

Ankle Bracing and the Neuromuscular Factors Influencing Joint Stiffness

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Context: Health care professionals commonly prescribe external stabilization to decrease the incidence and severity of ankle sprains. The mechanism for this decrease is not clearly understood. Examining the effects of ankle bracing on biomechanical stability and influencing factors may provide important information regarding the neuromuscular effects of bracing.

Objective: To study the effects of 2 different ankle braces on the neuromuscular factors influencing ankle stiffness.

Design: Mixed-model repeated-measures design.

Setting: Research laboratory.

Patients or Other Participants: Twenty-eight physically active participants composing 2 groups: 14 with unilateral functional ankle instability (age = 26.19 ± 6.46 years, height = 166.07 ± 12.90 cm, mass = 69.90 ± 13.46 kg) and 14 with bilaterally stable ankles (age = 23.76 ± 5.82 years, height = 174.00 ± 11.67 cm, mass = 68.60 ± 13.12 kg).

Intervention(s): Participants were fitted with surface electromyography electrodes over the peroneus longus, peroneus brevis, tibialis anterior, and soleus muscles. Each participant

received transient motion oscillations to his or her ankle on a custom-built medial-lateral swaying cradle in each of 3 conditions: no ankle brace (NB), lace-up brace (LU), and semirigid brace (SR).

Main Outcome Measure(s): Ankle stiffness as measured by the cradle and preactivation levels (percentage of maximal voluntary isometric contraction) of the 4 test muscles.

Results: Stiffness levels increased across brace conditions (NB = 24.79 ± 6.59 Nm/rad, LU = 28.29 ± 7.05 Nm/rad, SR = 33.22 ± 8.78 Nm/rad; $F_{2,52} = 66.185$, $P < .001$). No differences were found between groups for rotational stiffness (stable = 27.36 ± 6.17 Nm/rad, unstable = 30.18 ± 8.21 Nm/rad; $F_{1,26} = 1.084$, $P = .307$). Preactivation levels did not change for any of the tested muscles with the application of an ankle brace ($F_{2,52} = 1.326$, $P = .275$).

Conclusions: The increase in ankle rotational stiffness with the addition of an ankle brace and the lack of any demonstrable neuromuscular changes suggested ankle braces passively contributed to the stability of the system.

Key Words: stability, preactivation, reflexes, orthoses

Key Points

- As measured via transient oscillation, ankle rotational stiffness values were not different for participants with functional ankle instability compared with participants with stable ankles.
- In nearly every situation, ankle braces did not cause changes in neuromuscular attributes.
- Ankle braces primarily contributed to the stability of the ankle complex through passive, mechanical means.

Ankle sprains are common, are costly, and can result long-term disability.^{1–5} Freeman et al⁶ hypothesized that after a lateral ankle sprain, the afferent information generated by the mechanoreceptors in the joint and surrounding musculature is disrupted, causing functional instability. This deafferentation of the joint contributes to altered neuromuscular activity and decreased biomechanical stability that often leads to further joint injury.^{7,8} Bergmark⁹ theorized that this decrease in stability is a primary cause of musculoskeletal injury. Because patients lose this biomechanical stability with injury, medical professionals commonly implement external stabilization to decrease the incidence and severity of ankle sprains.^{10,11} The mechanism by which ankle braces reduce the incidence and severity of injury is unknown. The added mechanical benefits of external stabilizers are well documented, but their effects on the neuromuscular properties of the joint are not clearly understood.

