table 32.2, this is also available as a downloadable PDF file at <www.clinicalsportsmedicine.com>). This simple questionnaire takes less than 5 minutes to complete

Table 32.2 Victorian Institute of Sport Assessment (VISA-A) questionnaire

1. For how many minutes do you have stiffness in the Achilles region on first getting up? POINTS

100 min 0 min

0 1 2 3 4 5 6 7 8 9 10

2. Once you have warmed up for the day, do you have pain when stretching the Achilles tendon fully over the edge of a step? (keeping knee straight) POINTS

Strong severe pain no pain

0 1 2 3 4 5 6 7 8 9 10

3. After walking on flat ground for 30 minutes, do you have pain within the next 2 hours? (If unable to walk on flat ground for 30 minutes because of pain, score 0 for this question.) POINTS

Strong severe pain no pain

0 1 2 3 4 5 6 7 8 9 10

4. Do you have pain walking down stairs with normal gait cycle? POINTS

Strong severe pain no pain

0 1 2 3 4 5 6 7 8 9 10

Table 32.2 Victorian Institute of Sport Assessment (VISA-A) questionnaire (continued)

5. Do you have pain during or immediately after doing 10 (single leg) heel raises from a flat surface? POINTS

Strong severe pain	<input type="checkbox"/>	no pain	<input type="checkbox"/>										
	0	1	2	3	4	5	6	7	8	9	10		

6. How many single-leg hops can you do without pain? POINTS

0	<input type="checkbox"/>	10	<input type="checkbox"/>										
	0	1	2	3	4	5	6	7	8	9	10		

7. Are you currently undertaking sport or other physical activity? POINTS

0	<input type="checkbox"/>	Not at all	<input type="checkbox"/>
4	<input type="checkbox"/>	Modified training ± modified competition	
7	<input type="checkbox"/>	Full training ± competition but not at same level as when symptoms began	
10	<input type="checkbox"/>	Competing at the same or higher level as when symptoms began	

8. Please complete *either* A, B or C in this question.

- If you have no pain while undertaking Achilles tendon loading sports, please complete Q8A only.
- If you have pain while undertaking Achilles tendon loading sports but it does not stop you from completing the activity, please complete Q8B only.
- If you have pain that stops you from completing Achilles tendon loading sports, please complete Q8C only.

A. If you have no pain while undertaking Achilles tendon loading sports, for how long can you train/practise? POINTS

NIL	1–10 min	11–20 min	21–30 min	>30 min	<input type="checkbox"/>
<input type="checkbox"/>					
0	7	14	21	30	

B. If you have some pain while undertaking Achilles tendon loading sports but it does not stop you from completing your training/practise, for how long can you train/practise? POINTS

NIL	1–10 min	11–20 min	21–30 min	>30 min	<input type="checkbox"/>
<input type="checkbox"/>					
0	4	10	14	20	

C. If you have pain that stops you from completing your training/practise in the Achilles tendon loading sports, for how long can you train/practise? POINTS

NIL	1–10 min	11–20 min	21–30 min	>30 min	<input type="checkbox"/>
<input type="checkbox"/>					
0	2	5	7	10	

TOTAL SCORE (/100) %

and once patients are familiar with it they can complete most of it themselves.

Examination involves:

1. Observation
 - (a) standing
 - (b) walking
 - (c) prone (Fig. 32.2a)
2. Active movements
 - (a) plantarflexion
 - (b) dorsiflexion
3. Passive movements
 - (a) plantarflexion
 - (b) plantarflexion with overpressure (Fig. 32.2b)
 - (c) dorsiflexion
 - (d) subtalar joint (Fig. 32.2c)
 - (e) muscle stretch
 - (i) gastrocnemius (Fig. 32.2d)
 - (ii) soleus (Fig. 32.2e)
4. Resisted movements
 - (a) plantarflexion—calf raises
5. Functional tests
 - (a) single-leg calf raises
 - (b) hop (Fig. 32.2f)
 - (c) eccentric drop
6. Palpation
 - (a) Achilles tendon (Fig. 32.2g)
 - (b) retrocalcaneal bursa
 - (c) posterior talus
 - (d) calf muscle
7. Special test
 - (a) Prone inspection for tendon rupture
 - (b) Simmond's calf squeeze test⁸ (Fig. 32.2h)
 - (c) biomechanical assessment (Chapter 5)

Investigations

Plain radiographs are of limited value but, if symptoms are longstanding, they may reveal a Haglund's deformity, a prominent superior projection of the calcaneus (Fig. 32.3a), or spurs projecting into the tendon. This is associated with insertional tendinopathy and may also precipitate retrocalcaneal bursitis. Posterior impingement can be shown radiographically using functional views (see Fig. 32.14). X-ray may reveal calcification in the tendon itself but, unless severe (Fig. 32.3b), this can be asymptomatic.

In symptomatic patients, both ultrasound and MRI (Figs 32.3c–f) often reveal an abnormal signal in the Achilles tendon that generally corresponds with the histopathology of tendinosis described below (pp. 597–607). Ultrasound and MRI can help distinguish different causes of pain in the Achilles region (e.g. highlight whether the Achilles bursa or the



Figure 32.2 Examination of the patient with pain in the Achilles region

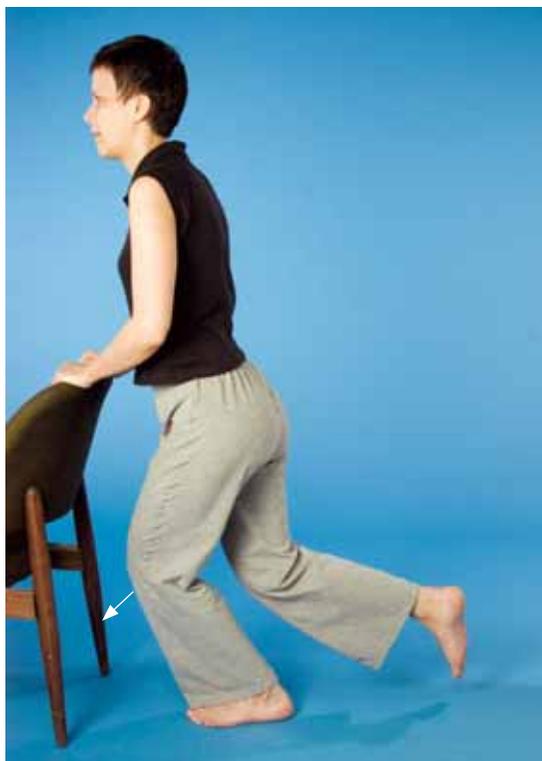
(a) Observation—prone. Look for swelling of the tendon and wasting of the calf muscle



