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Joint Stability Characteristics of the Ankle Complex in Female Athletes With Histories of Lateral Ankle Sprain, Part II: Clinical Experience Using Arthrometric Measurement

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Context: This is part II of a 2-part series discussing stability characteristics of the ankle complex. In part I, we used a cadaver model to examine the effects of sectioning the lateral ankle ligaments on anterior and inversion motion and stiffness of the ankle complex. In part II, we wanted to build on and apply these findings to the clinical assessment of ankle-complex motion and stiffness in a group of athletes with a history of unilateral ankle sprain.

Objective: To examine ankle-complex motion and stiffness in a group of athletes with reported history of lateral ankle sprain.

Design: Cross-sectional study.

Setting: University research laboratory.

Patients or Other Participants: Twenty-five female college athletes (age = 19.4 ± 1.4 years, height = 170.2 ± 7.4 cm, mass = 67.3 ± 10.0 kg) with histories of unilateral ankle sprain.

Intervention(s): All ankles underwent loading with an ankle arthrometer. Ankles were tested bilaterally.

Main Outcome Measure(s): The dependent variables were anterior displacement, anterior end-range stiffness, inversion rotation, and inversion end-range stiffness.

Results: Anterior displacement of the ankle complex did not differ between the uninjured and sprained ankles ($P = .37$), whereas ankle-complex rotation was greater for the sprained ankles ($P = .03$). The sprained ankles had less anterior and inversion end-range stiffness than the uninjured ankles ($P < .01$).

Conclusions: Changes in ankle-complex laxity and end-range stiffness were detected in ankles with histories of sprain. These results indicate the presence of altered mechanical characteristics in the soft tissues of the sprained ankles.

Key Words: ankle instability, joint laxity measurement, ankle sprains

Key Points

- Ankles with histories of lateral sprain showed more ankle-complex inversion rotation and less anterior and inversion stiffness than uninjured ankles.
- The mechanical property of stiffness might be important to understanding how lateral ankle sprain affects ligamentous elasticity and joint stability.
- These clinically important findings indicate that increased ankle-complex laxity is not the only identifiable mechanical tissue characteristic that changes after lateral ankle sprain.

Ankle sprain is one of the most common injuries encountered during sporting activity.[1] Lateral ankle sprain injury can result in changes to the ligaments and surrounding soft tissues that often lead to mechanical instability and functional insufficiencies.[2–7] Equally concerning is the recurrence rate after an initial sprain.[8] A search of epidemiologic and cohort studies identified *history of lateral ankle sprain* as a consistent risk factor associated with ankle sprain in sport.[8–12] Our understanding of the connection between history of ankle sprain and mechanical measures of ankle stability is unclear because not all ankles develop mechanical instability after 1 or more ankle sprains.[7,13]

Increased ligament laxity can result from a tear or lengthening of the involved ligamentous structures supporting the joint or less-than-optimal healing of the injured tissues.[2] Individuals with histories of ankle sprain present with increased joint laxity and persistent symptoms, such as the feeling of or actual giving way of the ankle during jumping and cutting activities.[14–16] However, some authors have not reported findings of increased laxity in the sprained ankles despite the presence of functional insufficiencies, such as impaired proprioception, altered neuromuscular control, strength deficits, and diminished postural control.[6,17]

The passive stiffness characteristics of a joint are created in part by the viscoelastic properties of the soft tissues that

surround and support the joint.[18] Leardini et al[19] reported that passive stiffness provided by the soft tissue structures is a vital component of joint stability. Thus, the mechanical property of stiffness may be important to understanding joint stability after injury. Only Wikstrom et al[20] have investigated passive ankle-joint stiffness in people who reported experiencing ankle sprains. They found no differences in anterior laxity or anterior stiffness of the ankle between individuals with or without reported functional ankle instability. In a later study, Wikstrom et al[7] reported that patients who had histories of ankle sprain and presented with no signs or symptoms of chronic ankle instability (CAI) and patients with CAI had increased anterior ankle-joint stiffness relative to uninjured control participants. When jointly examined, these previous reports appear specious because laxity and stiffness are inversely related. We wanted to build on the work of Wikstrom et al[7,20] and also examine the effects of previous lateral ankle sprain on inversion ankle-complex motion and stiffness. Therefore, the purpose of our clinically based study was to determine ankle-complex motion and stiffness in a group of athletes with a reported history of lateral ankle sprain. We hypothesized that ankles with histories of lateral sprain would demonstrate altered motion and stiffness characteristics when compared with the uninjured ankles.

