Journal of Bodywork and Movement Therapies (2007) 11, 203–213

Journal of
Bodywork and
Movement Therapies

www.intl.elsevierhealth.com/journals/jbmt

REVIEW

Neuromuscular contributions to functional instability of the ankle joint

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Received 17 January 2007; received in revised form 6 March 2007; accepted 19 March 2007

KEYWORDS
Ankle;
Ligaments;
Sprain;
Chronic ankle instability;
Mechanical instability;
Functional instability

Summary Sprain of the lateral ankle ligaments are one of the most common injuries encountered during athletic participation. Following initial injury there is an alarmingly high risk of re-injury and the development of residual symptoms such as pain, swelling and "giving way" of the ankle joint. These symptoms have been given the generic term chronic ankle instability (CAI). Two causes of CAI reported in the literature are mechanical instability (MI) and functional instability (FI). FI is a distinct phenomenon to MI with many neuromuscular contributing factors being reported. The purpose of this literature review is to describe those neuromuscular factors associated with FI.

Introduction

Inversion sprain of the lateral ligament complex of the ankle joint is one of the most common injuries experienced during sporting participation. In an epidemiological prospective study Hosea et al. (2000) examined the risk of ankle injuries in scholastic and collegiate basketball players during a 2-year period. Eleven thousand seven hundred and eighty athletes were recruited for the study. During the 2-year follow-up period there were 1052 ankle injuries. In a more recent study Beynnon et al. (2005) examined the incidence of ankle injuries in a 4-year study between 1999 and 2003. A total of 901 athletes were included in the study and of those included 43 (4.8%) suffered an inversion injury. Residual functional limitations often complicate a large proportion of initial injuries. It has been suggested that up to 70% of individuals involved in basketball with an initial ankle sprain could suffer from injury recurrence (Yeung et al., 1994). Another study has shown that following an initial ankle joint injury 55% of subjects can still suffer from symptoms at 6 weeks post injury with up to 40% reporting problems at 6 months (Gerber et al., 1998). The development of residual symptoms such as pain, swelling and "giving way" of the ankle joint as well as repeated inversion injury has been termed chronic ankle

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doi:10.1016/j.jbmt.2007.03.002

instability (CAI). Two factors contributing to the development of CAI are mechanical instability (MI) and functional instability (FI) (Hertel, 2002).

Traditionally MI due to sprain of the lateral ligament complex of the ankle has been associated with FI (Lentell et al., 1995). However Tropp et al. (1985) endeavoured to make a distinction between MI and FI, by defining MI as joint motion that exceeds normal physiological limits due to damage of the supporting ligamentous structures, while FI was defined as joint motion that did not necessarily exceed normal physiological limits but which was beyond voluntary control.

Various studies have been undertaken to determine the relationship of MI to FI of the ankle joint. Ryan (1994) performed an anterior drawer and talar tilt test on 45 subjects with unilateral FI to determine the presence or absence of MI. In this study subjects acted as their own controls, with the non-injured ankle acting as the control ankle. The presence or absence of MI was subjectively graded using a 5-point scale ranging from very hypomobile to very hypermobile. The functionally unstable ankle was considered to be mechanically unstable if it was graded very hypermobile or if it received a grading of at least two grades greater than the non-injured ankle on either instability test. On the basis of the study's definition of MI, there were 11 (24%) mechanically unstable ankles amongst 45 functionally unstable ankles. Lentell et al. (1990) in a study to determine the contribution of anatomical laxity of the ankle joint to the development of FI examined the amount of talar tilt in the involved and uninvolved ankles of 34 subjects with the subjective complaint of unilateral FI. Statistical analysis showed that 59% of the subjects had symmetrical talar tilt values, thus indicating that FI may be present in the absence of MI. Birmingham et al. (1997) used an ankle joint stress testing apparatus to determine the maximum inversion range of motion at the ankle joint in 30 subjects with unilateral FI. The non-injured ankle acted as the control ankle. The group mean value for inversion range of motion did not differ between the injured and non-injured ankles, again suggesting that FI may be a distinct entity to MI, and both conditions do not necessarily coexist.