(b) Passive movement—plantarflexion. This will be painful if posterior impingement is present. Overpressure can be applied



(c) Passive movement—subtalar joint. Restricted subtalar joint movement is a potential cause of Achilles region pain and a contributory factor to abnormal biomechanics



(e) Passive movement—muscle stretch (soleus). The patient stands upright and keeps the knee flexed. The foot should remain in a neutral position



(d) Passive movement—muscle stretch (gastrocnemius). The patient stands so that body weight causes overpressure. The knee must remain extended and the heel remains in contact with the floor. The foot remains in neutral by keeping the patella in line with the third metatarsal. Compare the stretch on both sides



(f) Functional tests. These can be used to reproduce pain, if necessary, or to test strength. Tests include double-leg and single-leg calf raises, hops forward (illustrated), eccentric drops and lunge



(g) Palpation—prone. Palpate the site of pain. Palpate the tendon and paratenon while the tendon moves to determine which structure is involved. Palpate the gastrocnemius, soleus and retrocalcaneal bursa(e)



(h) Special test—Simmond's (Thompson's) calf squeeze test for Achilles tendon rupture. The practitioner squeezes the fleshy part of the calf. The test is positive if the foot fails to plantarflex

Achilles tendon insertion is abnormal in patients with pain at the distal tendon). Color Doppler ultrasound is being used increasingly in tendinopathies; it provides information about the extent of the characteristic abnormal vascularity. It may also provide a target for treatment (see sclerotherapy p. 602). Because of the variability in imaging and its inconsistent clinical correlation,^{9,10} the results of imaging should not dominate

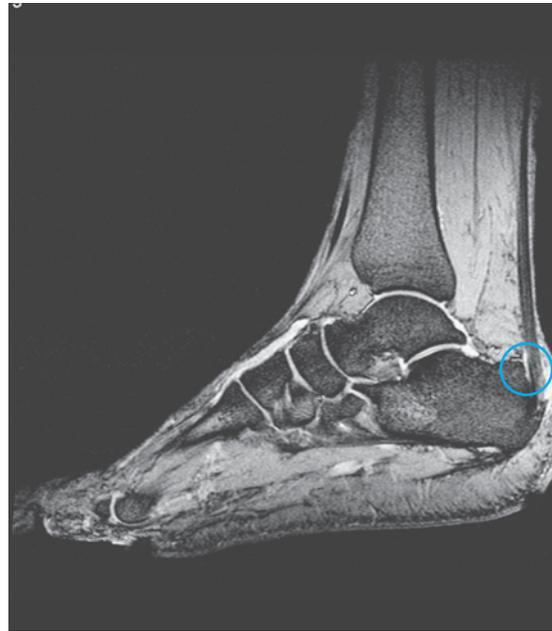
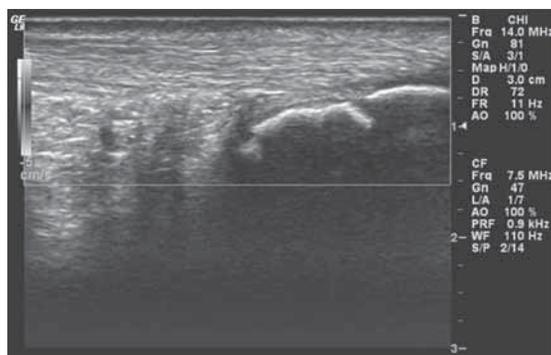


Figure 32.3 Imaging findings in patients presenting with Achilles pain

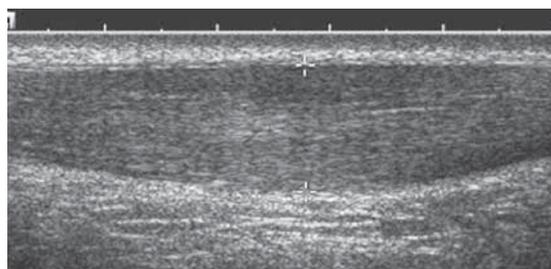
(a) MRI showing the prominent calcaneum of Haglund's deformity and associated tendon and bursal pathology



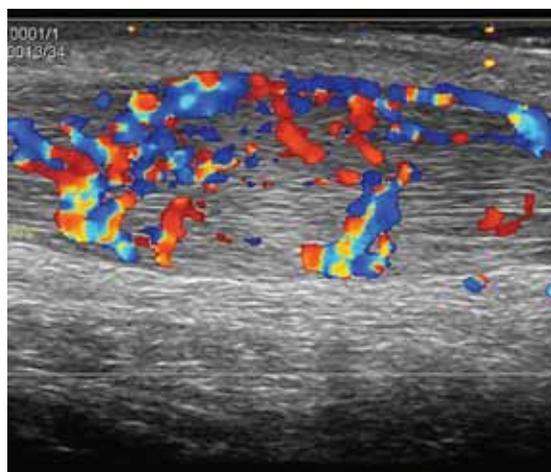
(b) Calcification in the insertion of the Achilles tendon. Patients can be managed according to the symptoms of tendinopathy (see p. 597)



(c) Gray-scale ultrasound appearance of a normal Achilles tendon



(d) Gray-scale ultrasound appearance of an Achilles tendon with mild morphological abnormality



(e) Color Doppler ultrasound appearance showing abnormal vessels in symptomatic tendinopathy

clinical decision making; variation in symptoms such as morning stiffness and load pain should direct treatment modification. Studies in many tendons have indicated that clinical outcomes are independent of imaging and change in imaging.^{11,12}



(f) MRI appearance showing mild morphological abnormality

Midportion Achilles tendinopathy

It is important to distinguish between midportion and insertional Achilles tendinopathy as they differ in their prognosis and response to treatment. We briefly review the pathology of Achilles tendinopathy, list expert opinion of the factors that predispose to injury, and summarize the clinical features of the condition. The subsequent section details the treatment of midportion tendinopathy.

