The *in vivo* kinematics of the tibiotalar joint after lateral ankle instability

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Abstract

BACKGROUND—Previous studies have suggested injury to the anterior talofibular ligament may be linked to altered kinematics and the development of osteoarthritis of the ankle joint. However, the effects of ATFL injury on the *in vivo* kinematics of the ankle joint are unclear.

HYPOTHESIS—Based on the orientation of the ATFL fibers, we hypothesized that ATFL deficiency would lead to increased anterior translation and increased internal rotation of the talus relative to the tibia.

STUDY DESIGN—Controlled laboratory study.

METHODS—The ankles of 9 patients with unilateral ATFL injuries were compared as they stepped onto a level surface. Kinematic measurements were made as a function of increasing load. Using magnetic resonance imaging and orthogonal fluoroscopy, the *in vivo* kinematics of the tibiotalar joint were measured in the ATFL deficient and intact ankles from the same individuals.

RESULTS—A statistically significant increase in internal rotation, anterior translation, and superior translation of the talus was measured in ATFL deficient ankles as compared to intact, contralateral controls. For example, at 100% body weight, ATFL deficient ankles demonstrated a statistically significant increase in anterior translation of 0.9 ± 0.5mm (p = 0.008). At 100% body weight, the ATFL deficient ankle was internally rotated relative to the intact ankle by $5.7 \pm 3.6°$ (p = 0.008). There was a slight increase of 0.2 ± 0.2mm in the superior translation of the ATFL deficient ankle compared to the intact ankle at 100% body weight (p = 0.02).

CONCLUSIONS—ATFL deficiency increases anterior translation, internal rotation, and superior translation of the talus.

CLINICAL RELEVANCE—Altered kinematics may contribute to the degenerative changes observed with chronic lateral ankle instability. These findings might help to explain the degenerative changes frequently observed on the medial talus in patients with chronic ATFL insufficiency and provide a baseline for improving ankle ligament reconstructions aimed at restoring normal joint motion.

Keywords

lateral ankle instability; anterior talofibular ligament; osteoarthritis; tibiotalar joint; ankle kinematics; ankle biomechanics

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Introduction

Lateral ankle ligament sprains are one of the most common injuries in sports and recreation[19, 23, 37, 44, 51] and are among the most frequently treated injuries in emergency departments [14, 21]. Although many patients have good clinical outcomes after conservative treatment, a significant number of patients experience chronic pain, instability, loss of range of motion, poor proprioception, and develop osteoarthritis [18, 20, 21, 23, 28, 32, 33, 42, 44, 48, 49, 51]. Between 10 and 40% of patients with lateral ankle sprains have been thought to experience chronic symptoms[1, 8, 11, 15, 22, 35, 39, 45, 49, 51].

Many investigators have reported a relationship between chronic lateral ankle instability and the development of degenerative changes [20, 23, 51, 52]. For example, one study reported that of 36 patients with chronic lateral ankle instability for at least 10 years, 72% had evidence of arthritic changes in the tibiotalar joint[20]. More recent patient studies attributed 13-16% of ankle arthritis to ligamentous injuries[51, 52], 85% of whom had sustained injury to the lateral ankle ligaments[51]. Many investigators have hypothesized that altered kinematics are a factor contributing to the development of osteoarthritis after lateral ankle ligament injuries [18, 20, 22, 23, 44, 48, 51, 52].

While these studies suggest a relationship between altered kinematics and the development of osteoarthritis, the effect of lateral ankle injuries on the *in vivo* weight-bearing kinematics of the ankle joint are unclear. Previous studies have measured the effects of rupture of the lateral ankle ligaments on joint biomechanics in cadavers [5, 16, 38, 44, 46]. However, it is difficult to reproduce the complex *in vivo* loading environment on cadavers[38]. Other studies have used skin markers and videographic analyses to study the altered biomechanics after lateral ankle ligament injury [10]. Although these studies provide valuable data on altered biomechanics after lateral ankle stability, it is difficult to measure directly the motion of the tibiotalar joint using skin markers.

Therefore, the objective of our study was to quantify the effects of lateral ankle instability on the motion of the tibiotalar joint under *in vivo* weight-bearing loading. Using a combination of a 3D magnetic resonance model and dual-orthogonal fluoroscopes, ankles with lateral instability were compared *in vivo* to contralateral controls under physiological loading conditions. We hypothesized that lateral ankle instability leads to increased anterior translation and increased internal rotation of the talus due to the orientation of the fibers of the anterior talofibular ligament (ATFL).

Methods

Patient Recruitment

A total of nine subjects (5 males, 4 females) were studied with IRB approval from our institution. All subjects had a body mass index of less than 30, and were aged 19-57 years. Subjects recruited in this study had unilateral ankle sprains which had been treated conservatively for at least 6 months. All subjects reported symptoms of chronic lateral ankle instability, including pain, feelings of instability, and recurrent ankle sprains. Patients with cartilaginous lesions (visualized during study MRI or after the study during surgery) were excluded. All subjects were required to have a healthy, normal contralateral ankle, confirmed by both physical examination and MRI, or they were excluded from study.

Each subject was clinically examined by a board-certified orthopaedic foot and ankle surgeon, who serves as a mentor to our foot and ankle fellows and has vast experience in foot and ankle. The surgeons defined the diagnosis of chronic instability as a history of recurrent ankle sprains and a sensation of instability in the setting of positive physical exam findings, including a

positive anterior drawer or talar tilt tests. To corroborate the clinical findings of instability, a fellowship-trained, board-certified musculoskeletal radiologist confirmed that all subjects had unilateral ATFL injury using magnetic resonance (MR) imaging. For patients that underwent surgery after the study, these findings were confirmed intraoperatively. Though the anterior talofibular ligament (ATFL) is the most frequently involved ligament in lateral ankle injuries, additional injury to the calcaneofibular ligament (CFL) is common [9, 14, 22, 28, 34, 51]. Patients with isolated ATFL injuries are difficult to recruit, so 4 patients included in this study had combined ATFL/CFL injuries.