The results of the above studies indicate that patients can thus have a history of recurrent inversion sprains with concomitant functional limitations and still present clinically with mechanically stable ankles. In those patients who complain of FI and a tendency of the foot to "give way" but in whom objective measures reveal a stable ankle, some pathological process distinct from MI is obviously present. Thus the purpose of this article is to describe those neuromuscular factors which have been reported in the literature to be associated with the development of FI (Figs. 1 and 2).

Balance deficits

Various mechanoreceptors are present in joint capsules, ligaments, muscles, and skin around the ankle (Wyke, 1967). Mechanoreceptors provide afferent impulses regarding joint movement and position as well as contributing to a complex reflex system, which acts to maintain a state of bodily equilibrium. It has been suggested that damage to

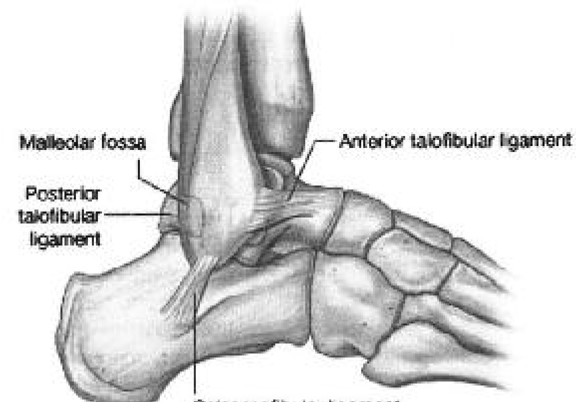

Figure 1 Lateral ligaments of the ankle. *Source*: Drake, R., Vogl, W., Mitchell, A., 2005. Gray's Anatomy for Students, p. 564, Fig. 6.98 A&B. Elsevier, Philadelphia.

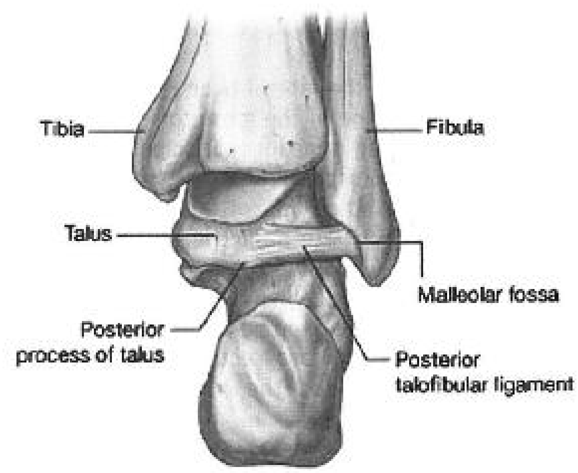

Figure 2 Posterior ligaments of the ankle. *Source*: Drake, R., Vogl, W., Mitchell, A., 2005. Gray's Anatomy for Students, p. 564, Fig. 6.98 A&B. Elsevier, Philadelphia.

mechanoreceptors following ankle sprain could interrupt the flow of these afferent impulses into the central nervous system, thus leading to balance deficits, and ultimately contributing to the development of FI (Freeman et al., 1965). This observation of balance deficits in individuals with FI observed by Freeman et al. (1965) was based upon the subjective opinion of the examiners and the patients' self-perception of balance performance when standing on the affected and unaffected legs.

Garn and Newton (1988) undertook a study to determine whether the one-legged standing balance test could be used to determine whether subjects with a history of recurrent ankle sprain exhibited greater balance deficits when standing on the injured leg as compared with the uninjured leg. They used the same test as described by Freeman et al. (1965). A one-legged standing balance test (eyes open and eyes closed) was used to compare balance when the subject stood on the uninjured ankle and then on the injured ankle. An independent observer was used to determine which side demonstrated impaired balance performance or whether balance performance appeared to be the same on both legs. There was a higher incidence of balance performance deficits noted while subjects stood on their injured leg. A similar testing protocol has been used by both Lentell et al. (1990) and Mulloy Forkin et al. (1996). In the study by Lentell et al. (1990) 15 (45%) subjects demonstrated no differences in balance from one limb to the other. However the remaining 18 (55%) subjects visually demonstrated notable balance performance differences from one side to the other. In 17 of the 18 subjects the deficit was noted while standing on the injured limb. Using a similar test protocol, Mulloy Forkin et al. (1996) reported that 63% of gymnasts (2 males, 9 females) with FI tested, exhibited balance performance deficits during eyes-closed single-leg stance.

