Managing Tendon Pathology of the Ankle

Kevin A. Schafer, Zijun Zhang, and Lew C. Schon

1 Tendon Anatomy, Pathologic Classifications, and Injury Risk Factors

1.1 Tendon Microstructure: Normal Anatomy and Changes in Diseased Tendons

Tendons are predominantly composed of water (nearly 55% by weight), with roughly 70% of their dry mass composed mainly of type I collagen (fibrous proteins) [1]. Type I collagen molecules are assembled into filamentous fibrils, which are arranged parallel to the long axis of the tendon in densely packed bundles to create a tissue with a high tensile strength [2]. While collagen is the primary molecule responsible for tendon strength, other non-collagenous components of the extracellular matrix play important roles. Proteoglycans help to resist compressive forces and help modulate collagen fibril formation [3]. Elastins (a protein forming the core of elastic fibers), oriented both longitudinally and transversely to collagen fibers, potentiate elastic deformation and enhance sliding between collagen bundles [4]. The production and degradation of these and other components of tendon extracellular matrix are the responsibility of tenocytes (tendon specific fibroblasts), which react to mechanical stimuli by remodeling the tendon and participating in the events of tendon repair [5]. The cellular processes contributing to tendon homeostasis are crucial to treating tendon pathology. While exercise can result in tendon adaptation, repetitive stimuli in chronic overuse settings may disrupt cellular homeostasis and ultimately lead to altered tissue composition and tissue breakdown [6]. When compared to healthy tendon, diseased tendons have an increased proteoglycan content, an increased ratio of type III to type I collagen, a decrease in total collagen content, molecular modifications to collagen molecules and their interconnections, and an increase in vascular and nerve ingrowth (Fig. 1) [2].

Alterations in tendon microstructure were thought to involve an inflammatory process, making treatment with steroid injections and oral anti-inflammatories seem logical. However, histologic studies have failed to identify a predominate inflammatory pathway in chronic overuse injuries, challenging these treatments [8]. More recent analyses however have described a complex relationship between inflammatory mediators and tendon degeneration [9]. While repetitive stresses can lead to disruption of tendon microfibers, it can also stimulate the release of inflammatory mediators that alter tendon repair pathways. These investigations suggest that ten-

K. A. Schafer (✉) · Z. Zhang · L. C. Schon
Institute for Foot and Ankle Reconstruction, Mercy Hospital, Baltimore, MD, USA
e-mail: kschafe2@mdmercy.com; zzhang@mdmercy.com; lschon@mdmercy.com

D. Herscovici Jr. et al. (eds.), *Evaluation and Surgical Management of the Ankle*,
https://doi.org/10.1007/978-3-031-33537-2_5

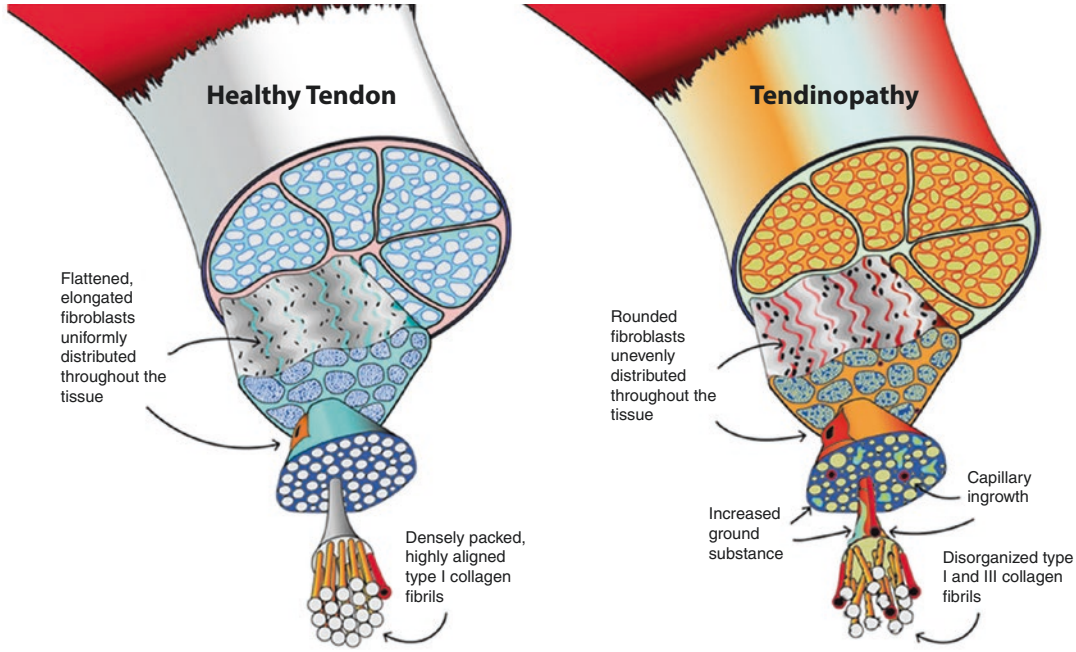

Fig. 1 Tendon microstructure in tendinopathy. Borrowed with permission from Mead et al. [7]

don degeneration and inflammation are intertwined processes at the cellular level and are not mutually exclusive.

1.2 Classifying Tendon Injury: Traumatic Injury, Tendinitis, and Tendinopathy

Tendon injuries occur frequently and can be classified into traumatic ruptures or injuries from mechanical overload. The term "tendinopathy" designates a pathologic tendon and overload injuries can be further described as a tendonitis, tendinosis, or tenosynovitis. Tendonitis indicates clinical and pathological inflammation of the tendon fibers, resulting in tendon microtears [10]. Tendinosis describes degeneration of the collagen bundle without clinical or cellular evidence of inflammation. Tenosynovitis indicates inflammation of the tendon fibers and its sheath and is only possible in tendons with a synovial sheath (peroneals, flexor hallucis longus) but not those with a paratenon (Achilles).

1.3 Extrinsic and Intrinsic Risk Factors for Injury

Contributing factors resulting in an injury can be classified into intrinsic variables that are inherent to the patient, and to extrinsic variables.

Intrinsic variables can be classified into local and systemic conditions and can make a patient more susceptible to developing tendinopathy (Fig. 2). Intrinsic conditions include anatomic, systemic, age related, and genetic conditions. Anatomic alignment and flexibility directly influence the loads experienced by tendons. For example, a cavovarus foot deformity (high arch and medially tilted hindfoot) increases stress on the peroneal tendons and lateral tissues, while a planovalgus foot (flattened arch and laterally tilted hindfoot) overloads the medial tissues and the posterior tibial tendon. Systemic conditions include diabetes, tobacco abuse, obesity, and rheumatologic disorders that result in impaired tendon microstructure and/or impaired tendon recovery after injury. Genetic conditions including Marfan syndrome and Ehlers-Danlos syndrome can result in pathologic laxity of soft tissues. Less joint

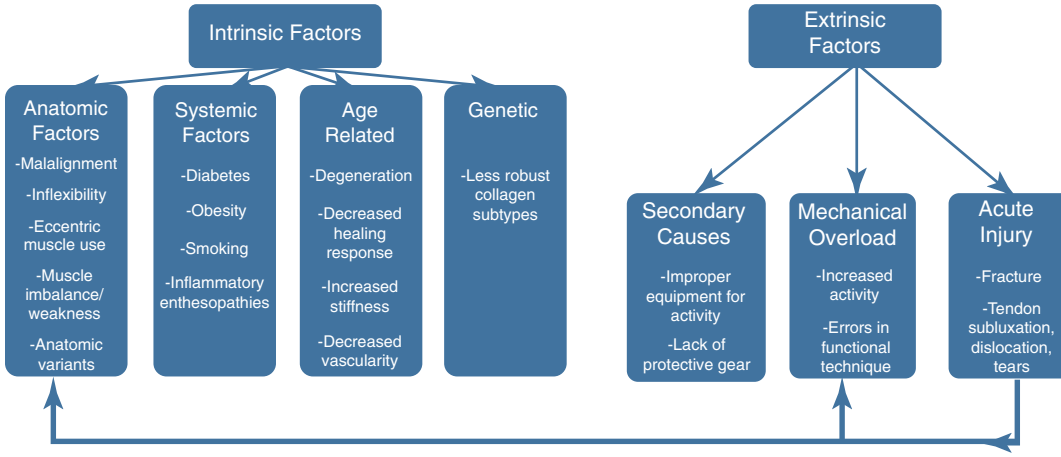

Fig. 2 Intrinsic and extrinsic factors in tendinopathy. Adapted from Federer et al. [11] and borrowed from Schafer et al. [91]. Overview of the various factors that can contribute to tendon injury. As the diagram depicts, acute injury can result in new intrinsic and extrinsic considerations

restraint from more elastic passive stabilizers can secondarily lead to tendon overload.

Extrinsic variables include acute injury, chronic mechanical overload, and environmental factors. An acute overload injury occurs with dynamic, high-impact activity where tendons experience higher loads in a shorter period of time, such as during running and jumping. Such an injury can sometimes permanently alter a patient's anatomy, secondarily resulting in change in a patient's intrinsic conditions (Fig. 2). For example, scar formation after a tendon tear can result in impaired tendon motion and decreased joint flexibility. A chronic mechanical overload condition can occur by increasing basic activities of daily living. The recurrent lower load cyclical stresses overwhelm the remodeling and repair capacity of the tendon. Lastly, environmental factors, including type of footwear, the training surface, and the type training activity, can all contribute to both mechanical overload and acute injury patterns [12, 13], and are important components of the patient history.

1.4 Biomechanics of Tendons

When discussing the different types of training activities, understanding the biomechanics of how tendons function is important as it may help explain how the injury occurred. The muscle-tendon unit is commonly injured during eccentric contraction. This is defined as contracting or tensioning the muscle while it is being lengthened. Concentric contraction indicates that the muscle is being shortened. The peak torque in the muscle-tendon unit is greater during eccentric contraction than during either isometric or concentric contractions where the muscle length stays a constant length or shortens, respectively [14].

After reviewing the general types of tendon injury and the mechanisms for tendon injury, we will now discuss commonly encountered tendon specific conditions. Each of the following sections will discuss the common presentations of each tendon condition and will review the key features of diagnosis and management.

