Ankle Injury Alters the Links between Joint Laxity, Peripheral Sensation, and Cortical Activation

Alan R. Needle*, C. Buz Swanik, FNATA†, Jochen Baumeister, William B. Farquhar†, Jody L. Greaney, Jill S. Higginson†, Thomas W. Kaminski, FNATA†

Appalachian State University, Boone, NC; †University of Delaware, Newark, DE; ‡University of Paderborn, Paderborn, Germany; §Pennsylvania State University, University Park, PA

INTRODUCTION

Approximately 60% of the general population has experienced an ankle sprain, and half of those patients report persistent symptoms of instability, despite extensive rehabilitation efforts. [1,2] These inconsistent results are limiting patient outcomes for many, and likely originate from our incomplete understanding of the neuromechanical pathology. Because previous studies have failed to consistently link laxity and proprioception with functional instability, the barriers limiting recovery is unknown. More sophisticated neurophysiologic technologies, such as microneurography recordings from intact muscle spindles [3] and electroencephalography of sensorimotor cortices [4], offer greater depth to our understanding of the long term disabilities associated with ankle sprains. These techniques can synchronously assess the precise mechanical events of ankle loading, while intercepting direct neural signals along afferent pathways. **By studying how these responses change *throughout* joint loading, it may reveal how this afferent activity becomes decoupled from joint laxity in patients with functional ankle instability and repeated sprains.**

PURPOSE

The purpose of this study was to investigate how the relationship between laxity and peripheral and cortical sensation throughout joint loading changes following ankle joint injury.

METHODS

Study Design: Correlational with group comparison

Dependent Variables:

- Joint laxity (anterior displacement) measured with *arthrometry*
- Muscle Spindle Afferent (MSA) Activity as measured through *microneurography*
- Somatosensory Cortical Activation (CP3/CP4 upper alpha event-related desynchronization) as measured through *electroencephalography (EEG)*

Independent Variables: Group: *Healthy control (CON), Functionally unstable (UNS), Coper (COP)*

PARTICIPANTS

42 able-bodied participants volunteered for this study, with 27 providing data for analysis (Table). Subjects were stratified into groups using the Cumberland Ankle Instability Tool and history of ankle injury. CON had no history of ankle sprain and a CAIT score ≥28; UNS had a history of one or more ankle sprains and a CAIT ≤24; and COP had a history of one or more ankle sprains, but a CAIT score ≥28.[5]

Subject Demographics

	CON	UNS	COP
N (M/F)	11 (5/6)	10 (4/6)	6 (3/3)
Age (yr)	21.9 ± 3.3	20.0 ± 1.1	23.0 ± 4.3
Height (cm)	168.7 ± 10.0	169.5 ± 64.5	172.7 ± 11.7
Mass (kg)	60.1 ± 11.4	64.5 ± 15.5	69.3 ± 12.8

PROCEDURES

Participants reported for a single test session. After providing university-approved informed consent, subjects were seated and fitted with a 32-channel EEG cap (NuAmps, Compumedics Neuroscan, Charlotte, NC) with linked earlobes serving as a ground. Electrodes were positioned in the international 10:20 system and impedance was tested to confirm adequate signal-to-noise ratio (<5kΩ).

Once the cap was fitted, participants were positioned supine on a padded table with the hip and knee of the test-side flexed approximately 30°. A customized instrumented ankle arthrometer (Blue Bay Research, Milton, FL) was affixed to the ankle (Fig 1). Peripheral nerve stimulation was used to identify the location of the common peroneal nerve. A microelectrode connected to a nerve traffic analyzer (Model 662c-3, Univ. Iowa Bioengineering, Iowa City, IA) was then inserted at this location and adjusted until a signal from muscle spindle afferents was confirmed. A reference electrode was placed in the nearby skin.[3]

With EEG and peripheral nerve signals acquired, a series of **50 anterior translations** were applied to the ankle joint (-30 to 130 N, 50N/sec). Five blocks of 10 trials were performed on each side, with one minute of rest provided between each block.[4]