Imaging

Subjects had both ankles MR imaged at the Center for Advanced Magnetic Resonance Development in the Department of Radiology using a 3.0T magnet (Trio, Siemens, Germany) and a dedicated 8 channel receive-only foot and ankle coil (Invivo, Orlando FL). For each subject, both ankles were imaged separately using a 3D double-echo steady state sequence (DESS, Flip angle 25 degrees, TE 6 msec, TR 17 msec) with a 15cm by 15cm field of view. The images produced had a resolution of 512 by 512 pixels and a slice thickness of 0.7 mm. Each ankle was scanned twice, once with water excitation and once without. In each sagittal image, the anatomy of the tibia and talus were outlined using solid modeling software (Rhinoceros, McNeel and Associates, Seattle WA). Comparison of water excitation and non-water excitation images allowed for accurate outlining of complex bone and soft tissue interfaces. Each contour was then placed in the appropriate plane in space, and the curves were used to generate a 3D surface model of the tibiotalar joint (Figure 1).

Next, each patient was imaged using two fluoroscopes (Pulsera, Philips, The Netherlands) positioned orthogonally above a platform. The fluoroscopes were wired to take images simultaneously with a resolution of 1024 by 1024 pixels. Subjects stood on the platform and stepped onto a level surface within the beam of both fluoroscopes. Subjects repeated this step for each ankle, increasing the load from 25 to 100 percent of their body weight as measured by a force plate. One fluoroscope obtained an anterolateral image, the other an anteromedial image (Figure 1).

Kinematics Measurements

Each pair of fluoroscopic images was imported into solid modeling software, where they were positioned in a 3D environment to reproduce the orthogonal orientation of the fluoroscopes during testing. Using a pixel density gradient matrix, edge detection software was used to outline the bone structures on the fluoroscopic images [12]. The 3D MR model of the tibia and talus was imported into this virtual dual-orthogonal fluoroscopic system. The 3D model was viewed from two directions corresponding to the location of the image source of the fluoroscopes. Then, the position of the tibial and talar models were individually manipulated in six degrees-of-freedom until their projections, as viewed from the two orthogonal directions, matched the outlines on the fluoroscopic images (Figure 1). Thus, the 3D models were used to reproduce the 3D motion of the tibiotalar joint during *in vivo* weight-bearing loading.

In order to quantify the motion at the tibiotalar joint, a Cartesian coordinate system was constructed for each subject based on the 3D anatomy of the tibia and talus. In order to reduce variability, the coordinate systems were created on both the intact and injured joints simultaneously. 12 Because the same coordinate system was drawn on both the intact and deficient ankles, the motion of the two joints was directly compared using the same anatomic coordinate systems. First, point clouds of approximately 5,000 points were created from the 3D models of each talus and tibia. The point cloud of the deficient ankle was then aligned to the position of the intact ankle using an iterative closest point technique [12, 31]. In this fashion, the position of the intact ankle under minimal load (approximately 10N) was defined as neutral

for both ankles. Next, the coordinate axes were drawn on the 3D models. To define the proximal-distal axis, a cylinder was fit to the shaft of the tibia. The medial-lateral axis was defined by a segment connecting the most medial and lateral extremes of the tibia. Finally, the anterior-posterior axis was positioned orthogonal to these two axes. To determine the origin of the axis, the talus was visualized in the sagittal plane and a circle was fitted to the curve of the talar dome. The center of this circle served to define the origin in the sagittal plane. The medial-lateral component of the origin was defined by the geometric center of the surface area of the talar dome.

The coordinate systems were used to measure the six degrees-of-freedom kinematics of the tibiotalar joint. Measurements included anteroposterior, mediolateral, and superoinferior translations as well as internal-external rotation, dorsiflexion-plantarflexion, and inversion-eversion. These rotations were represented by an Euler angle sequence, where plantarflexion-dorsiflexion is measured about a fixed axis in the tibia, internal rotation about a fixed axis in the talus, and inversion-eversion about an axis perpendicular to the other two.

Validation Study

To validate the accuracy and repeatability of this system to measure kinematics, a pilot study on a cadaveric ankle was conducted. First, the ankle was MR imaged with the protocol described above to construct a 3D model of the ankle joint. Next, sets of spherical, radio-opaque markers[13] with a diameter of 2.38 mm and a tolerance of 0.002 mm were implanted into the tibia, calcaneus, and talus. These markers were used to measure the motion of the ankle as it was moved from neutral to maximal dorsiflexion and maximal plantarflexion. Orthogonal images in each position were recorded using the two fluoroscopes. The orthogonal fluoroscopic images and 3D computer model of the ankle were imported into solid modeling software. The 3D models of the tibia, talus, and calcaneus were manipulated in six degrees-of-freedom to create a reproduction of the ankle in space as described above (Figure 2). Five independent matching trials were performed with the ankle in neutral, dorsiflexion, and plantarflexion.

The motions of the marker sets were tracked using edge detection algorithms based on the gradient of pixel intensity [12, 13] within each fluoroscopic image. The center of each bead on the two orthogonal fluoroscopic images was then used to calculate the position of each marker in 3D space. From these data, the relative motion of the ankle joint measured using the combined fluoroscopic and MR imaging techniques was compared to the motion measured using the marker sets. Orthogonal coordinate systems were created on the ankle, and the difference in translation and rotation between the markers and model-based matching technique were calculated. The average error in displacement across each of the five trials for each position was 0.04 ± 0.11mm, and the average error in rotation was $0.2 \pm 0.1°$. Therefore, we are confident in our ability to measure the changes in motion as attributed to lateral ankle instability.

