Sensorimotor Deficits Associated with Chronic Ankle Instability

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Abstract

Kim, Kyung-Min, Sensorimotor Deficits Associated with Chronic Ankle Instability, Exercise Science, 23(4): 287-296, 2014. Chronic ankle instability (CAI) is a common debilitating condition in sports medicine that contributes to dysfunction and disabilities. A significant portion of patients with ankle sprains develops CAI, resulting in recurrent injuries and an increased likelihood to develop a degenerative disease like ankle osteoarthritis. CAI may be due to either mechanical or functional insufficiencies, or both. The mechanical contributing factors are pathologic joint laxity, arthrokinematic restrictions, synovial inflammation and impingements, and degenerative changes. The functional insufficiencies include impairments in proprioception, alpha motoneuron pool excitability, reflex actions, strength, postural control, walking and running mechanics, and jumping and landing mechanics, which indicate alterations in the sensorimotor system. It may be intuitive that mechanical disruptions of the lateral ligaments in the ankle joint following an ankle sprain cause changes in joint mechanics, leading to the joint instability, but it is not clear how sensorimotor deficits arise from mechanical injury to the ankle. Recent evidence showed that deficits in postural control and alpha motoneuron recruitment might be more influential, however, each of the sensorimotor deficits play a role in CAI. Thus, the purpose of this review is to discuss the relevant literature surrounding chronic ankle instability with an emphasis on deficits in postural control and alpha motoneuron pool excitability, and to provide insight into rehabilitation for CAI patients suffering from sensorimotor dysfunction.

Key words: muscle activation, balance, ankle sprain
I. Introduction

Ankle sprains have been frequently reported as the most common sports injury in interscholastic and intercollegiate athletes (Fernandez et al., 2007; Fong et al., 2007; Hootman et al., 2007; Yard et al., 2008). These injuries accounted for 15% of all injuries in the 15 collegiate sports studied and are commonly found in athletes participating in basketball, soccer, football, and women’s volleyball and gymnastics (Hootman et al., 2007). Despite the high frequency of this injury 55% of patients with ankle sprains did not seek professional medical care (McKay et al., 2001) indicating a general perception of an ankle sprain as an innocuous injury. There exists, however, ample evidence that an ankle sprain is not a simple injury, and leads to a significant proportion (30-74%) of patients that report prolonged symptoms, self-reported disability, limited physical activity, and recurrent injury for months to years following the injury (Verhagen et al., 1995; Gerber et al., 1998; Braun, 1999; Konradsen et al., 2002; Anandacoomarasamy & Barnsley, 2005; van Rijn et al., 2008). These debilitating sequelae following ankle sprains have been termed as chronic ankle instability (CAI) (Hertel, 2002). It has been characterized by repetitive bouts of the ankle giving way and feeling of ankle joint instability during ankle activities that present for a minimum of one year post-initial sprain (Delahunt et al., 2010). It has been documented that 30% of patients suffering an initial ankle sprain are predisposed to develop CAI (Itay et al., 1982). CAI has been found to be associated with lower quality of life and an increased likelihood to develop ankle osteoarthritis (Anandacoomarasamy & Barnsley, 2005; Valderrabano et al., 2006; Sugimoto et al., 2009).

The development of CAI after initial ankle sprain has been extensively examined in the literature over the past few decades in an effort to understand the etiology of CAI and to develop effective treatment strategies, but there has been a large discrepancy in results from studies investigating CAI (Hertel, 2008; Hiller et al., 2011). A comprehensive review article by Delahunt et al. (2010) pointed out the inconsistent findings across studies assessing sensorimotor deficits in relation to CAI, which were largely attributed to high variability of inclusion criteria to determine whether the subjects have the condition of CAI. This inconsistent criteria may be derived from the lack of a universally agreed upon definition of ankle instability. Particularly, functional instability is typically determined with self-reported ankle symptoms although the definition of mechanical instability is universally accepted as a result of pathologic ligamentous laxity (Hubbard & Hertel, 2006; Hiller et al., 2011). In addition, the terminology describing the condition being investigated varies greatly such as functional instability, functional ankle instability, chronic instability, CAI, chronic lateral instability, chronic ankle sprain, multiple ankle sprains, and recurrent ankle sprain. It is suggested that CAI appears to be the most encompassing term and the most commonly used to describe patients who complain of on-going symptoms after ankle sprains (Delahunt et al., 2010; Gribble et al., 2014).