Researchers have suggested that ankle braces decrease range of motion,^{12,13} increase mechanical stability,^{11,14} and enhance proprioception^{15,16}; however, the effects of ankle bracing on foot and ankle dynamics have received less attention. Most research in this area has been centered on improvements in postural stability^{17–20} and attenuation of ground reaction forces.^{21,22} To our knowledge, no researchers have investigated the influence of ankle braces on ankle rotational stiffness, a biomechanical property that may help limit injurious ankle joint translations. An understanding of this influence is important, as stiffness has been shown to be a vital component of joint stability.^{9,23}

Joint stability is affected by several factors, including proprioception and muscle stiffness. Proprioceptive input from the joint provides feedback to the central nervous system, allowing it to modify the efferent output to adjust to the changing demands of the system.²⁴ The disruption of afferent information after a lateral ankle sprain interferes

with signals to the central nervous system, causing altered efferent responses and decreased biomechanical stability.⁶ *Muscle stiffness* has been described as the ratio of force response that results from and resists mechanical stretch.²⁵ The stiffness of the passive structures surrounding a joint contributes little to its biomechanical stability except at the end ranges.^{26,27} However, researchers^{28,29} have shown that the active stiffness properties of the muscles are essential to maintaining dynamic stability. Altered afferent information after an ankle sprain may interfere with the musculoskeletal system's ability to adequately stiffen and provide stability to the joint after a perturbation. Examining the influence of ankle braces on biomechanical stability may provide important information regarding the neuromuscular effects of bracing. Therefore, the purpose of our investigation was to study the effect of different ankle brace conditions on ankle stiffness. We hypothesized that participants with functional ankle instability would exhibit diminished rotational stiffness compared with participants with stable ankles and that the addition of an ankle brace would increase that stiffness.

METHODS

Participants

Participants included 28 physically active volunteers. The 14 participants with unilateral functional ankle instability (FAI) (age = 26.19 ± 6.46 years, height = 166.07 ± 12.90 cm, mass = 69.90 ± 13.46 kg) were assigned to the unstable group, and the 14 participants with bilaterally stable ankles (age = 23.76 ± 5.82 years, height = 174.00 ± 11.67 cm, mass = 68.60 ± 13.12 kg) were assigned to the stable group. Group assignment was determined by using the Ankle Instability Instrument oral questionnaire (score range, 1–12).³⁰ Participants who scored 5 or more on the survey and had a minimum of 1 giving-way episode per month were considered to have FAI and were assigned to the unstable group. No participant had a history of ankle disorders requiring treatment by a health care provider within 6 months before the study, had a known neuromuscular dysfunction, or was taking medication that would alter neuromuscular control at the time of the study. All participants gave written informed consent, and the study was approved by the Human Investigations Committee of the University of Virginia.

Instrumentation

Medial-Lateral Swaying Cradle. We designed and constructed a medial-lateral swaying cradle device (Figure 1) to measure ankle stiffness in participants who were administered transient motion oscillations. The device was designed to allow the participant to maintain a seated posture with hips and ankles at 90° while the ankle received a perturbation externally in an inversion-eversion direction with the axis of motion aligned with that of the subtalar joint. The perturbations were achieved by dropping a weighted ball onto the “wing” on the side of the cradle corresponding with the lateral side of the ankle. The energy of the perturbation was kept constant by dropping the ball from the same height (110 cm) each time. Reliability and validity information for the device was presented by Zinder et al.³¹ The intraclass correlation

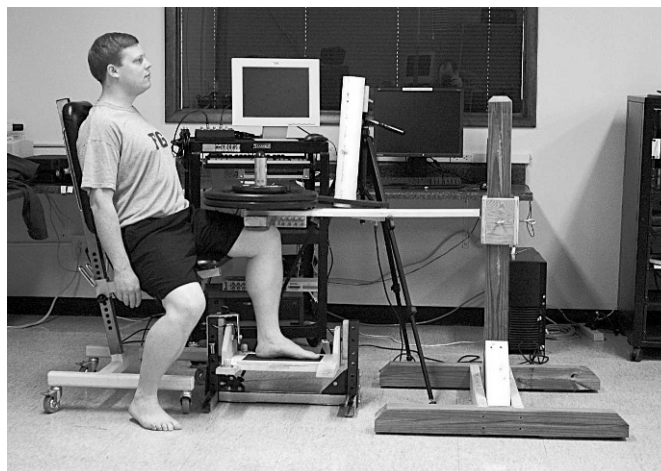


Figure 1. Experimental set-up.

