Functional Instability Following Lateral Ankle Sprain

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Contents

Abstract

Lateral ankle sprain (LAS) is an extremely common athletic injury. Despite extensive clinical and basic science research, the recurrence rate remains high. Functional instability (FI) following LAS is hypothesised to predispose individuals to reinjury because of neuromuscular deficits which result following injury. This paper provides an overview of the potential causes of FI which may manifest themselves clinically. The theoretical explanations of FI are discussed, as are implications for assessment and treatment of FI following LAS.

When LAS occurs, structural damage not only occurs to the ligamentous tissue, but also to the nervous and musculotendinous tissue around the ankle complex. While injury to the ligaments may result in laxity of the joints of the ankle complex, neuromuscular deficits are also likely to occur due to the injury to the nervous and musculotendinous tissue. These neuromuscular deficits may be manifested as impaired balance, reduced joint position sense, slower firing of the peroneal muscles to inversion perturbation of the ankle, slowed nerve conduction velocity, impaired cutaneous sensation, strength deficits and decreased dorsiflexion range of motion. Additionally, the abnormal formation of scar tissue after

injury may lead to sinus tarsi syndrome or anterolateral impingement syndrome, which may also lead to FI of the ankle complex.

Assessment of patients with LAS must address not only joint laxity and swelling, but should include examination for neuromuscular deficits as well. The treatment and rehabilitation goals must also address restoration of neuromuscular function, as well as restoration of mechanical stability to the injured joints.

Lateral ankle sprain (LAS) is one of the most common injuries experienced in sport. The recurrence rate for LAS among athletes has been reported to be as high as 80%.[1,2] Functional instability (FI) stemming from neuromuscular and proprioceptive deficits is hypothesised to be a major contributing factor to chronic ankle instability.[3-5] Researchers have demonstrated the existence of FI through a variety of assessment modalities, including impaired balance, diminished joint position sense, delayed peroneal muscle contraction to inversion perturbation, altered common peroneal nerve function and strength deficits. Prevention of recurrent LAS must begin with assessment of the neuromuscular and proprioceptive function of the injured extremity. Rehabilitation of LAS must emphasise restoration of any neuromuscular and proprioceptive deficits.

1. Overview of Functional Instability

Injury to the lateral ankle ligaments is extremely common in athletics.[6] The ligaments which support the lateral aspect of the talocrural and subtalar joints are often sprained following hypersupination of the ankle complex. Recurrence of LAS is very common. Mechanical instability (MI) and FI have both been hypothesised as causes of recurrent LAS. MI refers to laxity of a joint due to structural damage to ligamentous tissues which support the joint. MI may affect the talocrural, subtalar and/or inferior tibiofibular joints following LAS. However, a high proportion of individuals complaining of recurrent LAS do not exhibit gross laxity of any of these joints on physical examination. These individuals are suspected of having FI.

The lateral ligaments and joint capsule of the talocrural and subtalar joints have been shown to be extensively innervated by mechanoreceptors.[7-9]

A disruption of the sensory receptors within the lateral ligamentous structures is believed to result in a decreased ability to sense changes in joint position. Mechanoreceptors are most active in the sensation of joint movements near the ends of ranges of motion.[10] Mechanoreceptors sense increased tension in the ligaments and send an afferent message to the spinal cord. In response, an efferent response is sent to the muscles which can slow or reverse the direction of joint movement. For example, as the ankle is supinated close to its terminal range, the mechanoreceptors in the lateral ligaments become stimulated and an afferent message is sent to the spinal cord. In response, an efferent signal is sent to eccentrically contract the peroneal muscles in an effort to slow the rate of inversion. If the contraction is strong enough the ankle may actually begin to evert.[10]

The lateral ligaments of the ankle are generally thought to heal well as new collagen tissue is laid down following injury. This results in the appearance of a mechanically stable ankle after healing has taken place. However, little is known about the healing of mechanoreceptors and afferent nerve fibres following ligamentous injury. Nervous tissue is known to heal much more slowly than other body tissues. This may result in the presence of a mechanically stable ankle which lacks the ability to accurately assess joint position.

Receptors in muscles and tendons which cross the ankle joint can also sense joint movement and position, and they normally work in concert with the joint mechanoreceptors. Rehabilitation efforts for patients assessed with FI of the ankle have focused on the accentuation of the ability of musculotendinous receptors to sense joint position.[11]

1.1 Historical Perspective

First proposed by Freeman[3] in 1965, the most common reason hypothesised for the high recurrence rate of LAS is the development of proprioceptive deficits following LAS. Freeman[3] hypothesised that when ligament tissues are disrupted during LAS, the mechanoreceptors and afferent nerves are also disrupted because the nervous tissues possess less tensile strength than ligamentous tissues.[4] The definition of FI has broadened to include the occurrence of recurrent joint instability and the sensation of joint instability due to the contributions of any neuromuscular deficits.