METHODS

Design

We conducted a cross-sectional study to investigate individuals with histories of unilateral ankle sprain and to compare uninjured and sprained ankles using measures of ankle-complex motion and stiffness.

Participants

Fifty-three female National Collegiate Athletic Association volleyball, basketball, or soccer athletes completed an ankle-sprain injury questionnaire to assess eligibility for the study. Twenty-five athletes (age = 19.4 ± 1.4 years, height = 170.2 ± 7.4 cm, mass = 67.3 ± 10.1 kg) were identified and included because they reported experiencing at least 1 unilateral ankle sprain (15 right ankles, 10 left ankles) within the 24 months before the study. A volunteer was excluded if she reported any surgery or fracture to either ankle or a history of bilateral ankle sprains.

The average number of sprains reported per injured ankle was 2.44 ± 1.2 (range, 1–5). No incidence of initial or recurrent sprain was reported within the month before testing. Within 1 to 3 months before testing, 1 sprain was reported; 3 to 6 months, 3 sprains; 6 to 12 months, 7 sprains; and 12 to 24 months, 14 sprains. A total of 16 participants (64%) reported no pain in their sprained ankles with activity, 5 (20%) had minor pain after strenuous activity, and 4 (16%) reported moderate to substantial pain after strenuous activity. All ankles were asymptomatic for pain during the arthrometric testing. All athletes reported experiencing episodes of the ankle giving way or feelings of instability in the period between their initial injuries and arthrometric testing. All athletes participated in their respective sports at the time of testing, with 20 of the 25 athletes (80%) reporting wearing braces or tape support. Each participant provided written informed consent, and the study was approved by the Institutional Review Board of the University of South Alabama.

Ankle Arthrometer

Ankle-complex loading was performed with the Hollis instrumented ankle arthrometer (Blue Bay Research Inc, Navarre, FL; see Figure, p 194 of this issue).[21] The ankle arthrometer has been reported to be highly reliable for the examiner and a valid tool for assessing ankle ligamentous stability.[21–23]

Procedure

Athletes participated in 1 testing session, at which time ankle-displacement and -rotation measurements were obtained. Testing involving participant and ankle positioning replicated previously reported procedures.[21,24] The order of testing was randomly assigned between ankles, and 1 examiner (J.E.K.), who was blinded to the affected ankle, obtained all measurements.

The ankles were positioned in 10° of plantar flexion to permit isolation of the ankle capsuloligamentous structures and to reduce the influence of calf musculature tension on the ankle complex.[24] This *flexion angle* was defined as the measurement reference position. For AP displacement, the ankles were loaded manually to 100 N of anterior and posterior force. Starting at the neutral position, an anterior load was applied initially, followed by a posterior load.[24] The 100-N loading was selected as the standard test force to ensure that the magnitude of loading was both sufficient to detect joint laxity and tolerated by the participants.[21,24,25] For inversion-eversion rotation, the ankles were loaded manually to 4 N·m with inversion and eversion torque at 10° of plantar flexion. Starting at the neutral position, inversion loading was applied, followed by eversion loading.[21]

Data Reduction

Anterior displacement (millimeters) at the 100-N force load was recorded and defined as anterior motion. *Inversion rotation* (degree of range of motion [ROM]) at 4 N·m was recorded and defined as inversion motion. As the magnitude of the applied load increased beyond 50% of the force-displacement curve, the ankle complex displayed a linear increase in stiffness. To measure stiffness in this range, the data were plotted as applied load versus displacement and rotation. *Anterior end-range stiffness* was defined as force per displacement (newtons per millimeter) and was calculated by dividing 50 N (load difference between 50 and 100 N) by the anterior displacement between the 50- and 100-N force loads.[24] *Inversion end-range stiffness* was defined as torque (newton·meters) per degree of ROM and was calculated by dividing 2 N·m of torque (torque difference between 2 and 4 N·m) by the inversion rotation between the 2- and 4-N·m torque loads.

Statistical Analysis

We used 2-tailed dependent *t* tests to examine differences in joint motion and end-range stiffness between ankles (uninjured and sprained). The dependent variables were anterior displacement, anterior end-range stiffness, inver-

sion rotation, and inversion end-range stiffness (N·m per degree of ROM). The α level was set a priori at .05 for all analyses. The Cohen d, using pooled standard deviations, was calculated to determine effect size. The strength of the effect size was determined as small (0.20), medium (0.50), or large (0.80).[26] All statistical comparisons were performed with SPSS (version 18.0; SPSS Inc, Chicago, IL).