coefficient (ICC) (2,1) for trial-to-trial reliability was 0.96 with a standard error of measurement (SEM) of 2.05 Nm/rad, and the ICC (2,k) for day-to-day reliability was 0.93 with an SEM of 3.00 Nm/rad. Cradle angular displacement was recorded with a 270° single-turn potentiometer (Clarostat, Mexico City, Mexico) that was aligned with the cradle's axis of rotation.

Surface Electromyography. An 8-channel telemetric electromyography (EMG) system (TeleMyo 900; Noraxon USA Inc, Scottsdale, AZ) was used to record muscle preactivation. Unit specifications included an amplifier gain of 1 mV/V, a frequency bandwidth of 16 Hz to 500 Hz, common mode rejection ratio of 114 dB, and input resistance from 20 M Ω to 1 G Ω . Bipolar Ag/AgCl surface electrodes (Medicotest, Rolling Meadows, IL) measuring 10 mm in diameter with a center-to-center distance of approximately 2.0 cm were arranged parallel over the muscle bellies of the peroneus longus (PL), peroneus brevis (PB), tibialis anterior (TA), and soleus (SO). Muscle activity was collected from surface electrodes via a battery-operated FM transmitter/amplifier (Noraxon USA Inc) that each participant wore. From the transmitter, the signal was telemetered to the computer in which the raw EMG data were sampled at 1000 Hz and stored for analysis.

All data were sampled at 1000 Hz, acquired using DATAPAC III (version 2000 Lab Application Systems software; RUN Technologies, Mission Viejo, CA), and stored on a personal computer for analysis. All data were analyzed using MATLAB (version 7.2.0; The MathWorks, Natick, MA).

Procedures

We explained the entire testing protocol and gave participants the opportunity to question the examiner. Participants were fitted with the EMG electrodes and performed the maximal repetitions. Each participant was assigned a random order of test conditions for data collection.

Surface Electromyography Electrode Placement

Each participant's skin was shaved, cleaned with isopropyl alcohol, and lightly abraded with sandpaper

before application of surface electrodes. The PL electrodes were placed 3 fingerbreadths below and 1 fingerbreadth anterior to the fibular head, which corresponded with the junction of the proximal-third and middle-third of the fibula. The PB electrodes were placed 1 fingerbreadth anterior to the junction of the middle-third and distal-third of the fibula. The TA electrodes were placed 1 fingerbreadth lateral to the anterior ridge of the tibia over the thickest portion of the muscle belly. The SO electrodes were placed posterior to the peroneal tendons just distal to the junction of the middle-third and distal-third of the fibula. All electrode placements were performed by the principal investigator and were confirmed through manual muscle testing for each muscle with observation of real-time EMG. Surface electrodes and associated wires were further secured using an elastic bandage to prevent cable tensioning and movement artifact during the experimental tasks. When the surface electrodes were placed properly, the participant performed maximal voluntary isometric contractions (MVICs) for each test muscle. Electromyographic data collected during MVICs were used to normalize muscle preactivation values during the perturbations. Thus, these measures were expressed as a percentage of MVIC.

Transient Oscillation Perturbations

Inversion-eversion ankle stiffness was assessed across 3 brace conditions while participants received transient motion oscillations to their ankles. Brace conditions consisted of the application of no brace, a nonrigid lace-up brace (Ankle Lok; Swede-O Inc, North Branch, MN), and a semirigid brace (Aircast Air-Stirrup; DJO Inc, Vista, CA). The order of brace condition was randomized. Five transient oscillation perturbations were conducted in each condition. A real-time display of cradle platform position was used to ensure that the cradle was in a neutral starting position before each perturbation.

