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# Treatment of tendinitis: From basic science to clinical application

Schweizerische Zeitschrift für «Sportmedizin und Sporttraumatologie» 52 (1), 36–41, 2004

The importance of sports and exercise in western society has increased substantially in the past 20 years. Exercise now has a role in the management of hypertension, lipid disorders, peripheral vascular disease, diabetes mellitus, coronary artery disease, and host of other conditions [1]. Leisure time is also more than ever spent in the pursuit of athletic activity [2], and professional sports receive more attention in the media and require more intense training and commitment than previously. However, a commitment to exercise as part of a healthy lifestyle increases the risk of overuse injuries. Tendon overuse injuries are common in many sports and activities [3–7].

Anatomically, tendons connect muscles to bone, and function to transfer the force of the muscle contraction to the bone to create movement at joints. The structure of tendons allows them to with-

stand great tensile forces. These forces are present for both daily activities as well as sports. At the end of the normal stance phase of the walking cycle, muscle tension through the Achilles tendon is estimated at 250% of body weight [8]. In repetitive hopping in place, a force of about 4000 N has been measured in the Achilles tendon [9].

Tendons vary in shape and size, but their structure consists of closely packed collagen fibers parallel to one another and surrounded by an epitendon. These fibers form fibrils and several parallel fibrils embedded in the extracellular matrix are known as fibers. Fibers in turn group together to form fascicles, which are surrounded by an endotendon, which is an areolar connective tissue sheath, and these are enclosed in an epitendon [10] (*Fig. 1*). When patients undergo surgery for failure to respond to the usual

<i>Pathological Diagnosis</i>	<i>Concept (Macroscopic pathology)</i>	<i>Histologic Finding</i>
Tendinosis	Intratendinous degeneration (commonly due to aging, microtrauma, vascular compromise)	Collagen disorientation, disorganization and fiber separation by and increase in mucoid ground substance, increased prominence of cells and vascular spaces with or without neovascularization and focal necrosis or calcification
Tendinitis/Partial rupture	Symptomatic degeneration of the tendon with vascular disruption and inflammatory repair response	Degenerative changes as noted above with superimposed evidence of tear, including fibroblastic and myofibroblastic proliferation, hemorrhage and organizing granulation tissue
Paratenonitis	«Inflammation» of the outer layer of the tendon (paratenon) alone, whether or not the paratenon is lined by synovium	Mucoid degeneration in the areolar tissue is seen. A scattered mild mononuclear infiltrate with or without focal fibrin deposition and fibrinous exudates
Paratenonitis with tendinosis	Paratenonitis associated with intratendinous degeneration	Degenerative changes as noted in tendinosis with mucoid degeneration with or without fibrosis and scattered inflammatory cells in the paratenon alveolar tissue

[Sports Med. 27: 393–408, 1999. Kahn K.M., Cook J.L., Bonar F. et al.: Histopathology of Common Overuse Tendon conditions: Update and Implication for Clinical Management]

Table 1: Classification of Tendinopathies.

interventions, light microscopy findings demonstrate loss of the normal tendon structure, which is replaced by disorganized collagen fibers and necrotic tissue [11].

The events which take place when a tendon becomes painful are not known. Puddu et al. [12] initially noted that biopsy specimens taken at surgery rarely demonstrate inflammatory cells. Many authors [13–16] have reinforced this observation since the initial report. These pathologic observations have led to the belief that the term «tendonitis» does not adequately describe the process or pathology in painful tendons and should not be used. This has led several authors to classify tendon disorders by histopathologic criteria [17, 18] believing that lack of consistent nomenclature has limited progress in understanding the pathologic basis of tendon conditions. This school of thought holds that the loss of normal tendon architecture results not in inflammation, but in damage to both matrix and tenocyte and a lesion termed «tendinosis.» The difficulty with this model is the presence of tendinosis lesions in asymptomatic tendons. In a pathologic study of 1966 tendons, Jozsa et al. [19] noted that only 66% of control tendons were completely normal. Biopsy specimens from tendon surgeries demonstrated that 8% appeared completely healthy. Thus, the presence of tendinosis does not necessarily imply a painful injury. The paratenon can also be injured. The paratenon is a gliding layer of 6–8 thin membranes next to the epitenon. These membranes are rich in mucopolysaccharides and function as a lubricant [20]. Kvist et al. [21] performed histologic and histochemical analysis on Achilles tendons of 16 athletes with paratenonitis and compared them to 3 control tendons. They found a slight inflammatory cell reaction in all cases with fibrinous exudations, widespread fat necrosis and connective tissue proliferation and adhesion formation. They interpreted these findings as evidence of decreased gliding function and decreased oxygenation of the injured areas.