CAI can be quantified with the incidence rate of ankle joint “giving way” and “feelings of instability”. The giving way refers to “the regular occurrence of uncontrolled ankle joint inversion episodes that do not necessarily result in an ankle sprain” while the feelings of instability refer to “the situation whereby the ankle joint feels vulnerable during daily and sporting activities” (Delahunt et al., 2010). The use of ankle instability questionnaires like the Ankle Instability Instrument (Docherty et al., 2006) and Cumberland Ankle Instability Tool (Hiller et al., 2006) provides an objective way to quantify the perceived ankle instability. Individuals with CAI report limited ankle function, which can be quantified with a validated, reliable, and responsive ankle instrument like the Foot and Ankle Ability Measure (Eechaute et al., 2007; Carcia et al., 2008). Since CAI results from an initial ankle sprain regardless of its severity (Itay et al., 1982), it is requisite that individuals with CAI have a history of at least one ankle sprain. The initial ankle sprain should be older than one year to ensure that the ankle instability is considered as a chronic problem (Delahunt et al., 2010). Furthermore, the time frame elapsed since the most recent ankle sprain should be considered because subjects in an early stage of healing may be undergoing the acute responses of the most recent injury. Practically, anyone suffering a sprain within
6 weeks of the study should be excluded from CAI studies (Delahunt et al., 2010).

CAI has been described as having a multi-factorial etiology. The terms “mechanical instability” and “functional instability” have been widely used in the ankle literature to help describe the etiology of CAI (Hertel, 2002). Mechanical instability involves insufficiencies in pathologic joint laxity, arthrokinematic restrictions, synovial inflammation and impingement, and degenerative changes (Hertel, 2002). Functional instability is related to impairments in proprioception, motoneuron pool excitability, reflex actions, strength, postural control, walking and running mechanics, and jumping & landing mechanics (Hertel, 2008). The mechanical instability is commonly associated with pathologic ligamentous laxity about the ankle joint (Hubbard & Hertel, 2006) while the functional instability seems to be multifarious in nature (Santos & Liu, 2008). A study was conducted to identify specific sensorimotor measures that can predict the group membership of CAI (Selton et al., 2009). Twenty-five sensorimotor variables were measured within four constructs: joint kinesthesia, static balance, dynamic balance, alpha motoneuron pool excitability (αMNPE). The two constructs of static balance and αMNPE were found to accurately classified over 86% of participants with CAI (Selton et al., 2009). The result indicates that deficits in postural control and αMNPE might be more influential than the other factors; however, each sensorimotor deficit plays a role in CAI. Thus, the purpose of the review is to discuss the relevant literature surrounding chronic ankle instability with an emphasis on sensorimotor deficits in postural control and αMNPE, and to provide insights into rehabilitation for CAI patients suffering from sensorimotor dysfunction.

II. Sensorimotor Deficits

1. Postural Control

Postural control in single-leg stance has been recognized as a key risk factor that has been associated with CAI and recurrent ankle injuries. A systematic review found that poor postural control is most likely associated with an increased risk of sustaining an acute ankle sprain (McKeon & Hertel, 2008) and this association becomes more evident in individuals with a history of ankle sprain (McGuine & Keene, 2006). Conversely, this relationship between postural instability and risk of ankle sprain has been demonstrated in studies investigating the prophylactic effects of balance training on injury risk. It was found that improvement in postural control through balance training has substantially reduced the risk of ankle sprains, and it is more effective in individuals with a history of ankle sprain (McGuine & Keene, 2006; Hubscher et al., 2010). In addition, a rehabilitation program with an emphasis on balance training has produced better clinical outcomes such as improved postural control, gait mechanics, and self-reported function in patients with CAI (McKeon et al., 2008; McKeon et al., 2009). Collectively postural stability in single-leg stance appears to play an important role in CAI and recurrent injuries.