1.2 Relationship Between Functional Instability and Mechanical Instability

There are distinct relationships between the presence of FI and MI among individuals with recurrent LAS. Several studies have demonstrated that MI is related to various proprioceptive changes resulting in an accompanying FI,[12-14] while others have examined patients who complain of FI and also present with MI on physical and/or radiological examination.[15-18] As a joint develops MI, proprioceptive changes often occur which result in alterations in defence mechanisms to prevent injury. The resulting situation is a joint which continues to be stressed beyond its physiological limitations, further compounding the causes of both the MI and FI of the joint.[11,19] It must be noted that some individuals who develop MI will not develop FI, although these tend to be exceptional cases.

2. Evidence of Functional Instability After Ankle Sprain

Functional instability has been demonstrated in several ways following LAS.

2.1 Balance Deficits

Adverse changes in the ability to maintain balance during single leg stance following LAS have been reported by several researchers.[4,12,13,20-33] Numerous balance parameters have been found to be altered, the most common being an increased area of postural sway when balancing on the injured limb. Postural sway is defined as the deviation from the mean centre of pressure (COP) of the foot for a given trial.[34] In other words, injured patients distribute forces across a larger area of their foot than uninjured individuals. It has been hypothesised that this alteration of weight bearing in the foot may be a predisposition to recurrent LAS. However, little is known about the directional changes in weight bearing. There have also been reports which have not identified changes in balance following LAS.[16,17,35,36]

Balance has been assessed using both subjective and objective measurements. Subjective assessment has utilised judgement of impaired balance by both examiners and volunteers during performance of the modified Rhomberg test.[4] This test is performed by having the volunteers stand in a single leg stance and attempt to maintain their balance. The test is performed with eyes open and then repeated with eyes closed. Comparisons are then made between the injured and uninjured extremities. Both examiners and injured volunteers have reported greater balance impairments when standing on the extremity which experienced LAS compared with the uninjured extremity.[4,21,24,30]

Parameters of balance have been quantified by performing the modified Rhomberg test with volunteers standing on a piezoelectric force plate. These methods were first reported by Tropp and colleagues.[35] The magnitude of postural sway has been found to be increased when injured volunteers balanced on the limbs which had experienced LAS.[12,22,23,25-29,31-33] Increased impairment has been demonstrated in maintaining frontal plane stability during single leg stance.[20,23,28,29] It has been hypothesised that frontal plane assessment of postural control gives an indication of subtalar joint instability.[23]

Increased postural sway when balancing on uninjured limbs has been shown to be a predictor of LAS in soccer players.[36] Individuals with LAS have also been shown to utilise the hip strategy of balance maintenance more than the ankle strategy when balancing on their injured limbs.[37] The ankle

strategy occurs when muscle contractions first occur at the ankle and cause a torque which rotates the body towards the support surface following a perturbation. This strategy is generally used by healthy adolescents and young adults. The hip strategy occurs when hip flexion or extension are performed in the direction of a perturbation, thus causing a force to be generated against the support surface. The hip strategy is not as effective as the ankle strategy and is typically used by the elderly and those with balance disorders.[10]

Adding credence to the theory that FI is due to disruption of ligamentous mechanoreceptors, adverse changes in balance have been found after injection of an anaesthetic into the lateral ankle ligaments.[38,39]

2.2 Joint Position Sense Deficits

The ability to sense position of the ankle joint has been shown to be adversely affected following LAS.[15,21,30,40] Conflicting findings have been found by Gross.[41] Glencross and Thornton[40] were the first to demonstrate the impaired ability to actively replicate joint positions in individuals with a history of LAS. Volunteers were instructed to actively replicate various positions of plantar flexion in which they had been previously held by the examiner. Degrees of error from the correct position were measured and used as the dependent variable. The volunteers had greater error when tested on their injured limbs compared with their uninjured limbs. Greater degrees of error were also found in test positions closer to the ends of plantar flexion range of motion. The volunteers who had experienced severe LAS also demonstrated greater error than did those with mild LAS.[40]

The ability to detect very slow passive motion has also been shown to be impaired following LAS. Garn and Newton[21] found a decreased ability to detect passive plantar flexion at a rate of 0.3° per second in a population of 30 athletes with a history of multiple unilateral LAS. These findings were replicated in a similar population of 11 competitive gymnasts by Forkin and colleagues.[30] Lentell and colleagues[15] also demonstrated impaired ability

to detect passive inversion motion at a speed of 0.3° per second in a population of 42 recreational athletes with a history of unilateral FI.