Because so few cases of tendon injury result in surgery, researchers have resorted to alternative methods of studying tendon injury. Animal models, initially using bacterial collagenase were studied in horses [22] and demonstrated histologic changes similar to tendinosis, but the model was criticized because the injury did not result from an exercise stimulus. A second model [23] utilizing rabbits and species specific cytokines as the inflammatory stimulus, demonstrated more rapid histologic recovery, but also did not utilize exercise as a stimulus. In 1990 Backman et al. [24] created a model of tendon overuse injury in rabbits utilizing a «kicking machine» which electrically stimulated the gastrocnemius while passively dorsiflexing the ankle. The model produced an eccentric contraction of the muscle tendon unit, and histology of the kicking leg performed after 4 weeks of training demonstrated irregular thickening over the Achilles tendon with degenerative changes in the central portion of the tendon and thickening of the paratenon. An increased number of fibroblasts, lymphocytes, plasma cells and granulocytes were also encountered. Biopsy specimens were obtained the day after the last exercise, which may be why inflammatory cells were encountered. In 1992, Kraus-Hansen et al. [25] utilized ligatures in the vasculature of the equine superficial digital flexor tendon to isolate the blood supply of the tendon. At 35 days postoperatively, a marked paratendinous response involving the entire segment of devascularized tendon accompanied by changes in the tendon core. Histology demonstrated focal degeneration. The authors demonstrated that the lesions were similar to naturally occurring «tendonitis» and felt their work supported a vascular etiology of tendinosis. Soslowky et al. [26] developed an exercise model utilizing rats running for up to an hour a day 4–16 weeks on a treadmill. Supraspinatus tendons demonstrated loss of normal collagen fiber organization, with thickening and decreased biomechanical properties.

An alternative to animal models has been tissue culture studies. These studies have looked at alterations in tendon cell response to cyclic stretching, and found alterations in cell response. Arnoczky et al. [27] demonstrated that cyclic strain in canine tendon fibroblasts activates c-Jun N-terminal kinase (JNK), one of a group of stress activated protein kinases. Persistent activation of JNK has been linked to the initiation of apoptosis, a highly regulated form

of cell death. Yuan et al. [28] demonstrated evidence of increased apoptosis in torn rotator cuff tendons as compared to asymptomatic controls. Maffulli et al. [29] performed tissue culture on tenocytes from ruptured Achilles tendons, tendons operated on for symptomatic tendinosis, and asymptomatic tenocytes from patients undergoing amputation for peripheral vascular disease. When cells grown in culture were scraped with a pipette, increased type III collagen was noted in tenocytes from ruptured tendons and tendinopathy tenocytes than the normal controls. Type III collagen is usually produced early in the healing phase of tendons and later replaced by type I collagen as the wound heals. The authors postulated that this may make the previously injured tendon more susceptible to rupture. Murrell [30] has postulated that tendons degenerate and tear from a process of increased activity leading to activation of stress protein kinases, which if persistently elevated results in apoptosis, and alterations in the matrix, which is now more predisposed to tearing.