Multiple systematic reviews consistently reported that postural control is bilaterally impaired following acute ankle sprains (McKeon & Hertel, 2008; Wikstrom et al., 2010), and postural deficits were also present in the involved limbs of individuals with CAI (Arnold et al., 2009; Wikstrom et al., 2009; Munn et al., 2010; Wikstrom et al., 2010). Postural control impairments have been traditionally attributed to proprioceptive deficits derived from disrupted mechanoreceptors in the injured ligaments following an ankle sprain (Freeman, 1965; Freeman et al., 1965). Sensory structures like proprioceptors play an important role in postural control by providing necessary sensory information. It is intuitive that lack of sensory inputs from damaged mechanoreceptors could cause some level of postural instability. However, this perspective has been challenged with evidence showing postural stability was not impaired despite experimentally induced proprioceptive deficits (Konradsen et al., 1993; Riemann et al., 2004). Konradsen et al. (1993) investigated the effect of a proprioceptive deficit on single-leg stance balance performance. The ankle/foot complex was blocked with local anaesthetic to mimic proprioceptive deficits. It was shown that the magnitude of postural sway during single-leg
stance was unchanged by the anaesthesia. A similar finding was also reported in a study that anesthetized only lateral ankle ligaments (Riemann et al., 2004). The authors attributed the lack of postural impairments after the anaesthesia to the redundancy of sensory information available from other sensory receptors that were not influenced by the anaesthesia (Konradsen et al., 1993; Riemann et al., 2004). These findings suggest that impaired postural control in individuals with CAI may involve alterations in the neuromotor system along with proprioceptive deficits such as decreased alpha motoneuron activities (McVey et al., 2005; Palmieri-Smith et al., 2009) and a potential role of gamma motoneuron system (Hertel, 2008; Palmieri-Smith et al., 2009).

2. Alpha Motoneuron Pool Excitability

αMNPE of muscles stabilizing a joint has been extensively examined in the literature because it provides some insights into how the central nervous system responds to a joint injury or treatment. The αMNPE is defined as the proportion of αMNs available in a given motoneuron pool in the spinal cord (Palmieri et al., 2004a), Hoffmann reflex (H-reflex) is often used to estimate αMNPE in the literature. It is originally described by Paul Hoffmann in 1910 (Zehr, 2002; Palmieri et al., 2004a; Chen & Zhou, 2011), and is considered to be the electrical analogue of monosynaptic stretch reflex that is induced mechanically. Both reflexes are rapid muscular responses to activation of the Ia afferent pathway, but the H-reflex bypasses muscle spindle activity (Schieppati, 1987), allowing one to assess modulation of the monosynaptic reflex activity in the spinal cord. With the H-reflex measurement quantifying αMNPE and ease of methodology it has become common in studies investigating responses of the nervous system to a variety of conditions including neurologic diseases (Tokuada et al., 1991), musculoskeletal injuries (Hopkins et al., 2002; Hopkins & Stencil, 2002; McVey et al., 2005; Palmieri-Smith et al., 2009; Kim et al., 2010; Klykken et al., 2011), application of therapeutic modalities or exercise (Mynark & Koceja, 2002; Palmieri-Smith et al., 2007; Sefton et al., 2007a; Sefton et al., 2007b; Kitano et al., 2009; Chen et al., 2011; Grindstaff et al., 2011; Sefton et al., 2011), and performance of motor tasks (Katz et al., 1988; Llewellyn et al., 1990; Koceja et al., 1993; Koceja et al., 1995; Mynark et al., 1997; Mynark & Koceja, 1997; Koceja & Mynark, 2000; Chalmers & Knutzen, 2002; Bove et al., 2006; Sefton et al., 2008; Pinar et al., 2010).

Decreased αMNPE has been frequently documented in patients with joint injuries or surgery (McVey et al., 2005; Palmieri-Smith et al., 2009; Hart et al., 2010; Kim et al., 2010; Pietrosimone et al., 2011), and this phenomenon, clinically termed arthrogenic muscle inhibition, has been interpreted as on-going reflexive inhibition of the undamaged muscles surrounding a joint after distension or damage to the structures of that joint (Stokes & Young, 1984; Young, 1993; Hopkins & Ingersoll, 2000; Hart et al., 2010; Pietrosimone et al., 2011). This pathological muscle inhibition following a joint injury has been proposed as a limiting factor in joint rehabilitation because it seems to contribute to muscle dysfunction, such as muscle atrophy, weakness, altered gait mechanics, and diminished functional performance (Hopkins & Ingersoll, 2000; Hart et al., 2010; Pietrosimone et al., 2011).