Data Analysis

The kinematics data for each ankle (anteroposterior, mediolateral, and superoinferior translations as well as internal-external rotation, dorsiflexion-plantarflexion, and inversion-eversion) was averaged as a function of percent body weight. At each loading level, the motion of the ATFL deficient ankle was compared to the corresponding contralateral intact ankle using the Wilcoxon Signed-Rank Test. This nonparametric alternative to the paired t-test does not require that the data be normally distributed and therefore, in general, is more robust than the paired t-test for a small sample size [17]. Differences were considered statistically significant where $p < 0.05$.

Results

Anteroposterior Translation

On average, the talus of both ATFL deficient and intact ankles translated anteriorly under the applied load (Figure 3). In the intact ankles, the talus translated anteriorly from a mean of 0.2 ± 0.6mm at 25% body weight to 0.4 ± 1.0mm at 100% body weight. A similar trend was detected in the ATFL deficient ankles. However, there were statistically significant increases in anterior translation compared to the intact ankle at each loading level. For example, at 100% body weight the injured talus translated anteriorly by 1.3 ± 0.9mm. This represents an increase of 0.9 ± 0.6mm compared to the intact ankle (p = 0.008). All 9 of the injured ankles translated anteriorly relative to the intact ankle.

Mediolateral Translation

With weight-bearing loading, there was a slight lateral translation of the talus in both the intact and ATFL deficient ankles with weight-bearing loading (Figure 4). In the intact ankle, the lateral position of the talus ranged from 0.1 ± 0.6mm at 50% body weight to 0.4 ± 0.6mm at 75% body weight. Similar values were found for the deficient ankle, ranging from 0.4 ± 0.6mm at 25% body weight to 0.7 ± 0.7mm at 75% body weight. No statistically significant differences were detected between the intact and unstable ankles.

Superoinferior Translation

With increasing loading, the talus displaced superiorly relative to the tibia in both the intact and injured ankles (Figure 5). In the intact ankle the superior displacement ranged from 0.0 ± 0.3mm at 25% body weight to 0.3 ± 0.7mm at 100% body weight. A similar trend was observed in the injured ankle, where superior translation ranged from 0.4 ± 0.6mm at 25% body weight to a maximum of 0.6 ± 0.7mm at 100% body weight. There was a statistically significant increase in the superior translation of the talus in the injured ankle compared to the intact ankle at 100% body weight (p = 0.02). Eight ankles translated superiorly, and one ankle was within 0.1mm of the contralateral side.

Internal-External Rotation

The internal rotation of the talus relative to the tibia in the intact ankles did not change dramatically with increasing load (Figure 6). At 25% body weight, the talus was a mean of $0.6 \pm 2.7°$ internally rotated, compared to $0.2 \pm 3.5°$ of external rotation at 100% body weight. The talus in the deficient ankles rotated internally with increasing body weight, ranging from a minimum of $3.5 \pm 5.1°$ at 25% body weight to a maximum of $5.5 \pm 4.4°$ at 100% body weight. There was a statistically significant increase in the internal rotation of the talus at each loading level. For example, at 100% body weight, the injured talus was rotated internally by $5.7 \pm 3.7°$ compared to the intact talus (p = 0.008). All 9 of the injured ankles rotated internally relative to the intact ankle.

Inversion-Eversion

On average, the talus in both ATFL deficient and ATFL intact ankles showed increasing eversion with increased load (Figure 7). Intact ankles everted from $0.4 \pm 1.3°$ to $2.5 \pm 2.7°$ as the applied body weight increased from 25% to 100%. Similarly, the ATFL intact ankles everted from $0.6 \pm 2.3°$ to $2.2 \pm 2.2°$. The differences in inversion between intact and injured ankles were less than 0.4° under this loading. No statistically significant differences in inversion were detected between ATFL deficient and intact ankles.

Dorsiflexion-Plantarflexion

Both the intact and injured ankles dorsiflexed with increasing load (Figure 8). In the intact ankle, the talus was $1.3 \pm 4.3°$ plantarflexed at 25% body weight and dorsiflexed to $6.4 \pm 7.7°$ at 100% body weight. Similarly, the deficient ankle ranged from $0.0 \pm 5.0°$ of plantarflexion at 25% to $7.3 \pm 6.2°$ of dorsiflexion at 100% body weight. No statistically significant differences in plantarflexion-dorsiflexion were observed between injured and intact ankles.

Discussion

In this study, we used a combination of a 3D magnetic resonance and orthogonal fluoroscopic imaging to investigate the *in vivo* tibiotalar joint motion of patients with lateral ankle instability under physiological loading conditions. Six degrees-of-freedom kinematics were compared between the injured ankle and the intact, normal contralateral ankle. We found that ATFL deficiency increased anterior translation and increased internal rotation of the talus relative to the tibia. In addition, an increase in the superior translation of the talus was observed. All 9 of the ankles translated anteriorly and rotated internally. Regarding superior translation, 8 ankles translated superiorly, and one ankle was within 0.1mm of the contralateral side. The strength of these trends is reflected by the smaller p values for anterior translation and internal rotation, compared to superior translation.

Previous studies have focused on the role of the ATFL in resisting anterior translation and inversion [24-27, 32, 42]. However, some studies have reported that the ATFL resists internal rotation of the talus [38, 43, 47]. For example, Omori et al applied a 100N compressive load to cadaveric ankles and showed that transecting the ATFL and CFL caused increases in inversion and an increase in internal rotation only in 10° plantarflexion [38]. The function for the ATFL in the current study is somewhat different than that reported in previous studies in that we did not detect a large difference in inversion, but measured significant differences in internal rotation. However, our findings are in agreement with a cadaveric study by Stormont et al, who reported that under a compressive load of 670N, the ATFL and CFL did not contribute significantly to the stability of the ankle in resisting inversion. Rather, the articular surfaces were thought to constrain inversion. In their study, the ATFL also provided resistance to the internal rotation of the talus. Our finding that injury to the lateral ligaments has a greater affect on the internal rotation compared to inversion may be further explained by the differences between *in vivo* and *in vitro* loading conditions. As noted previously [38], the levels of loading applied *in vivo* might be larger than those applied during cadaveric studies. Furthermore, the active musculature present during *in vivo* loading might provide additional stability to help prevent the inversion of the talus during weight-bearing.