Participants were seated with their eyes closed, hips and knees flexed to 90°, and ankles in a neutral position. A load equal to 50% of body mass was placed on a pad directly above the knee along the longitudinal axis of the tibia (Figure 1). In previous studies in our laboratory, participants were positioned in a bipedal upright posture with their weight evenly distributed on each leg and their feet approximately 30 cm apart. A pilot study of 12 participants showed no difference in stiffness values between bipedal standing and seated posture with the load of 50% of body mass ($F_{1,10} = 0.343$, $P = .571$, $1-\beta = .083$). The 2 positions had a Pearson product moment correlation of 0.841 ($P < .001$) and an R^2 value of 0.707 for rotational stiffness. We chose the seated position for this study to allow for more experimental control during the test perturbations.

Calculation of Ankle Inertia and Stiffness

In a dynamic system, the rotational inertia, or resistance to rotational motion, can profoundly affect the overall stiffness of the system. In our study, we provided a dynamic perturbation, allowing for viscoelastic activity and reflex activation of the ankle musculature. Therefore, accurately assessing the system's rotational inertia was essential. In previous studies,^{32,33} the ankle inertia was

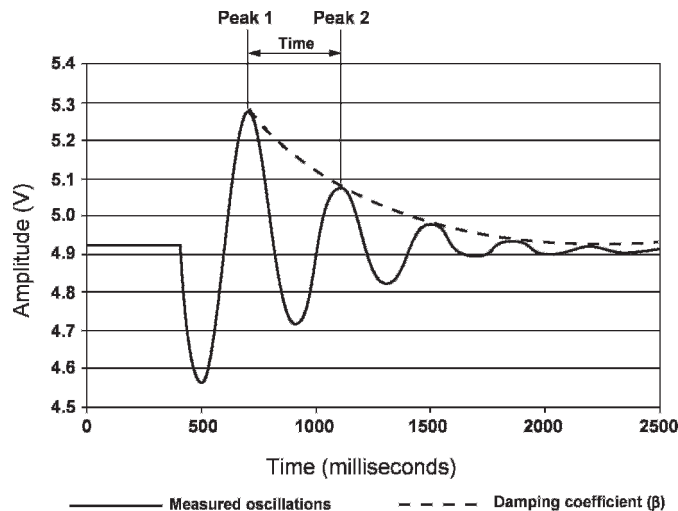


Figure 2. Stiffness calculation.

determined using estimates from anthropometric normative data. In our study, we added known amounts of mass at a fixed distance (inertia) to the system and used regression analysis to determine the stiffness of the ankle.

In calculating ankle-cradle inertia versus applied external mass (inertia), we added 0, 1, and 2 weights of equal magnitude (0.57 kg) the same distance from the axis of rotation to each side of the cradle device before normal data collection. The natural frequency of oscillation of a rotating system is influenced by both the inertia and the rotational stiffness of the system. In our study, we measured the damped frequency (ω_d) with the potentiometer and calculated the external inertia of the system and the natural frequency of oscillation (Figure 2). Next, we calculated the stiffness of the system as the slope of the regression line between the natural frequency of oscillation and the applied external inertia. Assuming second-order dynamic behavior, the formula

$$I_{Ext} = \frac{(k + mgl)}{\omega_n^2} - I_0 \quad \text{or} \quad I_{Ext} = (k + mgl) \left(\frac{1}{\omega_n^2} \right) - I_0$$

allowed us to plot the added external inertia (I_{Ext}) versus the inverse of the square of the natural frequency ($1/\omega_n^2$). The natural frequency of the oscillations (ω_n) was determined by measuring the damped frequency of the oscillations and combining it with the damping coefficient ($\omega_n = \omega_d + \beta$) (Figure 2). Failure to account for the amount of damping inherent to the system would have affected the magnitude of the natural frequency of oscillation, thus influencing the calculated stiffness value. It was determined that the pendulum behavior (mass of the system \times gravitational acceleration \times length of the radius [mgl]) had an effect of less than 1% on the ankle stiffness and, therefore, was ignored, making the equation resemble the equation of a line ($y = mx + b$). The stiffness (k) was the slope of the regression line, and the inertia of the ankle and cradle was the intercept (I_0).

