Injury of the Achilles Tendon: Diagnosis with Sonography

We determined the diagnostic accuracy of sonography for the assessment of injury to the Achilles tendon. After anatomic investigations in three human cadavers, we performed a clinical study in 24 healthy volunteers and 73 symptomatic patients referred for achillodynia or signs of heel thickening or both in whom a clinical diagnosis of acute total rupture was excluded. High-resolution real-time sonography was performed and the results were compared with final clinical diagnoses (55 patients) and surgical findings (18 patients). Fifty-two of the patients had been involved in various sporting activities (long-distance runners, jumpers, and basketball players), three patients had familial hypercholesterolemia, five patients had systemic inflammatory disease, and 13 patients had no known underlying cause. Anatomic investigation demonstrated accurate assessment of tendon structure and thickness. Sonograms were abnormal in 53 patients (sensitivity, 0.72; specificity, 0.83), and the extent of structural disorders of the tendon could be assessed properly. Abnormalities occurred in the form of tendon swelling (45%), abnormal tendon structure (42%), rupture (15%), and peritendinous lesions (47%). No changes were detected in low-grade disease of short duration, which suggests symptoms caused by functional disorders.

Sonography is valuable in the diagnosis of various lesions of the Achilles tendon and its surrounding tissue. Furthermore, it can be used to estimate the degree of tendon abnormality and to differentiate between functional and morphologic conditions.

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Acute total ruptures of the Achilles tendon are easily diagnosed clinically [1]. However, complex chronic trauma or partial ruptures hamper the clinical examination and the sensitivity of clinical diagnosis is only about 0.7–0.8 [2]. Tendinitis and peritendinitis may result in a painful thickening of the heel that cannot be accurately differentiated by using clinical examination alone [1, 2]. Considerable delays in surgical treatment have been shown to significantly worsen the eventual outcome in these patients [1]. Therefore, a sensitive imaging tool is needed to predict the risk for tendon rupture and to institute specific therapy and training programs in sports medicine [3].

Low-kilovoltage radiography and xeroradiography only outline the silhouette of the tendon and substantial fluid accumulation in the surrounding tissue [4]. MR imaging, with its excellent soft-tissue contrast resolution and multiplanar capabilities, shows structural abnormalities and fluid accumulations that may be differentiated into hematomatic and nonsanguineous types. Since 1979, sonography has been suggested to demonstrate tendon rupture, alterations of tendon structure, and abnormalities of the peritendinous soft tissues [4–13].

In several of these studies preliminary results were reported, some of them based on small sample populations. Our objective was to determine the value of sonography in detecting abnormalities of the tendo Achillis, using the results of surgery and clinical follow-up to establish the final diagnosis.
Subjects and Methods

We compared sonographic and macroscopic sagittal and transverse sections in three anatomic specimens. After obtaining the sonograms, anatomic sections were obtained with a cryomicrotome. Blocks of tissue were frozen and, as each millimeter of tissue was removed, a photograph was taken of the surface. Normal structure, contour, and diameters of the Achilles tendon and the peritendinous tissue and bursae were evaluated.

We investigated 73 symptomatic patients (54 men and 19 women 13–60 years old; mean age, 38 years) referred for achillodynia and/or thickening of the heel due to tendon enlargement or involvement of the peritendinous soft tissue. Achillodynia is defined as pain at the posterior part of the heel on flexion movements of the foot. Patients with a definite clinical diagnosis of acute total rupture were excluded from the study since clinical assessment is usually sufficient for starting proper therapy. The group consisted of 52 athletes (long-distance runners, jumpers, and basketball players) or patients involved in sporting activities when younger. Another three patients had extensive familial hypercholesterolemia, five patients had systemic inflammatory disease (rheumatoid arthritis in two, systemic lupus erythematosus in three), and 13 patients had no known underlying cause.

After informed consent, 24 healthy volunteers—most of them young athletes (17 men, seven women)—were examined as a control group.

We used McNemar’s test to establish the value of sonography in comparison with clinical and surgical data and analysis of variance to test the relationship of tendon thickness between normal and abnormal individuals.