The findings of increased anterior translation and internal rotation in ATFL deficient ankles may be explained by the orientation of the ATFL (Figure 9). The ATFL originates on the anterior border of the lateral malleolus and courses anteromedially and inserts on the talar body just anterior to the lateral malleolar articular surface [50]. The orientation of the ATFL suggests that it restricts the anterior translation of the lateral side of the talus, thereby countering the anterior translation of the talus that we observed with increasing weight-bearing. In addition, because the medial constraints are intact, the lateral side of the talus is preferentially unstable, which could explain our observation of increased internal rotation of the talus. Further support for the role of the ATFL in internal rotational stability can be found in measurements made by Taser et al[50]. They reported that as the ATFL runs from the fibula to the talus, it angles 25° distally from the horizontal plane and 47° medially from the sagittal plane [50]. These data suggest that the ATFL insufficiency might result in larger changes in rotational stability as compared to inversion stability.

Previous authors have reported an association between lateral ankle instability and the early onset of post-traumatic osteoarthritis[20, 48]. Many investigators have hypothesized that a change in kinematics and cartilage loading may explain osteoarthritic lesions on the medial talus in prolonged ankle instability. Harrington observed increased wear on the medial portion of the tibiotalar joint in patients who had experienced lateral ankle sprains ten years previously [20]. It was hypothesized that these changes were due to increased stresses on the medial portion of the joint as a result of abnormal kinematics [20]. Taga et al reported that patients with chronic ankle instability had an increased incidence of cartilage lesions, particularly in the anteromedial portion of the joint 48. They hypothesized increased anterior translation of the talus in chronic ankle instability might contribute to the increased incidence of cartilage lesions in the joint 48. Hinterman et al 22 noted that 72% of patients with lateral ankle injuries had cartilage damage, primarily on the medial portion of the talus. They hypothesized that the damage might be due to increased contact stresses in this region [22]. Whether the development of osteochondral defects is in fact a result of altered kinematics or due to other factors (e.g., initial trauma to the cartilage) remains unclear.

The findings of altered anterior translation, internal rotation, and superior translation of the talus in the current study might support the hypothesis that altered kinematics contribute to the degeneration of the tibiotalar joint that is observed in patients [20, 22, 48]. Previous authors have suggested that increased anterior translation of the talus might increase wear on tibiotalar joint[48]. There may also be a relationship between the increased internal rotation of the talus detected in this study and the development of osteoarthritis. Increased internal rotation may lead to increased cartilage loading on the medial side of the talus, due to the curved surface of the cartilage on the medial portion of the tibia (Figure 10). Furthermore, increased rotation might cause increased shear stresses in the cartilage and predispose the joint to degenerative changes [2-4]. These findings might help to explain the increased degeneration observed on the medial talus in patients with lateral ankle instability [20, 22, 48]. Finally, our observation of increased superior translation of the talus relative to the tibia might indicate that there are increased compressive strains in the cartilage of the ATFL deficient joint. Future studies will address the effects of altered motion on *in vivo* contact patterns in these patients.

The kinematic data elucidated by this study may also have important clinical applications to reconstruction of the lateral ankle ligaments. Understanding how ATFL deficiency affects the motion of the tibiotalar joint may provide direction in the design and evaluation of reconstructive techniques. A major goal of either repair or reconstruction is to restore normal motion to the joint, and hence prevent degenerative changes to the cartilage [30, 44, 48, 49]. Currently, the appropriate surgical treatment for ankle ligament injuries remains controversial [11, 21, 28-30, 34, 36, 40, 41, 44], with more than 80 different reconstruction and repair techniques described in the literature [7]. Many authors advocate anatomic reconstruction, arguing that tenodesis does not restore normal anatomy and normal motion [6, 30]. A recent long-term follow-up study by Krips et al advocated an anatomic reconstruction over tenodesis[30]. However, radiographic evidence of degenerative changes was observed in 60% of patients in the anatomic reconstruction group and 80% of patients in the Evans tenodesis group[30]. The ability of current surgical techniques to prevent the development of osteoarthritis remains unclear. The findings of this study may help surgeons to focus their efforts on reconstructive techniques that counter the increased internal rotation and anterior translation found with lateral ankle instability.

There are some limitations with this study. This study only examined quasi-static weight-bearing motion of the joint. Future studies should to evaluate the dynamic motion of the tibiotalar joint during gait. Furthermore, the independent contributions of ATFL, CFL, and other soft tissue injuries to the kinematics of lateral ankle injuries could not be elucidated in this study because of the small patient population. Future studies might investigate the differences between these types of injuries. Finally, future studies might also investigate ATFL-

deficient subjects who do not have ankle instability to see if they show the same changes in kinematics as those with ankle instability.

In summary, this study combined a 3D magnetic resonance model and dual-orthogonal fluoroscopes to compare chronically unstable ankles *in vivo* to contralateral controls. We found that under weight-bearing loading, ATFL deficient ankles showed a significant increase in anterior translation, internal rotation, and superior translation of the talus compared to intact controls. These data may provide insight into the mechanisms behind the development of osteoarthritis and provide baseline data for evaluating the surgical treatment of these injuries.

