A Method to Induce Navicular-Cuneiform/Cuneiform-First Metatarsal Sprain in Athletes

Rebecca E. Frimenko, W. Brent Lievers, Patrick O. Riley, Jeff R. Crandall, Richard W. Kent

Abstract   Injury to the tarsometatarsal (TMT) joint is of concern to athletes, but the mechanism of injury remains unknown. This limits the design of effective countermeasures. As a step toward elucidating the mechanics of these injuries, this paper details an experimentally produced TMT sprain that disrupted the first ray at the distal end of the medial cuneiform with additional navicular-cuneiform instability. The sprain was generated through both axial load and rotation. A material testing machine was used to axially load the specimen quasi-statically, to maintain that load during testing, and to impart a pre-determined axial rotation (twist) to the foot. A foot fixture was designed to hold the specimen in plantarflexion with the toes in hyperextenion. Rotation was imposed such that the hind-foot was pronated with respect to the fore-foot. Four male, cadaver feet (65 – 79 years old) were tested at axial loads ranging from 500 N – 800 N with rotation from 10° – 45°. All four feet were examined for injury post-test by a surgeon. Instability was found in three of the four specimens along the first ray at the proximal and distal ends of the medial cuneiform. Peak loads of 795 – 1103 N and moments of 13 – 27 Nm were recorded.

Keywords   Mid-foot, sports injury, sprain, tarsometatarsal joint

I. INTRODUCTION

Injuries to the mid-foot, commonly called Lisfranc injuries, are associated with significant morbidity. Athletes who experience a low-grade Lisfranc sprain typically lose a month of playing time, and those requiring surgery spend at least six months out of play [1]. In extreme cases an athletic Lisfranc injury can even be career-ending [2]. Given their severity, understanding the mechanics of Lisfranc injuries is critical if interventions are to be designed which can protect athletes from these devastating injuries.

The name Lisfranc has been applied to various anatomic structures. For example, some sources refer to the entire tarsometatarsal (TMT) joint complex as the "Lisfranc joint" [3]. The term Lisfranc has also been applied to the ligament which runs from the medial cuneiform (C1) to the base of the second metatarsal (M2) or specifically to its interosseous bundle [4] – [5]. The imprecision of these eponymous terms can lead to confusion. Rather than engage in imprecise terminology, this paper will refer to joints and injuries by their anatomical location.

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Unfortunately, even this attempt at precision is insufficient because there are a number of different TMT injury patterns, of varying severity, resulting from different injury causes and mechanisms [6]. The most general dichotomy for classifying the mechanisms of TMT injuries distinguishes between those resulting from a direct, crushing force and those caused by an indirect force applied elsewhere to the lower extremity (Figure 1). Indirect injuries can be further subdivided into "high-energy" loading of the foot, such as might occur during an automotive crash, and "low-energy" loading typical of falls and athletic injury. Indirect twisting and axial loading of a plantarflexed foot are the two most prominent low-energy indirect mechanisms identified in case studies and retrospective analyses of athletic TMT injuries [1],[7]. This category of trauma may also be subdivided into injuries with osseous fractures, injuries which are purely ligamentous, or those that are a combination of both osseous and ligamentous injuries. As may be gathered from these divisions, different mechanisms and loading conditions generate a variety of injury patterns, all of which are associated with TMT injury. This study focused on athletic-type TMT sprains without any associated osseous fractures. Specifically, this paper describes TMT injury which disrupts the ligaments connecting the first metatarsal (M1) and C1 as well as the navicular (NAV) and C1 joints (NAV-C1/C1-M1).
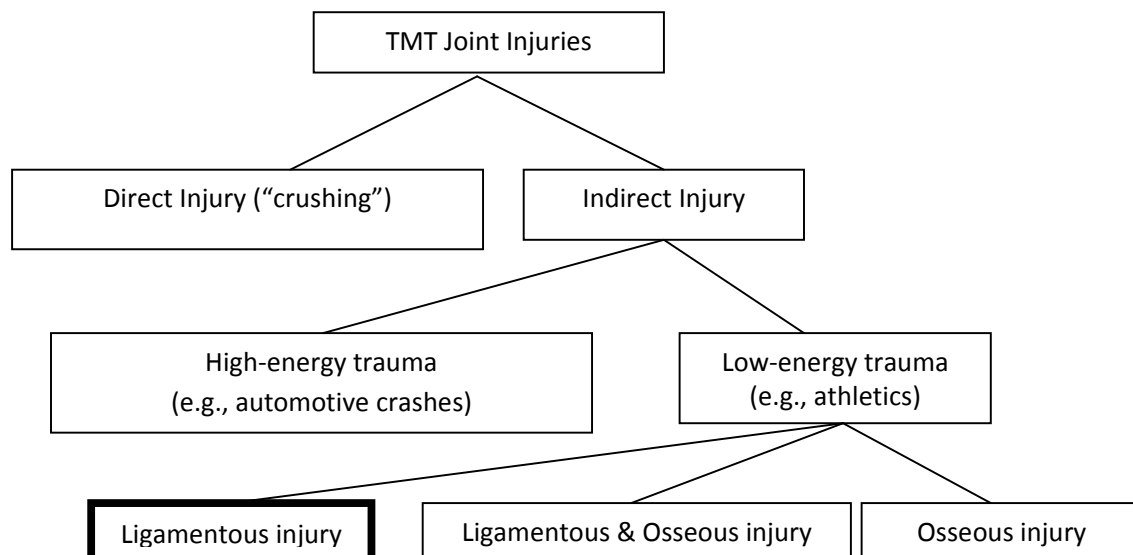