Decreased accuracy in assessing passive inversion joint position was seen up to 12 weeks after injury in a population of 44 individuals who sustained acute LAS and were assessed 1, 3, 6 and 12 weeks after injury. Significant differences between injured and uninjured ligaments were present at all four testing intervals. Position sense of the injured ankle improved between 1 and 6 weeks after injury. However, it plateaued and did not return to levels of the uninjured side within 12 weeks.[18]

Impaired joint position sense has also been shown to be a predictor of LAS in individuals with no history of LAS. Payne and colleagues[42] prospectively assessed the active joint position sense for dorsiflexion, plantar flexion, inversion and eversion in 31 female collegiate basketball players. Players with impaired position sense in inversion and eversion were found to experience more LAS in the following competitive season.[42]

Gross[41] did not find impaired active or passive joint position sense among 14 individuals with repetitive unilateral LAS. Testing was performed on an isokinetic dynamometer which allowed inversion and eversion movement at a speed of 5° per second. This speed was much faster than the 0.3° per second utilised by the other investigators and may have played a role in these conflicting results.

2.3 Delayed Peroneal Muscle Reaction Time

The peroneal muscles are the first muscles to contract in response to a sudden ankle inversion stress and thus are vital to controlling the dynamic stability of the ankle complex.[43,44] Delayed activation of the peroneal muscles in response to sudden inversion perturbations has been hypothesised as a cause of FI following LAS. There have been several studies which support this hypothesis,[14,43,45,46] but also several which refute it.[18,47-51] Considerable differences in methodology among these studies may have contributed to the conflicting findings.

Significantly slower reaction times to inversion perturbation in previously injured ankles have been

reported for the peroneus longus,[14,43,45,46] peroneus brevis[45] and tibialis anterior[46] muscles. Other authors have reported slowed peroneal muscle responses to inversion perturbation in ankles with a history of LAS, but not at statistically significant levels.[14,50]

The magnitude of peroneal muscle electromyogram (EMG) activity in response to inversion stress was found to increase following lateral ankle reconstruction or repair surgeries, suggesting that restoring mechanical stability to the ankle may also restore functional stability.[49] Muscle firing patterns favouring the ability to evert the ankle in response to inversion stress were seen in healthy volunteers following 8 weeks of ankle disc training.[52] Peroneal response has also been shown to slow when inversion stress is performed at increasing magnitudes of talocrural joint plantar flexion.[53]

2.4 Altered Common Peroneal Nerve Function

Injury to the common peroneal nerve has been reported as a complication accompanying LAS, which may play a role in FI of the ankle.[54-64] Traction placed on the common peroneal nerve and its deep and superficial branches during the hypersupination leading to LAS are suspected as the cause of nerve injury.[59,63]

Slowed nerve conduction velocity (NCV) along the common peroneal nerve has been hypothesised as a contributing factor to FI following LAS.[61,62] Nitz and colleagues[62] found slowed NCV of the common peroneal nerve in 17% of grade II LAS and 86% of grade III LAS in a population of 66 patients with serious LAS. Ten percent of grade II injuries and 83% of grade III injuries were accompanied by slowed NCV of the tibial nerve. It should be noted that many of these patients had concomitant injury to the lateral and deltoid ligaments.

Kleinrensink and colleagues[63] demonstrated slowed NCV along the superficial and deep branches of the peroneal nerve in a population of 22 patients who had experienced LAS. Initial testing performed between 4 and 8 days after injury revealed that NCV of the superficial branch in the injured limbs was significantly less than the uninjured limbs. Follow-up testing 5 weeks after injury revealed no significant differences. The initial NCV along the deep branch of the injured limbs was significantly slower than a control group, but not the uninjured limbs. No differences were noted between groups at the follow-up testing.[63]

Diminished sensation along the sensory distribution of the common peroneal and sural nerves has been reported as a complication following LAS which may be related to FI.[61,62,64] Nitz and colleagues[62] reported the inability to distinguish between crude touch and pin prick along the peroneal nerve distribution in 54% of patients who had experienced grade II and III LAS. Symptoms were present up to 6 weeks after injury. Bullock-Saxton[64] reported diminished sensation of vibration along the common peroneal nerve distribution in patients following severe LAS.