Acknowledgments

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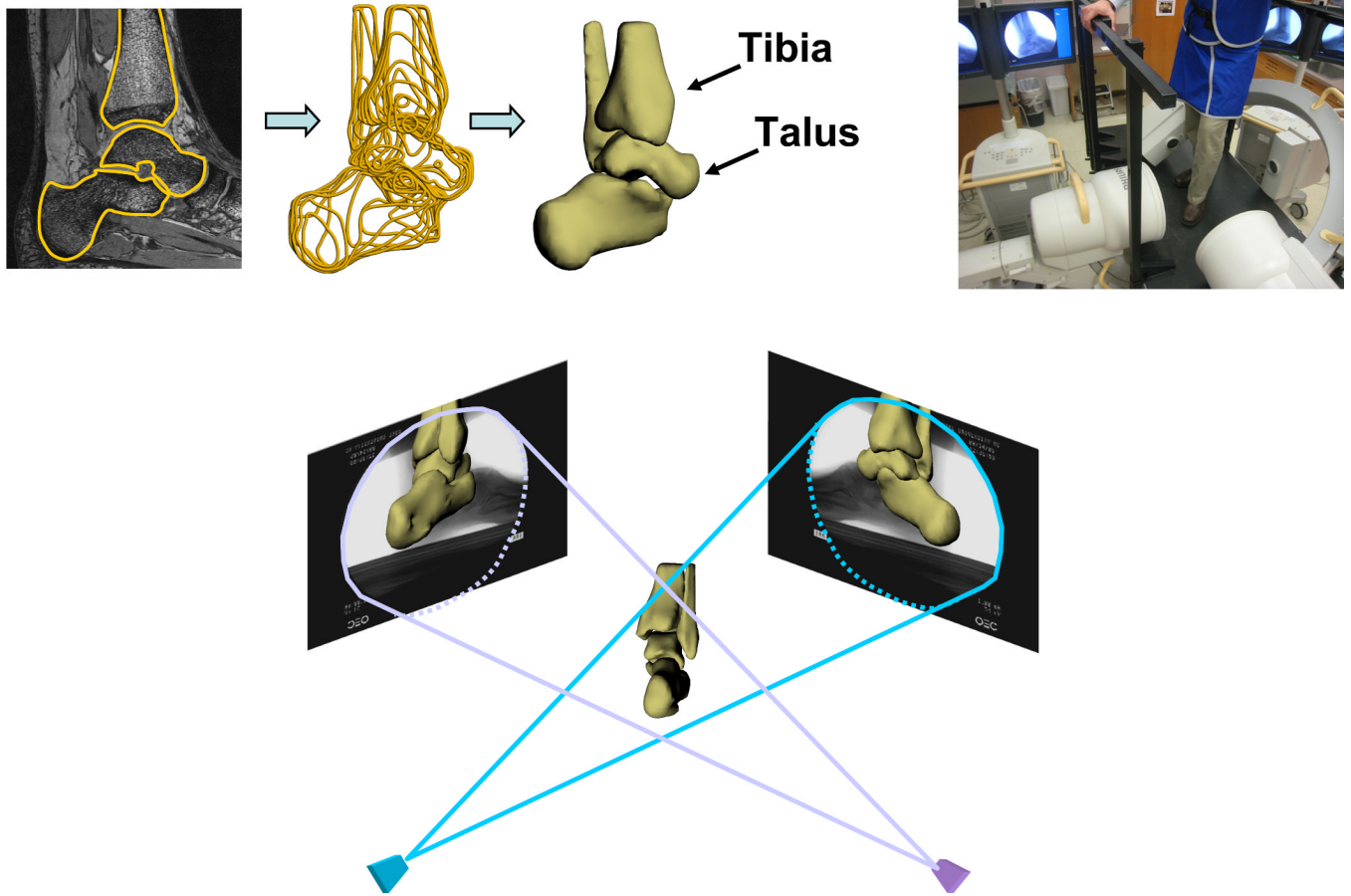
Figure 1.
3D models of the tibia and talus were created from high-resolution MR images (upper left). The patient then stepped onto a level surface with increasing body weight while being imaged using orthogonal fluoroscopy (upper right). The 3D models of the tibia and talus were manipulated in 6 degrees-of-freedom until their projection matched that of the orthogonal fluoroscopic images (bottom).
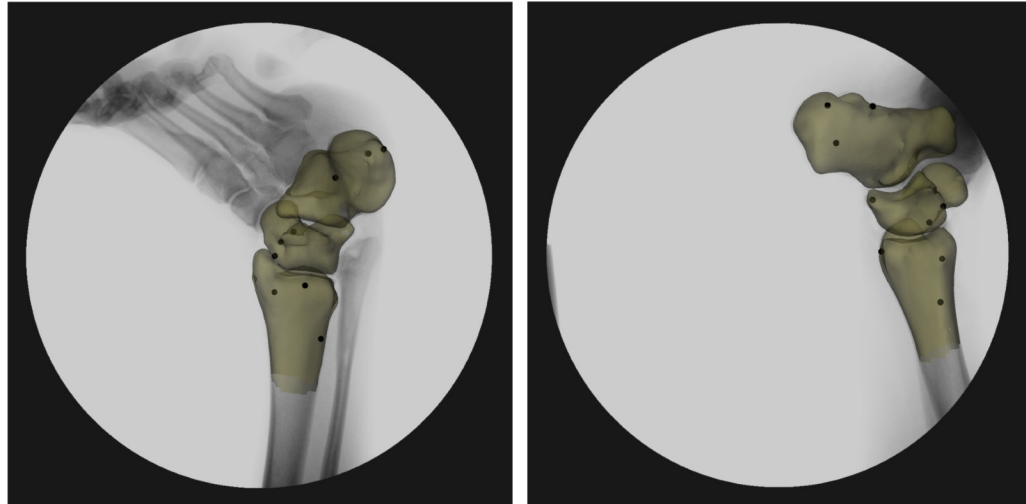
Figure 2.
Radio-opaque markers were implanted into a cadaver for a validation study. The accuracy of the system in measuring tibiotalar kinematics was within 0.04 ± 0.11mm and $0.2\pm0.1°$.