Levels of MVIC for the 4 muscles tested were established for each participant by collecting 3 maximal 5-second trials. The first and last seconds of the MVIC trials were removed from the data to ensure only steady-state results during MVIC trials. The peak activity across the 3 trials

was averaged for each muscle. Average peak muscle activity was used to normalize EMG data for each muscle tested. Thus, EMG data for preactivation were expressed as percentages of MVIC. Muscle activity was recorded before and during the perturbations. The preactivation period consisted of the 250 milliseconds before trigger activation. After acquisition, all EMG data were low-pass filtered at 250 Hz, high-pass filtered at 30 Hz, notch filtered at 60 Hz, and rectified. This was followed by integration using an 11-point Hanning window, which was chosen for its ability to smooth the data without changing the temporal characteristics of the EMG.

Statistical Analysis

A mixed-model repeated-measures analysis of variance with 1 between-subjects (stability group) and 1 within-subjects (brace) factor was performed between participants in the stable group and participants in the unstable group over the 3 brace conditions (no brace, lace-up brace, semirigid brace). Post hoc analyses using Tukey honestly significant differences tests were performed to interpret effects. The dependent variables were ankle stiffness and muscle preactivation in the PL, PB, TA, and SO. We set the α level a priori at .05 for all comparisons. All data were analyzed with SPSS (version 14; SPSS Inc, Chicago, IL).

RESULTS

The mean and SD of all ankle stiffness values were 28.77 ± 7.39 Nm/rad (Table 1). Statistical analysis revealed differences between brace conditions collapsed across stability groups ($F_{2,52} = 66.185, P < .001, 1-\beta = .171$; effect size [Cohen d] = 0.34) (Figure 3). Post hoc analyses showed that the lace-up brace condition produced 14% more stiffness than the no-brace condition and that the semirigid brace condition produced 17% more stiffness than the lace-up brace condition. No differences in stiffness between stability groups were observed (stable = 27.36 ± 6.17 Nm/rad, unstable = 30.18 ± 8.21 Nm/rad; $F_{1,26} = 1.084, P = .307$). In addition, no interactions for brace \times stability group were observed ($F_{2,52} = 1.326, P = .275$).

Means, SDs, and 95% confidence intervals for muscle preactivation are presented in Table 2. We found no differences by brace condition in the PL ($F_{2,52} = 0.864, P = .427$), PB ($F_{2,52} = 0.947, P = .394$), TA ($F_{2,52} = 0.730, P = .487$), or SO ($F_{2,52} = 7.751, P = .184$) muscle. We also found no differences in muscle preactivation between stability groups in the PL ($F_{1,26} = 0.713, P = .406$), PB ($F_{1,26} = 0.646, P = .429$), TA ($F_{1,26} = 0.430, P = .518$), or SO ($F_{1,26} = 1.637, P = .212$) muscle. In addition, no interactions for brace \times stability group in the PL ($F_{2,52} = 0.320, P = .727$), PB ($F_{2,52} = 0.202, P = .818$), TA ($F_{2,52} =$

