NEUROMECHANICS OF ANKLE INSTABILITY: REHABILITATION IMPLICATIONS

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Ankle sprains and subsequent chronic ankle instability are common among participants of recreational and competitive athletics. While the initial ankle sprain injury is caused by disruption of one or more of the lateral ligaments, much of the residual disability that patients have is due to sensorimotor deficits. This paper will explore the links between the neurologic and biomechanical sequelae of ankle sprains and provide clinical recommendations for the rehabilitation of these injureis.

Sprains to the lateral ankle ligaments are the most common injury incurred by competitive athletes. While these injuries are often perceived as being innocuous, almost half of individuals who suffer an initial sprain still have residual symptoms one year later.[1] Symptoms including pain, weakness, and sensations of the ankle "giving way" during functional activities are common. Up to one-third of first time ankle sprainers will go on to develop chronic ankle instability (CAI).[1] CAI is operationally defined as a patient having persistent symptoms and sensations of the ankle "giving way" more than 12 months after initial lateral ankle sprain. Repetitive sprains and CAI have been associated with an increased risk of the development of ankle osteoarthritis, diminished physical activity across the lifespan, and reduced self-reported quality of life.[2]

The mechanism of lateral ankle sprains is excessive supination of the rearfoot coupled with external rotation of the lower leg. While often described as "plantar flexion/inversion" sprains, it is important to recognize that excessive internal rotation of the foot is also part of the mechanism of many of these injuries as well. The anterior talofibular ligament (ATFL) is the most commonly sprained lateral ankle ligament, followed by the calcaneofibular ligament (CFL), and the rarely injured posterior talofibular ligament. Ligamentous injury frequently results in mechanical instability of the ankle with ATFL injuries exhibiting excessive anterior drawer and CFL injuries exhibiting excessive inversion talar tilt. Adding an internal rotation component to the anterior drawer maneuver also provides clinicians with an assessment of rotary instability of the talus.

While mechanical instability in the form of excessive joint laxity is evident in many patients after ankle sprain, sensorimotor deficits (i.e., functional instability) are even more prominent. Patients with frank instability of the talocural, subtalar, and/or inferior tibiofibular joints are candidates for surgical stabilization procedures, however the vast majority of acute ankle sprain and CAI patients are treated conservatively. The centerpiece of conservative treatment is neuromuscular training aimed at alleviating the sensorimotor deficits stemming from the ligamentous injury. The interactions between ligamentous injury and sensorimotor deficits have been the focus of considerable research over the past few decades. The purpose of this paper is to describe the neurophysiologic and biomechanical consequences of acute lateral ankle sprains and CAI, and to delineate how rehabilitation may be used to address these consequences and improve patient outcomes.

Neuromechanics of Ankle Instability

The concept of sensorimotor deficits associated with lateral ankle sprains was first described by Freeman and colleagues in the 1960's.[3] They hypothesized that proprioceptive deficits occurred after ankle sprain because of concomitant damage to the sensory receptors located in the injured ligaments, and that subsequently the dynamic control of joint stability by the musculature surrounding the joint was compromised because of inadequate sensory information stemming from the injured joint. It has since become evident that there is a wide

spectrum of sensorimotor changes that are associated with lateral ankle ligament injury. (See Figure 1)