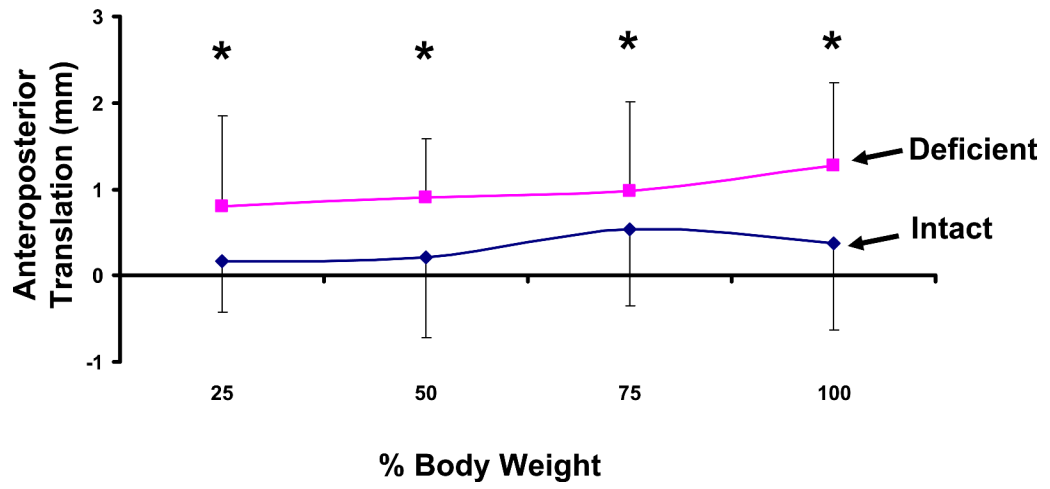
Figure 3.
Weight-bearing loads caused an anterior translation in both the intact and ATFL deficient ankles. Statistically significant increases were observed in the deficient ankle compared to the intact ankle (* $p < 0.05$).
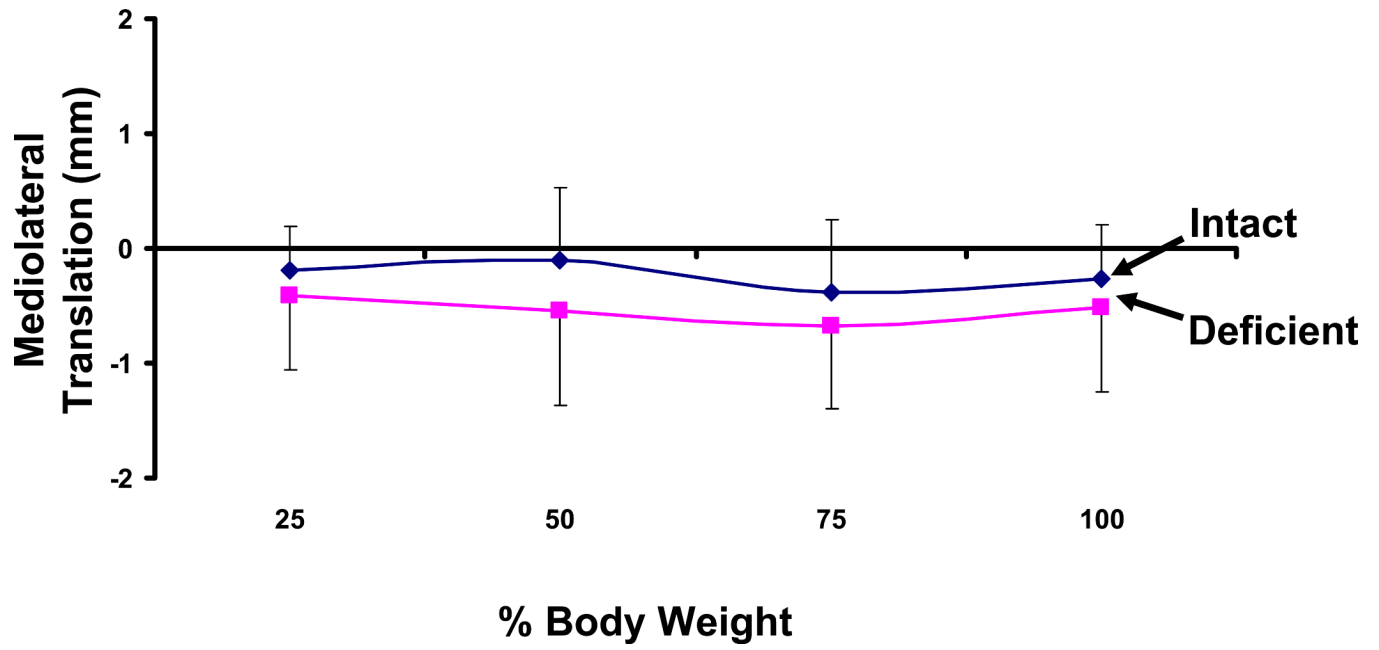
Figure 4.
Weight-bearing loads caused a lateral translation of the talus in both the intact and ATFL deficient ankles. No statistically significant differences were detected.
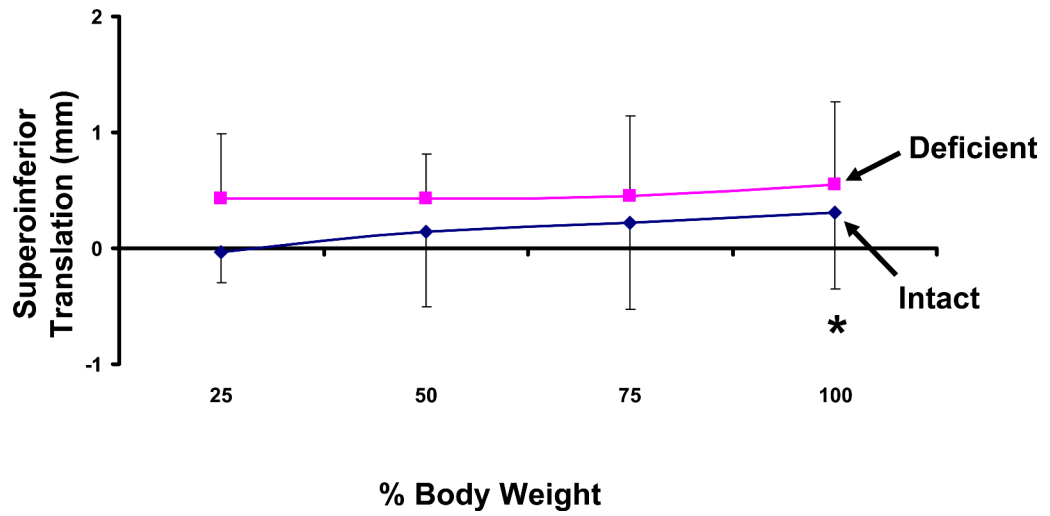
Figure 5.