2 Anterior Tibial Tendon

2.1 Pathology and Evaluation

The tibialis anterior tendon is the primary dorsiflexor of the ankle and plays a crucial role in controlling ankle plantarflexion during heel strike via eccentric contraction. These functions are crucial to normal gait and athletic activity.

As previously described, chronic tendinopathy or an acute rupture can occur. In chronic ten-

dinopathy, prior to rupture, combinations of intrinsic and extrinsic factors result in tendon overload and progressive tendon degeneration. Intrinsic conditions that can contribute to tendon degeneration include gout, inflammatory arthritis, diabetes, and underlying arthritis with bony exostoses that abrade the tendon. Many patients develop anterior tibial tendinopathy with minimal or no noticeable symptoms. As a result, it is not uncommon for these patients to present after they have sustained a rupture. However, patients may experience pain and swelling along the course of the tendon prior to rupture, with these symptoms exacerbated by basic repetitive activity.

When examining a patient with chronic tendinopathy, the tendon may appear enlarged. The provider should assess for tenderness along the course of the tendon, pain during resisted dorsiflexion, and power during resisted dorsiflexion. Subtle weakness can be difficult to detect, and it is crucial to compare dorsiflexion strength to the unaffected extremity. It is also important to assess ankle flexibility via the Silfverskiöld test [15] as a tendoachilles or gastrocnemius contracture can create greater resistance to dorsiflexion. Following exam, weightbearing radiographs are routinely obtained to examine for contributing pathology such as midfoot arthritis and bony exostoses. If the diagnosis is unclear, magnetic resonance imaging (MRI) can be obtained to further evaluate the condition of the tendon and detect tenosynovitis.

If the tendon ruptures in the setting of chronic tendinopathy, the typical location is 2–3 cm proximal to its insertion [16], which is defined as the avascular zone of the tendon [17]. These degenerative ruptures most commonly occur in males older than 45 years of age [18], usually without injury or antecedent pain. There is often a delay to diagnosis with patients frequently presenting with a poorly described gait disturbance. Findings on examination include a painless palpable mass at the anterior ankle representing the proximal tendon stump [19], a loss of the normal visual and palpable contour of the tendon during resisted dorsiflexion (Fig. 3), and a foot drop noticeable during gait (steppage gait). Comparing both extremities often demonstrate less supination and

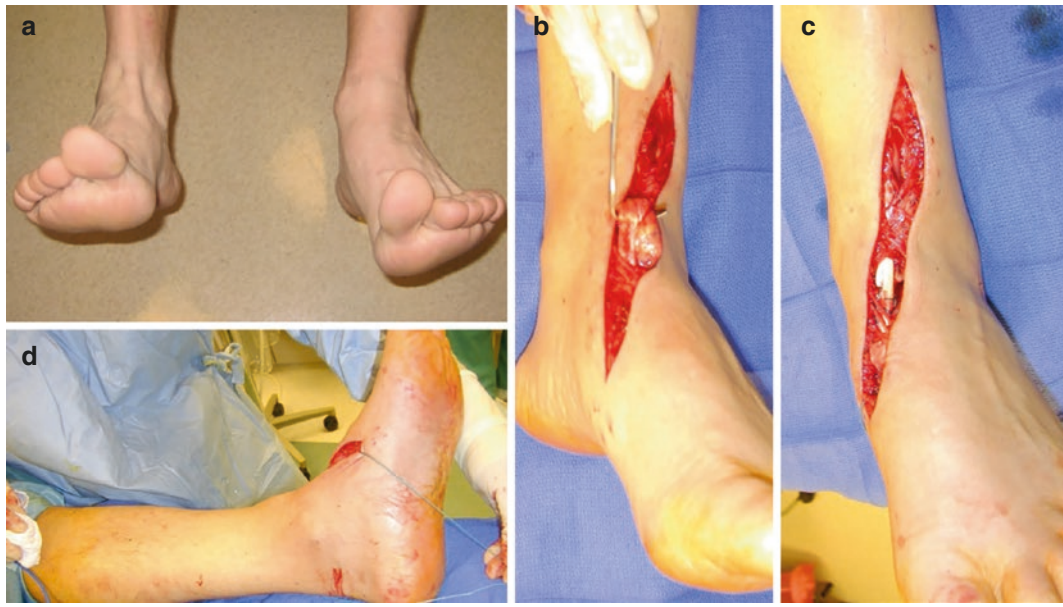

Fig. 3 Tibialis anterior ruptures. (**a**) Clinical photo showing the loss of tendon contour after tendon tear on the patient's left versus the intact right side. (**b**) After surgical incision, the ruptured tendon is localized. (**c**) A tendon repair was performed in this clinical example using a hamstring allograft. (**d**) The repair is tensioned so that the ankle rests in slight dorsiflexion

more eversion of the forefoot with active ankle dorsiflexion due to recruitment of the toe extensors. However, the patient will have less dorsiflexion strength when compared to the noninvolved extremity. In patients with delayed diagnoses, clawing of the toes may be noted, due to the sustained recruitment of the toe extensors for ankle dorsiflexion [20].

Acute ruptures are more apparent at the time of injury. They are less common than tendinopathic ruptures, with the former accounting for 20% of patient presentations [21]. Acute ruptures occur in younger, more active individuals resulting after an eccentric contraction with the ankle in plantarflexion, although penetrating trauma and injury associated with distal tibia fractures have also been described [22, 23]. Swelling, ecchymosis, and tenderness along the tendon are more impressive than in patients with attritional ruptures. In penetrating trauma, extension of the hallux and lesser toes should also be evaluated as these tendons can also be injured. A tendon rupture due to a tibial fracture is rare but can be easily overlooked as lack of dorsiflexion is attributed to antalgic guarding and/or deformity through the fracture [21, 22]. However, a nonpalpable tendon or palpable gap along with an inability to actively dorsiflex should raise suspicion for this injury, warranting additional advanced imaging, such as ultrasound and/or MRI.

In both acute and chronic ruptures, weightbearing radiographs of the ankle should be obtained routinely to evaluate for additional injury or contributing pathology. Even if the exam is consistent with tendon rupture, an MRI can be helpful to identify the level of the tendon tear and overall condition of the tendon to assist with surgical planning.

2.2 Treatment

In patients with symptomatic tendinopathy prior to rupture, the initial treatment depends upon the severity of symptoms. When the patient is acutely painful, the preferred initial intervention is immobilization in a walking boot during the day and in a night splint when they are resting, along with oral anti-inflammatories and icing for several weeks. The authors recommend against steroid injections given the risk of associated tendon rupture. When pain has improved, patients begin a stretching routine to address calf tightness. When pain is minimal, eccentric strengthening is introduced. If these modalities are ineffective after several months, extracorporeal shockwave therapy (ESWT), platelet rich plasma (PRP) or bone marrow aspirate concentrate (BMAC) injections can be considered in addition to the immobilization to trigger a biologic response, although clinical data is lacking to support routine use of these second line modalities. If these conservative approaches fail, surgical debridement, suture reinforcement, and the use of an Achilles lengthening, to eliminate equinus, can be considered. In low demand patients unable to undergo surgery, a dorsiflexion assist ankle-foot orthosis (AFO) can mitigate tripping risks and can lead to good outcomes when treating tendinopathy or an attritional tendon rupture.

The specific surgical intervention or reconstruction is dependent upon tendon quality, timing from injury, mechanism of rupture, the physical demands of each patient and their medical comorbidities. However, tendon repair or reconstruction is the preferred treatment to restore strength and range of motion in most patients [21]. Outcomes after tendon repair or reconstruction report a high level of satisfaction and marked functional improvement from their preoperative state [16, 23, 24]. In healthy tendons, primary repair is usually possible within 6 weeks of the injury. In injuries where the tendon has avulsed at the insertion, the tendon should be anchored to the medial cuneiform with either a bone tunnel and interference screw, suture button device, or suture anchor [20]. Regardless of the surgical timing, chronic weakness in dorsiflexion and inversion relative to the injured extremity is anticipated [21] and should be discussed preoperatively.

Beyond 6 weeks and in acute attritional ruptures, the feasibility of end-to-end repair should be assessed [20]. Significant tendon gaps after

tendon debridement often require reconstructive procedures. Reconstructive procedures have included Z-plasty lengthening [25], free tendon segment transposition/sliding grafts [11], turn down grafts [26], free tendon autograft or allograft (Fig. 3) [27], and tendon transfers [16]. For gaps less than 4 cm [20], local soft tissue reconstructions with tendon lengthening or sliding grafts are possible. For larger gaps, tendon autograft or allograft reconstructions and tendon transfers are favored. In the case of severe atrophy, tendon transfers that add a functional muscular unit are preferred over graft reconstructions.

Post-operatively, all patients are kept in a splint to maintain dorsiflexion for the first 2 weeks. After 2 weeks, they are immobilized in a walking boot during the day and in a night splint when they are sleeping or resting. Weightbearing in a boot brace is typically initiated at 2 weeks. The patients are given strict instructions to avoid active or passive plantarflexion for 3 months. Between 3–6 months, the boot can be gradually eliminated.

3 Achilles Tendon

The Achilles is different from other tendons as it does not have a true synovial sheath but rather a "paratenon." This facilitates tendon gliding and provides vascularization to the epitenon and endotendon [28]. The pathologies of the Achilles can be divided into tendinopathies and traumatic ruptures, which will be discussed in the following sections.

3.1 Tendinopathies: Insertional and Non-insertional

Achilles tendinopathies are the most frequent Achilles tendon disorder [29], and have two primary subtypes: insertional and non-insertional. Insertional tendinopathies involve the calcaneal insertion of the tendon, whereas non-insertional tendinopathies involve the tendon 2–6 cm proximal to its insertion [30].

3.1.1 Insertional Tendinopathy: Pathology and Evaluation

In insertional tendinopathy, patients describe pain at the posterior heel that is exacerbated by inactivity or limits their activity, difficulty with shoe wear, posterior ankle swelling, and often report the development of a "bump," due to the development of insertional osteophytes (enthesophytes). On examination, the tendon is often thickened and painful to palpation. Deep and proximal to the insertion, there may also be a painful retrocalcaneal bursitis, seen in conjunction with a prominent posterosuperior calcaneal prominence (Haglund's deformity). However, retrocalcaneal bursitis and a Haglund's prominence can be present in isolation without insertional tendinopathy. Additionally, one should also evaluate ankle dorsiflexion, with the knee flexed and extended, as decreased dorsiflexion contributes to pathologic tendon loading [31].