Figure 1 - Categorization for TMT sprains. This study examines athletic-type, low-energy, ligamentous injury.

As with any injury, before it can be prevented, the cause and mechanism of disruption must be understood. To date, many authors have proposed possible TMT injury mechanisms based on case studies and retrospective analyses of patients [1]-[3], [7], [10]-[12]. These anecdotal observations are supported by the qualitative results of manual manipulations performed on cadaver lower extremities [3], [9]. While a few researchers have quantitatively explored TMT injury, their studies have focused on the mechanisms and injury patterns which are typically seen in high-energy automotive crashes [13]-[15]. No studies to date have recorded engineering parameters while inducing athletic-type TMT injuries.

The mechanism of injury and the tolerance of the relevant anatomical structures must be known in order to develop interventions capable of preventing injury. To address this concern, this paper

presents a study which replicated an athletic-type first ray injury around the TMT joint. Specifically, this paper details a sprain of the NAV-C1 and C1-M1 joints. The method of load application, loading rates, and the resulting peak engineering parameters which occurred during testing are examined.

II. METHODS

Four lower extremities from two matched pairs were tested in accordance with the ethical guidelines established by the U.S. National Highway Traffic Safety Administration and with the approval of the University of Virginia Center for Applied Biomechanics Oversight Committee.

Test Fixture

Test conditions were guided by the previously mentioned case studies and retrospective analyses. From these, it was determined that axial loading and hind-foot pronation would be applied serially. Axial loading was applied quasi-statically prior to rotation. The peak rate of rotation was set at 150°/s, based upon an analysis of the performance data for elite athletes reported elsewhere [16]. A test fixture was designed for use in an Instron axial-torsion material testing system (Model 8874; Norwood, MA). The test fixture was designed in two parts: a mating assembly to apply load through the proximal end of the specimen and a platform on which the distal end of the specimen would rest (Figure 2 A).

The proximal assembly held the specimen to the Instron through bracing applied to the tibia and calcaneus. During specimen preparation, two threaded rods were drilled into these two bones in a medial-lateral orientation. These rods were secured to a mating fixture on the Instron, and load and rotation were applied through these rods during testing. A 3:1 planetary gear set linked the actuator piston of the Instron testing machine with the proximal fixture. The gear set was necessary to reach anticipated torque values based upon previous lower extremity work [17]. Without the gear set, the Instron testing machine had a range of 135°; the 3:1 gear ratio limited the rotation applied to the foot to 45°.