Weight-bearing loads caused a superior translation of the talus relative to the tibia. At 100% body weight, a statistically significant increase in superior translation was observed in the ATFL deficient ankle (*p < 0.05).
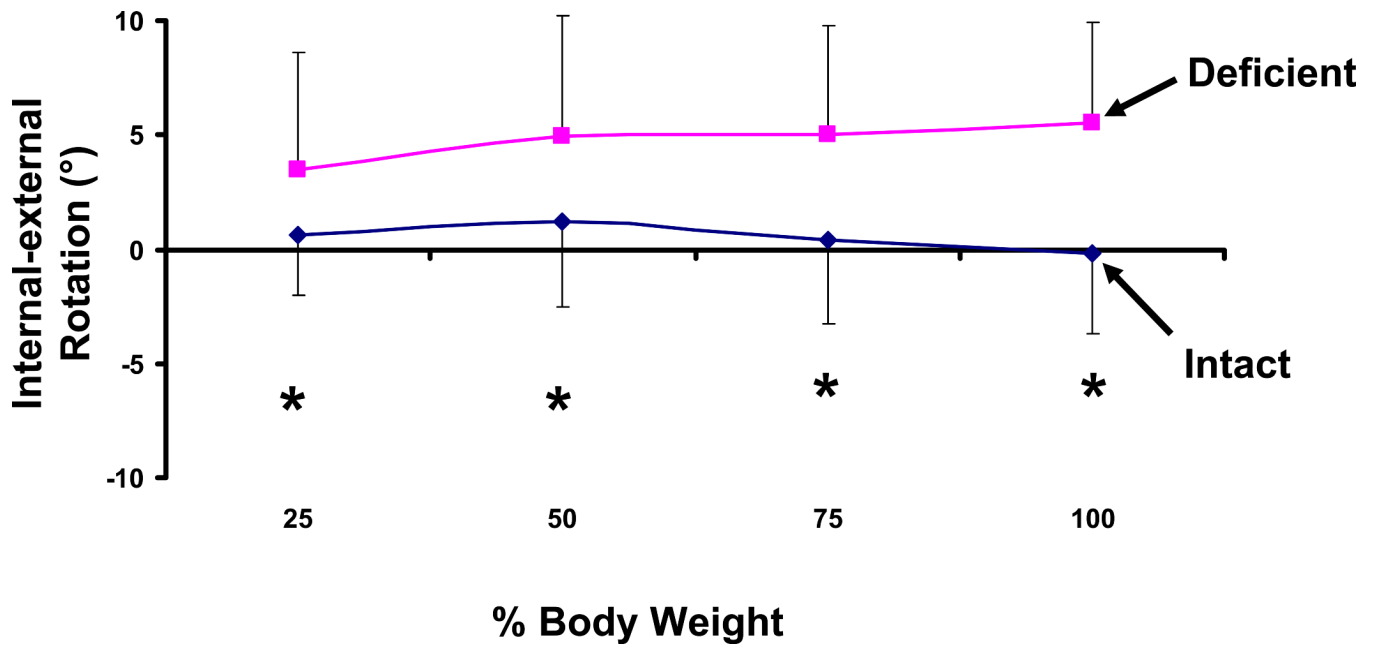
Figure 6.
ATFL deficient caused a statistically significant internal rotation of the ATFL deficient ankle relative to the intact ankle (*p < 0.05).
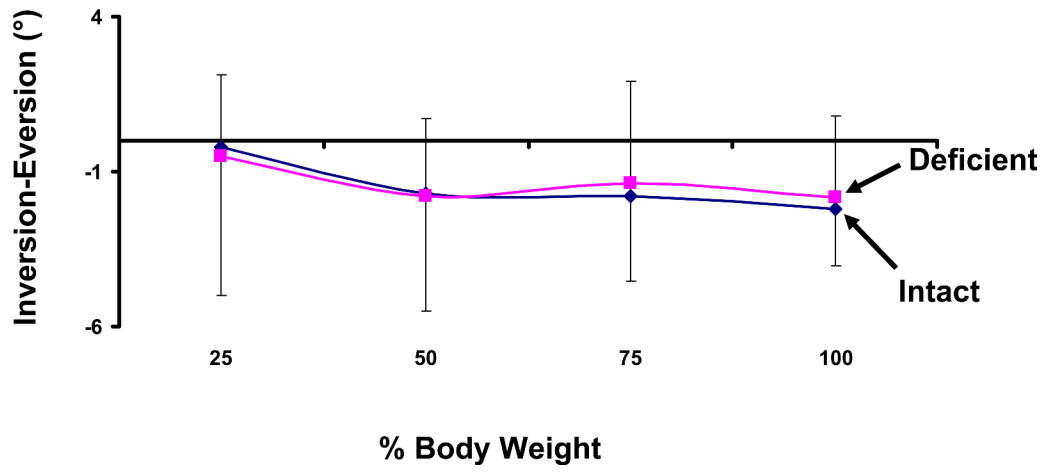
Figure 7.
Weight-bearing loads caused an eversion of the talus. No statistically significant differences were observed between intact and ATFL deficient ankles.
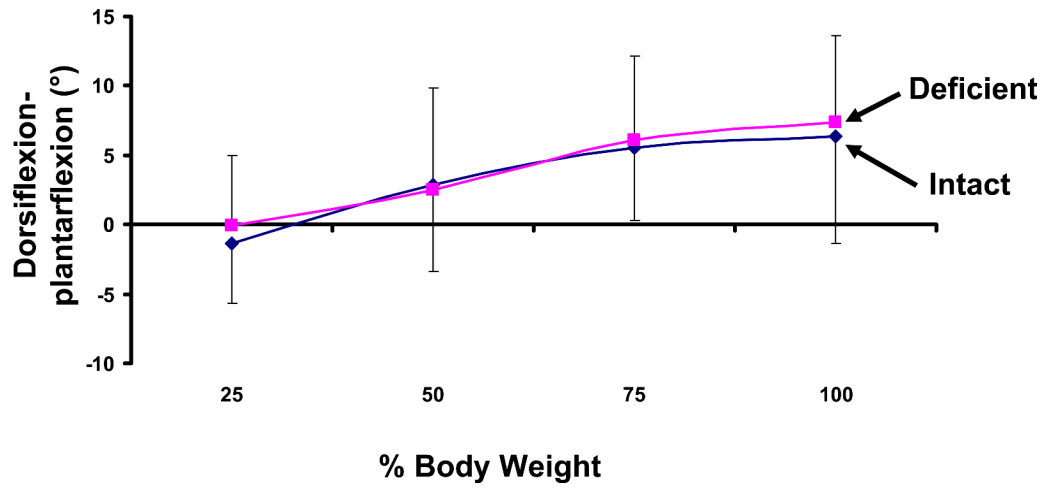
Figure 8.
