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The Relationship Between Joint Stiffness and Muscle Activity in Unstable Ankles and Copers

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Context: Rolling sensations at the ankle are common after injury and represent failure in neural regulation of joint stiffness. However, deficits after ankle injury are variable and strategies for optimizing stiffness may differ across patients. ***Objective:*** To determine if ankle stiffness and muscle activation differ between patients with varying history of ankle injury. ***Patients:*** Fifty-nine individuals were stratified into healthy (CON, n = 20), functionally unstable (UNS, n = 19), and coper (COP, n = 20) groups. ***Main Outcome Measures:*** A 20° supination perturbation was applied to the ankle as position and torque were synchronized with activity of tibialis anterior, peroneus longus, and soleus. Subjects were tested with muscles relaxed, while maintaining 30% muscle activation, and while directed to react and resist the perturbation. ***Results:*** No group differences existed for joint stiffness ($F = 0.07$, $P = .993$); however, the UNS group had higher soleus and less tibialis anterior activation than the CON group during passive trials ($P < .05$). In addition, greater early tibialis anterior activation generally predicted higher stiffness in the CON group ($P \leq .03$), but greater soleus activity improved stiffness in the UNS group ($P = .03$). ***Conclusion:*** Although previous injury does not affect the ability to stiffen the joint under laboratory conditions, strategies appear to differ. Generally, the COP has decreased muscle activation, whereas the UNS uses greater plantar-flexor activity. The results of this study suggest that clinicians should emphasize correct preparatory muscle activation to improve joint stiffness in injury-rehabilitation efforts.

Keywords: neuromuscular control, neuromechanical decoupling, reaction times, functional ankle instability

Joint instability is a commonly debilitating condition characterized by sensations of "giving way" at a joint, typically following ligamentous injury. Although instability occurs at the knee and shoulder joints, functional ankle instability develops in nearly one-half of individuals experiencing an ankle sprain.[1] As approximately 60% of the general population has experienced an ankle sprain[2] and functional ankle instability is associated with decreased physical activity and long-term disability secondary to osteoarthritis,[3,4] these injuries place a significant financial burden on the health-care industry.[5] The primary challenge in the prevention and treatment of joint instability is a poorly understood etiology, as clinicians and researchers do not understand why 50% of patients successfully "cope" with no residual instability in the ankle model of joint stability.[1] Excessive mechanical laxity as well as altered sensorimotor function have been proposed as causes for this problem, but findings have been inconsistent in establishing a clear reason for these sensations of instability.[6–9] Though rolling, or giving way, of the ankle represents a failure to use muscle activation to regulate the stiffness of the joint, few studies have attempted to simultaneously quantify how joint stiffness and muscle activation across various reaction conditions changes in this subset.

Stiffness is defined as the joint's resistance to load through a range of motion and is proportionate to the amount of energy absorbed.[10,11] This property has strong implications for injury prevention, as the ability to appropriately regulate this absorption of energy represents the stability of the joint. The majority of investigations into joint stiffness following injury has used arthrometry, applying a gradual passive perturbation to quantify changes to joint laxity, a reciprocal of joint stiffness.[9,12] Using this technique, investigators may draw conclusions regarding stiffness changes to the capsuloligamentous tissue, as well as resistance from changes in resting muscle tone.[13] However, as more rapid and dynamic perturbations are applied to the joint through a greater range, stiffness changes represent alterations in the series and parallel elastic components of the muscle as well as the regulation of reverse cross-bridge cycling.[14–16] Few studies have investigated these features at the ankle joint, with most studies exploring alterations in triceps surae

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stiffness.[17] This may not be entirely applicable to the study of functional ankle instability, as sensations of giving way occur primarily in the frontal plane. In addition, in a few studies that have measured frontal plane stiffness under a dynamic condition in these subsets, indirect stiffness measurement techniques have been used.[18,19] Utilizing a stiffness assessment technique in which a torque response is directly quantified during a simulated injurious event would expand our knowledge by enabling the distinct study of involuntary and voluntary muscle contractions and their mechanical effect on the joint.

Joint stiffness represents a mechanical measure of joint stability; however, it is entirely affected by the neuromuscular control of the surrounding muscles. It has been suggested that reactive mechanisms alone may not be enough to prevent joint injury, leading to an emphasis on preparatory muscle activation in studying joint instability.[20,21] This feed-forward activity, whether from an increase in involuntary muscle tone or from volitional contraction, modifies joint stiffness through several mechanisms, such as pretensioning the muscle (more linked actin-myosin cross-bridges) and facilitating alpha-gamma coactivation.[22,23] Combining simultaneous measures of stiffness and muscle activation in those with a history of injury could provide essential information regarding the neuromuscular control strategies that provide "optimal" joint stiffness, how preparatory and reflexive activation may become altered after injury, and how the link between mechanical stiffness and muscle activation may become decoupled after injury.[11]