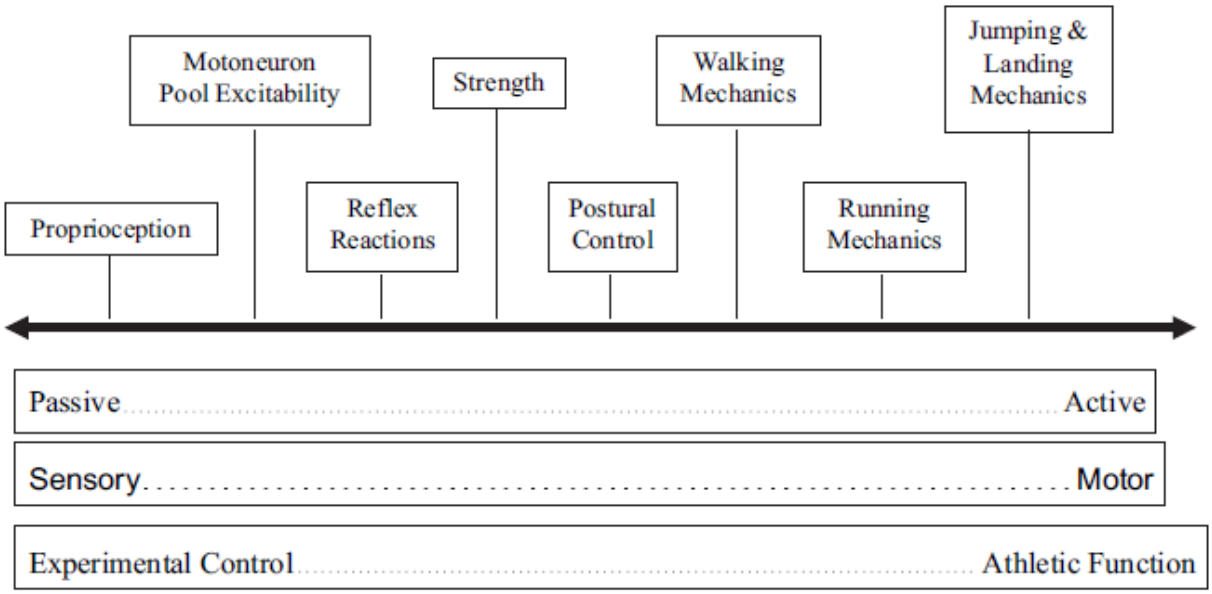
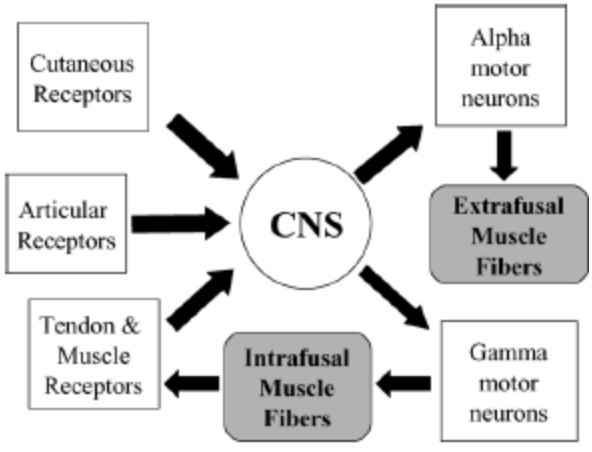


Figure 1. Spectrum of sensorimotor deficits identified in patients with chronic ankle instability.[2]

Proprioceptive deficits associated with ankle ligament injury have traditionally been demonstrated via measures of active or passive joint position sense.[4] Such measures are generally thought to assess the function of joint receptors in the joint capsule and ligaments around the ankle. Impairments of muscle spindle function in the muscles crossing the ankle joint, particularly the peroneal muscles, have also been implicated as source of sensory dysfunction associated with CAI. Most recently, deficits in cutaneous sensory function have also been demonstrated in patients with CAI via diminished vibrotactile sensation of the plantar cutaneous receptors[5] and altered cutaneous reflexes along the distribution of the sural nerve.[6] This range of sensory deficits is likely due to neural plasticity about the entire ankle complex in response to the lateral ligamentous injury. These sensory deficits are linked to alterations in both alpha- and gamma-motoneuron function.[7] (see figure 2)

Figure 2. Model of sensorimotor integration related to joint injury.[7]