Tropp et al. (1985–1986) in a series of investigations were one of the first investigators to try and advance the idea put forward by Freeman et al. (1965), by using stabilometry to objectively quantify the association between balance deficits and FI. They were concerned with the excursion of the centre of pressure (COP) during single-leg standing with the eyes open. One of their initial studies showed that soccer players with a history of FI showed significantly higher stabilometric results when compared to a group of soccer players without ankle instability (Tropp et al., 1985). In a subsequent study Tropp (1986) reported that there was no significant difference in stabilometric values between the affected and unaffected legs of soccer players with unilateral FI. However, a comparison of stabilometric values for both legs of the FI group with a healthy non-injured reference group revealed significantly higher values of COP excursion. In contrast to this result several studies have failed to detect differences in balance performance between FI subjects and healthy non-injured controls. Isakov and Mizrahi (1997) recorded the anteroposterior and mediolateral components of ground reaction force (GRF) during single-leg standing in a group of 8 competitive female gymnasts. They failed to demonstrate any differences between the injured and non-injured limb with either eyes open or eyes closed. Furthermore Baier and Hopf (1998) also did not find any significant differences in balance performance as measured by the anteroposterior and mediolateral components of the COP in a group of 22 athletes with FI when compared to a control group of 22 non-injured athletes. Studies by Kinsella and Harrison (1998) and Bernier et al. (1997) have also failed to show any differences between FI subjects and control subjects during single-leg stance.

When considering the results observed above one must be aware that the one-legged balance test is a relatively static task. It has been suggested that as most joint receptors are only active near the end range of motion, a more dynamic method may be necessary for neural discharge of joint mechanoreceptors (Wilkerson and Nitz, 1994). Thus when the supporting surface beneath the foot is level, the cumulative effect of gravity, momentum, GRFs, and muscle forces on ankle motion will not be equivalent to that produced on an unstable surface.

Various authors have endeavoured to address this issue. Olmsted et al. (2002) used the Star Excursion Balance Test (SEBT), a valid and reliable tool (Kinzey and Armstrong, 1998; Hertel et al., 2000) to detect deficits in subjects with ankle instability. They found that ankle instability subjects demonstrated significantly decreased reach while balancing on the injured side compared to the matched side of the non-injured group, and when compared with their own uninjured side. The authors concluded that static single-leg balance tests may not be sensitive enough to detect motor-control deficits related to balance performance, and that dynamic test such as the SEBT provide a means of identifying functional deficits related to balance performance in subjects with functional ankle instability (FAI). Gribble et al. (2004) have subsequently advanced the work of Olmsted et al. (2002) by also quantifying maximal ankle-dorsiflexion, knee-flexion and hip-flexion angles during the performance of the SEBT. The results of this study showed that the injured limb of the ankle instability group exhibited smaller reach distance values, knee-flexion and hip-flexion angles for all directions tested compared to their non-injured limb and the matched limb of the control group. Hiller et al. (2004) examined the ability of highly trained ballet dancers to regain their balance after an external unexpected perturbation in both a foot flat and a demi-pointe position. In the foot flat position, the time taken to regain baseline oscillation after a sudden unexpected perturbation of 15° of inversion was significantly longer for the FI subjects when compared to the control group. There was no difference between the groups for the demi-pointe position. A recent study by Ross et al. (2005) has shown that subjects with FI exhibit a longer time to stabilization while performing a single-leg jump landing compared to a non-injured control group. The authors concluded that subjects with FI do suffer from deficiencies in dynamic stabilization, and that measures of time to stabilization following a jump landing technique could be used in the clinical setting as an outcome measure to guide return to physical activity in subjects with FI. Hertel and Olmsted-Kramer (2007) have also shown that subjects with FI suffer from postural control deficits. In this study the authors examined time-to-boundary measures of postural control and consistently found that subjects with FI had lower scores compared to a non-injured control group, thus indicating that this measure of postural control may be more capable than traditional measures of postural control, such as the excursion of the COP in detecting balance deficits in subjects with FI.