Clinical symptoms and signs were evaluated by experienced orthopedic surgeons or rheumatologists. Depending on the duration of achillodynia, patients were grouped into those with a history of complaints lasting (1) 2 months or less, (2) 2–12 months, and (3) longer than 1 year. Sensations of pain were evaluated, as were signs of swelling, which were documented as diffuse or circumscribed (swelling at the level of insertion of tendon or palpable nodules). Diagnosis of rupture was made by means of the Thompson test (in which pressure on the calf does not result in plantar flexion of the foot), assessed by an inability to stand on tiptoe or palpation of a localized groove.

At the time of diagnostic imaging, the final diagnoses and the results of other imaging techniques (low-kilovoltage radiography in 34 patients) were not known to any of the four investigators. We used linear phased-array and real-time sector scanners with frequencies of 5–10 MHz (Acuson, Mountain View, CA, and Advanced Technology Laboratories, Bothell, WA). The optimal focal zone of the transducer was placed at the level of the tendon. Patients were placed prone with their feet overhanging the end of the scanning table. If necessary, an acoustic standoff was placed on the skin (Kiteck, 9M, St. Louis, MO) to decrease artifacts in the near field. Documentation included sagittal imaging of the entire tendon and transverse sections. Comparison with the contralateral Achilles tendon and dynamic investigation during plantar and dorsiflexion also were done.

Variables evaluated included tendon swelling, abnormal tendon structure, and peritendinous lesions. Tendon thickness was measured on the anteroposterior scan with a neutral position of the talocrural joint. We used the shorter diameter at the point where the tendon was most thickened. Tendon structure was interpreted as abnormal when there was any disturbance of homogeneous structure in the form of hypoechoic signals with vanishing internal echoes, hyperechoic signals with or without inhomogeneity, or echogenic spots with acoustic shadowing indicating calcification. Analysis of the peritendinous tissue included defects of tendon contours, fluid accumulations, and estimated thickness of the subcutaneous tissue. Contour defects were described as abrupt narrowing of tendon diameter with irregular acoustic shadowing, undulating defects, and/or vanishing borders. Depending on the anatomic location, hypoechoic fluid was interpreted as hematoma, bursitis, or fat pad necrosis. The superficial bursa of Achilles was not evaluated, as clinical findings are specific and diagnostic imaging is not needed.

We compared sonographic results with clinical outcomes in all 73 patients (one to four follow-up investigations within 1–17 months) and with operative diagnoses in 18 patients. A final clinical diagnosis of Achilles tendon injury due to mechanical stress was established if there was a typical clinical history with exaggeration of symptoms during walking or flexion maneuvers. Systemic inflammatory and metabolic diseases were confirmed by means of specific laboratory tests.

Repeated sonographic examinations were performed in 34 patients. Nine of the patients undergoing surgery had at least two postoperative sonographic examinations.

Results

Normal Anatomy

The Achilles tendon originates at the point where the posterior fascia of the triceps surae unites with the fibers of the soleus muscle. Its insertion at the midpoint of the posterior aspect of the os calcis can be depicted as a strong echogenic border against the bone. The tendon flattens and covers the calcaneous like a cuff. In children, a broad layer of hyaline cartilage covers the posterior aspect of the calcaneus. With high-resolution equipment, the structure of the hyaline cartilage is hypoechoic with speckled hyperechoic foci that correspond to the calcified posterior epiphysis calcanei.

In the group of normal control subjects, all tendons showed a homogeneous fibrillar structure on sagittal scans (Fig. 1), with six to eight characteristic undulated lines of internal echoes that reflect the acoustic borders between twisted collagen fibrils and loose connective tissue permeating between fasicles. On transverse scans, they appeared in a honeycomb pattern. Tendon echogenicity depends on the angle between the sound beam and collagen fibers. Because of the normal convex course of the tendon, focal artifactual changes in echo texture are almost always visible and should not be mistaken for localized abnormality. The tendon is demarcated against the surrounding fatty tissue by its epiten- denium, which is visible as an echogenic line. Its form is oval and may be ventrally concave and its orientation is generally oblique, with the transverse axis oriented from postero lateral to anteromedial. In some individuals, the tendon is subdivided by an echogenic septum, which may be visible sonographically and should not be misinterpreted as a sign of rupture.