After the initial examination, the authors recommend lateral weightbearing radiographs of the ankle. These best profile the development of enthesophytes, intratendinous calcifications, and a Haglund's deformity. It is important to recognize, however, that bone spurs are frequently found in asymptomatic patients [32]. An MRI can be useful to study the size and quality of the tendon, to identify any associated tears, and to detect any adjacent retrocalcaneal bursitis or bony edema. Although MRI studies have been used to determine whether earlier surgical intervention is warranted [33], high-level evidence is lacking to support routine use of MRI scans. Ultrasound assessment for tendon thickening, while more affordable than MRI, has less diagnostic utility in insertional pathology [34] than in noninsertional tendinopathy.

3.1.2 Insertional Tendinopathy: Treatment

Treatment begins with non-operative care. Patients with severe tenderness and marked pain with passive motion and weightbearing respond poorly to immediate strengthening and stretching exercises. Instead, the authors' preference is several weeks of immobilization in an off the shelf solid AFO with wedges or in a hinged brace,

placing the foot into an equinus position (approximately 20°). Immobilization is supplemented with oral anti-inflammatories and icing. Once severe pain and tenderness subside (typically 1–2 weeks), physical therapy is initiated. Eccentric strengthening exercises are then the primary intervention [35]. Using limited ankle range of motion eccentric programs (plantarflexion to neutral dorsiflexion) have higher patient satisfaction than a full eccentric motion programs (plantarflexion to below neutral dorsiflexion) [32]. A typical rehabilitation program lasts 12 weeks, although some patients may require longer treatment. The benefit of these exercises is that they are low risk, and affordable as a first line intervention [35]. Active and passive (night splints) stretching to improve range of motion is also a regular component of nonoperative therapy. Use of ESWT has also been studied, with both high energy (single treatment requiring anesthesia) and low energy (multiple in office treatments without anesthetic) proposed when eccentric training fails [35–37]. Additional described treatments include PRP and BMAC injections [35, 38], but high-quality evidence is lacking to support their routine use. The authors strongly recommend against steroid injections given the known risk of complete tendon rupture [39].

When nonoperative treatment fails, the risks and benefits of operative treatment are discussed with the patient. After tendon debridement and retrocalcaneal decompression, patient outcomes are favorable with an estimated satisfaction of 87% [32]. It is important however to discuss the course of recovery with the patient, as full recovery often takes close to 12 months [40]. It is also imperative to discuss potential surgical complications, with complication cited between 6-30% that include scar sensitivity and delayed wound healing [32].

When proceeding with surgical treatment, the most common procedure involves partial detachment of the tendon insertion to allow for tendon debridement (tendon fibers and insertional calcifications), a retrocalcaneal bursectomy, and a Haglund's prominence exostectomy. The tendon is then secured back down to the calcaneus with bony anchors. When >50% of the tendon is debrided (more commonly in revision cases), a tendon augmentation using the flexor hallucis longus (FHL) or flexor digitorum longus transfer (FDL) is recommended with low morbidity and good functional outcomes reported [41, 42]. The authors' postoperative protocol is identical for insertional tendinopathy, non-insertional tendinopathy, and acute ruptures (please see end of Sect. 3.2.2).

One final point is to address a gastrocnemius contracture. If a preoperative Silfverskiöld test demonstrates a contracture, a gastrocnemius recession can be performed at the start of the procedure to help improve ankle motion and reduce tension on the debrided and repaired tendon. Adjusting the tension on the repair and reconstruction is necessary for a successful functional outcome. Of note, an isolated gastrocnemius recession has been proposed as a stand-alone surgical treatment in patients with Achilles tendinopathy (insertional and noninsertional) with a coexisting gastrocnemius contracture, reporting faster recoveries and lower surgical risks [43]. However, patients should be told that plantarflexion power and endurance are significantly decreased after a recession [43], which may not be tolerated in more active individuals (laborers, athletes).

3.1.3 Noninsertional Tendinopathy: Pathology and Evaluation

Tendinopathy within the body of the Achilles tendon is termed noninsertional tendinopathy. It is the most common Achilles pathology in athletes and occurs with roughly equal frequency to insertional tendinopathy in nonathletes [44]. A tendinosis is the most frequent presentation, although an inflammatory tendinitis can occur with an acute overload event and can be treated like other acute strains. The causes of mechanical overload are similarly multifactorial (Fig. 2). While a hypovascular zone of the tendon has been described in cadaveric studies and suggested as a root cause of tendinopathy [45], this theory has been debated in an in vitro study [46].

Patients complain of pain 2–6 cm proximal to the insertion that increases with activity. It occurs

at the beginning and end of exercising, with an intermediate period of reduced pain and dysfunction [30]. There is visible swelling in this area and the tendon may feel thickened and fibrotic and is tender to palpation. A lateral ankle radiograph may aid in identifying intrasubstance calcifications and is obtained routinely by the authors. If the diagnosis is unclear or when surgical treatment is being considered, an MRI can be helpful for evaluating the thickness and length of the diseased tendon. While ultrasound can offer useful information regarding tendon size and tendon quality [47], it requires considerable technical skill and experience. The authors do not routinely use ultrasound and prefer MRI when initial nonoperative treatment fails or the diagnosis is unclear.

3.1.4 Noninsertional Tendinopathy: Treatment

The first line of treatment is nonoperative management with eccentric strengthening [48]. While other modalities have been investigated in combination with eccentric training (prolotherapy, laser therapy, ESWT), no study has shown a definitive benefit compared to eccentric training alone [48]. When eccentric training is ineffective, however, there is evidence to support ESWT [49]. Additional evidence for other treatments like PRP or BMAC injections is needed prior to routine use [50].

When nonoperative management fails, operative treatment, consisting of a tenotomy and debridement, can be considered. Both open and endoscopic debridement have reported good results with an overall patient satisfaction rate of 90% or higher [48]. Additionally, isolated gastrocnemius recession for patients with a gastrocnemius contracture has also been described with favorable outcomes [51]. When debridement results in >50% or complete loss of a segment of tendon, augmentation with a FHL or FDL transfer, attaching tendon to the calcaneus via an anchor, bone tunnel, or suture button is effective [52]. In addition, the authors also perform a tenodesis of any Achilles tendon remnant to the transferred tendon, to add additional power to the transfer.

3.2 Acute and Chronic Achilles Tendon Ruptures

3.2.1 Pathology and Evaluation

Traumatic Achilles tendon ruptures are most seen in males, typically in their 30s or 40s, who participate in recreational or sports intermittently [53]. They often present with complaints of posterior ankle pain, swelling and a “popping” sensation on the back of the leg described as if they were struck from behind.

The physical examination can confirm the diagnosis with a near 100% sensitivity [54]. The Thompson test, performed with the patient in the prone position or on their knees, involves the examiner squeezing the gastrocnemius muscle to elicit a plantarflexion response that occurs if the tendon is in continuity (Fig. 4). This examination maneuver has a sensitivity 0.96 and specificity 0.93 [54]. While the patient is positioned in this fashion, the resting tension of the tendon is also examined by observing the resting dorsiflexion angle of both ankles, as a torn Achilles tendon commonly results in increased resting dorsiflexion (Fig. 4). Other common findings include a palpable defect in the tendon and decreased plantarflexion strength with an inability to perform a single leg heel raise or weakened plantarflex against resistance [54]. If the diagnosis is unclear, plain radiographs, ultrasound, or an MRI study can help to confirm the presence of a partial or complete tear [55]. Radiographs can be helpful in identifying calcifications along the tendon or near the insertion suggesting a preexisting tendinopathy. An MRI also has increased utility in chronic injuries (>4–6 weeks from injury), as the body quickly fills the palpable gap with scar tissue such that a gap is not clearly palpable.

3.2.2 Treatment

In the acute injury, without pre-existing tendinopathy, several randomized trials have reported similar functional outcomes, strength, and rates of re-rupture for both operative and nonoperative care, when an accelerated (early motion and weightbearing) rehabilitation protocol was followed [56]. However, higher complication rates