It has been suggested that static conditions such as single-leg balance tests may fail to elicit postural control deficiencies due to the ease of the testing procedure (Riemann et al., 1999). Consequently, their value in assessing patients in the clinical setting must be questioned. More dynamic testing procedures may thus be necessary to detect balance performance deficits in subjects with FI. Furthermore static balance exercises for the rehabilitation of balance performance may not be an adequate challenge for the fine tuning of the postural control system, and thus more challenging exercises such as the SEBT and jump landing stabilization drills may be more valuable rehabilitation tools. A recent study by Ross and Guskiewicz (2006) has shown that dynamic coordination training performed on unstable surfaces (foam and wobble board) can enhance dynamic postural stability as measured by the time-to-stabilization in subjects with FI.

Strength deficits

A potential cause of FI, historically cited involves weakness of the peroneal musculature (Bosien et al., 1955; Staples, 1972). The peronei act as the primary evertors of the ankle and hence weakness of this muscle group may impair the ability to dynamically control inversion stresses, thus rendering the ankle vulnerable to inversion sprain. However, some of the evidence supporting weakness of the peronei is based on manual muscle testing which is a crude and subjective form of muscle strength evaluation (Munn et al., 2003). Strength training of the peronei forms a central component of treatment programs for FI (Caulfield, 2000), thus indicating that evertor weakness is regarded by many clinicians as a significant factor in the development of functional problems following lateral ligament complex ankle sprains.

Lentell et al. (1990) used an isokinetic dynamometer to investigate the peak torque of the ankle invertors and evertors at the speeds of 30°/s and 0°/s in the injured and uninjured legs of subjects with unilateral FI. They failed to show a difference either isometrically or isokinetically between sides. However, the authors did acknowledge the potential limitations of their study, conceding that, during activity most ankle sprains occur in a closed kinetic chain at velocities above 30°/s. Therefore there may be difficulty with extrapolating the results to the conditions under which the majority of ankle sprains occur. The issue of velocity was addressed by Lentell et al. (1995) in another study where the testing velocities utilized were 30°/s, 90°/s, 150°/s and 210°/s. The results of this study also indicated that evertor strength deficits were not prevalent in subjects with FI, which is further supported by the work of Bernier et al. (1997) and McKnight and Armstrong (1997). Ryan (1994) also failed to show the presence of evertor strength deficits in a group of subjects with unilateral FI. Interestingly, however, there was a decrease in the peak torque of the ankle invertors on the injured side compared to the non-injured side. The presence of invertor strength deficits in FI subjects has also been noted by Wilkerson et al. (1997), who demonstrated significantly greater invertor deficits than evertor deficits for both peak torque and average power using isokinetic dynamometry testing at speeds of 30°/s and 120°/s. The idea of the presence of evertor strength deficits in subjects with FI was refuted by Kaminski et al. (1999) who found no significant differences in concentric, eccentric or isometric eversion ankle strength in a group of subjects with FAI, when compared to a non-injured control group. Munn et al. (2003)

investigated concentric and eccentric ankle strength using isokinetic dynamometry at speeds of 60°/s and 120°/s to determine whether eccentric strength deficits may contribute to the development of FI. They like Ryan (1994) and Wilkerson et al. (1997) found significant deficits in eccentric invertor strength.

The literature seems to suggest that evertor strength deficits do not form a significant factor in the development of functional problems following lateral ligament complex ankle sprains and thus traditional strength training of the ankle joint evertors following an ankle sprain with resistance elastic may be of little clinical and functional importance. However ,it may be interesting in the future to investigate the nature of evertor musculature endurance as the evertors may undergo fatigue with extended periods of activity, thus leading to loss of dynamic support and thus a predisposition to inversion sprain. Perhaps of more importance than evertor strength is the timing of evertor muscle activity during functional activities in subjects with FI. Studies by Caulfield et al. (2004) and Delahunt et al. (2006a, b) have shown that subjects with FI have decreased pre-initial contact peroneus longus integral muscle activity during jumping. Furthermore the study by Delahunt et al. (2006a, b) has also shown that during a jump landing procedure, subjects with FI also concomitantly exhibit an increased inverted position of the ankle joint which leaves the ankle joint vulnerable to an unexpected initial contact (i.e. landing on an opponents foot or landing on an uneven surface) and subsequent inversion sprain. Another study by Delahunt et al. (2006a, b) has shown that during treadmill walking subjects with FI exhibit an increased inverted position of the ankle joint prior to, at, and immediately following heel-strike. However, during this study the subjects with FI were shown to have increased post heel-strike peroneus longus integral muscle activity. The authors concluded that this increase in peroneus longus muscle activity post heel-strike may be the results of changes in feed-forward neuromuscular control in an effort to protect the ankle joint against the increased inverted position observed at the ankle joint. The difference between the peroneus longus muscle activity observed in the aforementioned studies may be the result of differences in ankle joint loading rates. Walking is an everyday activity and the number of steps taken each day may be sufficient to train feed-forward protective and corrective mechanisms to counteract the potentially injurious inverted position of the ankle joint; while jump landing is not an everyday occurrence and consequently protective mechanisms may not be reinforced to counteract the inverted position of the ankle joint noted in subjects with FI during this activity.