Histopathology and basic molecular biology

When operating on patients with chronic Achilles tendinopathy, the surgeon generally finds a degenerative lesion characterized by an intratendinous, poorly demarcated, dull-grayish discoloration of the tissue with a focal loss of normal fiber structure (Fig. 32.4a).¹³ A partial tear or rupture, defined as a macroscopic discontinuity involving a small proportion of the tendon cross-section, is seen in approximately 20% of cases. These tears always occur in a region of pre-existing pathology and do not occur in normal tendon tissue.¹⁴ The paratendinous structures are either normal or contain edema or scarring. Importantly, when the symptomatic parts of such Achilles tendon tissue are examined under the light microscope, there is collagen fiber disarray (Fig. 32.4b). This applies equally

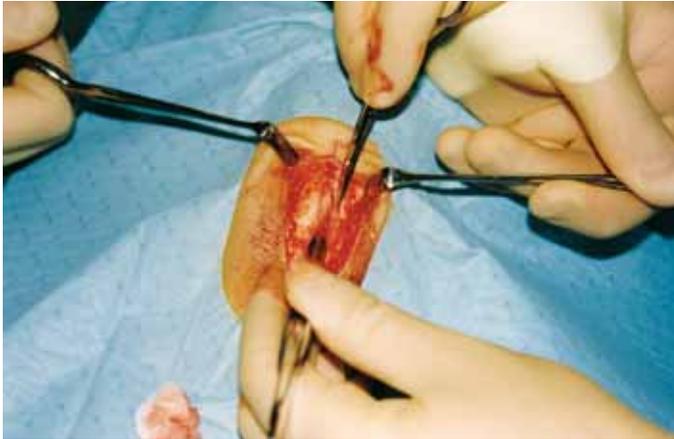
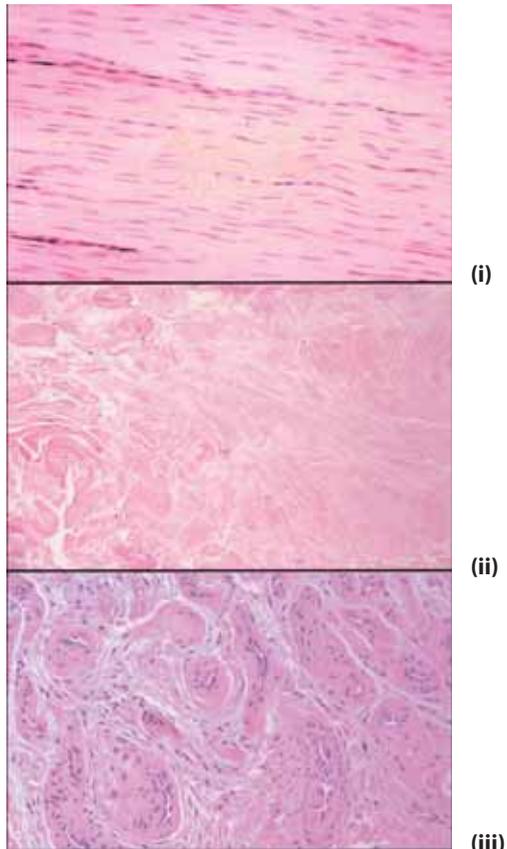


Figure 32.4 (a) Intraoperative photograph showing the appearance of tendinosis.



(b) Light microscopic appearance of this tissue: **(i)** appearance of normal tendon; **(ii)** collagen fiber disarray, loss of the characteristic parallel bundles, fewer cell nuclei; **(iii)** areas of abnormal vascularity and increasingly prominent cells (see also Figs 32.1c, 32.3e)

to areas of partial tear, which show hypervascularity without signs of tissue repair.¹³ This histopathological picture is called 'tendinosis' and is identical in tendons with macroscopically evident partial tears and those without.¹³ These regions of tendon disarray correspond with areas of increased signal on MRI and hypochoic regions on ultrasound.¹⁵

It is noteworthy that inflammatory cells are absent in tendinosis. Also, intratendinous microdialysis^{16,17} and contemporary molecular biology techniques (cDNA arrays, real-time quantitative polymerase chain reaction) of appropriately prepared biopsy tissue¹⁸ all failed to show evidence of prostaglandin-mediated inflammation. There are, however, signs of what Hart et al. have termed 'neurogenic inflammation'.¹⁹ This is characterized by neuropeptides, such as substance P and calcitonin gene-related peptide (CGRP). It appears that peptidergic group IV nerve fibers release peptides from their terminals, starting various pathophysiological, and presumably painful, processes. While this field awaits further research advances, clinicians can endeavor to limit the onset of pathology by attending to the likely risk factors for Achilles tendinopathy.

Predisposing factors

Injury to the Achilles tendon occurs when the load applied to the tendon, either in a single episode or, more often, over a period of time, exceeds the ability of the tendon to withstand that load. Factors that may predispose to Achilles tendinopathy include:

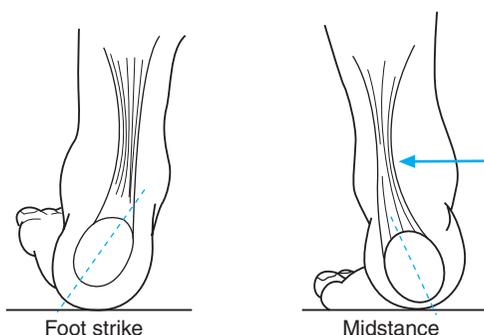
- years of running
- increase in activity (distance, speed, gradient)
- decrease in recovery time between training sessions
- change of surface
- change of footwear (e.g. lower heeled shoe, shoe with heel tab)
- excessive pronation²⁰ (increased load on gastrocnemius–soleus complex to resupinate the foot for toe-off) (Fig. 32.5)
- calf weakness
- poor muscle flexibility (e.g. tight gastrocnemius)
- joint range of motion (restricted dorsiflexion)
- poor footwear (e.g. inadequate heel counter, increased lateral flaring, decreased forefoot flexibility) (Chapter 6)
- genetic predisposition.²¹

Clinical features

The presentations of Achilles tendinopathy can vary as listed in Table 32.3.

Table 32.3 Clinical features associated with presentation of overuse Achilles tendinopathy (i.e. not a complete rupture)

Clinical feature or imaging finding	Variability in presentation with overuse Achilles tendinopathy
History	
Onset of pain	May be sudden, gradual but noticeable, or insidious
Severity of pain	May range from a minor inconvenience to profound pain with activity
Duration	May range from days to years
Disability	May be minimal, moderate or severe
Examination	
Extent of swelling/crepitus	Can range from being a major feature of the presentation to being absent
Extent of tenderness	May range from being pinpoint to extending throughout several centimeters of the tendon
Presence of a nodule	May or may not be present, and when present may vary in size

**Figure 32.5** Whipping action of the Achilles tendon produced by overpronation, a risk factor for midportion Achilles tendinopathy

Practise tips relating to imaging Achilles tendinopathy

There are various appearances of Achilles tendinopathy with imaging (Table 32.4). Thus, we recommend that the history and physical examination remain the keys to diagnosis. Until patients become familiar with the concept of tendinosis, imaging may help illustrate that the abnormality is one of collagen disarray and abnormal vasculature; this will help the patient understand the lengthy time course of healing.