Ligamentous injury has been linked to mechanical joint changes as well as alterations in neuromuscular control.[6,7,20] However, studies have not consistently associated ankle instability with altered stiffness nor have studies directly measured ankle stiffness under reactive conditions, with simultaneous measures of muscle activity. Therefore, it is unclear how mechanical stiffness and neuromuscular control may decouple and if this relationship may provide insight into the development of functional ankle instability. The purpose of this study was, therefore, to determine if short-range and total ankle inversion stiffness and muscle activation patterns differ between healthy uninjured controls, functionally unstable ankles, and ankle copers. We hypothesized that while functionally unstable ankles would have lower levels of stiffness and muscle activation compared with healthy and coper groups, these groups would be further discriminated by an altered correlation between stiffness and laxity in previously injured ankles. Our study design and analysis allowed us to determine if these group differences exist, as well as if decoupling between mechanical joint stiffness and the neuromuscular control may occur in these subsets.

Methods

Experimental Design

A cross-sectional design was used in this study, with a correlational analysis. Dependent variables included ankle joint stiffness and muscle activation levels. For stiffness, independent variables included group, stiffness range (short-range or total), and reaction condition. For muscle activation, independent variables included group, muscle, and reaction condition.

Participants

Fifty-nine physically active subjects were recruited for this study (Table 1). Subjects were stratified using history of ankle injury and the Cumberland Ankle Instability Tool (CAIT) into groups of healthy control (CON), functionally unstable (UNS), and copers (COP). CON subjects had no history of ankle sprain and a CAIT score above 27, COP subjects had a history of 1 or more ankle sprains and a CAIT score above 27, and UNS subjects had a history of 1 or more ankle sprains and a CAIT score below 25.[24] When subjects reported bilateral injury, the side with a lower CAIT score was included in the study. All subjects had no history of fracture or surgery to the legs as well as no neurological disorders, and CON and COP participants were free of all lower extremity injury for 12 months.[25]

Instrumentation

All testing was conducted using a custom-built stiffness and proprioception assessment device (SPAD, Figure 1).[26,27] The SPAD consists of a servomotor attached to an adapter arm and adjustable chair. The device motor was controlled using custom LabVIEW software (National Instruments, Austin, TX) and was capable of generating rapid perturbations to the joint while a torque sensor measures the joint's resistance. Analog signals of position and torque were collected in custom software and synchronized with surface electromyography (EMG) recordings (Konigsberg Instruments, Pasadena, CA) from the lower leg muscles.

Procedures

Testing consisted of a single session, and all procedures were approved by the university human subjects review board. After providing informed consent, self-adhesive rectangular Ag/AgCl surface electrodes (Phillips Medical

Table 1 Subject Characteristics

	CON	UNS	COP
n	20	19	20
Age (y)	22.5 ± 2.4	22.3 ± 4.1	23.1 ± 3.3
Height (cm)	170.1 ± 11.8	171.2 ± 9.8	172.6 ± 8.2
Mass (kg)	68.9 ± 15.8	73.6 ± 22.9	73.4 ± 14.6
CAIT	29.2 ± 1.0	18.3 ± 3.5	29.4 ± 0.8
# of sprains	0.0 ± 0	4.3 ± 4.5	1.4 ± 0.9

Abbreviations: CON, healthy control; UNS, functionally unstable; COP, coper; CAIT, Cumberland Ankle Instability Tool.

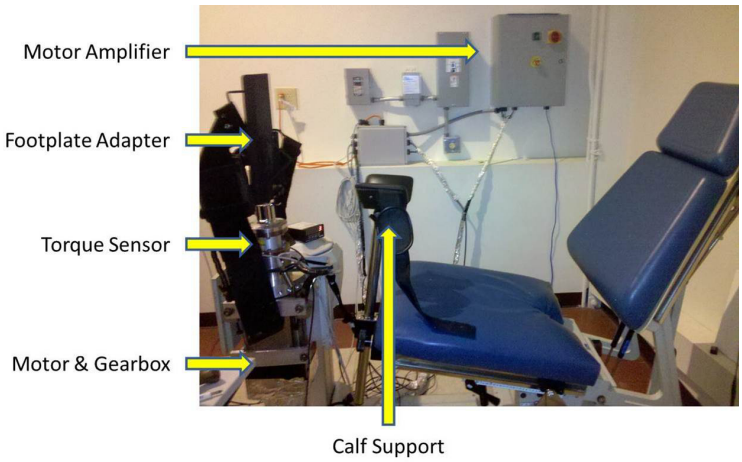

Figure 1 — The stiffness and proprioception assessment device (SPAD) used to apply standardized perturbations to the ankle joint.