Clinically, the diagnosis of tendinopathy is made by careful palpation of the tendon in question. The presence of thickening or nodularity should also be noted. In certain joints, accompanying conditions such as posterior impingement of the ankle, retrocalcaneal bursitis, patellafemoral pain syndrome at the knee, impingement syndrome of the shoulder, and radial tunnel syndrome of the elbow need to be ruled out. Williams et al. [31] described the painful arc sign for Achilles tendinopathy, in which an area of swelling moves with ankle dorsiflexion and plantarflexion. Maffulli et al. [32] studied the Royal London Hospital Test (*Fig. 1*), in which a painful lesion of the Achilles tendon will be less painful as the lesion moves away from the examiner's fingers as the tendon moves from dorsiflexion to plantarflexion. The arc sign produced more false negative results than the Royal London Hospital Test in Maffulli's study. The outcome of most case series of tendon injuries demonstrates a high response to non-surgical treatment. Ferretti et al. [13] operated on only 18 of 150 patients (12%) with patellar tendonitis. Nirschl et al. [33] performed surgery on only 88 of 1213 tendons (7.2%) in his series of lateral epicondylitis. However, Martens et al. [6] performed surgery on 33% of their patients with patella tendonitis, and Johnston et al. [34] reported only 51% of their patients responding to non-surgical interventions for Achilles tendinitis. In some studies [35–40] surgical cases were selected for discussion without any mention of the number of cases seen and treated without surgery. This emphasis on case series without controls has led Labelle et al. [41] to criticize the scientific literature for lack of scientific evidence of present treatments of lateral epicondylitis. Labelle et al. looked at ultrasound, ionization, steroid injections, oral non steroidal anti-inflammatory medications and stated that there was «not enough scientific evidence to favor any particular type of treatment for acute lateral epicondylitis.» While case series studies implied that some type of

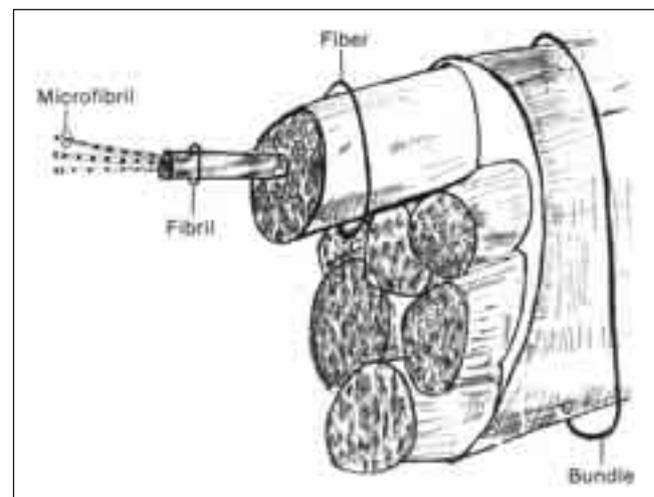


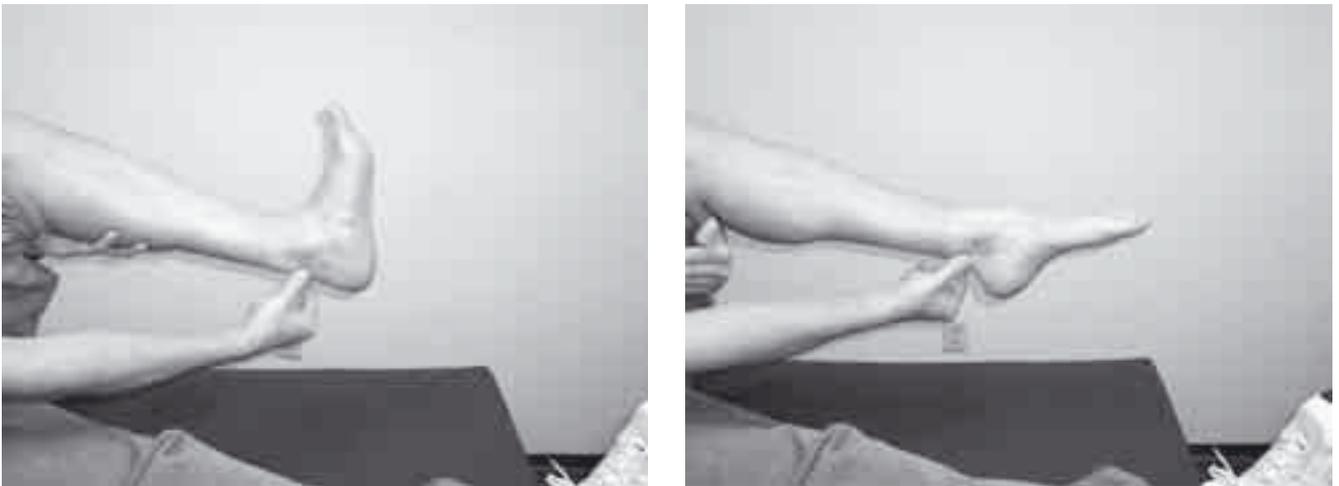
Figure 1: Schematic of tendon anatomy.