There is a growing body of research to suggest that the presence of eccentric invertor strength deficits may play a significant role in the development of residual symptoms following lateral ligament complex ankle sprains.

The presence of invertor strength deficits has been suggested to occur due to selective inhibition (Ryan, 1994). The process of selective inhibition was described by Swearingen and Dehne (1964), who postulated that decreased stress tolerance of an injured joint triggers reflexive mechanism which, inhibit muscles that are capable of increasing tensile stress on damaged ligaments. Thus the invertors of the ankle may be inhibited due to their ability to initiate movement in the direction of initial injury.

Wilkerson et al. (1997) have proposed that the invertor muscles may play a significant role in preventing loss of postural stability over a fixed foot. When the centre of mass is displaced over a fixed foot with both its medial and lateral borders anchored, the shank moves laterally resulting in closed chain eversion. When the centre of mass is displaced beyond the lateral border of the foot and the limit of closed chain eversion is reached, the medial border of the foot will begin to rise, subsequently resulting in a rapid inversion of the foot. Hence, eccentric activity of the invertor muscles, which control lateral postural stability, may play a significant role in the maintenance of dynamic ankle stability. Thus, if the invertors are weak there may be a predisposition to inversion sprains. The role of invertor strength deficits in the development of FI warrants further investigation.

Proprioceptive deficits

Proprioception as defined by Rowinski (1990) is the cumulative neural input to the central nervous system, from mechanoreceptors in the joint capsules, ligaments, muscles, tendons and skin. The coded proprioceptive information conveyed to the spinal cord from the mechanoreceptors passes through multi-synaptic interneuronal connections and eventually results in excitation or inhibition of motor neurons (Newton, 1982). Freeman et al. (1965) have hypothesized that because the tensile strength of the mechanoreceptors is less than the connective tissue within which they are embedded, these mechanoreceptors must be disrupted when ankle ligaments and capsules are torn or stretched. Subsequently Freeman et al. (1965) theorized that disruption of these mechanoreceptors results in decreased sensory input to the CNS which may in turn lead to faulty positioning and diminished reflex responses, thus leading to an increased incidence of recurrent ankle sprain. The assessment of joint proprioception can be divided into two components, namely kinaesthesia and joint position sense (Lephart and Henry, 1996; Lephart et al., 1998). Kinaesthesia is measured by assessing the threshold-to-detection of passive motion (TTDPM), while joint position sense is measured by assessing the reproduction of passive and active joint positioning (Lephart and Henry, 1996; Lephart et al., 1998).

Joint position sense

Gross (1987) undertook a study to examine the effects of recurrent lateral ankle sprains on subjects' active and passive judgements of joint position sense. The test procedure required subjects to actively and passively replicate predetermined ankle joint positions in the inversion–eversion range of motion. The results of this study found that ankle sprain had no significant effect on judgements of joint position. In contrast Jerosch and Bischof (1996) have reported that subjects with a history of recurrent ankle sprains exhibit a deficit in active replication of joint position in the inversion range of motion; while Boyle and Negus (1998) observed a deficit in passive joint position replication in the planter flexion–inversion range of motion.