Normal tendon thickness ranged from 4.0 to 6.7 mm (mean, 5.2 mm) in our group of 24 healthy adults. Those asymptomatic subjects with tendon thickness of more than 6 mm were involved in intensive sports activities suggesting physiologic adaptation to mechanical stress or beginning painless tendinitis.

The pretendinous fat pad, radiologically referred to as Kager’s triangle, shows a mottled echo texture with a broad
Fig. 1.—31-year-old healthy man.
A. Normal sonogram. Achilles tendon (arrows) originates from distal soleus muscle fibers (s), uniting with posterior fascia of triceps surae. With a sector scanner, which provides a larger field of view, typical amplification of tendon signals is visible in center of image. Tendon inserts at os calcis (c). Pre-Achilles fat pad structure (fp) varies among individuals. Anterior fascia of triceps surae muscle covers deep crural and peroneal muscles (pm), which run behind posterior aspect of tibia (t) and fibular joint (j).
B. Zoomed sagittal linear sonogram. Homogeneous linear structures within tendon correspond to collagen fibers. Tendon borders (arrows); normal sub-Achilles bursa (arrowheads).
C. Transverse sonogram. Oval tendon fibers (arrows) and loose connective tissue between fascicles appear in a honeycomb pattern.

spectrum of variation among people. Consequently, differentiation between normal structure and inflammatory swelling may not be possible. Together with the Achilles tendon it fills the distal part of the triceps surae compartment of the calf. Sometimes the tendon of the plantar fascia is visible as a linear structure running through the distal part of the pre-Achilles fat pad, with its shape and course varying considerably. Ventrally, it ends at the deep layer of the crural fascia, which covers the flexor hallucis longus and the peroneus brevis muscles.

A crescent-shaped deep sub-Achilles bursa forms between the tendon and the upper posterior edge of the calcaneus. It was visible as a thin hypoechoic structure in all of the subjects examined. Its form depends on the amount of flexion and varies from person to person.

As the posterior epitenon is the continuation of the superficial crural fascia, the Achilles tendon lies under the subcutaneous tissue. At the skin, an echogenic band delineates the acoustic border between coupling gel and skin. Echogenic structures below represent the epidermis and the corium with an indistinct border to the more hypoechoic subcutaneous tissue.

Pathologic Findings

Abnormalities (Table 1) were found in 53 patients. Sonographic detection had a sensitivity of 0.72 and a specificity of 0.83 (chi-square = 9375, p = .002). In 20 patients with Achilles tendon injury due to mechanical stress, no sonographic abnormalities were found. Retrospective analysis revealed that 14 of the 20 patients had achillodynia lasting not longer than 2 months. In none of these 14 cases were clinical signs of swelling detected or function tests abnormal, and in 11 patients the symptoms subsided within 4–8 weeks after the first onset of achillodynia. This suggests the complaints were due to functional disorders without structural derangements of the tendon and its surrounding tissue. Calculating the numbers excluding the group of patients with low-grade symptoms increased the sensitivity to 0.9. In nine cases, sonographic examination of the contralateral asymptomatic heel revealed abnormalities in tendon structure that were not detected clinically. Thorough reexamination disclosed the source of former pain in the contralateral heel in seven patients.

Tendinitis and tendon necrosis.—Changes were found in 32 patients who had one or more than one of the following abnormalities.

Focal or diffuse thickening of the tendon occurred in 24 (45%) of 53 patients. There was a significant difference (p = .0001 with alpha = 0.05) between tendon thickness in normal control subjects (4.0–6.7 mm; mean, 5.15), patients with Achilles tendon injury without operation (4.0–14 mm; mean, 7.58), and postoperative patients (7.0–15.0 mm; mean, 10.43 mm).