Treatment of midportion Achilles tendinopathy

Level 2 evidence-based treatments for Achilles tendinopathy include heel-drop exercises, nitric oxide

Table 32.4 Variations in imaging findings in patients with overuse Achilles tendinopathy (i.e. not a complete rupture)

Investigation	Variations seen in clinical practise
Ultrasound— extent of swelling	Tendon swelling can be associated with tendon fiber damage (see below) or it can occur without discontinuity and swelling (e.g. fusiform swelling). It is possible to have a normal ultrasound scan with symptoms and signs of Achilles tendinopathy but differential diagnoses must be fully evaluated and excluded.
Ultrasound— discontinuity of tendon fibers	Tendon fibers may appear intact or extensively damaged on ultrasonography ('hypoechoogenicity'). This is usually associated with tendon swelling.
MRI appearance	The MRI appearance can vary from essentially normal to a marked increase in abnormal signal, best seen on T2-weighted sequences. Another feature of tendinopathy is increased tendon diameter without signal.

donor therapy (glyceryl trinitrate [GTN] patches), sclerosing injections and microcurrent therapy (see below). In addition, experienced clinicians begin conservative treatment by identifying and correcting possible etiological factors. This may include relative

rest, orthotic treatment (heel lift, change of shoes, corrections of malalignment) and stretching of tight muscles. Whether these 'commonsense' interventions contribute to outcome is unlikely to be tested.

Figure 32.6 illustrates a commonly used progression of treatment. The sequence of management options may need to vary in special cases such as the elite athlete, the person with acute tendon pain unable to fully bear weight, or the elderly patient who may be unable to complete the heel-drops. As always, the clinician should respond to individual patient needs and modify the sequence appropriately.

Alfredson's painful heel-drop protocol

In 1984, Curwin and Stanish²³ pioneered what they termed 'eccentric training' as therapy for tendon injuries. From this base, Alfredson and colleagues made

three critical modifications.^{24,25} Firstly, they considered worsening pain as part of the normal recovery process; thus, they advised patients to continue with the full exercise program even as pain worsened on starting the program. Along those lines, if the patient experienced no pain doing the program, he or she was advised to increase the load until the exercises provoked pain (Fig. 32.7, Table 32.5). The second innovation was to incorporate two types of heel-drops into the program (see below); traditionally, only one type of heel-drop had been prescribed. Finally, they prescribed 180 heel-drops per day—a far larger number than had been recommended previously. Alfredson's 12-week program cured approximately 90% of those with midtendon pain and pathology.^{25–27} In addition to the good clinical results, ultrasound and MRI follow-up demonstrated that patients' tendons returned towards normal appearance and thickness.²⁸

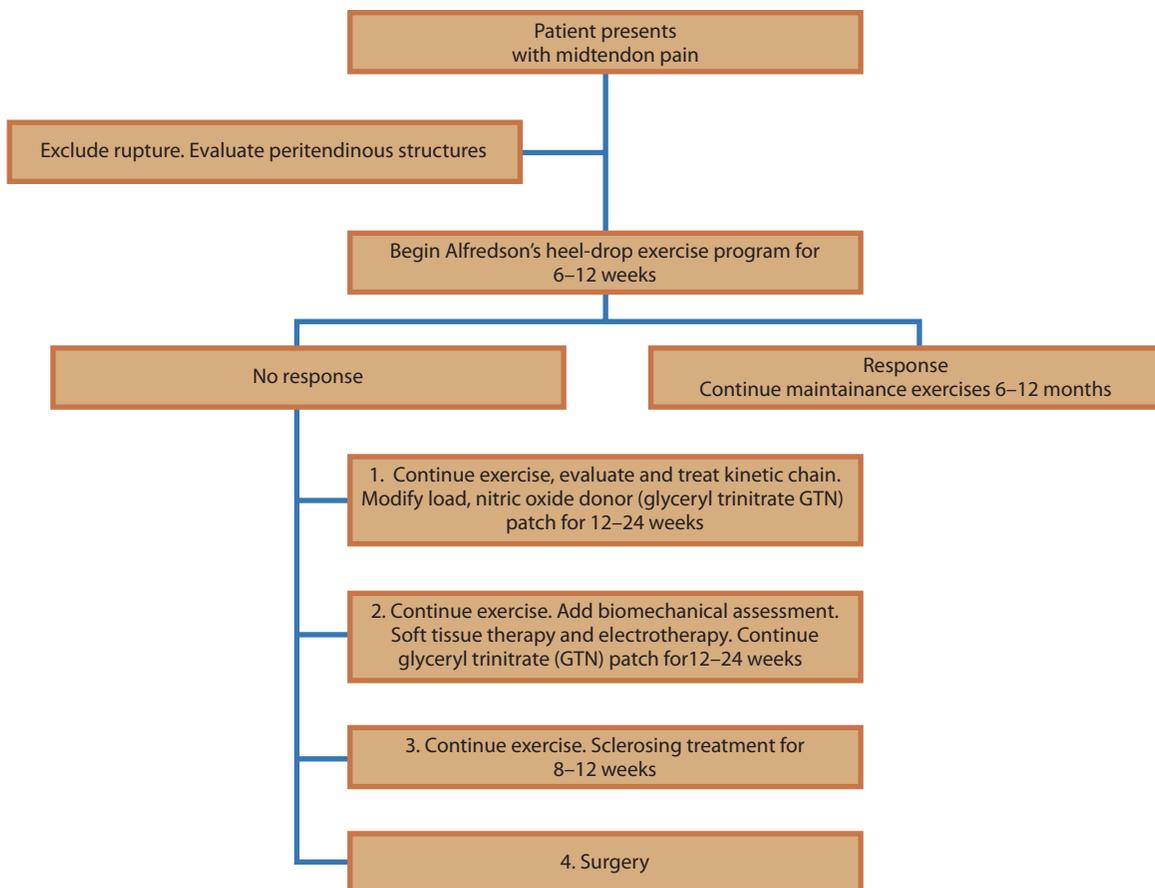
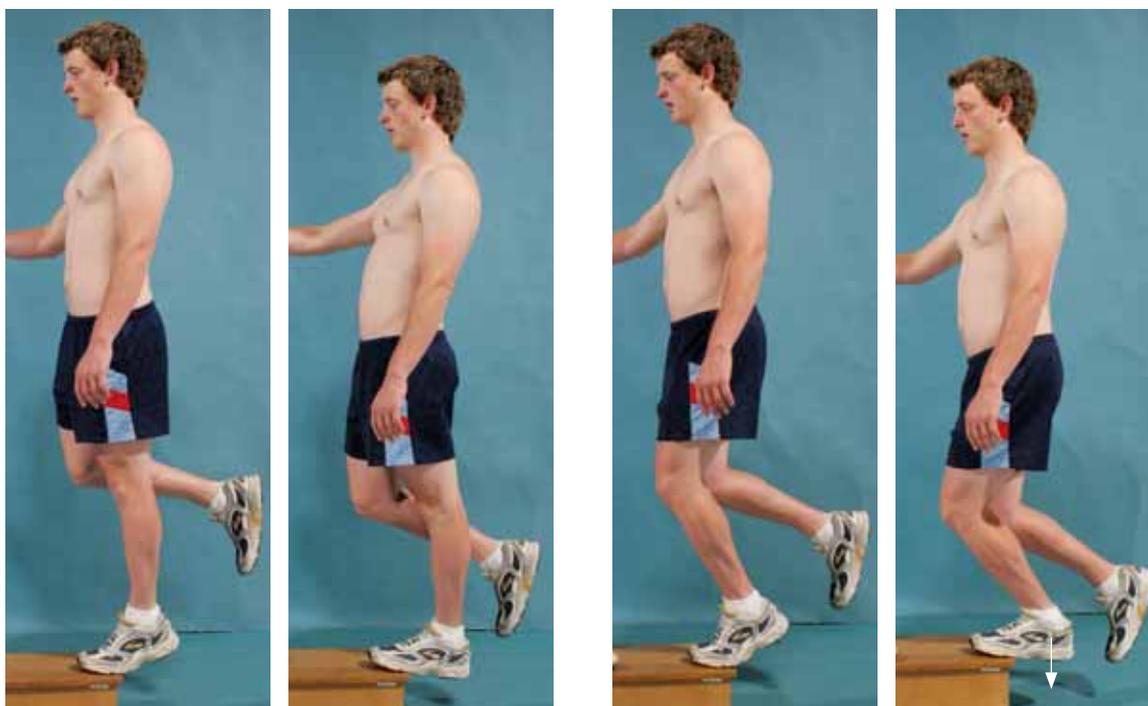