rehabilitation program was an important first step in the management of all types of tendon overuse injuries, regardless of the pathology involved, the details of the rehabilitation program are still somewhat controversial. Curwin and Stanish [42] had initially stressed the importance of eccentric strengthening in the management of tendonitis, but Alfredson et al. [43] demonstrated the importance of aggressive heavy load eccentric strengthening in recalcitrant Achilles tendonitis when compared to «ordinary» physical therapy programs. Recent updates on the success of this type of rehabilitation have demonstrated highly successful outcomes, although some patients still require surgery [44]. Smidt et al. [45] compared relative rest, corticosteroid injections given as one ml Triamcinolone, and one ml of lidocaine 1% given in all tender spots until pain free and physiotherapy consisting of nine treatments of pulsed ultrasound, deep friction massage, and an exercise program consisting of progressive, slow, wrist and forearm stretching and muscle conditioning and occupational exercises, intensified over four steps. Short term benefits were noted for the corticosteroid injection group, but long term benefits were noted for the physiotherapy program although the gains were small in comparison to relative rest.

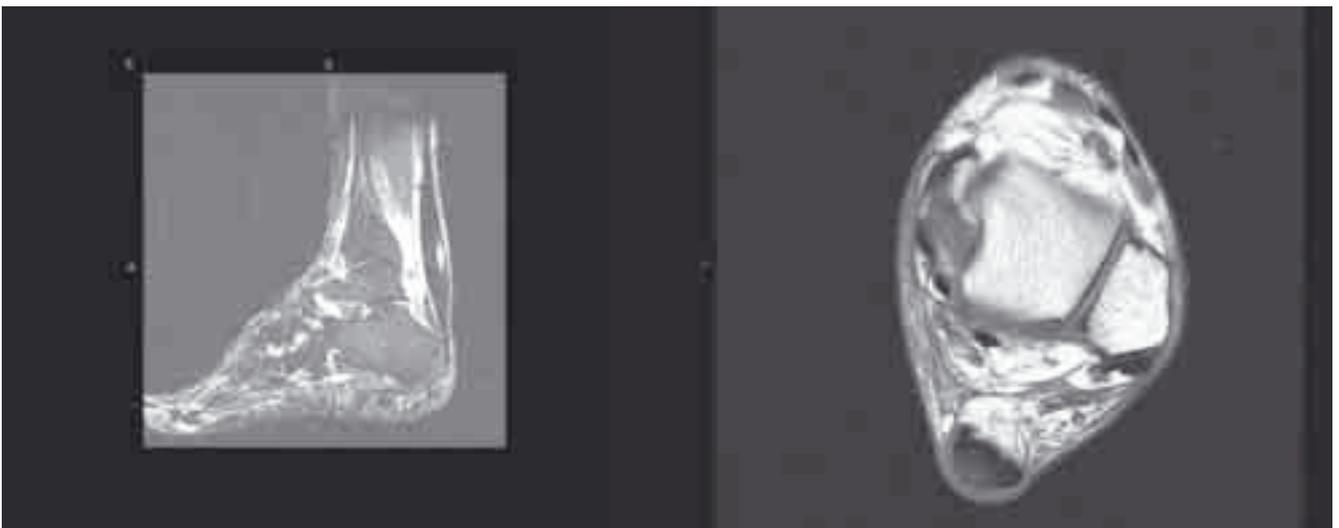
Risk factors for the development of tendon overuse injuries have been looked at by several authors. Milgrom et al. [46] prospectively studied military recruits in the Israeli infantry, evaluating shoe wear, height, weight, body mass index (BMI), external rotation of the hip, tibial intercondylar distance, arch type, phys-