System, Andover, MA) were placed in series over the midbelly of the subjects' tibialis anterior (TA), peroneus longus (PL), and soleus (SOL) muscles in line with previously defined locations.[28] A ground electrode was placed on the subjects' patella. Before electrode placement, the skin over each muscle was palpated, cleaned, shaved, and abraded. Correct placement of electrodes was confirmed with real-time monitoring of activity during isolated muscle contractions and throughout testing. Subjects were then positioned in the SPAD with the hip flexed approximately 110°, the knee flexed 90°, the calf supported in padding, and the foot secured in a foot plate (Figure 2).

Before stiffness testing, subjects provided maximum voluntary isometric contractions (MVICs) by initiating a contraction and holding it at maximal effort for 3 seconds in the directions of pronation, supination, and plantarflexion to allow for normalization of EMG signals. Ankle joint stiffness was assessed under 3 conditions: passively, actively, and reactively (Table 2). For all stiffness conditions, the same 20° supination perturbation was applied to the joint at a velocity of 240°/s and acceleration of 3000°/s^2. These values were selected to mimic the rate at which ankle sprains occur and because of the constraints of our testing apparatus.[29] Subjects were provided 2 trials to familiarize them with the perturbation before initiating test trials. The order of conditions was randomized for each subject, and 5 trials were collected for each condition, with a minimum of 30 seconds between each trial. Position, torque, and EMG were collected at 2400 Hz in custom LabVIEW software.

Data Reduction and Analysis

Joint quasi-stiffness was calculated as the change in torque divided by the change in rotation. Values were extracted for the short-range (0–3°) and for the total perturbation (0–20°).[30] All values were normalized to the subject's body mass, as size and subsequently strength of the ankle would affect generated torque.[30] Group differences were compared using a 3-way analysis of variance (ANOVA)

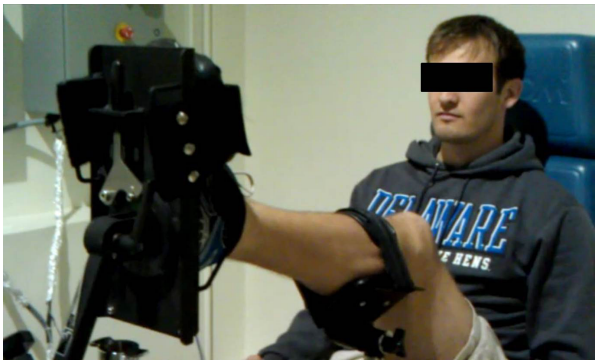

Figure 2 — Participant positioning in the stiffness and proprioception assessment device.

Table 2 Subject Instructions for Each Test Condition

Condition	Instructions
Passive	"Remain completely relaxed throughout the entire perturbation."
Active	"Push out to [30% MVIC] prior to the move. When you feel the perturbation, hold that amount of contraction without pushing any more or less."
Reactive	"Push out to [30% MVIC] prior to the move. When you feel the perturbation, resist it as hard and as fast as you can as if you're stopping your ankle from rolling in."

Abbreviation: MVIC, maximum isometric voluntary contraction.

with 1 between-subjects factor (group, 3 levels) and 2 within-subjects factors (condition, 3 levels; range, 2 levels). Pairwise comparisons were used for post hoc analysis. An a priori level of significance was set at.05.

All EMG data were band-pass filtered (20–400 Hz) using a zero-lag fourth-order Butterworth filter, full-wave

rectified, and then low-pass filtered (10 Hz) to create a complete linear envelope. The ensemble peak EMG from the 3 MVIC trials was used for normalization of stiffness trials. Peak EMG (%MVIC) was extracted for all stiffness trials. EMG onset (seconds) was calculated for each muscle by locating peak activity and searching backward to find the point EMG went below 10% of peak activity. Average EMG activity (%MVIC) was calculated for 250 milliseconds before the perturbation (PRE), 250 milliseconds from the start of the perturbation (POST-1), and 250 to 500 milliseconds from the start of the perturbation (POST-2). All trials were visually inspected for artifacts and correct contraction; dependent variables were averaged across trials for each condition. Group differences for peak and EMG onset were compared using a 3-way ANOVA with 1 between-subjects factor (group, 3 levels), and 2 within-subjects factors (condition, 3 levels; muscle, 3 levels). Separate 3-way ANOVAs with 1 between-subjects factor (group, 3 levels) and 2 within-subjects factor (muscle, 3 levels; time, 3 levels) were used to compare group differences for each stiffness condition. Pairwise comparisons were used for post hoc analyses. An a priori level of significance was set at .05.

Pearson product-moment correlation coefficients were calculated to determine relationships between stiffness and EMG variables within active and reactive conditions and groups. To assess between-group differences correlation coefficients were converted to *z*-scores, and the difference in *z*-scores was used to determine between-group differences.[31,32] Effect sizes for these findings were interpreted using Cohen *q*, whereby 0.1, 0.3, and 0.5 are considered cutoffs for small, medium, and large effect sizes, respectively.[33] Overall correlations (main effects) were assessed only in the absence of between-groups differences to ensure a single group was not affecting the overall correlation and to test our primary hypotheses.