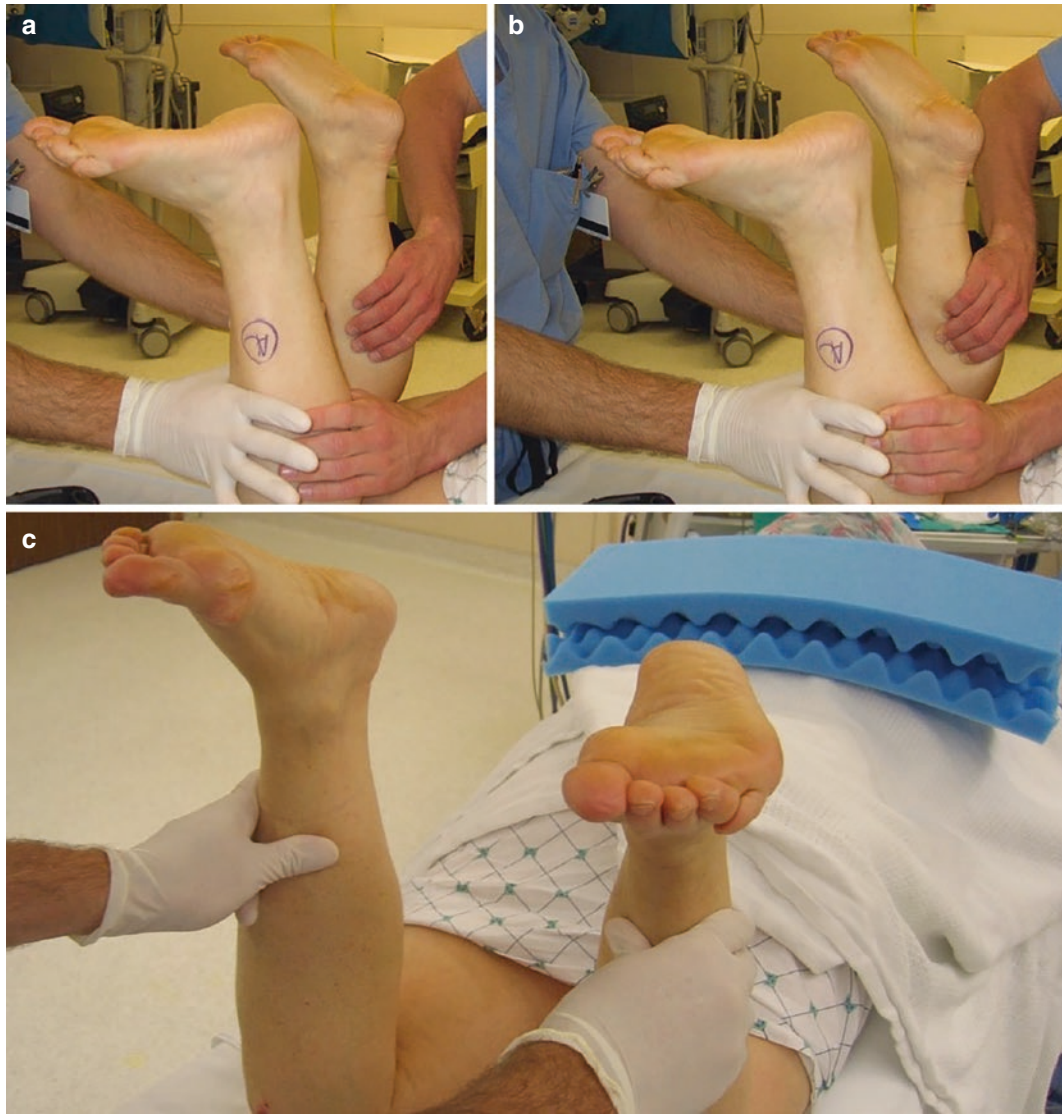

Fig. 4 Acute achilles tendon rupture examination. (**a**, **c**) On prone exam, there is increased resting dorsiflexion on the right side where the Achilles tendon is torn. (**b**) The gastrocnemius is stimulated with a calf squeeze, and the intact muscle tendon unit on the left creates a plantarflexion response on the left versus no response on the right

(wound complications, infection, and sural nerve damage) have been noted with surgery.

A recent multicenter, randomized controlled trial, however, has shown that surgical treatment reduces the risk of re-rupture (6.2% vs. 0.6% nonsurgical and surgical treatment, respectively), even when early functional rehabilitation is utilized for nonoperative care [57]. Other studies have shown that surgery results in a faster and greater recovery of strength, faster return to work, especially in the military population [58, 59], while avoiding a 2 cm Achilles lengthening seen in nonsurgical patients [60]. Although nonoperative care is a viable option, the authors favor surgical management unless the patient has a pre-existing condition(s) that may increase the risk of postoperative complications (e.g., poorly controlled diabetes or vascular disease).

The optimal rehabilitation protocol, for nonoperative care, has been debated. A meta-analysis

utilizing functional rehabilitation (early plantarflexion immobilization followed by controlled motion weightbearing 10 days after injury) was found to be superior to immobilization and delayed motion protocols, resulting in lower rates of re-rupture [56]. However, a meta-analysis comparing these two protocols did not find a difference in re-rupture rates, strength, or ability to return to sport or work [61]. When using nonoperative care, the authors prefer functional rehabilitation, given better patient compliance and satisfaction. The critical time between tendon rupture and beginning nonoperative care is debated, but expert opinion has suggested immobilization in plantarflexion must be initiated within 48 h of injury [62]. The authors favor operative repair when patients present beyond 48 h without plantarflexion immobilization. Our preferred protocol has been previously described by Glazebrook et al. [62].

The critical time for a repair is currently unknown. While no functional differences were detected in repairs performed within 1 week of the injury [63], no study has compared outcomes with later repairs. However, despite a high rate of return to activity with surgery, patients can expect a chronic strength deficit compared to the uninjured extremity [7, 64]. The options for surgical treatment include a traditional open or minimally invasive repair. The latter offers reduced rates of wound complications when compared to open repair, but has equal rates of re-rupture, return to preinjury activity level, time to return to work, and ankle range of motion [65, 66]. However, minimally invasive techniques have reported a greater risk of sural nerve injury and palpable suture knots. In a recent study, approximately one in nine patients undergoing surgery experienced a complication, with overall complication rates similar in open and minimally invasive repairs [67]. These included wound dehiscence, deep and superficial infections, symptomatic venous thromboembolism, sural nerve injury, and re-ruptures.

In chronic ruptures (>4–6 weeks), surgical treatment is indicated unless the patient has low functional demands and significant comorbidities that preclude surgical intervention. Other contraindications include patients with an active infection, severe peripheral vascular disease, and patients who are household or poor community ambulators. These patients are best managed with an AFO. If patients are candidates for a repair, a primary end to end repair is often not possible when the tendon gap exceeds 2 cm [68]. However, there is no consensus regarding the best reconstruction option for each size defect [68]. Techniques for reconstruction include Achilles tendon turndown flaps, a V-Y gastrocnemius fascial advancement, FHL and FDL transfers, bone block Achilles allografts, and free tendon autografts and allografts [68]. Good to excellent results have been described but there is a paucity of comparative studies amongst these techniques. The authors recommend the following algorithm. For chronic cases that have a mild difference in resting tendon tension or less than a 2 cm gap, an end-to-end repair or advancement of the tendon to the calcaneus is performed. For cases with a 2–5 cm gap or moderate difference in resting tendon tension, a V–Y advancement is performed. With a greater than 5 cm gap or severe difference in resting tension, a turndown procedure is performed. If there is inadequate tissue proximally to perform the turndown, an allograft semitendinosus is used to span the void. When any of these scenarios is associated with atrophy of the gastrocnemius-soleus complex, an FDL or FHL tendon transfer is added to augment plantarflexion strength.

The authors' postoperative care, for tendon repairs or reconstructions, is to place patients into a splint in equinus until sutures are removed at 10 days. They are then placed into a hinged boot brace, in 20° of equinus, until 6 weeks postoperatively. While in this plantarflexion boot brace, protected weightbearing with crutches is initiated. After 6 weeks, the ankle can be brought into a neutral position with full weightbearing. During the 3–6 months period, the boot can be gradually eliminated.

4 Peroneal Tendons

The peroneus longus and brevis tendons are the principal foot evertors, but also assist with ankle and forefoot plantarflexion. These tendons have

critical functions of opposing inversion to help maintain balance during gait, but also function as dynamic stabilizers of the lateral ankle during rapid inversion events.

4.1 Anatomical Considerations

In most individuals, the peroneus brevis muscle fibers extend more distally than peroneus longus muscle fibers, with brevis muscle extending approximately 16–20 mm above the tip of the fibula [69], or just proximal to the entrance of the tendons into the fibular groove [70]. Extension of the muscle beyond this point is termed a low lying muscle belly and has been hypothesized as a contributing factor in cases of tendon subluxation, tenosynovitis, and tendon tearing [69]. Other variations that may contribute to tendon pathology include accessory muscles within the peroneal sheath. These include the peroneus quartus and quintus and are found within the peroneal tunnel in 10–30% of patients [71]. These accessory muscles and their tendons can cause overcrowding of the peroneal tunnel and produce secondary tendon stenosis or retinacular attrition.

As the tendons course distally, regions of angular change or compression against bony prominences result in localized tendon stress. Prior to an abrupt change in their route at the distal tip of the fibula, the tendons reside posterior to the fibula in a retrofibular groove that is stabilized by a fibrocartilaginous ridge and the superior peroneal retinaculum (SPR). Traumatic injury to the SPR, a shallow fibular groove, and anomalous tendons overcrowding the tendon tunnel have been proposed as causes of SPR incompetence or laxity [70]. When the SPR is torn or lax, the brevis can subluxate laterally over the lateral corner of the fibula. The brevis may then be compressed against this bony prominence with contraction of the overlying peroneus longus [70]. Distal to the fibula, the tendons enter their own separate subsheaths, separated by the peroneal tubercle. At this location, tendon stability is achieved by the inferior peroneal retinaculum. However, the retinaculum and the tubercle may create another source of compression. This commonly occurs in the setting of a prominent or hypertrophied tubercle and most commonly affects the peroneus longus [72]. A final area of mechanical stress is at the cuboid, where the longus tendon undergoes an angular turn below the cuboid as it courses towards the first metatarsal.

While these mechanical stresses have been described as causes of tendon pathology, regions of tendon hypovascularity have also been suggested as secondary contributing factors. Older cadaveric studies have discussed avascular regions at the retromalleolar groove, affecting the peroneus brevis, and from the distal fibula to peroneal tubercle and at the tendon's entrance into the cuboid tunnel affecting the peroneus longus [73]. However, more recent publications have challenged this hypothesis, showing that both tendons are largely well vascularized by a vincular network [74].

4.2 Tendinopathy

4.2.1 Pathology and Evaluation

Peroneal tendinopathy often presents with pain and swelling at the lateral ankle and hindfoot. Many report no change in activity and cannot describe a precipitating injury. Others may report a history of an ankle inversion injury, which is often associated with patient perceptions of ankle instability. Symptoms are exacerbated by activity and abate with rest. Pain at the posterior aspect of the distal fibula commonly represents peroneus brevis pathology, whereas pain secondary to peroneus longus pathology is more commonly localized to the peroneal tubercle and cuboid tunnel [71]. However, these patterns are not mutually exclusive.

Examination requires careful inspection with patients standing and ambulating as patients with cavovarus alignment and/or metatarsus adductus deformities will chronically overload their peroneal tendons. The course of the tendons should be palpated to assess tenderness, swelling, and tendon thickening. Passive plantarflexion and inversion as well as active plantarflexion and eversion may elicit pain. Resisted eversion or having the

patient perform circumduction (circles) of the foot and ankle may result in palpable and/or visible tendon subluxation or dislocation with an incompetent SPR. The stability of the ankle should also be assessed, as lateral ligamentous laxity results in increased work of the peroneal tendons to stabilize the lateral ankle and hindfoot.