Tendon structure was altered in 22 patients (42%), and the degree of tendon abnormalities could be estimated. Focal hypoechoic lesions generally occurred 2–5 cm above the calcaneus in the medioventral part of the tendon. In four patients, surgery disclosed focal necrosis. Extensive inhomogeneity of tendon structure indicating chronic tendinitis was observed in 13 cases. It occurred in seven of the surgically treated patients and four of those with a ruptured tendon.

Calcifications at the site of insertion were found in three patients and could also be seen on lateral radiographs. In four patients, tiny intratendinous echogenicities with acoustic shadowing suggesting focal abnormalities with the beginning of little calcifications were invisible on corresponding radiographs.

Tendon lesions of patients with systemic metabolic or inflammatory disease could not be differentiated from tendinitis resulting from mechanical stress. In the two patients with rheumatoid arthritis, no calcaneal erosions were seen, nor
TABLE 1: Correlation of Sonography with Duration of Symptoms and Surgical Findings

<table>
<thead>
<tr>
<th>Sonographic Result</th>
<th>All Patients</th>
<th>With Symptoms of 2 mo or Longer</th>
<th>Surgical Correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td>True positive</td>
<td>53</td>
<td>53</td>
<td>17</td>
</tr>
<tr>
<td>False positive</td>
<td>4</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>True negative</td>
<td>20</td>
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<tr>
<td>False negative</td>
<td>20</td>
<td>6</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>97</td>
<td>83b</td>
<td>18</td>
</tr>
</tbody>
</table>

*Sensitivity = 0.72; specificity = 0.83.
*Sensitivity = 0.90; specificity = 0.83.

were erosions visible on other radiographs. A hypoechoic oval lesion was seen in one, connected to the tibiotarsal articulation, corresponding to joint effusion. In two of the three patients with familial hypercholesterolemia, type II, thickened echogenic tendons were found bilaterally.

Peritendinous lesions.—Changes in the peritendinous tissue were found in 25 (47%) of 53 patients. Defects in tendon contour indicating acute peritendinitis showed poorly defined borders with the surrounding tissue, with or without semicircular fluid accumulations. Peritendinous swelling could be estimated by the thickening of the subcutaneous tissue. However, in four patients, the clinical sign of edematous swelling of the heel was invisible on sonography, probably because of compression effects from the transducer during investigation. In one patient, a hypoechoic tumor shown compressing the tendon was surgically proved to be a cutaneous fibroma.

Anterior Achilles bursitis (Albert disease) was found in four patients. In two of them a round or oval hypoechoic lesion (Fig. 2) could be documented, suggesting an intrabursal fluid collection. In the other two patients only indirect signs of hypoechoic transformation of adjoining tendon fibers were seen and there was no enlargement of the bursa. In two patients, sonographically guided intrabursal injection of a soluble corticosteroid was effective in relieving the symptoms; sonographic follow-up studies after 1 month showed a normal tendon.

Tendon rupture and postoperative changes.—Incomplete tendon ruptures were diagnosed in eight (15%) of 53 patients. Six of them had recent rupture and showed one or more of the following sonographic signs: localized reduction of tendon diameter (five cases), contour defect (five cases), irregular acoustic shadowing (owing to rolled fibers in three patients), intra/peritendinous fluid (hematoma) of more than 1 cm in diameter (three cases), and thickening of the severed ends of the tendon (five patients).

All but two clinically diagnosed tendon ruptures were identified correctly with sonography. One partial rupture at the site of insertion of the soleus muscle was overlooked. In one patient, a false-positive diagnosis of rerupture was made on the basis of a bizarre hyperechoic strand of soft tissue that had been used by the orthopedic surgeon to bridge the defect. Conversely, two clinically occult ruptures were detected with sonography. They were overlooked by the clinician because a dense fibrous scar and granulomatous tissue masked the defect (Fig. 3).

Old ruptures were diagnosed in two of the eight patients who had a positive Thompson test and had experienced trauma 8 and 17 weeks earlier, respectively. With sonography, a characteristic cuff of hypoechoic and inhomogeneous echoes (Fig. 4) surrounded the torn tendon, indicating repair with granulomatous tissue.