There is emerging evidence that individuals with CAI have decreased αMNPE in ankle stabilizing muscles (McVey et al., 2005; Palmieri-Smith et al., 2009; Kim et al., 2010). McVey et al. (2005) first reported that people with unilateral CAI showed decreased αMNPE, as assessed with H-reflex in the soleus and peroneus longus, but not the tibialis anterior compared to healthy controls. The consistent findings were documented in subsequent studies (Palmieri-Smith et al., 2009; Kim et al., 2010) suggesting the decreased αMNPE in the ankle muscles may be a potential mechanism of muscle dysfunction seen in patients with CAI. On the other hand, one study reported lack of the group difference in the soleus αMNPE associated with CAI (Doeringer et al., 2009). It should be noted that this study compared unilateral measures of αMNPE, as measured with H-reflex between groups, but this group comparison may not be as accurate as a group comparison with bilateral measures due to the high variability in H-reflex measurement among subjects (McVey et al., 2005).
αMNPE of ankle muscles has been also examined in patients with acute lateral ankle sprains and those with artificially effused ankle joints (Palmieri et al., 2004b; Klykken et al., 2011). Palmieri et al. (2004b) assessed the effects of ankle joint effusion on αMNPE of ankle muscles. They hypothesized that ankle muscles become inhibited following the effusion just as the quadriceps do following knee effusion (Iles et al., 1990; Hopkins et al., 2001). Surprisingly, they found the opposite results in that all ankle muscles (soleus, peroneus longus, and tibialis anterior) considerably increased their αMNPE. The authors attributed the facilitation to the role of ankle musculature in stabilizing the foot/ankle complex in order to maintain posture and locomotion. In addition, patients with acute ankle sprains were also found to have increased αMNPE in the soleus, but not in other ankle muscles (Klykken et al., 2011). These findings implicate that αMNPE following an acute ankle sprain may involve different mechanisms following the artificial effusion that are largely influenced by affective activity of articular mechanoceptors. There was a moderate to strong relationship between tibialis anterior αMNPE and current visual analog pain scores in the ankle sprain patients, indicating that pain may play an important role in modulating αMNPE. However, more research is needed to confirm this relationship (Klykken et al., 2011). Collectively, there are conflicting findings on αMNPE of the ankle stabilizing muscles between patients with acute ankle sprains and CAI. Further research is warranted to clarify changes in αMNPE following an acute ankle sprain over the injury recovery.

### III. Clinical Implications

It is known that CAI is a pathological condition that arises from an initial ankle sprain, yet it is not known how CAI develops after the injury. Lack of information about the mechanism of functional instability of the ankle cause a number of treatment strategies, making it difficult to identify the most effective treatment protocol. Nonetheless, CAI patients may benefit more from balance training involving single-leg standing and hopping (McKeon et al., 2008; McKeon et al., 2009; Hubscher et al., 2010). Furthermore, an intervention aimed at restoring αMNPE may provide favorable outcomes on muscle function including joint cryotherapy, transcutaneous electrical nerve stimulation, neuromuscular electrical stimulation, and manual therapy (Hopkins & Ingersoll, 2000; Hopkins et al., 2002; Hopkins & Stencil, 2002; Palmieri-Smith et al., 2007; Palmieri-Smith et al., 2008; Pietrosimone et al., 2009; Pietrosimone & Ingersoll, 2009; Rice et al., 2009; Kim et al., 2011). It is suggested that decreased αMNPE be addressed with these modalities before active rehabilitative exercises begin in rehabilitation since active exercises would not be effective in muscle function when alpha motoneurons innervating muscles to exercise are inhibited (Hopkins & Ingersoll, 2000; Palmieri-Smith et al., 2008). Collectively, there may be a chance to further improve treatment outcomes when the disinhibitory intervention is incorporated into balance training. This possibility is based upon recent evidence that decreased αMNPE may be a neurophysiological mechanism responsible for deficits in postural control associated with CAI (Sefton et al., 2008; Kim et al., 2012; Kim et al., 2013).

### IV. Conclusion

CAI is a common pathological condition in sports medicine that affects one’s bodily function, resulting in disabilities and lower health-related quality of life. CAI may be due to either mechanical or functional insufficiencies, or both. The mechanical insufficiencies are pathologic joint laxity, arthrokinematic restrictions, synovial inflammation and impingements, and degenerative changes. The functional insufficiencies include impairments in proprioception, alpha motoneuron pool excitability, reflex actions, strength, postural control, walking and running mechanics, and jumping & landing mechanics, which indicates alterations in the sensorimotor system. The definition of mechanical instability is universally accepted as a result of pathologic ligamentous laxity while the sensorimotor aspects of CAI (functional instability) remain unclear. Nonetheless, static pos-
tural control and αMNPE may be key factors responsible for deficits in sensorimotor function seen in patients with CAI. It is conclusive that patients with CAI have impairments in postural control with emerging evidence of decreased αMNPE associated with CAI. Further research is warranted to clarify the link between postural control and αMNPE. This line of research would help clinicians and researchers gain a greater understanding of the condition and may lead to conceptualization of optimal treatment and rehabilitation strategies.

References


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