Radiographic evaluation should begin with weightbearing radiographs of both the foot and ankle. These are useful to help evaluate alignment, integrity of the neighboring joints, and identify contributing osseous pathology, including a prominent peroneal tubercle or os peroneum. Ultrasound may show a dynamic tendon subluxation, dislocation, tears and instrasheath snapping [75]. An MRI is obtained when the diagnosis is unclear or when symptoms are refractory to nonoperative care. However, a standard ankle MRI with the patient supine may result in increased tendon signal without true pathology secondary to the so-called *magic angle effect*. This occurs when the tendon collagen fibers are oriented 55° relative to the magnetic field, producing an imaging artifact [76]. These effects can be mitigated when the patient is imaged in 20° of plantarflexion [77], or in the prone position [76]. There is less suspicion for imaging artifact if the abnormal tendon signal is appreciated in multiple planes and multiple sequences (T1 and T2 images), and if there is additional pathology including thickening of the tendon or adjacent tenosynovitis.

4.2.2 Treatment

Nonoperative treatment begins with a short period of rest from activity, ice, oral nonsteroidal anti-inflammatories, compression, and immobilization in a boot or stirrup brace limiting inversion and eversion. If a foot deformity exists and is flexible, orthotics or wedges can be used to help correct the deformity and offload the peroneals. Once symptoms have begun to improve with rest and immobilization, the authors begin physical therapy to strengthen the peroneals and other dynamic ankle and hindfoot stabilizers. When rest, immobilization, and therapy fail beyond 3 months, ESWT, PRP, or BMAC injections are low risk nonsurgical alternatives currently lacking supporting evidence for routine use [78]. Despite reports of intrasheath injection of steroid with modest pain relief and without frequently observed tendon rupture [79], the authors do not offer this treatment to their patients given this potential risk.

After failed nonoperative treatment, surgical treatment can be considered. While tendoscopy can be a useful technique for diagnosis and limited surgical debridement, its use for more involved debridement and tendon repair remains to be defined [71]. Standard treatment consists of an open debridement of the sheath and tendons, with the excision of inflamed tenosynovium and frayed or unhealthy appearing tendon. Pathologic malalignment (cavovarus foot, ankle joint varus, and metatarsus adductus) deformities contributing to tendon overload along with anatomic variants (prominent peroneal tubercle, low lying muscle belly, accessory tendons) may also contribute to pathology and should be addressed.

The traditional teaching has reported that debridement of >50% of either tendon may require a tenodesis to the intact tendon or the use of an allograft/autograft tendon [80]. However, a recent biomechanics study showed that two-thirds of the tendon can be debrided and the intact tendon can still resist physiologic tensile loads [1]. After debridement, the recommendation is to tubularize the remaining tendon, without supplemental augmentation or tenodesis, even if less than 50% remains, as long as the tendon can resist a substantial intraoperative longitudinal stress by the surgeon [80]. When insufficient tendon remains, tenodesis to the noninvolved tendon or an allograft/autograft reconstruction may be needed. Advocates of tenodesis argue that the procedure is simple and does not rely on allograft/autograft healing [81]. Proponents of allograft/autograft reconstruction argue that tenodesis does not restore adequate tension to the tenodesed tendon and that reconstruction is less likely to result in chronic foot imbalance [82].

If there is insufficient tendon for repair or a gap after debridement, the authors' preferred approach is a turndown procedure. In this tech-

nique, a long section of the proximal tendon is harvested and flipped distally to fill the void (Fig. 5). Suturing the turned down segment from its distal attachment to the most proximal zone where the turn down was harvested is critical to maintain continuity between the turned down graft and the proximal segment. The authors prefer to preserve the roles of the longus and brevis and perform a tenodesis only in patients with low functional demands. When the longus cannot be salvaged, due to its distal segment, a suture anchor into the fifth metatarsal tuberosity can allow the peroneus longus to function as a second brevis. In the relatively rare scenario when both tendons are irreparable, interposition tendon grafting can be performed if there is adequate tendon excursion and healthy muscle [83]. If the remaining muscle/tendon unit does not have excursion or the muscle is significantly atrophied, an interposition graft will have minimal function. As such, these conditions require treatment with either an FDL or FHL tendon transfer, resulting in good functional outcomes albeit with residual strength and balance deficits [84]. After any of the above procedures are performed, it is crucial to repair or reconstruct the superior peroneal retinaculum to avoid tendon instability and dysfunction. In addition, it may also be necessary to perform a groove deepening of the fibula. This will unload the tendons, provide adequate space for the reconstruction, and avoid stenosis or potential tendon subluxation.

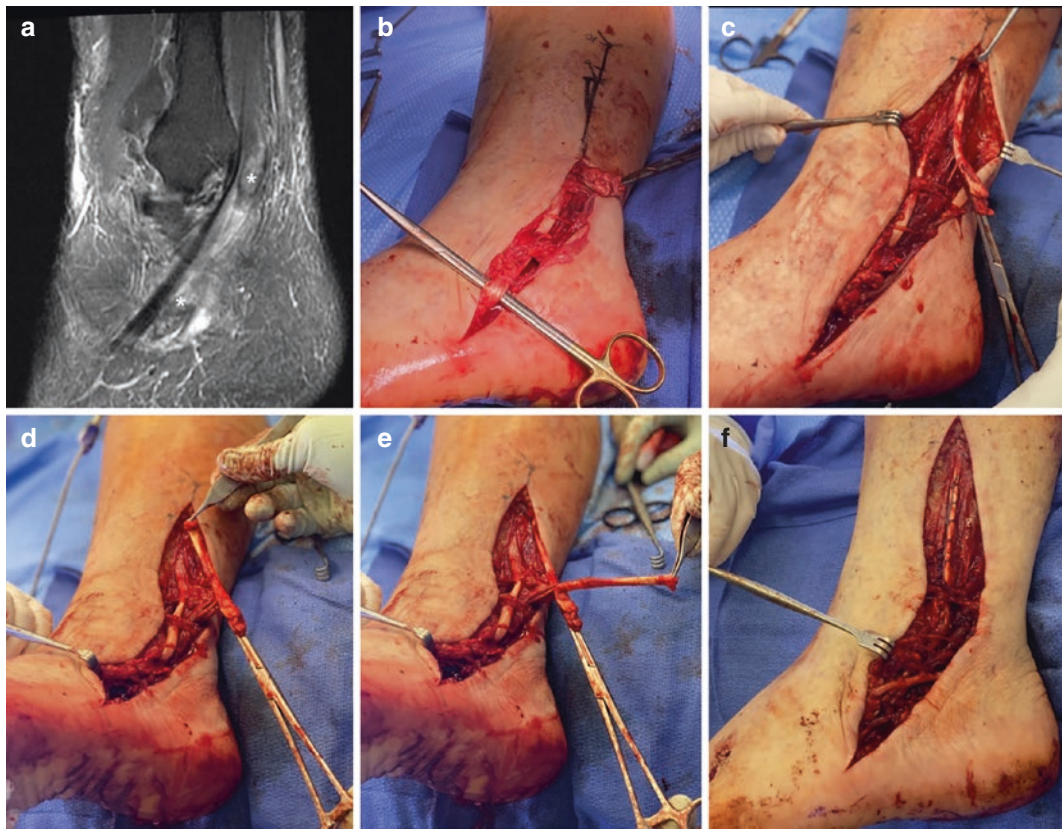

Fig. 5 Peroneal tears and turndown repair. (**a**) A select sagittal MRI showing distal intrasubstance fibrillations of the peroneus brevis and completely torn peroneus longus with a large gap between the proximal and distal tendon stumps. (**b**) Surgical photo showing the intrasubstance tearing with preserved continuity of the frayed peroneus brevis overlying the dissection forceps, and a torn peroneus longus. (**c**) The intact peroneus longus tendon proximally is dissected and isolated. (**d**–**f**) 50% of the tendon is transected from proximal to distal, allowing the tendon to then be turned down for distal repair

The authors' postoperative protocol is to immobilize the patient in a plantarflexion splint for 10 days after surgery. Patients are then transitioned into a hinged boot brace in equinus for 6 weeks and begin progressive weightbearing and protected motion. In addition, a night splint or brace that protects against passive inversion during sleep is important for the first 3 months. Early protected range of motion is essential to prevent tendon adhesions and involves restricting active and passive dorsiflexion beyond neutral, and avoiding active eversion and passive inversion, to prevent excessive tension on repaired or reconstructed tendons. After 6 weeks, the ankle can be brought into neutral position and progressed to full weightbearing. During months 3–6 the boot can be gradually eliminated. The authors use this protocol after all peroneal tendon reconstruction and repairs (following section). Self-directed physical therapy versus formal therapy is discussed on a patient-by-patient basis.

4.3 Tendon Tears

Peroneal tendon tears can occur acutely during inversion injuries or can develop as part of more chronic tendinopathies. The peroneus brevis is more commonly torn, with longitudinal tears primarily noted at the posterior lateral malleolus [81]. As with previous tendon evaluations, MRI and ultrasound are useful modalities to localize the location and extent of a tear. If a tear is identified but the patient is asymptomatic, no further treatment is required. If symptomatic, however, the treatment algorithm is identical to that discussed above for tendinopathy. When nonoperative care fails and tears treated surgically are deemed repairable, both absorbable and nonabsorbable suture have been used without any clinical difference [85]. If tendon repair isn't possible and reconstruction is required, the same treatment algorithm detailed above is followed. In general, surgery improves outcomes with high patient satisfaction and high rate of return to work, although athletic patients may not return to their prior sporting level [86].

4.4 Instability

4.4.1 Pathology and Evaluation

Peroneal instability, from an incompetent SPR, permits subluxation or dislocation of both tendons. Acute SPR injuries occur during forced dorsiflexion with the hindfoot inverted with simultaneous contraction of the peroneal tendons [71], with forced dorsiflexion during eversion also described as a cause. Patients report a popping sensation at the lateral ankle followed by swelling. This is commonly misdiagnosed and treated as an ankle sprain. In contrast, retinacular attenuation occurs when anatomic variants overcrowd the peroneal tunnel [81], or in the setting of lateral ligament insufficiency with instability [87].

There are five different patterns of instability. The first, and most common, is an elevation of the retinacular attachment from the lateral fibula without complete detachment of either the SPR or fibrocartilaginous rim from the fibula. This creates a potential space for anterior peroneal tendon dislocation [88]. Second, and less common, the SPR elevates from the fibula and the fibrocartilaginous rim and is disconnected from the fibula. Third and even less common, the SPR avulses from the fibula with a cortical fragment producing the so called "fleck sign" [80]. Rarely, in a fourth type of instability, the SPR avulses from the calcaneus [71]. The fifth type of instability, described by Raiken et al., is an intrasheath subluxation injury where either the tendons swap their normal orientation or where the longus tendon displaces through a longitudinal tear of the brevis [75].