Figure 32.6 Flow chart showing one approach to the clinical management of midportion Achilles tendinopathy (modified from Alfredson et al.²²)



(a)

(b)

(c)

(d)

Figure 32.7 Alfredson's painful heel-drop protocol for Achilles tendinopathy. The heel-drop protocol consists of two key exercises: the 'gastrocnemius drop' and the 'soleus drop'.

(a) For the gastrocnemius drop, the patient begins in a demipointe position, with the heel raised and the knee fully extended.

(b) From the starting position, the patient lowers the heel so that the foot is parallel with the ground.

Variations on the heel-drop program may also be effective²⁹ but have not been as rigorously evaluated as have those used in patients in Alfredson's program.

PRACTISE PEARL There is evidence to suggest that heel-drops promote superior clinical recovery than do heel-raises.³⁰

(c) For the soleus drop, the patient again adopts the demipointe position with the heel raised, but for this exercise the knee should be flexed to 45° so that the soleus is engaged.

(d) The patient lowers the heel so that the foot is parallel with the ground.

In a study that compared these two types of exercises, 82% of patients who did the heel-drops were back to their previous activity level at the completion of treatment, whereas only 36% of those doing heel-raises achieved that outcome.³¹

Nitric oxide donor therapy

There is level 2 evidence to support nitric oxide donor therapy (glyceryl trinitrate [GTN] patches

Table 32.5 Alfredson's painful heel-drop protocol (180 drops/day)

Number of exercises	Exercise specifics	Exercise progression
3 × 15 repetitions 2 times daily 7 days/week for 12 weeks	Do exercise both with knee straight (fully extended) (Fig. 32.7a) and knee bent (flexed 45°) (Fig. 32.7b) over edge of a step Lower only (heel-drop) from standing on toes (i.e. raise back onto toes using unaffected leg or arms)	Do exercises until they become pain-free Add load until exercises are again painful Progressively add load up to 60 kg



(e) To increase the load, additional weight can be added using a backpack or, where necessary, a weight machine

applied locally 1.25 mg/day).³³ Compared with the control group, the glyceryl trinitrate group showed reduced pain with activity by 12 weeks. In this group, 28 (78%) of 36 patients' tendons were asymptomatic with activities of daily living at six months, compared with 20 (49%) of 41 patients' tendons in the control group (Fig. 32.9b).

Glyceryl trinitrate (GTN) patches come in varying doses. A 0.5 mg patch should be cut in quarters and applied to the site of maximum pain for 24 hours at a time and then replaced (Fig. 32.9a). A 0.2 mg patch would best be cut in half and applied similarly.

Corticosteroid injection around and into tendon

The Cochrane database of systematic reviews concludes that peritendinous injection of corticosteroids in Achilles tendinopathy has short-term pain-relieving effects but no effect or detrimental effects in the longer term.^{34,35} Although intratendinous injection is generally contraindicated,³⁵ Danish researchers³⁶ injected corticosteroid intratendinously in six tendons with promising results. This, however, requires further research before being recommended.

How do heel-drops reduce pain in tendinopathy?

There are several possible explanations for the effectiveness of heel-drops but none have yet been proven. Heel-drops have an immediate and long-term influence on tendon. In the short-term, a single bout of exercise increases tendon volume and signal on MRI.²⁸ Heel-drops affect type 1 collagen production and, in the absence of ongoing insult, may increase tendon volume over the longer term.³² Thus, heel-drops may increase tensile strength in the tendon over time. Repetitive loading and a lengthening of the muscle–tendon unit may therefore improve the capacity of the musculotendinous unit to affectively absorb load (Fig. 32.8).



Figure 32.8 How heel-drops might work to improve Achilles tendon metabolism

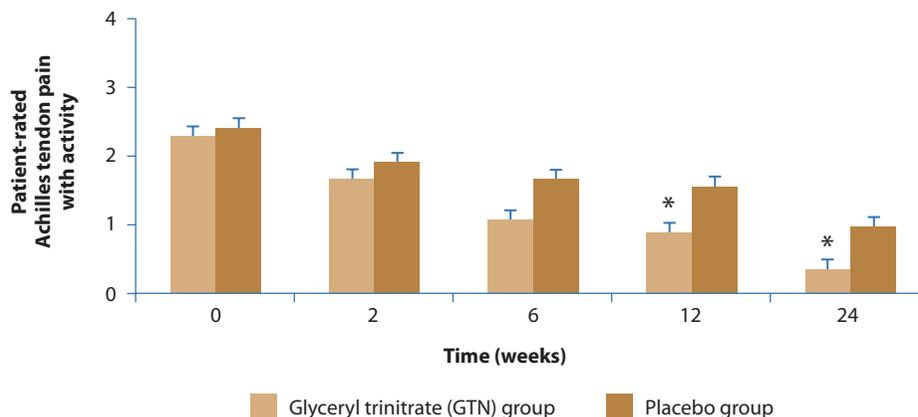
Sclerosing injections

This treatment consists of using ultrasound guidance (Fig. 32.10a) while injecting a vascular sclerosant (polidocanol, an aliphatic, non-ionized, nitrogen-free substance with a sclerosing and anesthetic effect) in the area of neovascularization anterior to the tendon (Figs 32.10 b, c). Short-term (six month) evaluation of



Figure 32.9 Nitric oxide donor therapy.