RESULTS

For the ankle-complex–motion measures, anterior displacement did not differ between sprained and uninjured ankles ($t_{24} = 0.916$, $P = .37$, Cohen d = 0.27). However, the sprained ankles displayed greater inversion rotation than the uninjured ankles ($t_{24} = 2.37$, $P = .03$, Cohen d = 0.77). For the ankle-complex–stiffness measures, the sprained ankles displayed less anterior end-range stiffness ($t_{24} = 3.14$, $P = .004$, Cohen d = 0.64) and inversion end-range stiffness ($t_{24} = 2.67$, $P = .01$, Cohen d = 0.54) than the uninjured ankles (Table).

DISCUSSION

The ankles with histories of lateral ankle sprain showed altered load-displacement characteristics. We hypothesized that when compared with uninjured ankles, ankles with histories of sprain would demonstrate increased anterior displacement and less end-range stiffness when loaded in anterior drawer and increased rotation and less end-range stiffness when loaded in inversion. Our findings showed that the sprained ankles demonstrated greater inversion rotation, decreased anterior end-range stiffness, and decreased inversion end-range stiffness. Anterior displacement was only 0.54 mm greater in the sprained ankles than in the uninjured ankles. This lack of increase indicates that the anterior talofibular ligament (ATFL) supporting the anterior aspect of the joint may not have been damaged enough to cause increased laxity and likely maintained near-normal length at the time of testing. It is also possible that at the time the sprain or sprains occurred, the ATFL was not completely torn (ie, none of the athletes reported being informed by a physician or athletic trainer that her ankle sprain resulted in a ligament being completely torn). Despite the absence of increased anterior laxity in the ankles we studied, anterior drawer testing to identify ankle-complex laxity is considered important not only at the time the injury occurs but also throughout the follow-up period. Increased anterior laxity has been found immediately after lateral ankle sprains and in 3% to 31% of participants 6 months to 1 year after injury.[3,4,13] This illustrates the importance of objective assessment of ankle-complex laxity immediately after an ankle sprain and for at least 1 year after injury so clinicians can know how long to immobilize, protect, and rehabilitate a sprained ankle.

Observed differences in ankle-complex stiffness after sprain could be due to changes in joint arthrokinematics, capsular adhesions, limited talar mobility, or increased muscle tone. All are possible factors for increased stiffness with or without changes in ligament laxity.[7,19,20,27] Wikstrom et al[7] found increased anterior stiffness in both individuals with ankle sprains but without CAI (14.98 ± 2.0 N/mm) and individuals with CAI (14.95 ± 2.04 N/mm) relative to an uninjured control group (14.01 ± 2.6 N/mm). Wikstrom et al[20] also reported no difference in anterior stiffness between control participants (11.8 ± 1.9 N/mm) and individuals with reported functional ankle instability (12.8 ± 3.8 N/mm). In addition, they found no group difference for anterior laxity (mean values = 5.9–6.2 mm).[20] In contrast, the sprained ankles we studied showed low anterior end-range stiffness (14.95 ± 6.4 versus 19.85 ± 8.8 N/mm) along with no change in anterior laxity (mean values = 6.74–7.28 mm) when compared with the uninjured ankles. We also observed less inversion end-range stiffness in the sprained (0.148 ± 0.03 N·m/° ROM) than uninjured (0.175 ± 0.06 N·m/° ROM) ankles along with greater inversion rotation in the sprained (33.12° ± 6.7°) than uninjured (28.21° ± 6.1°) ankles.

The increased inversion rotation, decreased anterior end-range stiffness, and decreased inversion end-range stiffness observed in the sprained ankles may have been caused by improper ligament healing or by the ligaments' healing in an elongated state.[3,13,28] A reason for the decreased stiffness observed in the sprained ankles cannot be determined directly from our findings, but initial tissue damage and associated composition and morphologic changes subsequent to the injury may have contributed to the stiffness differences observed. Researchers[29] have shown that torn ligaments do not recover completely and that healing tissue must stretch farther before all disorganized fibers strengthen and bear load. It is not clear why anterior end-range stiffness was less when anterior laxity was not increased in the sprained ankles. Whereas overall anteroposterior laxity was maintained over a load range, end-range stiffness possibly decreased partly because of a shift in the starting point of the load-displacement curve caused by elimination of the toe or low-stiffness region.[29] This indicates that the injured ankles responded to loading in a reproducible manner, with modification of the soft tissue structures likely occurring and leading to the observed changes in the biomechanical and microstructural behavior of the ankle complex.