Kinaesthesia

Garn and Newton (1988) undertook a study to determine whether subjects with a history of recurrent ankle sprain displayed deficits in the detection of passive plantar flexion in the injured ankle joint when compared to the non-injured ankle joint. Results of the study indicated that subjects had significantly greater difficulty detecting passive motion in the injured ankle compared to the non-injured ankle, a finding which has been supported by Mulloy Forkin et al. (1996). A similar result was reported by Lentell et al. (1995) for the inversion range of motion. In contrast to these studies, two more recent investigations have shown that the ability to detect passive plantar flexion and dorsiflexion and inversion and eversion is not impaired in subjects with FAI (Refshauge et al., 2000; Hubbard and Kaminski, 2002).

When considering the results of the studies outlined above one must be aware of the following limitations. All of these studies, except that of Boyle and Negus (1998) investigated uniplanar movements. Ankle and foot motion rarely involves uniplanar motion and thus these studies may not be accurate indicators of joint position sense during high-speed functional activities. All of the studies outlined above have involved assessment of the ability to perceive open-chain passive joint position or the ability to actively reproduce a chosen open-chain joint position. The most prevalent mechanism of injury of the lateral ligament complex of the ankle is one of inversion, plantar flexion and internal rotation (Garrick, 1977). This position involves a compressive loading of the joint surfaces in a weight-bearing position; hence the studies outlined above have not replicated this injury mechanism in terms of injurious range of motion, and thus must be interpreted with caution. Furthermore the angular velocities utilized in the studies do not replicate the angular velocities, which occur during functional activities and hence the results of the studies must be interpreted with caution. Actively contracting muscles provide kinaesthetic information that is not present when a joint is moved passively (Garn and Newton, 1988). Thus even if an individual presents with kinaesthetic deficits in passive movements, compensatory kinaesthetic mechanisms may exist to enable the individual to participate in functional activities without apparent movement or balance problems. Evidence for this compensatory mechanism has been reported by Konradsen et al.(1993) whom have shown that anaesthesia of the ankle and foot complex rendered assessment of passive ankle position impossible, fully blocking afferent input to the CNS from capsular and ligamentous mechanoreceptors. However, active joint position sense persisted, indicating that this sense was sub-served by receptors in the musculature proximal to the anaesthetized area.

The role proprioceptive deficits in the development of FI has yet to be fully elucidated and thus further studies are warranted to determine the exact contribution of proprioceptive deficits to functional performance deficits in subjects with FI. A study by Eils and Rosenbaum (2001) has shown that a 6-week multi-station proprioceptive exercise program enhanced joint position sense as measured by a passive angle reproduction test in subjects with FI. The same exercise program was also shown to improve postural sway measures and muscle response times to an inversion perturbation. Perhaps of more interest was that a 1-year follow-up, FI subjects reported a reduced frequency of ankle joint inversion injury of almost 60%. The results of this study are further supported by a study by Clark and Burden (2005) who have shown that a 4-week wobble board exercise program produced a significant improvement in FI subjects' perception of ankle stability when measured by the Ankle Joint Functional Assessment Tool questionnaire as well as a reduction in the response time of the peroneus longus muscle to inversion perturbation.

Peroneal reflex stabilization deficits

Freeman et al. (1965) have proposed that the basic mechanism of ankle instability following ankle injury develops due to the lesion of mechanoreceptors in the joint capsule and ligaments surrounding the ankle. This theory proposed by Freeman et al. (1965) is known as articular deafferentation. According to this theory, dynamic stability of the ankle joint is dependent upon the ability of the evertors (peronei) to react quickly to sudden inversion perturbations, to develop sufficient tension to prevent injurious ranges of ankle motion, and thus prevent sprain of the lateral ligament complex of the ankle. Individuals with FI could have delayed and diminished reflex responses in the evertor muscle of their affected ankle in reaction to an inversion stress due to altered capsular and ligamentous afferent input. It has been suggested that an increase in the response time of the peronei to sudden inversion may have highly significant consequences in terms of risk of injury to the lateral ligament complex of the ankle (Wilkerson and Nitz, 1994). Numerous studies have investigated this hypothesis, with the main focus of the studies being the analysis of the reaction of the peronei to sudden unexpected inversion stresses.