In either acute or chronic injury, subluxation or dislocation of the peroneal tendons can be elicited on exam with ankle circumduction or forced dorsiflexion and eversion from a plantarflexed and inverted position. Weightbearing radiographs may reveal the fleck sign, while an MRI and/or ultrasound can help to identify concomitant tendon tears.

4.4.2 Treatment

Treatment of peroneal instability is based upon the patient's level of activity and the chronicity of the injury. In an acute injury, and in a nonathletic patient, where the tendons are reduced into the

retromalleolar groove, 6 weeks in a nonweight-bearing cast in slight plantarflexion and inversion can allow the SPR to heal with variable reported success [80, 89]. When acute dislocation occurs in a young and active patient, when the tendons are not reducible, when nonoperative treatment fails, or when peroneal instability is chronic, surgical treatment is recommended.

The surgery consists of reduction of the tendons into the peroneal groove with repair of the SPR to the fibula. In general, repair of the SPR with or without groove deepening has high patient satisfaction [71] and low rates of recurrent instability [90]. Supplemental surgical deepening of the retromalleolar groove for added tendon stability is somewhat controversial, with recent literature recommending that acute injuries in athletes and cases of chronic instability be treated with groove deepening, due to higher rate of return to sport [80]. This reflects the authors approach in which the groove is deepened at the posterior fibula via a three-limb osteotomy that allows the cortical surface of the groove to be reflected on a posteromedial cortical hinge. With the cortex reflected, intramedullary bone is removed with a bur, allowing for replacement of the reflected cortical flap into a recessed position that creates a deepened groove (Fig. 6). The

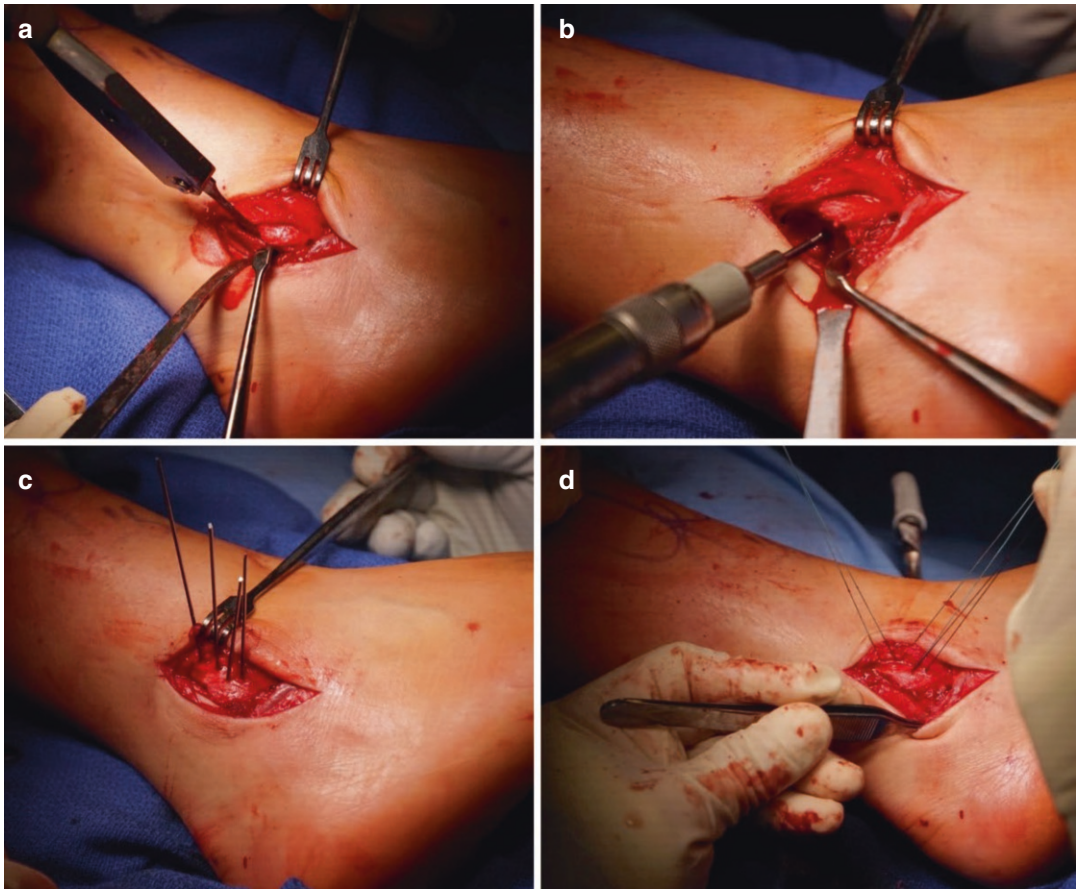

Fig. 6 Technique for fibular groove deepening. (**a**) An osteotome is used to create a cortical window into the medullary cavity of the distal fibula. (**b**) With the cortical window reflected inferiorly, a bur is used to remove intramedullary bone and create a groove. (**c**) With the cortical window replaced and tamped into a depressed position to create a groove, bone tunnels are created with k-wires. (**d**) Sutures are passed through the bone tunnel, into the superior peroneal retinaculum, and then back through the bone tunnel, allowing repair of the SPR to the newly created groove

authors' preferred technique is to then repair the SPR to its anatomic attachment using bone tunnels in the lateral fibula and nonabsorbable suture. In addition, concomitant peroneal tendon tears are addressed as described above. If a peroneal tendon tear is present, the above postoperative protocol is followed. If a groove deepening is performed and a tendon repair or reconstruction is not needed, the patient is splinted in neutral rather than plantarflexion. Similarly, a night splint or brace that protects against passive inversion or plantarflexion during sleep is important for 3 months. After 2 weeks, the patient can be progressed to weightbearing in a boot brace in neutral dorsiflexion. During 3–6 months postoperatively, a cloth brace can be used as the patient transitions from the boot brace.

5 Conclusion

In summary, the spectrum of tendon pathologies affecting ankle function is broad. While each tendon has unique characteristics, there are many common principles in tendon pathophysiology, clinical evaluation, and treatment. Tendinopathy can occur in all tendons, and often involves a change in tendon microstructure secondary to chronic stresses overwhelming the intrinsic repair pathways. While there is no documented evidence of cellular inflammation, inflammatory mediators are likely intimately involved in tendon remodeling and degeneration. In injuries of chronic tendon overload, it is crucial to consider the spectrum of intrinsic and extrinsic factors that are contributing to the patient's presentation. Modifiable factors should be addressed during both nonoperative and operative treatment of the injured tendon. In general, nonoperative care, consisting of a period of immobilization, icing, and anti-inflammatories followed by physical therapy, is appropriate for most tendinopathies. When these treatments fail, debridement and repair, reconstruction, or tendon transfers may be needed to help reduce pain and restore function. In the setting of traumatic injuries resulting in tendon rupture or tendon instability, acute surgical treatment is commonly indicated.

References

1. Thorpe CT, Screen HRC. Tendon structure and composition. Adv Exp Med Biol. 2016;920:3–10. https://doi.org/10.1007/978-3-319-33943-6_1.
2. Xu Y, Murrell GAC. The basic science of tendinopathy. Clin Orthop Relat Res. 2008;466(7):1528–38. https://doi.org/10.1007/s11999-008-0286-4.
3. Parkinson J, Samiric T, Ilic MZ, Cook J, Handley CJ. Involvement of proteoglycans in tendinopathy. J Musculoskelet Neuronal Interact. 2011;11(2):86–93.
4. Grant TM, Thompson MS, Urban J, Yu J. Elastic fibres are broadly distributed in tendon and highly localized around tenocytes. J Anat. 2013;222(6):573–9. https://doi.org/10.1111/joa.12048.
5. Milz S, Ockert B, Putz R. Tenocytes and the extracellular matrix: a reciprocal relationship. Orthopade. 2009;38(11):1071–9. https://doi.org/10.1007/s00132-009-1490-y.
6. Riley G. Tendinopathy–from basic science to treatment. Nat Clin Pract Rheumatol. 2008;4(2):82–9. https://doi.org/10.1038/ncprheum0700.
7. Lantto I, Heikkinen J, Flinkkila T, et al. Early functional treatment versus cast immobilization in tension after achilles rupture repair: results of a prospective randomized trial with 10 or more years of follow-up. Am J Sports Med. 2015;43(9):2302–9. https://doi.org/10.1177/0363546515591267.
8. Khan KM, Cook JL, Kannus P, Maffulli N, Bonar SF. Time to abandon the "tendinitis" myth. BMJ. 2002;324(7338):626–7. https://doi.org/10.1136/bmj.324.7338.626.
9. Millar NL, Murrell GAC, McInnes IB. Inflammatory mechanisms in tendinopathy - towards translation. Nat Rev Rheumatol. 2017;13(2):110–22. https://doi.org/10.1038/nrrheum.2016.213.
10. Bass E. Tendinopathy: why the difference between tendinitis and tendinosis matters. Int J Ther Massage Bodywork. 2012;5(1):14–7. https://doi.org/10.3822/ijtmb.v5i1.153.
11. Reb CW, Stenson JF, Daniel JN. Tibialis anterior tendon reconstruction using augmented half-thickness tendon segment transposition. Foot Ankle Spec. 2017;10(2):144–8. https://doi.org/10.1177/1938640016685825.
12. Calloway SP, Hardin DM, Crawford MD, et al. Injury surveillance in major league soccer: a 4-year comparison of injury on natural grass versus artificial turf field. Am J Sports Med. 2019;47(10):2279–86. https://doi.org/10.1177/0363546519860522.
13. Rowson S, McNally C, Duma SM. Can footwear affect achilles tendon loading? Clin J Sport Med. 2010;20(5):344–9. https://doi.org/10.1097/JSM.0b013e3181ed7e50.
14. Griffin JW. Differences in elbow flexion torque measured concentrically, eccentrically, and isometrically. Phys Ther. 1987;67(8):1205–8. https://doi.org/10.1093/ptj/67.8.1205.