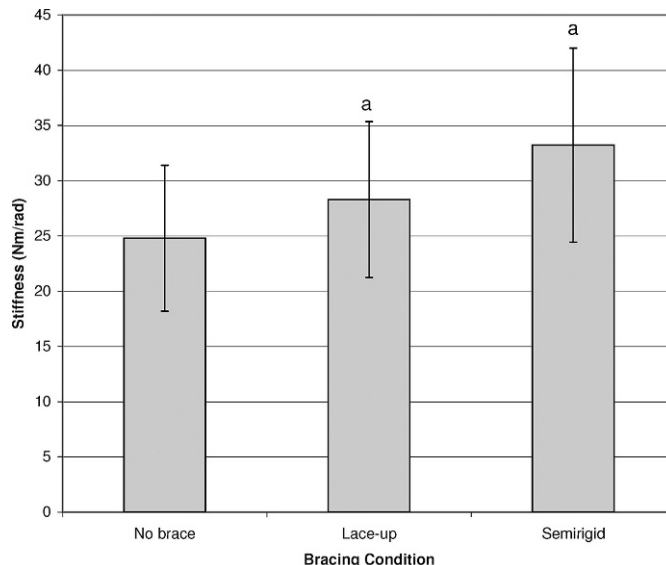


Figure 3. Comparison of stiffness over 3 brace conditions. ^a The semirigid brace condition was greater than the lace-up condition, and both were greater than the no-brace condition ($P < .05$).

0.365, $P = .696$), or SO ($F_{2,52} = 0.916, P = .407$) muscle were observed.

DISCUSSION

Our objective was to examine the contribution of ankle bracing to ankle stiffness among participants with stable and functionally unstable ankles. Ankle bracing was investigated because clinicians have successfully used ankle braces to decrease the incidence and severity of ankle injuries.^{11,14} However, the mechanism behind these decreases is unknown.

Our findings did not support our primary hypothesis that participants with FAI would exhibit lower rotational stiffness values compared with participants with stable ankles. Overall, rotational stiffness values of unstable ankles did not differ from stable ankles (27.36 ± 6.17 Nm/rad and 30.18 ± 8.21 Nm/rad), as measured via transient oscillation. These findings were similar to those of other investigators who compared rotational stiffness values between participants with stable and unstable knees³⁴ and shoulders.³⁵ They showed that participants with anterior cruciate ligament deficiency³⁴ and participants with anterior shoulder instability³⁵ exhibited the same rotational stiffness values as healthy control participants.

One possible explanation for the lack of difference between stability groups may lie in the body's attempt to compensate for the instability. Following Freeman's³⁶

Table 1. Stiffness (Nm/rad) Across Brace and Stability Conditions

| Group | No Brace | | | | Lace-Up Brace | | | | Semirigid Brace | | | |
|----------|------------------|--------------|--------------|------------------------|------------------|--------------|--------------|------------------------|------------------|--------------|--------------|------------------------|
| | Mean \pm SD | 95% CI | $F_{1,26}^a$ | P Value ^a | Mean \pm SD | 95% CI | $F_{1,26}^a$ | P Value ^a | Mean \pm SD | 95% CI | $F_{1,26}^a$ | P Value ^a |
| Stable | 23.65 \pm 5.14 | 20.02, 27.28 | 0.842 | .367 | 27.30 \pm 5.84 | 23.40, 31.21 | 0.544 | .467 | 31.12 \pm 7.22 | 26.35, 35.89 | 1.633 | .213 |
| Unstable | 25.94 \pm 7.80 | 22.31, 29.57 | | | 29.28 \pm 8.18 | 25.38, 33.19 | | | 35.31 \pm 9.93 | 30.54, 40.08 | | |

Abbreviation: CI, confidence interval.

^a Values are given for comparisons between the stable and unstable groups for each brace condition.

Table 2. Muscle Preactivation (Percentage of Maximal Voluntary Isometric Contraction) Across Brace and Stability Conditions