Lack of appropriate postinjury treatment and rehabilitation, along with increased mechanical stress placed on the soft tissues of the ankle due to participating in physical

Table. Summary of Ankle-Complex–Motion and –Stiffness Values for Sprained and Uninjured Ankles (Mean ± SD)

	Anterior		Inversion	
Ankle Condition	Displacement, mm	End-Range Stiffness, N/mm	Rotation, ° Range Of Motion	End-Range Stiffness, N·m/° Range of Motion
Sprained	7.28 ± 1.9	14.95 ± 6.4[a]	33.12 ± 6.7[b]	0.148 ± 0.03[c]
Uninjured	6.74 ± 2.0	19.85 ± 8.8	28.21 ± 6.1	0.175 ± 0.06

[a] Indicates reduced compared with uninjured ankle ($P = .004$).
[b] Indicates increased compared with uninjured ankle ($P = .03$).
[c] Indicates reduced compared with uninjured ankle ($P = .01$).

activity, could negatively affect the biophysical-strength properties of the ligaments and secondary constraints of the joint.[19,30] These changes could explain the increased inversion laxity and lowered anterior and inversion end-range stiffness values observed in the injured ankles. Given the cross-sectional design of this study, we do not know whether the observed changes in the mechanical characteristics of the ankle complex were due to the initial sprain or a recurrent sprain. We also could not determine from our data whether these differences were predisposing factors to recurrent lateral ankle sprain.

Researchers[28,31,32] have shown a general consensus to manage grade I and II lateral ankle ligamentous injuries with functional modalities, such as flexibility exercises, strength and balance training, ankle-joint proprioception, and muscular strength training. Kerkhoffs et al[33] conducted a systematic review of acute ankle ligamentous sprain management and reported that functional exercise was more effective than immobilization for reducing persistent swelling, restoring ROM, restoring ankle stability, and returning to sport participation. In contrast, O'Driscoll and Delahunt[34] conducted a systematic review and reported limited to moderate effectiveness of neuromuscular training to enhance sensorimotor and functional deficits in individuals with CAI. Conflicting findings such as these indicate a need to prospectively study the clinical management of ankle sprain to determine if the benefits of immediate postinjury and postexercise interventions are maintained and whether a carryover to long-term changes in laxity and stiffness exists. If ligaments do not heal and mechanical maladaptations develop, then alterations in the passive tension (elasticity) characteristics of the soft tissues surrounding the joint could develop. These sequelae could progress to the development of other ankle conditions, such as CAI, which is sometimes observed in individuals with histories of lateral ankle sprain.[13,16]

Authors of other systematic reviews and meta-analyses have reported on the associations among mechanical joint laxity, history of ankle sprain, and CAI. Cordova et al[4] examined the association between mechanical ankle laxity and CAI and showed that the influence of CAI on anterior drawer and inversion laxity was consistent with the primary mechanism of injury. They identified small to very large effect sizes for anterior and inversion joint laxity in patients with CAI when compared with healthy control participants. Cordova et al[4] reported that the greatest influence was with inversion laxity. This finding is not surprising given the high incidence and persistent effects of recurrent lateral inversion ankle sprains[2–5,14–17] and given that individuals with histories of ankle sprain are more likely to exhibit increased laxity, especially in inversion.[8–12] Normative data comparisons in the literature for inversion ankle-complex motion have shown that the uninjured ankles we studied (inversion ROM = 28.21° ± 6.1°) were within the normal mobility (–1 to +1 SD range, 19.28°–31.70°) reference range.[25] However, inversion ROM (33.12° ± 6.7°) of the sprained ankles was within the hypermobility (+1 to +2 SD range, 31.71°–37.91°) reference range of ankle-complex motion.[25] If return to activity is permitted before a ligament is fully healed, the ligament could heal in an altered or elongated state, resulting in greater laxity or lowered stiffness, such as observed in the athletes we studied. Given the high percentage of recurrence and chronic symptoms (eg, ankle osteoarthritis after ankle sprain), longitudinal studies must be developed to examine the effects of initial treatment and rehabilitation in restoring joint stability.[35]