Motor control and behavior alterations due to ankle instability have been demonstrated across a wide range of measurement domains indicating the presence of arthrogenic muscle responses to lateral ankle ligament injury. Motoneuron pool excitability deficits, as measured by H:M ratios, have been demonstrated in the peroneus longus and soleus muscles in patients with CAI[8] as has the diminished ability to modulate H-reflexes with increasingly difficult postures.[9] Delayed stretch reflex responses of the peroneal musculature in response to inversion perturbation during quiet standing[10] and walking[8] indicate a delayed dynamic defense mechanism. Not surprisingly, ankle eversion strength deficits have been

clearly documented[11], but weakness of other muscle groups at the ankle as well as proximal strength deficits in the form of hip extension and hip abduction deficits have also been observed in CAI patients.[2]
Balance deficits during quiet unipedal standing have been demonstrated in patients recovering from acute ankle sprain or CAI with a variety of force plate-based measures. Following unilateral acute ankle sprain, an increase in center of pressure velocity has shown bilaterally compared to pre-injury baseline measures indicating centrally-mediated changes in motor control.[12] Similarly, bilateral postural control deficits have also been identified in patients with unilateral CAI utilizing time to boundary measures.[7] Postural control deficits associated with lateral ankle instability have also been demonstrated using the Star Excursion Balance Test, a collection of non-instrumented balance and reach tasks.[13]

Several alterations in gait patterns have been demonstrated among patients with CAI that may indicate why they are at an increased risk of recurrent ankle sprains and episodes of ankle giving way during ambulation. Specifically, we have demonstrated that compared to healthy controls, during treadmill jogging patients with CAI exhibit: 1) a more inverted and plantar flexed rearfoot position in multiple portions of the jogging gait cycle[14], 2) increased plantar pressures on the lateral aspect of the foot[15], and 3) altered coupling relationships between transverse plane shank and frontal plane rearfoot motion as assessed with continuous relative phase analysis.[16] We have also recently found differences in peroneus longus activation during treadmill walking that elucidate adaptations made by CAI patients.[17] The CAI group activated their peroneus longus prior to initial contact whereas the control group activated after initial contact. As a consequence the peroneus longus was activated for a larger percentage of the gait cycle in the CAI group compared to the controls. Similar alterations in kinematic, kinetic, and EMG patterns have been demonstrated in jump landing studies as well.[2]

Rehabilitation Implications
It is apparent that multiple levels of sensorimotor impairment exist in patients with lateral ankle instability. Challenges remain, however, as clinical assessment techniques often lack the sensitivity to identify deficits that have been detected with biomechanics instrumentation such as force plates and motion capture systems. The questions that clinicians must ask are whether specific rehabilitation interventions are necessary for each individual impairment or whether the sensorimotor system as a whole can be restored from a single intervention. While the latter option would be ideal from a time and effort perspective for both patients and clinicians, at this time there is not adequate evidence to support such an approach. Thus, a rehabilitation approach that is based on a systematic assessment of specific impairments and appropriate treatment of identified impairments is recommended.

We have proposed a paradigm based on the tenet of “Assess-Treat-Reassess” in the domains of range of motion (arthrokinematic, osteokinematic), strength (concentric, eccentric), balance (static, dynamic) and functional activities (walking, running, jumping/landing, cutting).[18] There is quality evidence to demonstrate the specificity of rehabilitation interventions specific to each of these domains. For example, in CAI patients passive accessory joint mobilizations are useful for improving dorsiflexion range of motion[19], targeted strength training can improve muscle force production in patients[20], and balance training exercises can improve postural control in patients.[21,22] While the effects of balance training on gait kinematics have been assessed in CAI patients[23], there is currently less evidence for changing gait and landing mechanics than there is for the other assessment domains mentioned previously. Therapeutic exercises that challenge a patient’s postural control are thought to improve sensorimotor function by taking advantage of redundancy in the sensory system while simultaneously improving muscle function about the ankle. Most importantly, however, is the consistent finding that supervised rehabilitation improves

patient-oriented outcome measures such as self-reported function in patients with CAI.[21,22] Continued efforts are needed to better understand the links between the neuromechanical sequelae of lateral ankle instability to clinical interventions to improve patient outcomes.

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