Subject Set-Up

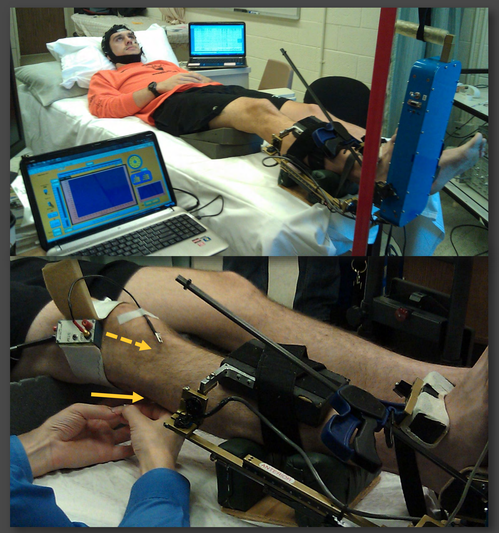

Figure 1. Top: Subject positioning for simultaneous recordings of joint laxity, MSA activity, and cortical activation. **Bottom**: Insertion of microelectrodes (arrows) for recordings of MSA activity.

DATA REDUCTION

Joint Laxity was partitioned from the start to end of each joint load. Peak anterior displacement was extracted for the first (Lax_E) and second (LAX_L) 1000ms of loading.

MSA Activity was amplified (80,000x), bandpass filtered (0.7-2Hz), rectified, and integrated (0.1s). The filtered signal was synchronized with force and displacement data at 100 Hz. Data was partitioned from the start to the end of each joint load and normalized to the ensemble peak. Mean MSA activity was extracted for the first (MSA_E) and second (MSA_L) 1000ms of loading.

Laxity, MSA, and Cortical Activity Across Groups

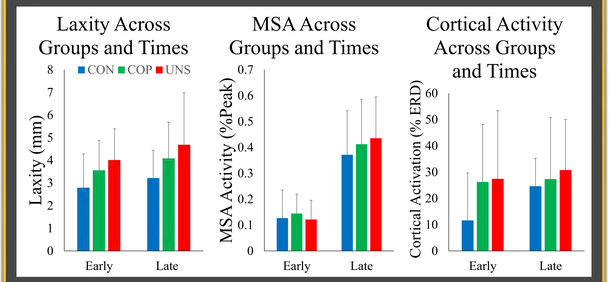

Figure 2. Group results for joint laxity (left), muscle spindle afferent activity (middle) and cortical activation (right) as the joint is loaded. No differences were observed across groups (p>0.05).

Electroencephalography signals were band-pass filtered (0.5 to 30 Hz) and artifacts were corrected and/or removed. EEG data was cut into 4-second epochs surrounding the start of joint loading. Event-related desynchronization (ERD) in the upper alpha (α-2) frequency band was calculated for the CP3 or CP4 electrodes (corresponding with contralateral somatosensory cortex) for the first (ERD_E) and second (ERD_L) 1000ms of loading. **Increased ERD indicates higher somatosensory cortex activity** in these frequencies from a period of quiet baseline (-2000 to -1000 ms prior to loading).

DATA ANALYSIS

Pearson correlation coefficients were used to investigate the relationships between all dependent variables in the first and second 1000ms of loading. Correlation coefficients for each group were converted to z-scores and used to determine groups differences.

RESULTS

Mean values for laxity, MSA, and EEG activity are presented in Fig 2. Lax_E was positively correlated with MSA_L (r=0.58, p=0.04), ERD_E (r=0.55, p<0.01), and ERD_L (r=0.47, p=0.01) across all groups. **UNS ankles displayed a positive correlation between Lax_E and MSA_E and MSA_L, that was significantly different from CON (p=0.02, Fig 3).** CON ankles displayed a positive correlation between LAX_L and ERD_E (r=0.65, p=0.02), and ERD_L (r=0.73, p<0.01, Fig 4).

Significant Correlations between Laxity & MSA Activity

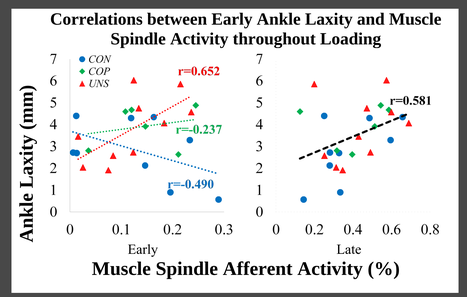

Figure 3. Correlations between early anterior displacement and MSA activity during early (left) and late (right) joint loading. A significant difference between groups was observed for early laxity and early MSA between CON and UNS (p=0.018).