Results

Joint Stiffness

Stiffness values are presented in Table 3. No significant 3-way interaction effect was detected for group × condition × range ($F_{4,112} = 0.07$, $P = .993$). A significant condition × range interaction effect was observed ($F_{2,112} = 64.35$, $P < .001$). Pairwise comparisons revealed that under all conditions, short-range stiffness was significantly higher than total stiffness ($P < .001$). Short-range stiffness was significantly lower in the passive condition than in the active condition ($P = .022$). Total stiffness was significantly lower in the passive condition than both active and reactive conditions ($P < .001$). No effect of group was detected ($F_{2,56} = 0.43$, $P = .650$).

Muscle Activation

Peak EMG and muscle onset is presented in Table 4, and mean EMG activity is displayed in Table 5. No significant group effect was observed for peak EMG ($F_{2,56} = 0.165$, $P = .849$); however, a significant condition × muscle interaction was observed ($F_{4,224} = 26.667$, $P < .001$). Pairwise comparisons revealed greatest activity in PL during active and reactive conditions ($P < .001$) and least activity in SOL during the reactive condition, compared with other muscles ($P < .001$). No main effect of group was observed for EMG onset ($F_{2,48} = 1.292$, $P = .284$); however a significant condition × muscle interaction was observed ($F_{4,192} = 2.878$, $P = .024$). Pairwise comparisons revealed SOL activation was faster than TA ($P = .006$) and PL ($P = .044$) in the passive condition. Although no differences were observed in the active condition, TA muscle activation was slower than PL ($P = .001$) and SOL ($P = .010$) in the reactive trials.

In the passive condition, mean muscle activation revealed a significant 3-way interaction effect (time × muscle × group; $F_{8,224} = 2.473$, $P = .014$). No group differences were observed at PRE; however, the UNS group displayed higher SOL activity than COP at POST-1 ($P = .031$) and lower TA activity than the CON group at POST-2 ($P = .044$). In addition, although both CON and UNS groups increased EMG activity significantly from PRE to POST-1, no change was observed among the COP group ($P < .05$).

Mean activation throughout the active condition had no 3-way interaction effect; however, significant time × group ($F_{4,108} = 5.783$, $P < .001$) and time × muscle ($F_{4,216} = 18.923$, $P < .001$) interaction effects were observed. Pairwise comparisons revealed no differences at PRE; however, the COP had less activation than the UNS group at POST-1 ($P = .040$) and POST-2 ($P = .021$). A statistical trend suggesting the UNS group had higher POST-2 activation than CON was also observed ($P = .058$). PL activation maintained the highest throughout all time blocks ($P < .001$), whereas TA remained higher than SOL at PRE ($P = .021$) and POST-2 ($P = .039$). No differences between TA and SOL were observed at POST-1 ($P = .088$).

Table 3 Short Range (0–3°) and Total (0–20°) Normalized Stiffness Values (Nm · degree^{-1} · kg^{-1}) Across Groups and Conditions

	Group	Short range	Total
Passive	CON	0.094 ± 0.05	0.007 ± 0.04
	UNS	0.084 ± 0.03	0.014 ± 0.01
	COP	0.102 ± 0.04	0.012 ± 0.01
Active	CON	0.099 ± 0.04	0.039 ± 0.02
	UNS	0.094 ± 0.04	0.048 ± 0.02
	COP	0.107 ± 0.03	0.041 ± 0.02
Reactive	CON	0.098 ± 0.04	0.039 ± 0.01
	UNS	0.090 ± 0.03	0.047 ± 0.02
	COP	0.104 ± 0.04	0.040 ± 0.02

Abbreviations: CON, healthy control; UNS, functionally unstable.
Note: Short-range stiffness was significantly higher than total stiffness under all conditions ($P < .001$).