15. Herzenberg JE, Lamm BM, Corwin C, Sekel J. Isolated recession of the gastrocnemius muscle: the Baumann procedure. Foot Ankle Int. 2007;28(11):1154–9. https://doi.org/10.3113/FAI.2007.1154.
16. Ellington JK, McCormick J, Marion C, et al. Surgical outcome following tibialis anterior tendon repair. Foot Ankle Int. 2010;31(5):412–7. https://doi.org/10.3113/FAI.2010.0412.
17. Petersen W, Stein V, Bobka T. Structure of the human tibialis anterior tendon. J Anat. 2000;197(4):617–25. https://doi.org/10.1046/j.1469-7580.2000.19740617.x.
18. Sammarco VJ, Sammarco GJ, Henning C, Chaim S. Surgical repair of acute and chronic tibialis anterior tendon ruptures. J Bone Joint Surg Am. 2009;91(2):325–32. https://doi.org/10.2106/JBJS.G.01386.
19. Lee MH, Chung CB, Cho JH, et al. Tibialis anterior tendon and extensor retinaculum: imaging in cadavers and patients with tendon tear. AJR Am J Roentgenol. 2006;187(2):161–8. https://doi.org/10.2214/AJR.05.0073.
20. Chen J, Kadakia R, Akoh CC, Schweitzer KM. Management of anterior tibialis tendon ruptures. J Am Acad Orthop Surg. 2021;29(16):691–701. https://doi.org/10.5435/JAAOS-D-20-00802.
21. Tickner A, Thorng S, Martin M, Marmolejo V. Management of isolated anterior tibial tendon rupture: a systematic review and meta-analysis. J Foot Ankle Surg. 2019;58(2):213–20. https://doi.org/10.1053/j.jfas.2018.08.001.
22. Ebrahimi FV, Tofighi M, Khatibi H. Closed tibial fracture associated with laceration of tibialis anterior tendon. J Foot Ankle Surg. 2010;49(1):86. https://doi.org/10.1053/j.jfas.2009.08.013.
23. Johansen JK, Jordan M, Thomas M, Walther M, Simatira AM, Waizy H. Surgical treatment of tibialis anterior tendon rupture. Foot Ankle Surg. 2021;27(5):515–20. https://doi.org/10.1016/j.fas.2020.06.011.
24. Fraissler L, Horas K, Bölch S, Raab P, Rudert M, Walcher M. Extensor hallucis longus-transfer for tibialis anterior tendon rupture repair. Oper Orthop Traumatol. 2019;31(2):143–8. https://doi.org/10.1007/s00064-018-0571-7.
25. Goetz J, Beckmann J, Koeck F, Grifka J, Dullien S, Heers G. Gait analysis after tibialis anterior tendon rupture repair using Z-plasty. J Foot Ankle Surg. 2013;52(5):598–601. https://doi.org/10.1053/j.jfas.2013.01.007.
26. Kopriva JM, Cheesborough J, Frick SL. Tendon turndown to bridge a tibialis anterior gap and restore active dorsiflexion after degloving foot injury in a child: a case report. JBJS Case Connect. 2020;10(3):e19.00445. https://doi.org/10.2106/JBJS.CC.19.00445.
27. Karnovsky SC, Desandis B, Papson AK, et al. Tibialis anterior reconstruction with hamstring autograft using a minimally invasive approach. Foot Ankle Int. 2018;39(5):535–41. https://doi.org/10.1177/1071100717750883.
28. Wu F, Nerlich M, Docheva D. Tendon injuries: basic science and new repair proposals. EFORT Open Rev. 2017;2(7):332–42. https://doi.org/10.1302/2058-5241.2.160075.
29. Järvinen TAH, Kannus P, Maffulli N, Khan KM. Achilles tendon disorders: etiology and epidemiology. Foot Ankle Clin. 2005;10(2):255–66. https://doi.org/10.1016/j.fcl.2005.01.013.
30. Singh A, Calafi A, Diefenbach C, Kreulen C, Giza E. Noninsertional tendinopathy of the achilles. Foot Ankle Clin. 2017;22(4):745–60. https://doi.org/10.1016/j.fcl.2017.07.006.
31. Whitting JW, Steele JR, McGhee DE, Munro BJ. Dorsiflexion capacity affects achilles tendon loading during drop landings. Med Sci Sports Exerc. 2011;43(4):706–13. https://doi.org/10.1249/MSS.0b013e3181f474dd.
32. Chimenti RL, Cychosz CC, Hall MM, Phisitkul P. Current concepts review update: insertional achilles tendinopathy. Foot Ankle Int. 2017;38(10):1160–9. https://doi.org/10.1177/1071100717723127.
33. Nicholson CW, Berlet GC, Lee TH. Prediction of the success of nonoperative treatment of insertional Achilles tendinosis based on MRI. Foot Ankle Int. 2007;28(4):472–7. https://doi.org/10.3113/FAI.2007.0472.
34. Kim DH, Choi JH, Park CH, Park HJ, Yoon KJ, Lee YT. The diagnostic significance of ultrasonographic measurement of the achilles tendon thickness for the insertional achilles tendinopathy in patients with heel pain. J Clin Med. 2021;10(10):2165. https://doi.org/10.3390/jcm10102165.
35. Jarin IJ, Bäcker HC, Vosseller JT. Functional outcomes of insertional achilles tendinopathy treatment: a systematic review. JBJS Rev. 2021;9:6. https://doi.org/10.2106/JBJS.RVW.20.00110.
36. Mansur NSB, Matsunaga FT, Carrazzone OL, et al. Shockwave therapy plus eccentric exercises versus isolated eccentric exercises for achilles insertional tendinopathy: a double-blinded randomized clinical trial. J Bone Joint Surg Am. 2021;103(14):1295–302. https://doi.org/10.2106/JBJS.20.01826.
37. Pinitkwamdee S, Laohajaroensombat S, Orapin J, Woratanarat P. Effectiveness of extracorporeal shockwave therapy in the treatment of chronic insertional achilles tendinopathy. Foot Ankle Int. 2020;41(4):403–10. https://doi.org/10.1177/1071100719898461.
38. Thueakthong W, de Cesar NC, Garnjanagoonchorn A, et al. Outcomes of iliac crest bone marrow aspirate injection for the treatment of recalcitrant Achilles tendinopathy. Int Orthop. 2021;45(9):2423–8. https://doi.org/10.1007/s00264-021-05112-3.
39. Csizy M, Hintermann B. Rupture of the Achilles tendon after local steroid injection. Case reports and consequences for treatment. Swiss Surg. 2001;7(4):184–9. https://doi.org/10.1024/1023-9332.7.4.184.

40. Yontar NS, Aslan L, Can A, Öğüt T. Mid-term results of open debridement and reattachment surgery for insertional Achilles tendinopathy: a retrospective clinical study. Acta Orthop Traumatol Turc. 2020;54(6):567–71. https://doi.org/10.5152/j.aott.2020.18426.
41. de Cesar NC, Chinanuvathana A, da Fonseca LF, Dein EJ, Tan EW, Schon LC. Outcomes of flexor digitorum longus (FDL) tendon transfer in the treatment of Achilles tendon disorders. Foot Ankle Surg. 2019;25(3):303–9. https://doi.org/10.1016/j.fas.2017.12.003.
42. Hunt KJ, Cohen BE, Davis WH, Anderson RB, Jones CP. Surgical treatment of insertional achilles tendinopathy with or without flexor hallucis longus tendon transfer: a prospective, randomized study. Foot Ankle Int. 2015;36(9):998–1005. https://doi.org/10.1177/1071100715586182.
43. Nawoczenski DA, DiLiberto FE, Cantor MS, Tome JM, DiGiovanni BF. Ankle power and endurance outcomes following isolated gastrocnemius recession for achilles tendinopathy. Foot Ankle Int. 2016;37(7):766–75. https://doi.org/10.1177/1071100716638128.
44. Li HY, Hua YH. Achilles tendinopathy: current concepts about the basic science and clinical treatments. Biomed Res Int. 2016;2016:6492597. https://doi.org/10.1155/2016/6492597.
45. Chen TM, Rozen WM, Pan WR, Ashton MW, Richardson MD, Taylor GI. The arterial anatomy of the Achilles tendon: anatomical study and clinical implications. Clin Anat. 2009;22(3):377–85. https://doi.org/10.1002/ca.20758.
46. Knobloch K, Kraemer R, Lichtenberg A, et al. Achilles tendon and paratendon microcirculation in midportion and insertional tendinopathy in athletes. Am J Sports Med. 2006;34(1):92–7. https://doi.org/10.1177/0363546505278705.
47. Tillander B, Gauffin H, Lyth J, Knutsson A, Timpka T. Symptomatic achilles tendons are thicker than asymptomatic tendons on ultrasound examination in recreational long-distance runners. Sports. 2019;7(12):E245. https://doi.org/10.3390/sports7120245.
48. Jarin I, Bäcker HC, Vosseller JT. Meta-analysis of noninsertional achilles tendinopathy. Foot Ankle Int. 2020;41(6):744–54. https://doi.org/10.1177/1071100720914605.
49. Rompe JD, Furia J, Maffulli N. Eccentric loading versus eccentric loading plus shock-wave treatment for midportion achilles tendinopathy: a randomized controlled trial. Am J Sports Med. 2009;37(3):463–70. https://doi.org/10.1177/0363546508326983.
50. Krogh TP, Ellingsen T, Christensen R, Jensen P, Fredberg U. Ultrasound-guided injection therapy of achilles tendinopathy with platelet-rich plasma or saline: a randomized, blinded, placebo-controlled trial. Am J Sports Med. 2016;44(8):1990–7. https://doi.org/10.1177/0363546516647958.
51. Molund M, Lapinskas SR, Nilsen FA, Hvaal KH. Clinical and functional outcomes of gastrocnemius recession for chronic achilles tendinopathy. Foot Ankle Int. 2016;37(10):1091–7. https://doi.org/10.1177/1071100716667445.
52. Drakos MC, Gott M, Karnovsky SC, et al. Biomechanical analysis of suture anchor vs tenodesis screw for FHL transfer. Foot Ankle Int. 2017;38(7):797–801. https://doi.org/10.1177/1071100717702848.
53. Erickson BJ, Mascarenhas R, Saltzman BM, et al. Is Operative treatment of achilles tendon ruptures superior to nonoperative treatment? A systematic review of overlapping meta-analyses. Orthop J Sports Med. 2015;3(4):2325967115579188. https://doi.org/10.1177/2325967115579188.
54. Kou J. AAOS Clinical practice guideline: acute achilles tendon rupture. J Am Acad Orthop Surg. 2010;18(8):511–3. https://doi.org/10.5435/00124635-201008000-00008.
55. Dams OC, Reininga IHF, Gielen JL, van den Akker-Scheek I, Zwerver J. Imaging modalities in the diagnosis and monitoring of Achilles tendon ruptures: a systematic review. Injury. 2017;48(11):2383–99. https://doi.org/10.1016/j.injury.2017.09.013.
56. Soroceanu A, Sidhwa F, Aarabi S, Kaufman A, Glazebrook M. Surgical versus nonsurgical treatment of acute Achilles tendon rupture: a meta-analysis of randomized trials. J Bone Joint Surg Am. 2012;94(23):2136–43. https://doi.org/10.2106/JBJS.K.00917.
57. Myhrvold SB, Brouwer EF, Andresen TKM, et al. Nonoperative or surgical treatment of acute achilles' tendon rupture. N Engl J Med. 2022;386(15):1409–20. https://doi.org/10.1056/NEJMoa2108447.
58. Lantto I, Heikkinen J, Flinkkila T, et al. A prospective randomized trial comparing surgical and nonsurgical treatments of acute achilles tendon ruptures. Am J Sports Med. 2016;44(9):2406–14. https://doi.org/10.1177/0363546516651060.
59. Renninger CH, Kuhn K, Fellars T, Youngblood S, Bellamy J. Operative and nonoperative management of achilles tendon ruptures in active duty military population. Foot Ankle Int. 2016;37(3):269–73. https://doi.org/10.1177/1071100715615322.
60. Heikkinen J, Lantto I, Flinkkila T, et al. Soleus atrophy is common after the nonsurgical treatment of acute achilles tendon ruptures: a randomized clinical trial comparing surgical and nonsurgical functional treatments. Am J Sports Med. 2017;45(6):1395–404. https://doi.org/10.1177/0363546517694610.
61. Zhang YJ, Long X, Du JY, Wang Q, Lin XJ. Is early controlled motion and weightbearing recommended for nonoperatively treated acute achilles tendon rupture? A systematic review and meta-analysis. Orthop J Sports Med. 2021;9(9):23259671211024604. https://doi.org/10.1177/23259671211024605.
62. Glazebrook M, Rubinger D. Functional rehabilitation for nonsurgical treatment of acute achilles tendon rupture. Foot Ankle Clin. 2019;24(3):387–98. https://doi.org/10.1016/j.fcl.2019.05.001.
63. Park YH, Jeong SM, Choi GW, Kim HJ. How early must an acute Achilles tendon rupture be repaired?