| Muscle and Group | No Brace | | | | Lace-Up Brace | | | | Semirigid Brace | | | |
|--------------------------|--------------|-------------|--------------|------------------------|---------------|-------------|--------------|------------------------|-----------------|-------------|--------------|------------------------|
| | Mean ± SD | 95% CI | $F_{1,26}^a$ | P Value ^a | Mean ± SD | 95% CI | $F_{1,26}^a$ | P Value ^a | Mean ± SD | 95% CI | $F_{1,26}^a$ | P Value ^a |
| Peroneus brevis | | | | | | | | | | | | |
| Stable | 3.57 ± 6.64 | 0.80, 6.30 | 0.985 | .330 | 3.30 ± 6.80 | 0.60, 6.00 | 1.166 | .290 | 3.81 ± 6.90 | 1.00, 6.60 | 0.863 | .361 |
| Unstable | 1.70 ± 2.23 | -1.00, 4.40 | | | 1.27 ± 1.66 | -1.40, 4.00 | | | 2.01 ± 2.26 | -0.80, 4.80 | | |
| Peroneus longus | | | | | | | | | | | | |
| Stable | 4.13 ± 8.33 | 0.80, 7.50 | 1.043 | .316 | 3.81 ± 8.38 | 0.40, 7.20 | 0.912 | .348 | 3.37 ± 8.27 | 0.10, 6.70 | 0.580 | .453 |
| Unstable | 1.79 ± 2.03 | -1.50, 5.10 | | | 1.59 ± 2.30 | -1.80, 5.00 | | | 1.64 ± 1.85 | -1.60, 4.90 | | |
| Soleus | | | | | | | | | | | | |
| Stable | 6.28 ± 11.28 | 1.80, 10.80 | 1.858 | .185 | 3.67 ± 5.12 | 1.50, 5.80 | 1.083 | .308 | 3.02 ± 4.89 | 1.10, 5.00 | 1.281 | .268 |
| Unstable | 2.08 ± 2.36 | -2.40, 6.60 | | | 2.14 ± 2.03 | 0.00, 4.30 | | | 1.51 ± 1.00 | -0.40, 3.40 | | |
| Tibialis anterior | | | | | | | | | | | | |
| Stable | 1.18 ± 2.18 | 0.20, 2.10 | 0.238 | .630 | 1.14 ± 2.24 | 0.20, 2.00 | 0.353 | .558 | 1.16 ± 2.22 | 0.30, 2.10 | 0.462 | .502 |
| Unstable | 0.86 ± 1.11 | -0.10, 1.80 | | | 7.62 ± 7.07 | -0.10, 1.70 | | | 0.74 ± 0.65 | -0.20, 1.60 | | |

Abbreviation: CI, confidence interval.

^a Values are given for comparisons between the stable and unstable groups in each condition

deafferentation hypothesis, it would seem logical that neuromuscular responses, including rotational stiffness, would be altered after joint injury. In functional situations, joint instability and poor joint kinematics could result in continued repetitive injury and further instability as described by the functional stability paradigm suggested by Lephart and Henry.⁷ In biomechanically unstable joints, the body may attempt to minimize further joint injury by increasing stiffness to relatively normal preinjury levels, effectively eliminating the difference between stability groups.

It is important to note that even though the statistical power in the group comparison was low ($1-\beta = .171$), the 95% confidence intervals for each condition (stable = 23.42, 31.30, unstable = 26.24, 34.12) contained considerable overlap. Combined with the small effect size (Cohen $d = 0.34$), this would suggest that the lack of power was due to no real clinical effect and, thus, has no clinical importance. If the effect size was larger and the 95% confidence intervals were more divergent, the lack of power could have been attributed to the small number of participants (14 per group).

Our data supported our second hypothesis that the application of an ankle brace would result in higher rotational stiffness values. Stiffness increased consistently from the no-brace condition (24.79 ± 6.59 Nm/rad) to the lace-up brace condition (28.29 ± 7.05 Nm/rad) and to the semirigid brace condition (33.22 ± 8.78 Nm/rad). Few studies of the effect of bracing on stiffness are available, but our data concurred with the findings of researchers³⁷⁻³⁹ who studied the effect of lumbar bracing on the neuromuscular response patterns of the lumbar stabilizers. Similarly, using the same instrumentation as we used, You et al⁴⁰ demonstrated trends toward increased ankle stiffness values after adding circumferential ankle pressure with a blood pressure cuff; however, their findings were not significant.