One to four postoperative sonographic follow-up studies were performed in nine patients. A thickened inhomogeneous tendon was found in seven patients and did not change during 4–16 months. The two patients without tendon thickening had undergone surgery for peritendinous abnormalities (one focal necrosis due to instillation of a corticosteroid, as reported previously [5]), and complete restitution could be demonstrated. Intratendinous sutures were visible in two cases in the form of echogenic lines and spots. In five patients, a flattened upper posterior edge of the calcaneus indicated operative removal of a prominent bursal projection.

Discussion

The most common cause of achillodynia is degeneration of tendon fibers due to mechanical stress. Metabolic diseases (hypercholesterolemia and disorders in steroid metabolism, particularly Cushing syndrome) and weakening due to systemic inflammatory diseases such as rheumatoid arthritis, psoriasis, Reiter syndrome, ankylosing spondylitis, juvenile chronic arthritis, and gout are uncommon causes [1]. Experimental and clinical studies [1] show that a normal tendon ruptures only from an extreme pulling force (4.3–9.12 kN) or after pretraumatic degeneration. Therefore, most ruptures occur because of predisposing destruction of tendon texture, as suggested by pathologists. Localized hypovascularization [14] and asynchronous rubbing of inadequately woven collagen bundles [15] have been proposed to cause circumscribed degeneration and increase the risk of rupture. The clinical term achillodynia summarizes at least five different

Fig. 2.—53-year-old man. Sagittal sonogram shows enlarged sub-Achilles bursa containing some fluid (arrows) in angle between os calcis (c) and Achilles tendon (arrowheads).
morphologic entities (tendinitis, peritendinitis, inflammation of the deep or superficial bursa of Achilles, and enthesopathy at the tendon insertion). Differentiation among them is necessary for adequate treatment but is not always possible with clinical investigation alone [1, 4].

Tendon structure is seen sonographically in the form of linear echoes representing acoustic borders between collagen fibers and loose connective tissue permeating between fascicles. On the basis of the phenomenon of acoustic fiber anisotropy as described by Dussik et al. [16], tendon echogenicity is angle-dependent. Crass et al. [17] found that tendons appear hypoechoic when scanned at an angle greater than 2–7°. They would become hyperechoic at smaller angles. With a sector transducer or when studying the somewhat curved Achilles tendon, circumscribed, angle-dependent, artificial inhomogeneities of tendon structure almost always occur. They should not be mistaken for focal tendinitis. At the site of interdigitation of soleus muscle fibers with the tendon, small hypoechoic spots may appear [10] that reflect a special form of angle-dependent tendon echogenicity. All non-artificial disturbances of homogeneous tendon structure indicate abnormality. We interpret small hypoechoic nodules, corresponding to “nodular tendinitis” [4], to be the correlate of areas of focal degeneration with unmasking of collagen fibers [1, 18]. Their typical echogenic appearance may be influenced by the amount of water stored by the proteoglycans [18]. The hypoechoic nodules are the earliest form of tendinitis without tendon thickening and are the possible site of initiation of tendon rupture. Tiny calcifications may be detected earlier with sonography than with plain film radiology.

Eleven of the 20 symptomatic patients without evidence of abnormal sonographic findings had had achillodynia for less than 2 months. This suggests the diagnosis of tenalgia, which is defined as functional disorders of the dorsal muscle apparatus of the leg without evidence of morphologic alterations [19], predicting a good result with conservative therapy. Our results correspond to those reported by Mathieson et al. [10], who describe all patients with normal sonographic findings as recovered completely with conservative treatment in less than 4 months.

In xanthomatosis, large amounts of cholesterol may be deposited in the tendon. In two of our patients, tendon thickening and inhomogeneity of tendon structure could be documented. Although these findings seem to be nonspecific for hyperlipoproteinemia, type II, sonography can be used to monitor patients with this disease [13].