Table 4 Peak and Onset of Muscle Activity Across Groups and Conditions

		Peak (% MVIC)			Onset (s)		
		TA	PL	Soleus	TA	PL	Soleus
Passive	Control	5.1 ± 8.0	5.2 ± 7.0	4.2 ± 3.0	0.734 ± 0.52	0.611 ± 0.39	0.436 ± 0.44
	UNS	3.2 ± 3.0	2.8 ± 3.0	5.6 ± 5.0	0.641 ± 0.56	0.544 ± 0.40	0.342 ± 0.45
	Coper	2.4 ± 3.0	2.9 ± 4.0	3.7 ± 3.0	0.649 ± 0.61	0.660 ± 0.58	0.582 ± 0.54
Active	Control	24.9 ± 34.0	39.4 ± 16.0	14.4 ± 16.0	0.396 ± 0.25	0.388 ± 0.25	0.452 ± 0.30
	UNS	17.4 ± 13.0	48.8 ± 20.0	17.9 ± 14.0	0.324 ± 0.27	0.352 ± 0.37	0.273 ± 0.14
	Coper	29.0 ± 45.0	38.3 ± 23.0	13.5 ± 9.0	0.481 ± 0.33	0.414 ± 0.37	0.445 ± 0.35
Reactive	Control	56.9 ± 54.0	70.8 ± 33.0	25.0 ± 16.0	0.602 ± 0.48	0.502 ± 0.404	0.557 ± 0.45
	UNS	43.9 ± 37.0	87.4 ± 35.0	35.7 ± 19.0	0.451 ± 0.43	0.326 ± 0.27	0.292 ± 0.29
	Coper	49.2 ± 36.0	81.1 ± 27.0	32.2 ± 26.0	0.465 ± 0.39	0.326 ± 0.26	0383 ± 0.33

Abbreviations: MVIC, maximal voluntary isometric contraction; TA, tibialus anterior; PL, peroneus longus; UNS, functionally unstable.

In the reactive condition, no group effects were observed ($F_{2,56} = 0.251$, $P = .779$); however a significant time × muscle interaction effect was observed ($F_{4,224} = 21.551$, $P < .001$). PL activation was highest across all time blocks ($P < .001$), whereas SOL activation was lower than TA across all time blocks ($P < .02$).

Stiffness-EMG Correlation Main Effects

Correlations are presented in Table 6. Both active and reactive conditions revealed significant correlations between short-range stiffness and muscle onset ($P \leq .02$) as well as between short-range stiffness and peroneal activation at PRE and POST-1 ($P < .001$). For the active condition, higher total stiffness positively correlated with POST-1 and POST-2 SOL activation ($P < .001$) and POST-2 PL activation ($P = .04$). In the reactive condition, increased short-range stiffness also correlated with higher PL activation at POST-2 ($P = .01$). Total stiffness was observed to correlate with faster and greater SOL activation ($P \leq .01$) and faster PL activation ($P = .02$).

Stiffness-EMG Correlation Group Differences

For the active condition, group differences were observed in the relationship between TA activation and total stiffness, where higher TA activation indicated higher total stiffness in UNS ankles, but not CON ankles ($P = .03$; $q = 0.70$). Higher peak and PRE SOL activation also correlated with better total stiffness among UNS ankles, but not in COP ankles ($P = .03$; $q = 0.72$). Higher PL preactivation correlated with higher total stiffness among CON ankles, but not UNS ankles ($P = .03$, $q = 0.72$). For the reactive condition, group differences were observed as a positive correlation between short-range stiffness and TA activation (peak, PRE, POST-1, POST-2) in CON ankles, but not in UNS ($P < .01$; $q \geq 1.0$) or COP ankles ($P = .03$; $q = 0.75$). TA activation at POST-1 positively correlated with total stiffness in UNS ankles, but not in CON ankles ($P = .03$; $q = 0.75$).

Discussion

In the current study, we assessed if differences in joint stiffness and muscle activation patterns existed among healthy ankles, functionally unstable ankles, and ankle copers. In addition, we aimed to determine if neuromechanical decoupling occurs following ankle joint injury, where individuals with a history of ankle sprain no longer appropriately couple their muscle activation to their joint's stiffness. Our results suggest that no overall group differences in joint stiffness exist between these subject samples; however, these groups regulate their joint stiffness using different muscle activation strategies.

Joint Stiffness

A primary finding of this study was that joint stiffness was similar for every group, indicating that under a controlled laboratory setting both healthy and previously sprained ankles are able to stiffen joints equally. Although we observed high variability across all groups despite normalization to body mass and controlling for levels of muscle preactivation, our data suggest that differences may exist in the strategies through which these subjects stiffened their joints. This indicates that what discriminates these groups may not be their ability to stiffen the joint but the selected muscular strategy to achieve that stiffness. While our procedures used a unique device for directly measuring joint stiffness, previous investigations using other stiffness assessment techniques also failed to discriminate functionally unstable ankles from healthy ankles.[9,18] Stiffness differences between functionally unstable ankles have been reported using joint

Table 5 Muscle Activity (Mean/MVIC) Across Groups and Within Each Perturbation Condition