The distal fixture consisted of a platform to force the toes into 90° extension. The platform also had a low wall which contained two side-guides to align the foot during testing and a bar with which to load the second – fifth metatarsal heads. This bar was implemented to stress the ligaments between the first and second rays in order to create TMT ligamentous injury. Pipe clamps around the side guides and dorsal aspect of the foot were used to secure the foot to the distal testing platform (Figure 2 B, C).
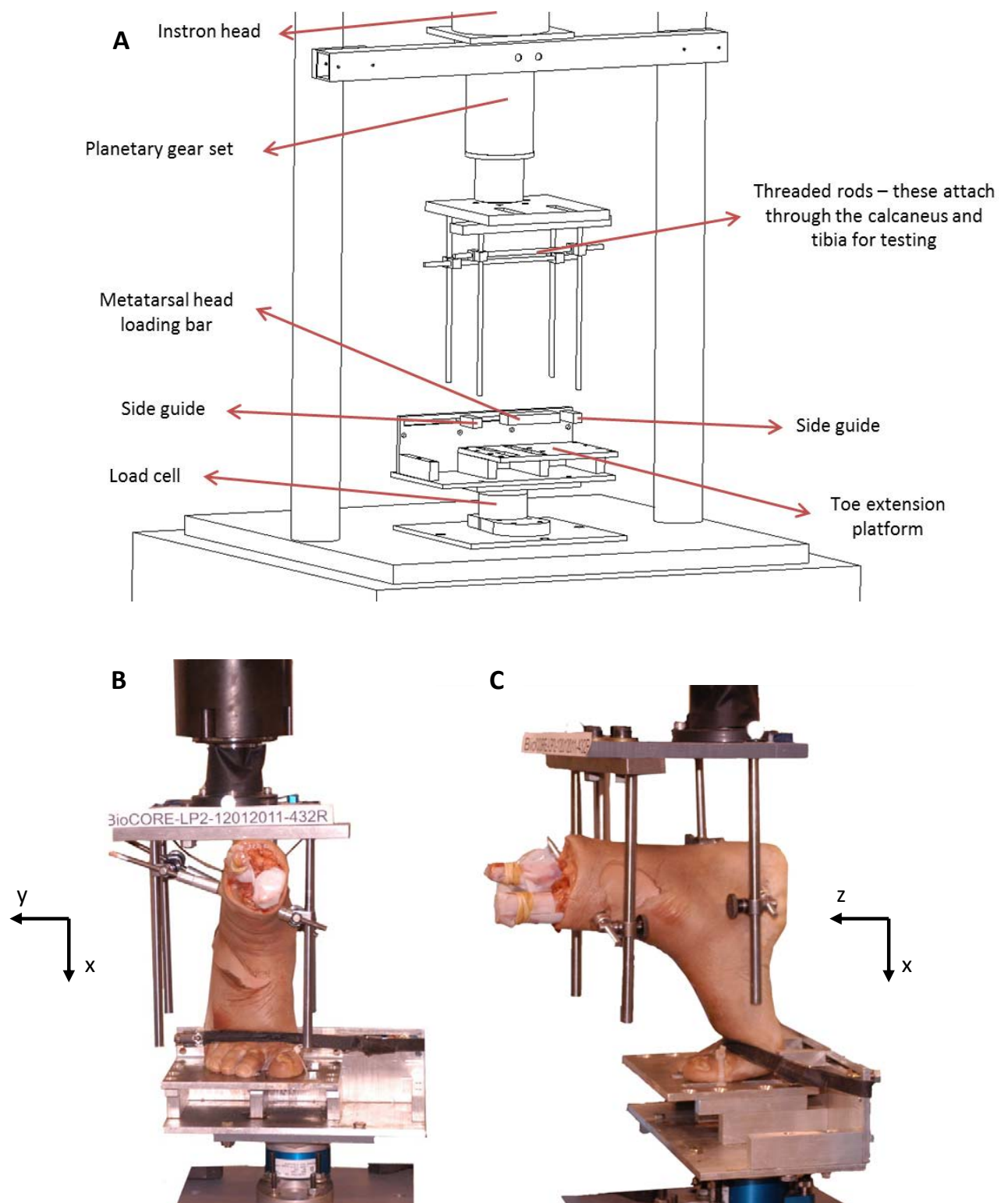
Figure 2 - Depictions of test fixture. A.) Schematic with labeled components. B.) Dorsal view of specimen loaded in test fixture immediately pre-test. C.) Medial view immediately pre-test.

A 13 kN six-axis load cell was fixed beneath the distal platform (Denton; Rochester Hills, MI, USA). An angular rate sensor and linear accelerometer were attached to the proximal assembly to provide information on rotation rate and axial position. Data were sampled at 10,000 Hz using a 16 channel data acquisition system (Dewetron Inc., Wakefield, RI).