Significant Correlations between Laxity & Cortical Activity

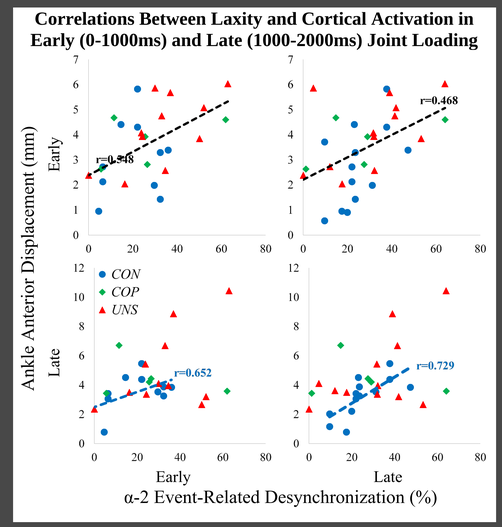

Figure 4. Correlations between joint laxity and cortical activation during early (first 1000ms) and late (second 1000ms) phases of joint loading. Early laxity significantly correlated with early and late cortical activation across all groups (p≤0.011). Late laxity correlated with early and late cortical activation only in CON (p≤0.016).

CONCLUSIONS

- **More lax joints have greater neural activity at peripheral and cortical levels.**
  - While this link was present across all groups for early laxity, the relationship was stronger for previously uninjured ankles throughout the load.
- **Greater early laxity in injured (unstable) ankles correlated with more muscle spindle activity; while the opposite was observed in uninjured ankles.**
  - This may imply fusimotor dysfunction as activation of the muscle spindles should be associated with increases in joint stiffness (α-γ co-activation).[6]
- As deficits following injury appear to be individualized, clinicians should aim to target this dissociation of neuromechanical joint properties for each patient.
- Future research efforts should address how specific rehabilitation techniques may induce beneficial adaptations to re-couple the nervous system's sensation of load with the stiffness properties of the joint.

REFERENCES

1. Hiller, C. E., Nightingale, E. J., Raymond, J., Kilbreath, S. L., Burns, J., Black, D. A., & Refshauge, K. M. (2012). Prevalence and impact of chronic musculoskeletal ankle disorders in the community. *Arch Phys Med Rehabil, 93*(10), 1801-1807.
2. Anandacoomarasamy, A., & Barnsley, L. (2005). Long term outcomes of inversion ankle injuries. *Br J Sports Med, 39*(3), e14; discussion e14. Needle, A. R., Swanik, C. B., Farquhar, W. B., Thomas, S. J., Rose, W. C., & Kaminski, T. W. (2013). Muscle Spindle Traffic in Functionally Unstable Ankles During Ligamentous Stress. *J Athl Train, 48*(2), 192-202.
3. Needle, A. R., Swanik, C. B., Farquhar, W. B., Thomas, S. J., Rose, W. C., & Kaminski, T. W. (2013). Muscle Spindle Traffic in Functionally Unstable Ankles During Ligamentous Stress. *J Athl Train, 48*(2), 192-202.
4. Needle, A. R., Swanik, C. B., Schubert, M., Reinecke, K., Farquhar, W. B., Higginson, J. S., . . . Baumeister, J. (2014). Decoupling of Laxity & Cortical Activation in Functionally Unstable Ankles During Joint Loading. *Eur J Appl Phys*.
5. Hiller, C. E., Refshauge, K. M., Bundy, A. C., Herbert, R. D., & Kilbreath, S. L. (2006). The Cumberland ankle instability tool: a report of validity and reliability testing. *Arch Phys Med Rehabil, 87*(9), 1235-1241. doi: 10.1016/j.apmr.2006.05.022
6. Bergenheim, M., Johansson, H., & Pedersen, J. (1995). The role of the gamma-system for improving information transmission in populations of Ia afferents. *Neuroscience research, 23*(2), 207-215.