Injury. 2017;48(3):776–80. https://doi.org/10.1016/j.injury.2017.01.020.
64. Goren D, Ayalon M, Nyska M. Isokinetic strength and endurance after percutaneous and open surgical repair of Achilles tendon ruptures. Foot Ankle Int. 2005;26(4):286–90. https://doi.org/10.1177/107110070502600404.
65. Grassi A, Amendola A, Samuelsson K, et al. Minimally invasive versus open repair for acute achilles tendon rupture: meta-analysis showing reduced complications, with similar outcomes, after minimally invasive surgery. J Bone Joint Surg Am. 2018;100(22):1969–81. https://doi.org/10.2106/JBJS.17.01364.
66. Rozis M, Benetos IS, Karampinas P, Polyzois V, Vlamis J, Pneumaticos SG. Outcome of percutaneous fixation of acute achilles tendon ruptures. Foot Ankle Int. 2018;39(6):689–93. https://doi.org/10.1177/1071100718757971.
67. Stavenuiter XJR, Lubberts B, Prince RM, Johnson AH, DiGiovanni CW, Guss D. Postoperative complications following repair of acute achilles tendon rupture. Foot Ankle Int. 2019;40(6):679–86. https://doi.org/10.1177/1071100719831371.
68. Schweitzer KM, Dekker TJ, Adams SB. Chronic achilles ruptures: reconstructive options. J Am Acad Orthop Surg. 2018;26(21):753–63. https://doi.org/10.5435/JAAOS-D-17-00158.
69. Mirmiran R, Squire C, Wassell D. Prevalence and role of a low-lying peroneus brevis muscle belly in patients with peroneal tendon pathologic features: a potential source of tendon subluxation. J Foot Ankle Surg. 2015;54(5):872–5. https://doi.org/10.1053/j.jfas.2015.02.012.
70. Sobel M, Geppert MJ, Olson EJ, Bohne WH, Arnoczky SP. The dynamics of peroneus brevis tendon splits: a proposed mechanism, technique of diagnosis, and classification of injury. Foot Ankle. 1992;13(7):413–22. https://doi.org/10.1177/107110079201300710.
71. van Dijk PAD, Kerkhoffs GMMJ, Chiodo C, DiGiovanni CW. Chronic disorders of the peroneal tendons: current concepts review of the literature. J Am Acad Orthop Surg. 2019;27(16):590–8. https://doi.org/10.5435/JAAOS-D-18-00623.
72. Bruce WD, Christofersen MR, Phillips DL. Stenosing tenosynovitis and impingement of the peroneal tendons associated with hypertrophy of the peroneal tubercle. Foot Ankle Int. 1999;20(7):464–7. https://doi.org/10.1177/107110079902000713.
73. Petersen W, Bobka T, Stein V, Tillmann B. Blood supply of the peroneal tendons: injection and immunohistochemical studies of cadaver tendons. Acta Orthop Scand. 2000;71(2):168–74. https://doi.org/10.1080/000164700317413148.
74. van Dijk PAD, Madirolas FX, Carrera A, Kerkhoffs GMMJ, Reina F. Peroneal tendons well vascularized: results from a cadaveric study. Knee Surg Sports Traumatol Arthrosc. 2016;24(4):1140–7. https://doi.org/10.1007/s00167-015-3946-4.
75. Raikin SM, Elias I, Nazarian LN. Intrasheath subluxation of the peroneal tendons. J Bone Joint Surg Am. 2008;90(5):992–9. https://doi.org/10.2106/JBJS.G.00801.
76. Mengiardi B, Pfirrmann CWA, Schöttle PB, et al. Magic angle effect in MR imaging of ankle tendons: influence of foot positioning on prevalence and site in asymptomatic subjects and cadaveric tendons. Eur Radiol. 2006;16(10):2197–206. https://doi.org/10.1007/s00330-006-0164-y.
77. Latt LD, Kuyumcu G, Taljanovic MS. High-resolution ultrasound and MRI imaging of peroneal tendon injuries. In: Sobel M, editor. The peroneal tendons. Cham: Springer; 2020. p. 97–123. https://doi.org/10.1007/978-3-030-46646-6_5.
78. Unlu MC, Kivrak A, Kayaalp ME, Birsel O, Akgun I. Peritendinous injection of platelet-rich plasma to treat tendinopathy: a retrospective review. Acta Orthop Traumatol Turc. 2017;51(6):482–7. https://doi.org/10.1016/j.aott.2017.10.003.
79. Fram BR, Rogero R, Fuchs D, Shakked RJ, Raikin SM, Pedowitz DI. Clinical Outcomes and complications of peroneal tendon sheath ultrasound-guided corticosteroid injection. Foot Ankle Int. 2019;40(8):888–94. https://doi.org/10.1177/1071100719847629.
80. van Dijk PA, Miller D, Calder J, et al. The ESSKA-AFAS international consensus statement on peroneal tendon pathologies. Knee Surg Sports Traumatol Arthrosc. 2018;26(10):3096–107. https://doi.org/10.1007/s00167-018-4971-x.
81. Danna NR, Brodsky JW. Diagnosis and operative treatment of peroneal tendon tears. Foot Ankle Orthopaedics. 2020;5(2):247301142091040. https://doi.org/10.1177/2473011420910407.
82. Pellegrini MJ, Glisson RR, Matsumoto T, et al. Effectiveness of allograft reconstruction vs tenodesis for irreparable peroneus brevis tears: a cadaveric model. Foot Ankle Int. 2016;37(8):803–8. https://doi.org/10.1177/1071100716658469.
83. Redfern D, Myerson M. The management of concomitant tears of the peroneus longus and brevis tendons. Foot Ankle Int. 2004;25(10):695–707. https://doi.org/10.1177/107110070402501002.
84. Seybold JD, Campbell JT, Jeng CL, Short KW, Myerson MS. Outcome of lateral transfer of the FHL or FDL for concomitant peroneal tendon tears. Foot Ankle Int. 2016;37(6):576–81. https://doi.org/10.1177/1071100716634762.
85. Steel MW, DeOrio JK. Peroneal tendon tears: return to sports after operative treatment. Foot Ankle Int. 2007;28(1):49–54. https://doi.org/10.3113/FAI.2007.0009.
86. Demetracopoulos CA, Vineyard JC, Kiesau CD, Nunley JA. Long-term results of debridement and primary repair of peroneal tendon tears. Foot Ankle Int. 2014;35(3):252–7. https://doi.org/10.1177/1071100713514565.
87. Geppert MJ, Sobel M, Bohne WH. Lateral ankle instability as a cause of superior peroneal retinacu-

lar laxity: an anatomic and biomechanical study of cadaveric feet. Foot Ankle. 1993;14(6):330–4. https://doi.org/10.1177/107110079301400604.
88. Oden RR. Tendon injuries about the ankle resulting from skiing. Clin Orthop Relat Res. 1987;216:63–9.
89. Bakker D, Schulte JB, Meuffels DE, Piscaer TM. Non-operative treatment of peroneal tendon dislocations: a systematic review. J Orthop. 2020;18:255–60. https://doi.org/10.1016/j.jor.2019.08.031.
90. van Dijk PAD, Gianakos AL, Kerkhoffs GMMJ, Kennedy JG. Return to sports and clinical outcomes in patients treated for peroneal tendon dislocation: a systematic review. Knee Surg Sports Traumatol Arthrosc. 2016;24(4):1155–64. https://doi.org/10.1007/s00167-015-3833-z.
91. Schafer KA, Adams SB, McCormick JJ. Peroneal tendonitis and tendonopathy. I: Sobel M, ed. The Peroneal Tendons: A Clinical Guide to Evaluation and Management. Springer International Publishing. 2020:183–191. https://doi.org/10.1007/978-3-030-46646-6_9.