The first two specimens were also fitted with motion capture markers (Vicon; Centennial, CO, USA) to record bone motion (Figure 3). Marker arrays, each consisting of four motion-capture markers, were rigidly attached to M1, C1 and second metatarsal (M2). Single markers were also attached with adhesive to both the stationary and moving parts of the test fixture. A recent assessment of the Vicon motion-capture system for high-rate events suggests that the accuracy of motion-capture system is less than half a millimeter [18]. The motion-capture system sampled data at 1,000 Hz.
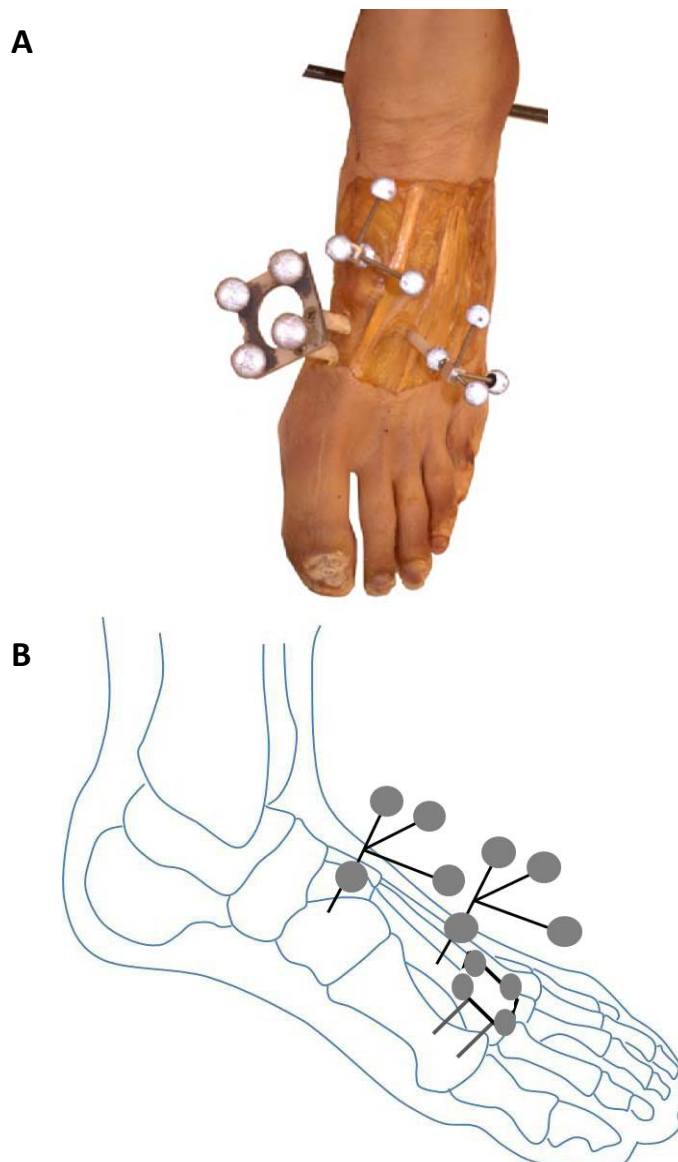

Figure 3 - Motion capture markers used during the first two tests. A.) Picture of arrays on the specimen during pre-test preparation. B.) Schematic of rigid attachment to the first metatarsal, first cuneiform and second metatarsal.

Specimen Preparation and Test Procedure

Pre-test preparation consisted of severing the specimen mid-tibia and inserting the threaded rods. A custom-made jig was used to ensure correct placement of the rods and to guide their medial-lateral orientation during drilling.   The Vicon marker arrays were also inserted during pre-test preparation. Both pre- and post-test CTs were taken to map the placement of the marker arrays relative to the bones.

Immediately prior to testing, the specimen was attached to the test fixture with the ankle in a slightly plantarflexed position, with the Instron axis of rotation located between the third and fourth rays. Placement was confirmed by visual inspection. A pre-determined axial load was applied, and this load was maintained throughout the test. The specimen was then rotated through 10, 20, 30, 40 and 45° of hind-foot pronation using a triangular waveform. Testing was discontinued before the final 45° test if force discontinuities became apparent in the data traces, if the foot showed obvious signs of deformity, or when the rotation limits of the Instron testing machine were reached.