(a) A glyceryl trinitrate (GTN) patch is worn over the most painful site 24 hours a day for six months.



(b) Glyceryl trinitrate patches provided superior outcomes than did the placebo patch for Achilles tendinopathy⁴

* = statistically significant difference ($P < 0.05$)

this treatment showed that eight of 10 tendons were pain-free after a mean of two treatments.³⁷ Tendons that were pain-free had no neovascularization outside or inside the tendon (Fig. 32.10d), but in the two unsuccessfully treated patients, vessels remained. Two-year follow-up of these patients showed that the same eight patients remained pain-free with no vessels in the tendon (unpublished data). Ultrasonographically, tendon thickness had decreased and the structure looked more normal.

In a small double-blind randomized controlled study³⁸ comparing the effects of injections of a sclerosing and a non-sclerosing substance (lignocaine [lidocaine] plus adrenalin [epinephrine]), two doses of the sclerosing substance led to five of 10 participants being satisfied with treatment; a further open-label treatment (injection) led to all remaining patients being satisfied. The placebo group, on the other hand, saw no patients satisfied after two placebo injections, and nine of 10 participants satisfied after open-label cross-over to the active agent.

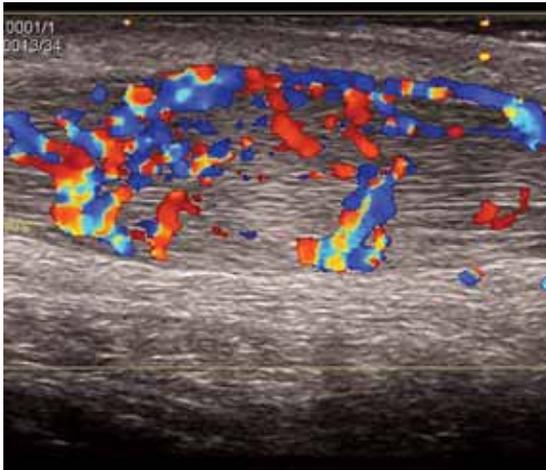
Rehabilitation after sclerosing injection includes one to three days of rest, then gradually increased tendon-loading activity while being careful to avoid jumping, fast runs and heavy strength training during the first two weeks. After two weeks, such activities that load the tendon maximally are permitted.

After treating 500 patients with Achilles tendinopathy, the pioneers of this treatment (Alfredson et al.) reported three complications. Two patients have sustained total ruptures (one at the end of an 800-m track race eight weeks after treatment) and another patient who was treated in the midportion sustained a partial rupture in an area in which he previously

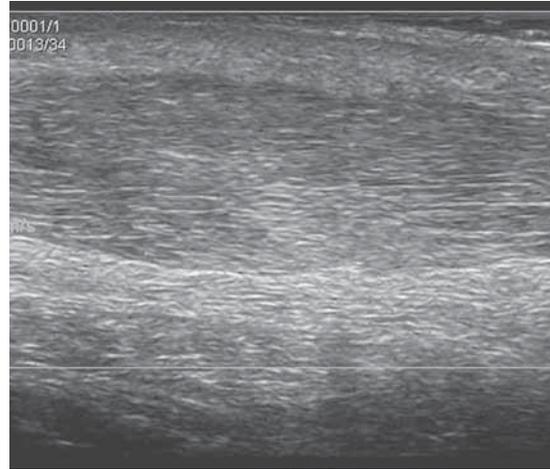


Figure 32.10 Sclerotherapy

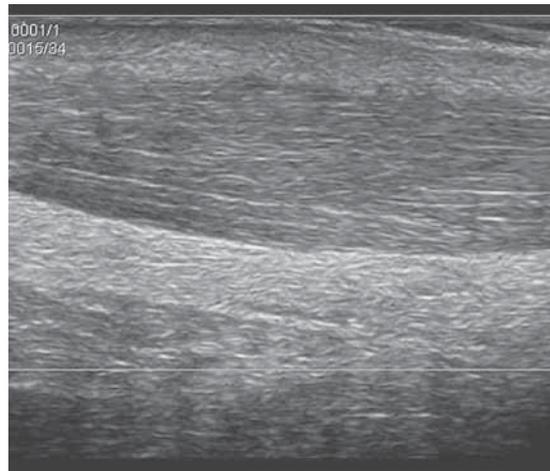
(a) Ultrasound-guided sclerotherapy



(b) Abnormal vessels—neovessels—before sclerotherapy



(c) Vessels absent immediately after sclerotherapy



(d) Vessels are still absent 12 months after treatment in a pain-free patient

had four intratendinous corticosteroid injections. In summary, sclerosing therapy may have a role in patients who fail to respond to heel-drops and nitric oxide donor therapy. As with all innovative therapies, further research is needed to determine the long-term safety of this procedure. If the technique proves successful when used in other centers and at other tendon sites, it would strengthen the argument for more routine use.³⁹

Electrophysical agents

There is little quality evidence to support the use of electrophysical agents, including extracorpor-

real shock wave therapy,⁴⁰ in treating tendinopathy. Therapeutic ultrasound increases protein synthesis in tendons,⁴¹ but there is an oversupply of poor quality protein in tendon suffering from overuse, so it may not improve the clinical outcome to further increase this substance.

A randomized trial⁴² showed a superior outcome after treatment and one month later in patients who had undergone hyperthermia with low-frequency microwave compared with those treated with traditional ultrasound. Microcurrent applied for two weeks decreased pain at 12 months compared with conventional treatment.⁴³

Adjunct conservative treatments

Biomechanical evaluation of the foot and leg is a clinically important part of Achilles tendon management. Although there is little empirical evidence to support the association between static foot posture and Achilles tendinopathy,⁴⁴ modification of foot posture in some patients can reduce pain and increase the capacity to load the tendon.²⁰

Similarly, soft tissue therapy of the calf complex can assist rehabilitation (Fig. 32.11), as can tendon mobilization.⁴⁵ Frictions have been shown to increase the protein output of tendon cells,⁴⁶ however, similar to the effect seen with ultrasound, greater amounts of collagen and ground substance may not improve pain or pathology.