Isakov et al. (1986) used a uni-axial tilting platform to induce sudden unexpected inversion while concomitantly recording peroneal EMG activity. The results of this study found no difference in the peroneal reaction times when control group profiles were compared to those of a group of subjects with recurrent ankle sprains. Furthermore no significant differences in mean reaction times between the involved and uninvolved ankles in the subjects with recurrent ankle sprains were found. Konradsen and Ravn (1990) utilized a similar testing method to that of Isakov et al. (1986) to examine and compare peroneal reaction time to sudden joint motion in a group of subjects with FI and a group of control subjects without ankle instability. The FI group exhibited significantly longer peroneus longus and peroneus brevis reaction times to the inversion stress. Thus the authors concluded that the increase in peroneal reaction time points to a reflex deficit in the peripheral reflex stabilization of the ankle. Hence the results of this study substantiate the theory of a proprioceptive deafferentation being responsible for FI as proposed by Freeman et al. (1965). Karlsson and Andreasson (1992) when comparing the reaction times of the peronei to sudden inversion found that the involved limbs of individuals with unilateral CAI demonstrated significantly longer peroneus longus and peroneus brevis reaction times when compared to the uninvolved limb, thus supporting the results of Konradsen and Ravn (1990). Ebig et al. (1997) examined the EMG response time of the peroneal and tibialis anterior muscles in response to sudden plantar flexion/inversion stress in a group of subjects with FI—subjects acted as their own controls. The results of this study indicated no significant differences between the stable and unstable ankles for the peroneal and tibialis anterior muscles. The authors concluded that the results of the study suggest that the self-reported functional ankle instability may not result in a diminished reflex response time of the peroneal and tibialis anterior muscles to sudden plantar flexion/inversion stress.

The studies of Konradsen and Ravn (1990) and Karlsson and Andreasson (1992) support the theory of articular deafferentation put forward by Freeman et al. (1965), while the studies by Isakov et al. (1986) and Ebig et al. (1997) refute this theory. Thus there appears to be little agreement in the literature as to whether there is really a delayed peroneal response time to sudden unexpected inversion stress.

More recent studies suggest that Freeman et al.'s (1965) theory of articular deafferentation may not be the main physiological mechanism underlying the development of FAI. Konradsen et al. (1993) investigated the peroneal reflex reaction time to sudden ankle inversion before and after regional block of the ankle and foot with local anaesthetic. Anaesthesia of the ankle totally blocked the afferent input from mechanoreceptors in the ligaments and capsule of the ankle. The peroneal reaction time to sudden ankle inversion was not altered. Articular deafferentation made no difference to response time. The authors concluded that afferent input from the active calf musculature is responsible for dynamic ankle protection against sudden ankle inversion.

The ability of the peroneal musculature to provide dynamic ankle protection against sudden unanticipated ankle inversion has been questioned (Ashton-Miller et al., 1996; Konradsen et al., 1997). After the delay due to neural latencies, which typically range from 85 to 90 ms until myoelectric activity is first observed, there is an additional delay because muscle contractile mechanics dictate that a further 90 ms is required by a muscle to develop contractile force to even half-maximal levels (Ottaviani et al., 1995). Konradsen et al. (1997) has shown that unlimited subtalar inversion from a standing position would put the lateral ligament complex at risk of sprain after approximately 100 ms. Thus Konradsen et al. (1997) have concluded that the ankle musculature cannot react fast enough to protect an ankle from injury in the case of sudden unanticipated inversion stress. It therefore seems that FI is not simply the result of a disorder of peripheral reflex control of ankle stability. In the case that ankle inversion occurs in less than 100 ms, it is clear from the two consecutive time delays aforementioned (85 and 90 ms) that the evertor musculature must be activated prior to the onset of the external forces during ground contact to provide dynamic ankle stability, thus indicating that a higher level of motor control exists to afford dynamic ankle joint stability.