Injury Diagnosis and Motion-Capture Analysis

Injuries were diagnosed through post-test necropsy by an experienced foot and ankle surgeon. Post-test CTs confirmed that bony fractures did not occur at either the motion-capture or threaded rod attachment sites.

To analyze the motion-capture data, the CT scan of each foot was segmented using Mimics software (Materialize; Leuven, Belgium). Coordinate systems were imposed on C1 and M1. The C1 z-axis was defined by the most distal junction of the medial and intermediate cuneiforms, the x-axis was perpendicular to the z-axis in the plane of the lateral side of the medial cuneiform, and the y-axis was mutually orthogonal.   The M1 x-axis ran parallel to the long axis of the metatarsal, the z-axis was perpendicular to the x-axis in a plane parallel to the lateral surface of the C1, and the y-axis was mutually orthogonal. Further coordinate transformations and analyses were conducted using Magics software (Materialize; Leuven, Belgium). Dorsal ligament origin and insertion are not well-defined for the TMT joint. As a surrogate to assess M1-C1 ligament extension, a ligament origin was created on the dorsal surface of C1 along the centerline of the bone at the most distal point. Similarly, a ligament insertion was created on the dorsal surface of M1 along the centerline of the bone at the most proximal point. Extension of the defined ligament was used to assess deformation and resulting injury.

III. RESULTS

Four specimens were tested under combined axial load and hind-foot pronation loading conditions. During testing, the Instron required approximately 0.016 s to reach a steady state angular velocity of 108°/s, with a peak rotation of 126°/s. Though less than the target rotation rate of 150°/s, the peak and steady state rotations were within the athletic range of loading and deemed acceptable by the testing group. Force discontinuities were not immediately apparent during review between testing trials. Two tests (1 L and 2 L) were halted due to gross collapse of the longitudinal arch, and the remaining two tests were performed to the maximum 45° of rotation.

Post-test necropsy revealed injury in three specimens (Table 1). Joint laxity was determined through qualitative evaluation by an experienced foot and ankle surgeon. First ray laxity was observed both proximal and distal to the medial cuneiform. No other abnormal ligament attenuation or laxity was

observed. No factures were apparent in any specimens through either the dissection or review of the CT results. Injury was not found at either the threaded rod or motion-capture array attachment sites.

Table 1 - Text matrix and specimen information.

Specimen Number	Age of Specimen (years at death)	Cause of death	Axial Pre-load (N)	Motion Capture?	Injury
1 L	79	Pneumonia	750	Yes	NAV-C1/C1-M1 laxity
2 L	65	Hemorrhagic stroke	500	Yes	NAV-C1/C1-M1 laxity
1 R	79	Pneumonia	500	No	NAV-C1/C1-M1 laxity
2 R	65	Hemorrhagic stroke	500	No	None

Further post-test review of the data traces did not reveal any discontinuities or indications of injury. Because of this, the injuries were identified as left-censored values, and peak values for force, moment and rotation angle are presented (

Table **2**). Maximum axial loads are higher than the initially applied preload. Though the Instron testing system was set to load control, feedback was not instantaneous. Because of this, peak loading varied from the initially applied load.

Table 2 - Maximum values recorded during testing.

Specimen Number	Maximum Axial Load (N)	Maximum Moment (Nm)	Maximum Rotation (deg)
1 L	860	13	10
2 L	795	27	42
1 R	838	27	45
2 R	1103	17	45