Surgical treatment

Surgical treatment procedures range from simple percutaneous tenotomy⁴⁷ to removal of tendon pathology via an open procedure. Percutaneous tenotomy resulted in 75% of patients reporting good or excellent results after 18 months.⁴⁸ The outcome of open surgery for Achilles tendinopathy was superior among patients whose tendons had diffuse disease, compared with those whose tendons had a focal area of tendinopathy.⁴⁹ At seven months post-surgery, 88% of those with diffuse disease had returned to physical activity, as had 50% of those with a focal lesion.

All Achilles tendon surgery requires early post-operative rehabilitation and this needs to continue for six to 12 months as final clinical results rely on the return of strength and functional capacity. Wise patients will continue with a maintenance program of

physiotherapist-prescribed rehabilitation exercises even after having returned to training and competition.

Insertional Achilles tendinopathy, retrocalcaneal bursitis and Haglund's disease

These three 'diagnoses' are discussed together as they are intimately related in pathogenesis and clinical presentation.⁵⁰⁻⁵³

Relevant anatomy and pathogenesis

The Achilles tendon insertion, the fibrocartilaginous walls of the retrocalcaneal bursa that extend into the tendon (Fig. 32.1b) and the adjacent calcaneus form an 'enthesis organ' (Fig. 32.12).⁵⁴ The key concept is that at this site the tendon insertion, the bursa and the bone are so intimately related that a prominence of the calcaneus will greatly predispose to mechanical irritation of the bursa and the tendon. Also, there is significant strain on the tendon insertion on the posterior aspect of the tendon with dorsiflexion.⁵³ This then leads to a change in the nature of those tissues, consistent with the biological process of mechanotransduction (physical response to a mechanical load, Chapter 2).

We distinguish between the term 'Haglund's deformity', a descriptive label for prominence of the posterolateral calcaneus, and 'Haglund's disease', a clinical label for the clinical syndrome of a prominent, painful lateral portion of the retrocalcaneal bursa



Figure 32.11 Soft tissue therapy to the belly of the calf muscles

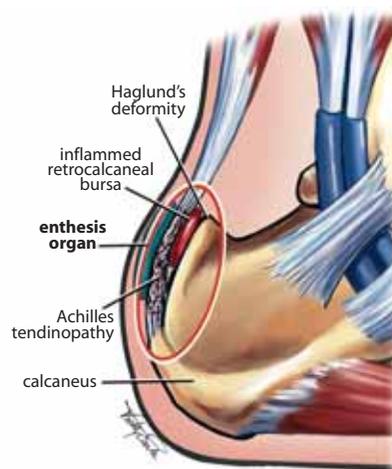


Figure 32.12 The 'enthesis organ' illustrates why insertional Achilles tendinopathy is often associated with retrocalcaneal bursitis and calcaneal prominence

associated with a prominent superolateral superior calcaneus (the Haglund's deformity).⁵ Thus, an asymptomatic patient may be found to have a Haglund's deformity on radiographs taken for another reason. This is important as the deformity, per se, is not an indication for treatment.

Insertional Achilles tendinopathy is not as common, or as well-researched, as midportion tendinopathy. The pathology is tendinosis, not inflammation,⁵⁵ and there is some local revascularization inside and outside the distal tendon.

Clinical assessment

Good clinical practise includes evaluation of the tendon, bursa and calcaneum by careful history, inspection of the region for bony prominence and local swelling as well as palpation of the area of maximal tenderness. Biomechanical abnormalities, joint stiffness and proximal soft tissue tightening can exacerbate an anatomical predisposition to retrocalcaneal bursitis; they warrant correction if present. Ultrasound and MRI can help to assess the extent of pathology in the tendon and the bursa. Radiography can complement clinical assessment of the calcaneum and will reveal tendon calcification, if present. If the pathology is truly in the tendon insertion, it is important to alert the patient that this is more challenging to treat than midportion tendinopathy. We iterate that symptoms of insertional Achilles tendinopathy, as with any enthesopathy, should raise suspicion about the possibility of the diagnosis of rheumatoid arthritis or spondyloarthropathy (Chapter 50).

Treatment

Treatment must consider the entheses organ as a unit. Isolated treatment of insertional tendinopathy is generally unsuccessful. For example, Alfredson's painful heel-drop protocol (very effective in midportion tendinopathy) only achieved good clinical results in approximately 30% of cases of insertional tendinopathy.⁵⁶ However, in a pilot study of 11 patients with more than two years of chronic insertional tendinopathy, sclerosing of local neovessels with polidocanol cured eight patients at eight-month follow-up. Pain during tendon-loading activity, recorded on a visual analog scale, decreased from 82 mm (3.2 in.) before treatment to 14 mm (0.6 in.) after treatment. This success rate was encouraging as nine patients had multiple pathology as is usually the case (thickened retrocalcaneal bursae, calcification, loose fragment). A heel lift worn inside both shoes (0.5–1.0 cm [0.25–0.5 in.]) is a good practical way of unloading the region.

Sometimes symptoms appear to arise mainly from the retrocalcaneal bursa itself. In these cases the symptoms may respond to NSAIDs or intrabursal corticosteroid injection (0.5 mL of corticosteroid and 0.2 mL of local anesthetic agent). Abolition of pain after local anesthesia helps confirm the diagnosis. Following injection, the patient should rest for 48 hours and then slowly resume activity, building up to full activity over a period of two to three weeks.

If conservative management fails in cases of Haglund's disease where a deformity is present, surgery is indicated. Thus, surgery may be indicated in a larger proportion of patients with insertional Achilles tendinopathy and retrocalcaneal bursitis compared with in those with midportion Achilles tendinopathy.

In summary, insertional Achilles tendinopathy is commonly associated with retrocalcaneal bursitis and Haglund's disease—it is a condition of the 'entheses organ'. This challenging condition should be distinguished from 'non-insertional' tendinopathy, which we call 'midportion' tendinopathy for simplicity. Accurate diagnosis is a key to identifying underlying conditions that may need to be treated (e.g. biomechanical abnormality, Haglund's deformity). The heel-drop program is not particularly effective in this enthesopathy. Surgical treatment and novel therapies, such as the sclerosant polidocanol, may be warranted in this condition. Spondyloarthropathy needs to be considered as a differential diagnosis.

Achilles tendon rupture (complete)

WITH JON KARLSSON

Complete rupture of the Achilles tendon classically occurs in athletes in their 30s or 40s. The typical patient is a 40-year-old male, and the male:female ratio is 10:1. The location of rupture is not associated with a 'watershed' area of poor blood supply.⁵⁷

The patient describes feeling 'as if I was hit or kicked in the back of the leg'; pain is not always the strongest sensation. This is immediately followed by grossly diminished function. A snap or tear may be audible.