		Tibialus Anterior			Peroneus Longus			Soleus		
		PRE	POST-1	POST-2	PRE	POST-1	POST-2	PRE	POST-1	POST-2
Passive	Control	0.017 ± 0.02	0.027 ± 0.04	0.018 ± 0.02	0.012 ± 0.02	0.024 ± 0.03	0.014 ± 0.02	0.016 ± 0.02	0.022 ± 0.02	0.018 ± 0.02
	UNS	0.010 ± 0.01	0.014 ± 0.01	0.012 ± 0.01[a]	0.007 ± 0.00	0.012 ± 0.01	0.008 ± 0.01	0.012 ± 0.01	0.027 ± 0.02[b]	0.012 ± 0.01
	Coper	0.010 ± 0.01	0.013 ± 0.02	0.011 ± 0.01	0.011 ± 0.01	0.013 ± 0.02	0.010 ± 0.01	0.013 ± 0.01	0.019 ± 0.02	0.014 ± 0.01
Active	Control	0.098 ± 0.10	0.109 ± 0.10	0.104 ± 0.10	0.182 ± 0.11	0.230 ± 0.12	0.217 ± 0.12	0.057 ± 0.03	0.075 ± 0.04	0.068 ± 0.04
	UNS	0.081 ± 0.07	0.110 ± 0.09	0.111 ± 0.09	0.203 ± 0.10	0.291 ± 0.14	0.307 ± 0.15	0.072 ± 0.04	0.106 ± 0.08	0.105 ± 0.08
	Coper	0.083 ± 0.09	0.100 ± 0.10	0.102 ± 0.10	0.151 ± 0.08	0.198 ± 0.11	0.195 ± 0.12	0.054 ± 0.04	0.069 ± 0.05	0.063 ± 0.06
Reactive	Control	0.154 ± 0.19	0.233 ± 0.26	0.314 ± 0.33	0.176 ± 0.08	0.300 ± 0.16	0.423 ± 0.26	0.061 ± 0.04	0.137 ± 0.09	0.101 ± 0.06
	UNS	0.085 ± 0.07	0.185 ± 0.16	0.235 ± 0.22	0.207 ± 0.11	0.407 ± 0.21	0.535 ± 0.30	0.077 ± 0.05	0.211 ± 0.15	0.156 ± 0.12
	Coper	0.090 ± 0.08	0.172 ± 0.14	0.277 ± 0.23	0.177 ± 0.09	0.338 ± 0.16	0.492 ± 0.24	0.068 ± 0.07	0.197 ± 0.19	0.127 ± 0.12

Abbreviations: MVIC, maximal voluntary isometric contraction; PRE, 250 ms before the perturbation; POST-1, 250 ms from the start of the perturbation; POST-2, 250–500 ms from the start of the perturbation; UNS, functionally unstable.

[a] Significant difference from control group. [b] Significant difference from coper group. UNS had greater activation than copers during the active condition.

Table 6 Pearson Product–Moment Correlation Coefficients (*r*) Between Stiffness and EMG Variables Across All Groups

			Peak EMG			EMG Onset			Mean PRE			Mean POST-1			Mean POST-2		
			TA	PL	Soleus	TA	PL	Soleus	TA	PL	Soleus	TA	PL	Soleus	TA	PL	Soleus
Active stiffness	SR	Control	–.325	.325	.197	**–.543**	**–.578**	**–.586**	–.272	**.508**	.259	–.280	**.587**	.219	–.286	**.465**	.218
		UNS	.188	.082	–.096	**–.579**	**–.507**	**–.445**	.181	.359	–.043	.148	.283	–.054	.152	.131	–.074
		Coper	.161	–.118	–.158	**–.578**	–.350	**–.598**	.252	.374	.117	.266	**.461**	.244	.262	**.447**	.222
		All	–.176	.071	–.077	**–.509**[a]	**–.453**[a]	**–.503**[a]	.068	**.377**[a]	.059	–.013	**.368**[a]	.045	–.017	.251	.019
	Total	Control	–.209	.430	.343	–.072	–.261	–.198	–.131	**.486**	**.457**	–.208	**.495**	.435	–.191	.435	.391
		UNS	**.455**[b]	–.142	**.531**[c]	–.328	–.201	–.362	.415	–.190[b]	**.673**[c]	**.473**[b]	–.023	**.619**	**.489**[b]	.064	**.565**
		COP	.048	–.117	–.135	–.211	–.016	–.205	.149	.214	.087	.128	.291	.136	.130	.261	.160
		All	–.047	.085	**.380**[a]	–.135	–.070	–.216	.196	.162	**.477**[a]	.274	.247	**.497**[a]	**.281**	**.273**[a]	**.484**[a]
Reactive stiffness	SR	Control	**–.634**	.212	–.066	–.352	**–.338**	–.363	**–.610**	.240	–.137	**–.624**	**.510**	.041	**–.566**	.379	.109
		UNS	.435[b]	.293	.261	–.319	**–.605**	**–.470**	.366[b]	.368	.091	.401[b]	**.486**	.182	.371[b]	.435	.225
		Coper	–.013[b]	.276	.146	–.435	**–.590**	**–.500**	.078[b]	.405	.186	.022[b]	**.503**	.206	–.023[b]	.392	.225
		All	–.174	.251	.087	**–.297**[a]	**–.453**[a]	**–.380**[a]	–.121	**.337**[a]	.045	–.105	**.423**[a]	.096	–.171	**.350**[a]	.102
	Total	Control	–.234	.120	.336	–.309	–.370	–.377	–.203	.238	.256	–.198	.333	.250	–.144	.169	.431
		UNS	.390	.141	**.454**	**–.560**	**–.505**	–.522	.402	.202	**.652**	**.498**[b]	.409	**.652**	**.461**	.233	**.546**
		Coper	.022	.188	.161	.020	–.208	–.144	.211	.375	.310	.022	.272	.227	.004	.231	.241
		All	.044	.200	**.339**[a]	–.258	**–.316**[a]	**–.349**[a]	.212	**.303**[a]	**.468**[a]	.242	**.382**[a]	**.477**[a]	.082	.215	**.397**[a]