Tendon thickness can be determined exactly. Consequently, palpable nodules may be assigned as intratendinous or originating from the epiteninum. In accordance with Fornage [4], we measured tendon size by using the shorter diameter on transverse scans in order not to overestimate the true transverse diameter of the obliquely oriented Achilles tendon. As focal swelling may occur [1, 4], we measured tendon thickness at the level of its widest diameter. Measuring at the standard level 1 cm above the superior calcaneal surface may lead to false-negative findings. We assume divergent interpretations in the literature concerning the value of tendon diameter are due to different ways of taking the measurements [4, 10, 13].

Peritendinitis manifests itself as tendon contour alteration and soft-tissue thickening, which may be estimated by the amount of subcutaneous tissue swelling. However, compression effects from the transducer may hide peritendinitis. Several of the false-negative sonographic findings in our series may be from peritendinous edema or tiny fibrous strands at the tendon border. Fluid accumulation around the tendon may occur with peritendinitis, as a result of tendon rupture or as an indication of necrosis of the pre-Achilles fat pad. Cutaneous lesions may be demonstrated and their extension, such as compression of the tendon surface or invasiveness, determined. With modern high-resolution equipment, the normal retrocalcaneal bursa is always visible. Variations between subjects may account for difficulties in demonstrating inflammation in the generally flattened or wrinkled bursa [20]. There-
fore, bursitis will be detectable only with considerable fluid accumulation or on the basis of accompanying focal tendinitis in the vicinity of the bursa. Sonographically guided injection of a corticosteroid into the bursa with exact positioning of the needle may avoid intratendinous installation, which has a high risk of tendon necrosis [1].

Our results and those of others show that tendinitis, peritendinous lesions, and rupture may be determined properly with the help of sonography (Table 2). Signs of focal tendinitis were found accidentally in nine cases in the contralateral heels. This supports the high value of sonography in detecting abnormalities of tendon structure. Peritendinous changes can be diagnosed only on the basis of semicircular fluid accumulation or considerable fibrous scars. Edema of the peritendinous tissue is assessed to better advantage with clinical examination; tiny fibrous strands may be missed by either method. Equivocal symptoms and signs of incomplete or old rupture can be specified with the help of sonography.

With plain films and xeroradiographs, internal anatomy of the tendon is poorly delineated [4]. The sensitivity of these methods in depicting ruptures is very low, and diagnosis of tendons at higher risk of rupture is based merely on indirect signs [3, 20]. Opacification of Kager’s triangle is visible only with considerable fluid accumulation. However, films should be exposed to prove or rule out bony fracure with accompanying ruptures. Furthermore, radiographic techniques may be useful in evaluating Haglund syndrome, which is the result of a prominent bursal projection of the calcaneus, and in detecting calcaneal spurs or calcifications [3, 20].

Therefore, high-resolution sonography should play a central role in imaging the Achilles tendon. It helps in classifying different nosologic entities included under the term achillodynia. Sonography specifies the clinical diagnosis and may reduce delay in treatment. A normal sonogram in a symptomatic patient with a short duration of achillodynia will reliably predict a successful response to conservative treatment and aid in establishing proper training programs. Clinically occult ruptures may be detected [4, 10]. Because of the possibility of a false-negative result in diagnosing peritendinous edema, a combined clinical and sonographic evaluation is mandatory.

## REFERENCES


## TABLE 2: Summary of Sonographic Findings in the Literature

<table>
<thead>
<tr>
<th>Study</th>
<th>Year</th>
<th>n</th>
<th>Rupture (%)</th>
<th>Tendon Swelling</th>
<th>Abnormal Structure</th>
<th>Peritendinous Lesions (%)</th>
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<tr>
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<td>63</td>
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<td>30</td>
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<td>20</td>
<td>15</td>
<td>+</td>
<td>+</td>
<td>35</td>
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<tr>
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<td>97</td>
<td>15</td>
<td>45</td>
<td>42</td>
<td>47</td>
</tr>
</tbody>
</table>

Note.—The discrepancies in demonstrated ruptures are attributed to different study design. NE = not evaluated; + = positive findings.

*a* Includes defects of tendon contour.

*b* Includes both tendon swelling and abnormal structure.