Motion-capture results of the test fixture reveal that 1 L rotated to a maximum of 10° while 2 L rotated to 42°. In light of the injury results, motion-capture description of ligament extension was calculated for M1 motion relative to the C1 (Figure **4**). Due to the limited rotation imposed on 1 L, only small displacements, less than 2 mm each, were seen in the motion-capture data. These results were not plotted due to small relative displacements. During 2 L rotation, a maximum ligament extension of 8 mm was recorded. Maximum ligament extension coincided with maximum rotation. Furthermore, although injury was noted at the NAV-C1 joint, motion for this joint is not available as the navicular was not equipped with a motion-capture array.
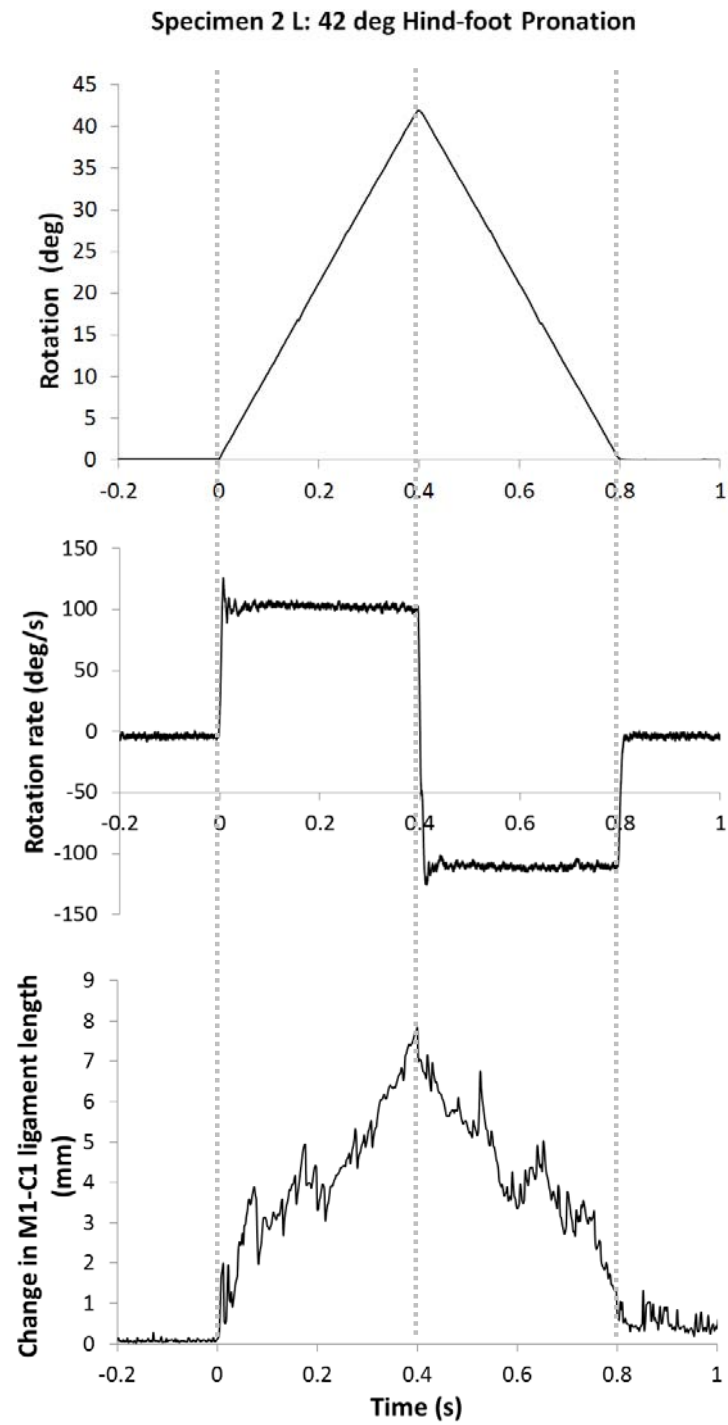
Figure 4 – Test results for Specimen 2L. *Top*: test fixture rotation as reported by the motion-capture system. M*iddle*: test fixture rotation rate as reported by the angular rate sensor. *Bottom*: change in M1-C1 ligament length during testing from motion-capture analysis.

IV. DISCUSSION

Mid-foot sprains are a debilitating athletic injury. This paper presents a method to induce purely ligamentous NAV-C1/C1-M1 sprains using an indirect, low-energy mechanism. The injuries created are relevant to athletics due to the loading rates during testing and the resulting purely ligamentous injury, consistent with most case studies of TMT sprain in athletes. Peak forces, moments and rotations are presented with injury results in order to guide further testing and fixture design.

As previously stated, from retrospective analyses of injury, axial load and a hind-foot pronation together were thought to produce TMT sprains in a low-energy scenario. Three of the four specimens tested in this low-energy loading condition resulted in TMT sprain. The results presented herein show that the M1-C1 ligament extended as much as 8 mm during rotation. Thus, the applied loading put the ligaments connecting the NAV-C1 and C1-M1 joints in tension, thus resulting in laxity and sprain.