ical fitness parameters, and training season. He found only cold weather training to be a risk factor for the development of Achilles paratendonitis. Witvrouw et al. [47] studied intrinsic risk factors for patella tendonitis over a 2 year period of time in undergraduate physical education students. They performed isokinetic strength evaluations of the quadriceps and hamstrings, Q-angle measurements, height, weight, leg length evaluations, and medial tibial intercondylar distance measurements. Only quadriceps and hamstring flexibility showed a significant association with patella tendonitis. 13.8% of subjects developed patella tendonitis during the study.

Imaging studies for tendon overuse injuries are controversial. Both ultrasound and MRI have been utilized to demonstrate tendinosis lesions, but all studies must be correlated with clinical findings [48]. Ultrasound is usually performed with high resolution transducers. Ultrasound has been criticized as too user dependant, often because of difficulty with image documentation [48]. For optimal imaging it is necessary to keep the probe in a position that sound waves reach the tendon perpendicularly (*Fig. 2*). False negative tests are avoided by performing dynamic studies. MRI offers high spatial and contrast resolution, and its multiplanar imaging capability allows any tendon to be imaged parallel and perpendicular to its course (*Fig. 3*). Artifact may appear on T1 weighted images if the course of the tendon forms an angle of 55 degrees with the static magnetic field. Referred to as «magic angle phenomena» it is a source of misdiagnosis on tendon degen-



*Figure 2:* Royal London test is designed to demonstrate a change in pain as the ankle moves from dorsiflexion to plantarflexion. This is consistent with a lesion of the tendon substance, rather than a paratenon lesion.



*Figure 3A:* Sagittal image of Achilles acute partial tear by MRI. Note fluid signal consistent within tendon. substance.

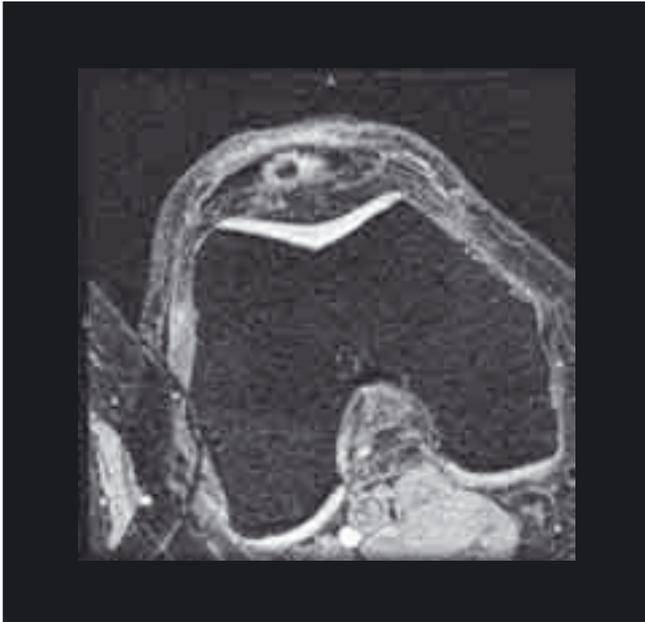


Figure 3B: Coronal image of patellar tendon. The grey signal within the patellar tendon consistent with a tendinopathy.