Abbreviations: EMG, electromyography; PRE, 250 ms before the perturbation; POST-1, 250 ms from the start of the perturbation; POST-2, 250–500 ms from the start of the perturbation; TA, tibialus anterior; PL, peroneus longus; SR, short-range stiffness; UNS, functionally unstable.

Note: Bold numbers indicate significant correlation at .05 level.

[a] Significant overall main effect. [b] Significant difference from control. [c] Significant difference from coper group.

arthrometry under passive conditions[12,34]; however, this measurement differs greatly from the rapid supination perturbation used in this study. Our stiffness assessment technique allows for greater control of joint acceleration and velocity and higher levels of muscle activation than other measures, although the loads measured may be less than those reported during injury.[29] Furthermore, research has suggested that "optimal" joint stiffness is a highly individualized measure and, therefore, may not be detected as a group effect.[13] Therefore, it may be imperative for clinicians to evaluate movement strategies rather than laxity or stiffness alone.

Although no overall group differences were detected, changes were observed between stiffness ranges and conditions. This was the first study to investigate ankle inversion short-range stiffness, and, consistent with other joints, short-range stiffness was always greater than total stiffness, with no differences in short-range stiffness across conditions. As short-range stiffness represents the initial stretch applied to the capsuloligamentous tissue, existing actin/myosin cross-bridges, and series/parallel elastic components of the musculotendinous structures, this value would not be affected by stiffness condition as the neural response would occur later than the first 3°.[15,16] In agreement with previous research, active and reactive conditions presented stiffness values more than 4 times that of the passive condition; although following the initial gain from precontraction, no additional benefit of reactive muscle activation was observed.[27] Although peak torque and muscle activity should be highest when a volitional response is added to the preactivation, the entire perturbation would last less than 500 milliseconds. Our findings are consistent with previous studies where muscle onset did not occur until 400 to 500 milliseconds, minimizing the contribution of volitional responses toward resisting the perturbation and making it difficult to discriminate between active and reactive conditions.[21] Although unstable ankles are able to stiffen the joint equal to healthy ankles and copers under a controlled laboratory setting, it remains unclear why this does not translate to functional activity as well how reflexive and volitional muscular contraction is regulating this stiffness.

Muscle Activation

Involuntary Responses. During the passive perturbation, subjects were instructed to remain relaxed such that resistance from the ankle came only from resting muscle tone and small amounts of involuntary reflexive muscle activation. Whereas no group differences were observed for EMG peak and onset, the fastest reflexive muscle activation was observed in the SOL across all subjects. Previous studies have quantified SOL activation as having the largest reflexive component during sagittal plane perturbations, and our results suggest that it also has the largest reflexive component in the frontal plane.[15] No group differences were observed before the perturbation; however, COP subjects demonstrated the smallest response over time to the perturbation, as they did not significantly increase muscle activation at any time point, suggesting a degree of reflexive inhibition.[35] This decreased muscle activation may serve to allow the nervous system to better predict joint stiffness by minimizing its variability in this subset, although subsequent analysis of biomechanical variability would be needed to further demonstrate this.[36] UNS ankles differed from COP ankles with higher SOL activation in the first 250 milliseconds of the perturbation and differed from CON ankles with less TA activation from 250 to 500 milliseconds. These findings suggest that COP ankles may have greater inhibition leading to suppression of ankle reflexes, although it is unclear if this is beneficial for preventing injury. Previous studies have suggested that some level of compliance may be more optimal for absorbing loads and maintaining joint stability at the knee joint, and these data suggest that COP ankles may demonstrate a better ability to use this strategy in protecting the joint.[37] Therefore, it may be beneficial to incorporate ankle rehabilitation techniques that encourage muscle relaxation or inhibition, such as biofeedback.[38]

