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

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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 limited patient outcomes for many, and likely originate from our incomplete understanding of the neuromechanical pathway. Because previous studies have failed to consistently link ankle laxity and proprioception with functional ankle load instability, the barrier limiting recovery is unknown. More sophisticated neuropsychologic technologies, such as microencephalography recordings from intact muscle spindles [3] and electromyography of sensory motor cortices [4] offer greater depth to our understanding of the long term disabilities associated with ankle sprains. These techniques can synchronously assess the mechanical events of ankle loading, while interpreting direct neural signals alongafferent pathways. By studying how these responses change throughout joint loading, it may reveal how this aberrant activity becomes decoupled from joint laxity in patients with functional ankle instability and repeated sprains.

METHODS

Study Design: Correlational with group comparison

Dependent Variables:

• Joint laxity (anterior displacement) measured with arthrometer
• Muscle Spindle Afferent (MSA) Activity as measured through microencephalography
• Somatosensory Cortical Activation (CPS/CPU upper alpha event-related desynchronization) as measured through electromyography (EEG)

Independent Variables:

• Health 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.

RESULTS

Mean values for laxity, MSA, and EEG activity are presented in Fig 2. Laxity was positively correlated with MSAa, (r=0.58, p=0.048), ERDα (r=0.55, p=0.001), and ERDβ (r=0.47, p=0.001) across all groups. UNS ankles displayed a positive correlation between Lax and MSAa, and MSAγ, that was significantly different from CON (p<0.02, Fig 3). CON ankles displayed a positive correlation between LAX and ERDα (r=0.65, p=0.02), and ERDβ (r=0.73, p=0.01, Fig 4).

Data Analysis

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

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 than for ankles with a history of exacerbation.
• Greater early laxity in injured (unstable) ankles correlated with more muscle spindle activity; while the opposite was observed in uninjured ankles.
• This may imply motor 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


Figure 1. Laxity, MSA, and Cortical Activity Across Groups

Subject Set-Up

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.

Figure 1. Top. Subject positioning for simultaneous recordings of joint laxity, MSA activity, and cortical activation. Bottom: Function of microelectrodes (corresponding channel numbers shown in m/s).

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

Figure 3. Correlations between Early ankle laxity and muscle spindle afferent activity throughout loading.

Figure 4. Correlations between Early ankle laxity and muscle spindle afferent activity across groups.

Figure 5. Correlations between joint laxity and cortical activation during early (0-1000ms) and late (1000-2000ms) phases of joint loading. Early laxity significantly correlated with early and late cortical activation across all groups (p<0.001). Late laxity correlated with early and late cortical activation only in CON (p=0.016).

Electromyographic 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 Contralateral (CL) electrodes (corresponding to contralateral somatosensory cortex) for the first (ERDα) and second (ERDβ) 1000ms of loading. Increased ERD indicates higher somatosensory cortical activity in these frequencies from a period of quiet baseline (~2000 to ~1100ms prior to loading).