eration [48]. It remains an extremely expensive test. Fritschy and de Gautard [49] reported excellent outcomes for the management of patella tendonitis using ultrasound to evaluate the patella tendon for painful tendinosis lesions. However, Khan et al. [50] evaluated the appearance of patella tendons before and after surgery on MRI and ultrasound and found that postoperatively, the images remained abnormal in both short and long term follow-up. MRI images also remained abnormal regardless of the clinical outcome. Shalaby and Almekinders [51] performed a small study of 10 patients with patella tendonitis and compared them to a group of patients undergoing MRI for reasons other than patella tendonitis. Only 58% of the patients with tendonitis exhibited unequivocal intratendinous lesions. 25% had no defect present. In the control group 24% had unequivocal intratendinous lesions, and had no changes in the patella tendon. In an animal study of forelimb superficial digital flexor tendons in thoroughbred race horses, performed over a 4 month training program, tendons were found to increase in cross-sectional area, and decrease in echogenicity although the results were not statistically significant. The authors felt the changes were the result of a training effect, and not pathology [52]. Fredberg and Bolvig [53] performed an ultrasound study on Achilles and patellar tendons in Danish soccer players. Abnormalities were present in 29% of 54 players although all were asymptomatic. In players with Achilles tendon lesions, 36% remained asymptomatic, and the lesion resolved. In 45% the lesion became symptomatic during the season. In 18% the lesion persisted but did not become symptomatic. For the patella tendon, 33% of the ultrasounds normalized over the course of the season and remained symptomatic, 17% of the subjects became symptomatic, and 50% remained asymptomatic with persistence of the abnormality on ultrasound. For both groups, the presence of the lesion was a significant predictor of the development of symptoms, but the authors stated that the changes in the tendon were not an indication for surgery. These studies demonstrate that the presence of a lesion on MRI or ultrasound does not imply the need for surgery, and most importantly, emphasizes the need for non-surgical interventions before surgery.

The outcome of surgery in the management of tendinosis unresponsive to non-surgical treatments is generally good, although long term follow-up is lacking in most studies. It is not clear why surgery works, and theories such as denervation of the tendon, improved tissue repair, and improved local circulation have been offered as explanations [44]. Ferretti et al. [13] in an early study in which other knee procedures were combined with surgery on the

patella tendon, noted satisfactory results in 11 of 18 cases of patella tendonitis, with follow-up from 18–80 months. Maffulli et al. [54] looked at 28 patients retrospectively after patella tendon surgery, and found 23 patients pain free at follow-up, with two failures and three patients who returned at a slightly lower level of play. The average time to return to play was 7 months. Patients who did not improve underwent further paratenon stripping and longitudinal tenotomy. The authors felt that the paratenon may have only been partially stripped or had become involved in a new pathologic process. Panni et al. [55] compared surgical and non-surgical outcomes in a prospective study of 42 subjects. Clinical results were good or excellent in all 42 cases. 33 patients responded to rehabilitation, with 24 excellent and 9 good results, at an average of 4.6 months. In the surgical cases, 5 patients had excellent results, and 4 patients good results, however, they were not able to return to sports until 9–12 months after surgery. Kettunen et al. [56] looked at 15 year follow-up of patella tendonitis in 18 patients and compared them to 14 controls. He noted more subjective symptoms and functional limitations at follow-up in the patella tendonitis group at follow-up, but symptoms were mild. Nonetheless, 28% had reduced training for more than one year on follow-up examination. The subjects with a higher Insall-Salvati ratio had a higher visual analog scale, and lower Kujala (outcome) score. Coleman et al. [57] compared open and arthroscopic tenotomy in a retrospective study and found no difference in outcome. However, although both groups improved in 96% of cases, 54% of the patients in the open surgical procedure group and 46% of the patients in the arthroscopic procedure group were able to return to full athletic competition. Patients did not appear to get symptomatic improvement after they were 12 months post surgery, and the authors recommended further surgery at 12 months if symptoms remained disabling. However, when patients achieved good relief with surgery, they appeared to maintain those results over the length of the study, with few patients who got relief initially requiring a reoperation.