The presence of mechanical ankle instability and functional ankle instability has been reported in individuals with CAI symptoms.[5,14,17,35] Mechanical ankle insufficiencies include pathologic laxity, impaired arthrokinematics, and synovial and degenerative changes.[16] Functional ankle insufficiencies include impaired proprioception, strength deficits, and altered neuromuscular control.[16] Athletes with mechanical instability may not always present clinical symptoms because their neuromotor capabilities can provide the necessary supportive restraint, especially in the presence of near-normal ankle ROM and stiffness.[7,20,25] In contrast, pain resulting from other conditions caused by anatomic changes around the foot and ankle can produce symptoms similar to functional instability in patients without mechanical laxity.[16] Therefore, one must maintain a high index of suspicion for associated injuries when evaluating these patients, as this will affect treatment recommendations.[29] Developing a better understanding of the effects of ankle sprain on joint mechanical characteristics is important because poorly managed ankle sprains may lead to substantial and disabling long-term problems, including instability and functional deficits associated with recurrent ankle sprain.[4–7,14,17,20,36,37] This could lead to other, as yet unknown, sequelae that may affect healing, recovery, and function.

The literature is unclear about why the percentage of reinjury in athletes with histories of lateral ankle sprain is so high.[8] In addition, the relationships among premature return to sport participation; the degree of healing; and vulnerability to developing long-term disabling effects, such as ankle osteoarthritis, are generally unknown.[34] Researchers should prospectively assess the influence of documented injury and instability on the mechanical characteristics of the ankle–subtalar-joint complex. If clinicians understand more about ankle instability on a mechanical basis, they can implement new and better techniques for evaluating injury.[36,37] In addition, rehabilitation programs could be improved to restore joint stability using enhanced techniques and by providing therapeutic exercise regimens that restore joint ROM while promoting passive and dynamic joint stability, muscle strength, and sensorimotor control.

A limitation of this study includes the participants' self-reporting the number of ankle sprains in the 24 months before data collection. Knowing that information about the injury would rely on participant recall with no additional documentation, we attempted to reduce the effect of this limitation by restricting the initial injury recall to the 24 months before completing the survey and testing. We could not obtain medical documentation of sprain severity for the initial or any subsequent sprains. In addition, no information was obtained from the participants about the type and length of immediate care or rehabilitation after any of the reported ankle sprains. The athletes we studied were cleared medically and were participating in their respective sports at the time of testing. We did not survey the athletes about their postinjury care or rehabilitation, so these are unknown. They reported experiencing at least 1 episode of the injured ankle's giving way or having a feeling of giving way in the period between their injuries and the

arthrometric testing. In addition, most athletes reported wearing ankle tape or bracing during athletic participation to support their ankles.

Despite including only women athletes in our study, we believed that the normal hormone fluctuations that occur during the menstrual cycle did not affect the ligamentous laxity measurements in the women we tested. Beynnon et al[38] found that cyclic estradiol and progesterone fluctuations that occur during the menstrual cycle did not produce cyclic fluctuations in ankle- or knee-joint laxity and concluded that investigators using joint laxity to identify a participant at risk for ligamentous injury only need to consider making measurements at a specific point, such as during a preseason screening evaluation, which is what we did. More recently, Ericksen and Gribble[39] examined potential hormone contributions to ankle laxity and postural control and reported that hormonal fluctuations during the menstrual cycle did not affect laxity or dynamic postural control, which are 2 factors associated with ankle instability.

Differences in our findings and those reported by others may be explained by the experimental setup and type of test device used to load the ankle. Given that ankle motion and stiffness are altered by the ankle-flexion angle and load range, joint position and loading need to be considered when comparing results from different studies.[24] Finally, during testing, we tried to ensure muscle relaxation by supporting the leg and ankle so muscle force was not required to maintain the desired joint angle.

CONCLUSIONS

Ankles with histories of lateral sprain showed more ankle-complex inversion rotation and less anterior and inversion end-range stiffness than uninjured ankles. The mechanical property of stiffness appears important to understanding how lateral ankle sprain affects ligamentous elasticity and joint stability. Our findings are clinically important because they indicate that increased ankle-complex laxity is not the only identifiable mechanical tissue characteristic that changes after lateral ankle sprain.

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