Effect of Muscle Preactivation. Although reflexes are important for maintaining joint stability, most functional activities are associated with some level of muscle preactivation. During the active perturbation, a general trend was observed where PL had the highest muscle activation and TA had the lowest muscle activation at PRE and POST-2; however, the initial response to the perturbation eliminated these differences at POST-1. All muscles displayed increased activity during this early load, which would be beneficial as this is within the period of time when injury is likeliest to occur (50–150 milliseconds).[21] When examining group differences, COP ankles demonstrated decreased muscle activation at POST-1 and POST-2. Similar to the passive trials, this finding provides evidence that COP ankles were better at maintaining stiffness without compensatory muscle activation and suggests the central nervous system in this subset is better able to negotiate the perturbation and down-regulate spinal reflexes.[35] When compared with stiffness measures, correlation main effects demonstrated that higher short-range stiffness was associated with faster muscle activation and subsequently greater PL activity. These data support rehabilitation efforts to improve short-range stiffness, as it appears to facilitate reflexive responses. Alternately, SOL activity was observed to better correlate with total joint stiffness, suggesting that to maintain stiffness throughout the entire perturbation, additional recruitment from SOL may be required.

Correlations between muscle activation and joint stiffness during the active condition revealed several differences in stiffness modulation between CON and UNS ankles. In the modulation of total stiffness, CON ankles relied on higher PL preactivation for higher total stiffness, whereas UNS ankles used higher peak TA activation at POST-1 and POST-2 to achieve higher total stiffness. These data suggest an important difference between these groups, as higher preactivation from a primary stabilizing

muscle contributes to joint stiffness in uninjured ankles, whereas unstable ankles appear to depend on greater postactivation from the dorsiflexors—potentially in an attempt to pull the joint into a more close-packed position. UNS also differed from COP ankles, using higher SOL peak and PRE activation to maintain higher joint stiffness. In contrast to healthy ankles, it appears that synergy between the TA and SOL may be most important in discriminating UNS ankles from CON and COP ankles.

Volitional Response. A third component present in the muscular response to a perturbation is the volitional reaction occurring after preactivation and reflexive responses. Under this condition, no group differences were observed, although PL activation was highest and SOL activation was lowest. It was also observed that TA had the slowest activation in the reactive trials. Correlation main effects were observed where higher short-range stiffness was associated with faster onsets in all muscles and higher PL activation, whereas total stiffness correlated with faster SOL and PL activation and greater SOL activation. As the task required subjects to do more than maintain a level of muscle contraction, it is important to note that greater SOL activation appeared to most greatly affect the stiffness in the later ranges of the perturbation.

Group differences emerged in the correlations between stiffness and muscle activation, as short-range stiffness was associated with *lower* TA activation in CON ankles, but not in COP or UNS ankles. In addition, higher TA activity was associated with higher total stiffness in UNS ankles, not CON ankles. These data imply that regulation of TA to optimize reactive joint stiffness may be a key discriminating factor between healthy and unstable ankles. As healthy ankles with less short-range stiffness used greater TA activity, they may be optimizing the close-packed position of the ankle joint, whereas those with higher innate stiffness do not require this activation. Several studies have supported a key role of regulating ankle dorsiflexion in discriminating healthy and unstable ankles in studies investigating gait and landing mechanics, as well as those performing a simple perturbation.[39,40] As the data support these findings, it seems imperative for interventions designed to improve dorsiflexion throughout functional movement tasks be implemented during rehabilitation.

Limitations

There are several limitations that must be addressed with the current study. First, the testing set-up used in this study was designed to isolate the ankle joint and muscles and, therefore, does not represent a functional position that an individual may be at risk for rolling their ankle. In addition, this study used subsets of healthy, unstable, and coper ankles, using 1 ankle per subject. Further studies might investigate these factors using patients with only unilateral injury, so that the uninjured side may serve as a better control group to minimize variability. Finally, all measurements in this study were taken a minimum of 6 months after injury so that subjects were cleared for return to activity, thereby preventing us from drawing conclusions as to whether our observations were the basis of a predisposition for injury or an adaptation following injury.

Conclusion

Our findings suggest that the ability to stiffen the joint is not affected by previous ankle injury; however, patients with functional ankle instability and copers appear to use separate strategies from healthy ankles to regulate joint stiffness. While uninjured subjects stabilized their ankles primarily through the peroneus longus muscle, unstable ankles demonstrated higher plantar flexor activity to stabilize the joint, and copers used greater dorsiflexor activity. In addition, copers demonstrated greater reflexive inhibition, potentially allowing the nervous system to better predict the joint's stiffness throughout a perturbation. The data support the notion that although maintaining joint stiffness is essential for preventing injury, the muscle activation strategy to achieve that stiffness may be of greater importance. These findings could represent a potential neuromechanical decoupling, where the joint becomes dependent on the wrong muscles to stabilize the joint, potentially leading to functional instability. As short-range stiffness correlated with faster muscle activation, it may be beneficial for clinicians to use measures for improving this stiffness following injury to potentially improve outcomes, as well as emphasizing preparatory muscle activation for preventing subsequent joint injury. Future research is required to determine what specific interventions (ie, balance, strength, or perturbation training) would be best at improving these factors.

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