Increasing weight-bearing loading caused an increase in dorsiflexion of the talus. No statistically significant differences were observed between the intact and ATFL deficient ankles.
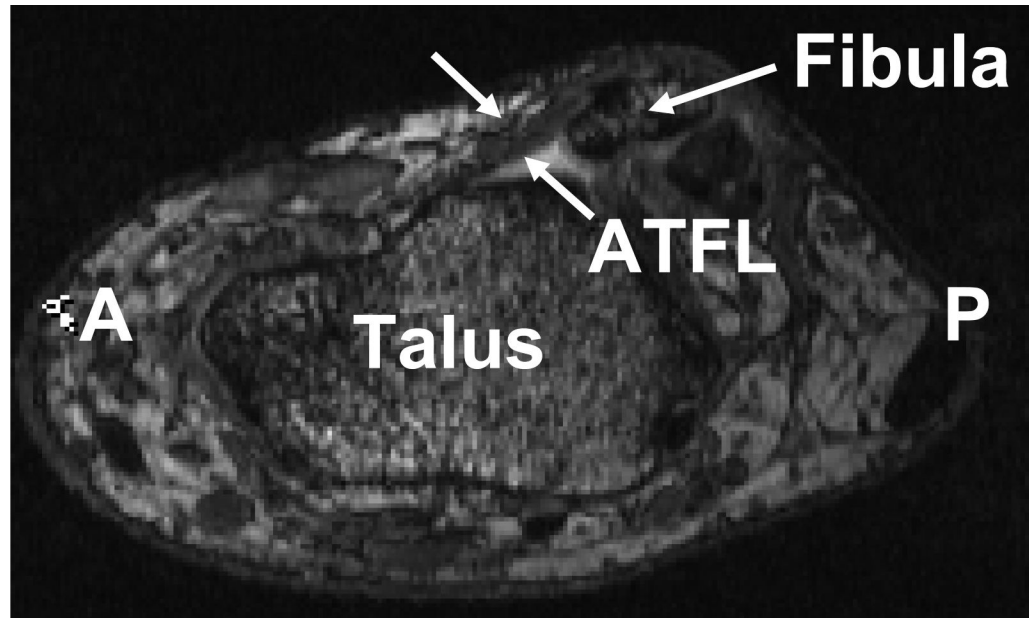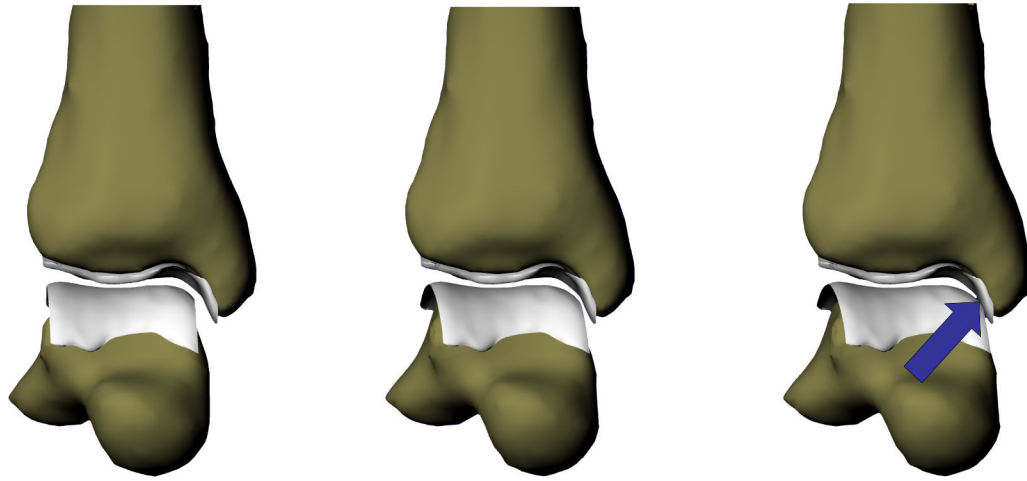
Figure 9.
The oblique orientation of the anterior talofibular ligament in the transverse plane suggests that it resists the anterior translation and internal rotation of the talus. (A = Anterior, P = Posterior)

Internal rotation of talus

Figure 10.
Increased internal rotation may lead to increased cartilage loading on the medial side of the talus, due to the curved surface of the cartilage on the medial portion of the tibia.