In our study, the level of axial load tested was 50% of body mass to replicate bipedal standing. Investigators^{41,42} have documented that loads on the body as high as 1200% of body mass are observed in functional situations.

Experimentally inducing such loads in the laboratory would be difficult, so inferences from our data to stiffness behavior in functional situations must be made with caution.

In an attempt to understand how ankle braces affect the biomechanical stability of the ankle, we evaluated the influence of ankle braces on ankle stiffness and the neuromuscular factors affecting it. In summary, the observed effects of bracing on stiffness have implications for the contribution of ankle braces to the reduction of incidence and severity of ankle injuries. We found that ankle braces increased rotational stiffness of the ankle. Increases in rotational stiffness have 3 basic causes: (1) increases in preactivation levels (intrinsic stiffness), (2) increases in reflex gain (reflex-mediated stiffness), and (3) increases in general passive, mechanical stiffness of the system. In nearly every situation, the ankle braces failed to cause changes in any of the neuromuscular attributes. This contradicts previous research showing that the cutaneous input from the application of an ankle brace or athletic tape facilitated neuromuscular changes as demonstrated by improved joint position sense^{15,16} and improved postural stability.^{17,18,20,43} Our data showed that braces caused no changes in the intrinsic stiffness of the system as evidenced by no differences in preactivation levels for any of the muscles tested. In essence, the only contribution to ankle stiffness and stability from the application of an ankle brace appeared to be passive.

Prospective and retrospective research has shown that ankle braces reduce the incidence and severity of ankle injuries.^{11,14,44,45} We attempted to critically analyze some of the contributions that ankle braces made to the biomechanical stability of the system in an effort to provide a mechanism for that reduction. Our data only supported passive, mechanical benefits from ankle brace use. The passive increase in stiffness caused the medial-lateral rotation of the ankle to decrease. This was evidenced by a decrease in the displacement amplitude across brace conditions. A decrease in joint rotation-translation would minimize the strain on the passive tissues surrounding the joint and reduce joint injuries.

Limitations

Several limitations may have contributed to the lack of significant findings in our investigation. First, we only looked at the acute changes in stiffness and muscle activity with bracing. We did not acquire data on prolonged use of ankle braces. Long-term ankle brace usage possibly would have more measurable effects on the neurologic properties of the system, as Cordova and Ingersoll⁴⁶ showed that long-term ankle brace usage may facilitate an increase in PL reflex amplitude. Second, the inclusion criteria we selected for FAI did not include mechanical assessment. Whereas researchers commonly know that FAI can occur without mechanical instability, information regarding the degree of laxity in the participants may have been warranted.⁴⁷ Third, the EMG data had considerable inherent variability, as evidenced by the large SDs. This variability could have implications for finding mean differences. Fourth, the seated task and the perturbation may not have been sufficient to elicit differences in EMG contributions. A more aggressive perturbation or demanding task potentially could have exacerbated any group differences.

Implications

Our study also had implications for the biomechanical stability of the ankle complex. To maintain equilibrium in response to unexpected perturbations, the system must be able to effectively modulate itself through adaptations in intrinsic and reflex-mediated responses. The relative lack of neuromuscular changes across all brace conditions implied that the ankle system is inherently unstable. In practice, however, the system does not behave in as unstable a manner as our results would imply. The neuromuscular activity that was present possibly was adequate for biomechanical stability but simply did not differ across brace conditions.

CONCLUSIONS

We found that the addition of ankle braces, from lace-up to semirigid, systematically increased the rotational stiffness of the ankle. Although stiffness increased, few demonstrable changes occurred in the neuromuscular properties of the system. This finding suggested that the primary contributions of an ankle brace to the stability of the ankle complex are through passive, mechanical means. More investigation into defining ankle stability and the contribution of ankle orthoses is needed.

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