Results for Achilles tendon surgery have also been reported. Schepesis and Leach [58] performed a retrospective review of 45 surgical cases in a predominantly competitive long distance running population. They found that 92% of their patients were satisfied with the outcome when procedures were done on the Achilles tendon and tendon sheath. However, when procedures on the retrocalcaneal bursa were performed, success rates dropped to 71%. A longer follow-up of these and other patients demonstrated that 16% of the initial group that reported success required reoperation, whereas in patients followed for less than 5 years, only one of 34 patients required re-operation [59]. Patients who had procedures for paratenonitis had 87% satisfactory results, however, patients with tendinosis had only 67% satisfactory long term results. The authors felt that the long term prognosis for tendinosis of the Achilles tendon was not as good as for paratenonitis and retrocalcaneal bursitis. Nelen et al. [60] performed a study on 170 patients who had Achilles tendon surgery only involving the Achilles tendon segment 2–6 cm proximal to the insertion of the tendon. 143 of the cases were available for follow-up 2–7 years postoperatively. In 24 cases requiring substantial debridement, they reinforced the Achilles tendon with a turned down flap. In less severe cases they excised the diseased tendon. In results similar to Schepesis et al. [59] they had 87% good or excellent results with procedures on the paratenon, but only 73% good to excellent results on cases with tendinosis. However, the group with tendon flap reinforcement had 87% good to excellent results. Maffulli et al. [61] reported on a group of patients who had symptoms for 2 years before surgery and who had undergone surgery a minimum of 2 years before being studied. The group consisted of 14 patients, with lesions in the central body of the Achilles tendon. Only 5 of the 14 patients had a satisfactory outcome, and two of these patients underwent a second procedure. It was the authors' recommendation that surgery would be better performed sooner in these patients, and they postulated a failed healing response. Paavola et al. [62] reported on a prospective long term outcome of Achilles tendinosis. They performed surgery

on 29% of their patients, and followed them for 8 years. 84% returned to full activity, and 94% were asymptomatic or had only mild discomfort with strenuous exercise. Complications from Achilles tendon surgery are uncommon. Paavola et al. [63] looked at complications from 432 consecutive patients undergoing surgery for Achilles tendinopathy surgery, and found an 11% complication rate. The most common was skin edge necrosis, superficial wound infections, hematomas, and seroma formation. While Nelen et al. noted that complications were rare and did not affect the outcome of surgery, 14 of the patients in the Paavola et al. study required re-operation. Alfredson et al. [64] demonstrated that there was no difference between short and long term immobilization (2 vs. 6 weeks) in recovery of strength after Achilles tendon surgery, and subjects required a year to return isokinetic strength to equal the non-operated side. Alfredson et al. [65] also demonstrated that bone mass was equal remained decreased postoperatively 26 weeks after surgery, but generally recovered by one year. Tallon et al. [66] reviewed the literature on Achilles tendinopathy surgery, and found significant flaws in the methodology. They noted that the literature did not address whether surgery offered any benefit over forced rest, and which surgical procedure was best. Most importantly, there was a negative correlation between and a high rate of good surgical results and overall method scores.

Several alternatives to surgery have been mentioned in the literature. Steroid injections, once considered an important adjunct to a therapeutic exercise program are increasingly less relied upon. Shrier et al. [67] in a review of corticosteroid injections noted that there was insufficient published data to determine the comparative risks and benefits of corticosteroid injections. Johnston et al. [34] performed brisement on 9 patients who did not improve with a rehabilitation program. They injected 5 ml of 0.25% bupivacaine into the tendon/peritendon interface. Two of the patients had audible squeaking of the tendon with ambulation. Three of the nine patients had permanent resolution of symptoms. Extracorporeal shock wave therapy (ECSWT) has recently been shown to be effective in several types of tendinopathy. Wang and Chen [68] found ECST safe and effective for lateral epicondylitis. They treated 57 patients and performed 6 sham treatments. The sham patients did not improve, but 61.4% of patients were pain free and 29.5% were significantly improved. The length of the study is short, with only 1–2 year follow-up. Peers et al. [69] studied ECSWT and found comparable results to surgical intervention. The authors examined the six month and two year follow-up of patella tendinopathy with ECSWT and compared it retrospectively with surgical treatment.

In summary, tendon overuse injuries are common, and generally easily diagnosed. Much remains to be learned about them and many controversies exist as to their management. Rehabilitation remains the cornerstone of treatment, but the details of the rehabilitation program are still somewhat uncertain. Indications for surgery are still unclear, but generally performed when both patient, therapist (or athletic trainer) and physician are satisfied that an exercise program will not resolve the problem. Surgery is generally effective, but controversy still exists over the optimal surgical procedure, and the reasons why surgery is effective. Several alternatives to surgery exist, but most are not well studied, or there is only short term follow-up on their effectiveness.

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