Altered arthrokinematics and arthrokinetics during dynamic activity

It has been suggested that inappropriate positioning of the foot and ankle joint complex could be a potential contributing factor to the development of inversion sprain of the lateral ligament complex of the ankle joint (Tropp, 2002). Garrick (1977) has suggested that the most common mechanism of sustaining an inversion injury involves a compressive loading of the foot/ankle complex in a position of inversion, plantar flexion and internal rotation. Using a muscle model driven computer simulation Wright et al. (2000) have shown that an increased touchdown plantar flexed position of the ankle joint increases the susceptibility of inversion sprain. However, the limitation of this study lies in the fact that it was a computer simulation model and extrapolation to in vivo conditions is purely conjecture. Caulfield and Garrett (2002) examined the lower limb sagittal plane movement during a single-leg drop jump in a group of non-injured control subjects and a group of subjects with FI. Results of this study showed that FI subjects had a greater time averaged dorsiflexed position of the ankle joint during the time period from 10 ms pre landing to 20 ms post landing. FI subjects also had a greater time averaged knee flexion than controls during the time period from 20 ms pre landing to 60 ms post landing. Based on the timing of the differences noted the authors concluded that the observed differences noted in the FI group were likely to be the result of changes in central patterning rather than reflex control. In a later study the same authors investigated patterns of GRF in subjects with FI compared to a non-injured control group during a single-leg drop jump (Caulfield and Garrett, 2004). FI subjects differed from control subjects in terms of peak lateral and anterior GRF components as well as time averaged vertical, frontal and sagittal components of GRF. The observed differences were manifested immediately post landing and too early for reflex correction to take place. Thus the authors concluded that the observed differences in the various components of GRF could lead to abnormal stress being applied to the ankle joint leading to repeated injury to the supporting structures of the ankle joint. From the data presented above it appears that during jump landing procedures that subjects with FI suffer from deficits in arthrokinematics and arthrokinetics, which could render the functionally unstable ankle joint more susceptible to further injury.

Ankle sprains do not only occur during landing from a jump. It is common place for subjects with FI to experience episodes of "giving way" of their ankle joint during activities with slight or no external provocation, such as during gait (Konradsen, 2002). Using a cadaver study of simulated gait Konradsen and Voigt (2002) showed that if the foot is inverted to such an extent that causes the lateral edge of the foot to collide with the ground, the propulsive energy of the lower extremity could force the ankle/foot complex to rotate into 40° of inversion 40° of plantar flexion and 30° internal rotation, resulting in a hyperinversion injury and subsequent sprain of the lateral ligament complex of the ankle joint. In a more recent study Monaghan et al. (2006) have shown that subjects with FI exhibited altered ankle joint kinematics and kinetics during overground walking at their self-selected velocity. In this study the subjects with FI were found to have a more inverted position of the ankle joint prior to and immediately following heel-strike compared to a non-injured healthy control group, as well as a concentric evertor moment, while control subjects exhibited an eccentric invertor moment following heel-strike. The observed differences in inversion range of motion between the FI and control group could leave the FI group vulnerable to inversion sprain, as Tropp (2002) has suggested that following heel-strike, the line of action of the GRF is dependent upon the position of the foot in relation to the centre of gravity and inertia. Thus if the ankle joint is held in a more inverted position when heel-strike occurs, an external inversion load is placed upon the ankle joint, thus increasing the potential for a hyperinversion injury and sprain of the lateral ligaments.

During the rehabilitation stage following an inversion sprain, gait training may be an overlooked component of rehabilitation. Braces may be an important component of rehabilitation protocols following injury to the lateral ligament complex of the ankle joint if they can function to help prevent the potentially injurious excessive inversion range of motion noted in subjects with FI.

Conclusion

FI may occur without any objective signs of MI of the ankle. Dynamic balance deficits do seem to be a consistent finding in individuals with FI, and thus rehabilitation protocols should endeavour to incorporate exercises which will challenge the postural control system. Recent reports suggest that dynamic postural control exercises such as training on wobble boards and uneven surfaces can produce significant improvements in dynamic balance in subjects with FI. The role of proprioceptive deficits in the development of FI has yet to be fully elucidated and thus warrants further investigation. There seems to be no specific strength deficits in the evertors of the ankle joint but strength deficits are apparent in the invertor musculature. Thus rehabilitation protocols should incorporate eccentric strength training of the invertor musculature. There is mounting evidence to suggest that the timing of evertor muscle activity may be critical in the development of residual symptoms following an inversion injury episode. Furthermore the physiological mechanisms underlying these deficits do not appear to be simply a disorder of articular deafferentation leading to decreased peroneal reflex stabilization, and thus the role of changes in central motor programmes warrants further intensive study. Rehabilitation protocols should place emphasis of activities that can enhance pre-programmed feed-forward synergistic muscle activity. Activities such as training correct timing and execution of landing techniques could be successful in achieving this objective.

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