The patient will usually have an obvious limp but may have surprisingly good function through the use of compensatory muscles. That is, the patient may be able to walk, but not on the toes with any strength.

PRACISE PEARL Four clinical tests can greatly simplify examination of complete Achilles tendon rupture:

1. On careful inspection with the patient prone and both ankles fully relaxed, the foot on the side with the ruptured tendon hangs straight down (because of the absence of tendon tone); the foot on the non-ruptured side maintains a little plantarflexion.
2. Acutely, there may be a palpable gap in the tendon, approximately 3–6 cm (1.5–3 in.) proximal to the insertion into the calcaneus.
3. The strength of plantarflexion is markedly reduced.
4. Simmond's (also known as Thompson's) calf squeeze test is positive (Fig. 32.2h).⁸

Treatment of the acutely ruptured Achilles tendon may be either surgical (Fig. 32.13) or conservative.

Surgical management

Open surgical treatment of Achilles tendon rupture is associated with a 27% lower risk of rerupture compared with non-surgical treatment.⁵⁸ However, open operative treatment is associated with an 11% risk of complications, including infection, adhesions and disturbed skin sensitivity.⁵⁹ Another approach to reduce these operative complications is to perform surgery 'percutaneously' but this does not eliminate the risk of complications. Early post-operative mobilization with a functional brace reduced the complication rate compared with in those who had been managed with post-operative cast immobilization for eight weeks. Post-operative management depends on the type of surgery and the surgeon's post-operative protocols. Because range of movement and strength can be difficult to regain after rupture repair, the earliest possible mobilization and rehabilitation is recom-



Figure 32.13 Intraoperative photograph showing the ruptured Achilles tendon. The surgeon is showing that the gap between the torn tendon end exceeds > 5 cm

mended. A protocol consisting of open surgical end-to-end repair, a brief period of post-operative cast immobilization (one to two weeks), followed by controlled range of motion training until the eighth post-operative week provided excellent outcomes.⁶⁰

Non-surgical management

Non-surgical management of an Achilles tendon rupture may be indicated in older patients, or patients with a low level of activity.⁵⁸ This involves cast immobilization, initially in a position of maximal plantarflexion to protect the tendon for four weeks, then after four weeks gradually reducing the amount of plantarflexion. The total immobilization time is eight weeks. Some, but not all, studies of this treatment method have reported residual lengthening of the Achilles tendon as well as the higher rerupture rate mentioned above. A recent meta-analysis of Achilles tendon rupture treatments concluded that there were insufficient data to draw conclusions about different non-operative treatment regimens.⁵⁸ It should, however, be borne in mind that non-surgical treatment leads to a high success rate provided that no rerupture occurs; thus, the main drawback of a non-surgical treatment protocol is the increased risk of rerupture. Recent studies have also discussed the possibility of treating the ruptured Achilles tendon with early range of motion without any surgical intervention. We look forward to those results with interest.

Posterior impingement syndrome

Posterior impingement syndrome of the ankle refers to impingement of the posterior talus by the adjacent aspect of the posterior aspect of the tibia in extremes of plantarflexion. An enlarged posterior tubercle of the talus (Fig. 32.14a) or an os trigonum (Fig. 32.14b) may be present. This condition is commonly found in ballet dancers, gymnasts and footballers, all of whom maximally plantarflex their ankles. It is also seen secondary to ankle plantarflexion/inversion injuries.

The os trigonum represents an unfused ossific center in the posterior process of the talus. This is a normal anatomical variant present in approximately 10% of the population. The pain arises because of the space-occupying nature of the bone; it does not depend on whether the bone is fused or not.

The diagnosis of posterior impingement syndrome is suggested by pain and tenderness at the posterior aspect of the ankle and confirmed by a positive posterior impingement test.



(a)



(b)

Figure 32.14 Posterior impingement. (a) Prominent posterior process of the talus (arrow) and (b) the os trigonum (arrow), both of which can be associated with posterior impingement

PRACISE PEARL Pain is reproduced on passive plantarflexion of the ankle (Fig. 32.2b).

If further confirmation is required, a small amount of a local anesthetic agent can be injected around the posterior talus and the impingement test performed without pain. Ideally, this test would be done under radiographic guidance so that there is certainty about the location of the injection. In practice, this is not always feasible and the test relies on the clinical accuracy of the practitioner.

Treatments that expert clinicians have used for posterior impingement syndrome include relative rest, manual mobilization of the subtalar, talocrural and midfoot joints, NSAIDs and electrotherapeutic modalities. In ballet dancers, forcing turnout and/or 'sickling' the foot can predispose to this condition, so technique assessment is essential. If the condition persists, a corticosteroid injection around the area of maximal tenderness may reduce pain. This is best done from the lateral side, as the medial aspect of the ankle contains the neurovascular bundle. Frequently, this condition does not respond to conservative management and requires surgical removal of the posterior process or the os trigonum. This can be done arthroscopically.

Sever's lesion

Sever's lesion or calcaneal apophysitis is a common insertional enthesopathy among adolescents (Chapter 40). It can be considered the Achilles tendon equivalent of Osgood-Schlatter lesion at the patellar tendon insertion.

Less common causes

Accessory soleus

Although considered a 'rare' cause of Achilles region pain, anatomical studies suggest that an accessory soleus is present in about 6% of people. This was mirrored in an Italian study of 650 athletes; 18 (2.7%) had an accessory soleus.⁶¹ The condition is more common among men than women and the average age of presentation is 20 years. The primary presenting patterns are pain in the Achilles region during exercise (a 'compartment' type pain) with swelling, or painless swelling. When pain is present it arises in the Achilles tendon. Imaging findings are characteristic; plain radiographs show a soft tissue shadow posterior to the tibia obscuring the pre-Achilles fat pad. Ultrasound, CT and MRI can each confirm a mass with

the same texture as muscle. In cases that are symptomatic, observation is an appropriate treatment but, if symptoms warrant, surgical removal of the accessory soleus is probably the best treatment.⁶²

Other causes of pain in the Achilles region

Achilles bursitis (Fig. 32.1b) is generally caused by excessive friction, such as by heel tabs, or by wearing shoes that are too tight or too large. Various types of rather stiff boots (e.g. in skating, cricket bowling) can cause such friction, and the pressure can often be relieved by using a punch to widen the heel of the boot and providing 'donut' protection to the area of bursitis as it resolves.

Referred pain to this region from the lumbar spine or associated neural structures is unusual and always warrants consideration in challenging cases (Chapters 3, 35).

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