Previous to this set of tests, forces and moments required to induce mid-foot sprains had never been recorded. As an initial estimate, anticipated values were taken from testing of the ankle and subtalar ligament in relation to low ankle sprain [17]. As a result, a planetary gear set was integrated with the test setup to ensure injurious levels of torque were produced, but which also limited the test's range of rotation. However, moments recorded during injury creation were less than one third of the anticipated values.

The injuries created during this study are referred to as athletic-type injuries due to the purely ligamentous injury and because the test conditions replicated the loading mechanism, loading rate, and boundary conditions observed in athletic performance. However, the specimens are not representative of a young, athletic population. Differences in age, osseous and soft-tissue geometry, and muscle activation will undoubtedly result in different injury thresholds for cadavers versus athletic, living subjects. Nevertheless, a cadaver-based injury threshold is expected to be more conservative than the threshold for living athletes, thus ensuring that the athlete is protected.

Though TMT sprains are often referred to as "Lisfranc injuries," this term is ambiguous due to the many anatomical structures and the spectrum of injuries – which vary in pattern and severity – associated with the name Lisfranc. The soft tissue laxity resulting from this study occurred around the first ray, both immediately proximal to and distal to C1. However, it should be noted that the C1-M2 ligament, the Lisfranc ligament, was intact in all specimens during post-test necropsy.

Information regarding athletic TMT injuries is largely derived from case studies and retrospective analyses of injury, either from athletes themselves or athletic trainers, physical therapists, or physicians. Unfortunately, these reports are often vague when describing the exact nature of Lisfranc injuries. This is likely due to the difficulty of imaging intra-TMT ligaments. Historically, unless the injured foot is examined surgically or has large diastasis due to gross ligament rupture, it has been difficult to document all the disrupted anatomic structures. As a result, the epidemiologic data regarding TMT injuries are limited and incomplete. Though the current body of knowledge shows long recovery times for mid-foot sprains in athletes, there is very little detail given as to the exact location and nature of these sprains. The injuries produced in this study are relevant to the study of athletic TMT dislocation; however, it is unknown exactly how often this particular TMT sprain occurs.

Each of the many joints of the foot has its own axis of rotation. The use of the Instron device for this test series imposed a fixed axis of rotation upon each foot and thus generated a kinematic behavior that is a simplification of motions that could occur during athletics. The axis was positioned laterally on the specimen to place greater stress along the first ray. Future research should explore the sensitivity of injury to variation in the imposed axis of rotation.

In all four tests, the foot was in either a neutral or plantarflexed position with respect to the tibia. Variability in ankle position (i.e. tibial attachment to Instron above or below the calcaneus attachment) was deemed acceptable because the calcaneus was fixed and load was applied distal to the ankle. Additionally, in normal gait, the windlass mechanism is engaged partly via activation of the triceps surae muscles and subsequent rotation of the calcaneus. The windlass mechanism raises the arch of the foot and helps to load the TMT joint [19]. In this test fixture, the calcaneus was fixed via the treaded rod attachment to the Instron, and the windlass mechanism was engaged though full extension of the phalanges.

Future work should focus on two areas of athletic-type TMT sprain: epidemiology and further experimental, *in vitro* creation of injury. Epidemiologic data specific to athletics are needed to identify the ligaments most commonly injured and the patterns of injury which require the longest recovery times. Player case studies and injury history data can then guide specific *in vitro* testing programs aimed at replicating the documented injuries. Particular attention should be given to the development of tests capable of rupturing the C1-M2 (Lisfranc) ligament.

V. CONCLUSIONS

The experimental results presented herein describe the loads, moments and angular displacements used to produce a ligamentous sprain of the navicular-first cuneiform and the first cuneiform-first metatarsal joints. This report represents the first phase of an on-going project aimed at developing a quantitative injury criterion which could be used by equipment manufacturers to design interventions aimed at reducing the incidence of Lisfranc injuries. The current results provide valuable initial estimates for loads and rotations needed to create other TMT sprains. Future work must examine additional testing conditions such as off-loading the first ray and varying forefoot abduction/adduction under axial load. Once the exact mechanisms of TMT sprains have been determined, quantitative testing results may be formulated into injury criteria and used to guide the design of protective sports equipment.

VI. ACKNOWLEDGEMENT

VII. REFERENCES

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