2.5 Strength Deficits

Weakness of the muscles which evert or pronate the ankle complex has been demonstrated to be a contributing factor to FI following LAS.[18,65-68] Several authors have also reported no differences in eversion strength following LAS.[15,17,24,69,70] The peroneus longus and brevis muscles are the primary movers during concentric eversion. The importance of eversion strength to ankle stability was demonstrated by Ashton-Miller and colleagues,[71] who showed that the evertor muscles were able to generate greater moments than ankle braces or taping worn in conjunction with three-quarter top shoes when the ankle was exposed to 15° of inversion.

When assessed experimentally, conflicting results have been found regarding the torque production of the peroneal muscles following LAS. Most strength assessments have been performed in the open kinetic chain using isokinetic dynamometers at speeds much slower than functional activities. The validity of these methods relating to the closed kinetic chain eccentric function of the peroneal muscles has been previously questioned, and interpretation of these results should be made with caution.[72]

Bosien and colleagues[65] first reported the high prevalence of peroneal weakness in individuals having residual disability following LAS. Using manual muscle testing to assess eversion strength, 66% of patients presenting with residual symptoms over 2 years following LAS were found to have peroneal weakness.[65] Weakness in concentric pronation among the injured ankles of 15 patients with FI was demonstrated at 30 and 120° per second using an isokinetic dynamometer.[66]

Wilkerson and colleagues[68] demonstrated peak torque deficits ranging from 5 to 18% between the ankles of patients with unilateral LAS. Ankle eversion strength was found to be significantly less in both ankles of 25 individuals who had experienced unilateral LAS when compared with those of healthy control individuals.[67] Decreased eccentric eversion moments were found in the initial 6 weeks following injury in 44 patients with acute LAS.[18]

Conversely, Lentell and colleagues[24] found no significant differences in the strength of injured and uninjured ankles in 33 patients with a history of LAS. The patients were assessed for eversion strength isometrically and concentrically at 30° per second on an isokinetic dynamometer. No differences were found between the injured and uninjured ankles of 42 recreational athletes with unilateral FI when concentric eversion strength was measured at 30, 90, 150 and 210° per second.[15] Ryan[70] found no significant differences in concentric eversion torque at 30° per second between previously injured and uninjured ankles. No significant differences in eccentric eversion torque at 90° per second were found between 9 individuals with history of LAS and 9 healthy controls individuals.[17] Likewise, no significant differences in peroneal EMG activity were found during ankle disk exercises in volunteers with and without history of chronic LAS.[69] No predictive value of eversion weakness to LAS was observed in a study utilising collegiate basketball players with no history of LAS.[42]

Inversion strength deficits following LAS have been demonstrated by several authors.[68,70] Ryan[70] found significant invertor weakness in individuals with unilateral FI. Wilkerson and colleagues[68] demonstrated greater inversion deficits than eversion deficits in the concentric isokinetic testing of 30 individuals with a history of unilateral LAS. The authors suggested that reflexive inhibition of the muscles which can produce the motion which caused the initial injury may result following joint injury. The importance of the agonist/antagonist cocontraction in the maintenance of dynamic joint is not entirely understood.[68]

Higher eversion to inversion torque ratio measured concentrically at 60° per second was found to be a predictor of LAS in a prospective study of risk factors to LAS utilising 145 collegiate athletes.[73] This finding suggests that weaker invertors may predispose athletes to LAS. Studies showing no differences in inversion strength following LAS have also been reported.[17,24]

2.6 Decreased Dorsiflexion Range of Motion

Diminished dorsiflexion following LAS is thought to contribute to FI following LAS. Inflexibility of the triceps surae prevents the ankle from reaching full dorsiflexion and as a result the ankle is held in a more plantar flexed position throughout the gait cycle. The talocrural joint is in its closed pack position in full dorsiflexion, thus the talus is able to invert and internally rotate more when it is not in full dorsiflexion at heel strike. This excess motion may predispose individuals with diminished dorsiflexion to recurrent LAS.

A population of professional basketball players with a history of multiple episodes of bilateral LAS was found to have a mean of 3.6° of passive dorsiflexion, while healthy control individuals exhibited 17.9° of dorsiflexion.[28] Collegiate dance students with history of lower extremity injuries, including LAS, were shown to have significantly less dorsiflexion range of motion than their previously uninjured counterparts.[73] Wilson and colleagues[74] noted an overall loss of sagittal plane movement 3 and 10 days following LAS in 13 college athletes, although they did not distinguish between losses of plantar flexion or dorsiflexion. While limited dorsiflexion range of motion has been shown to be pres-

ent following LAS, it has not been shown to be a predictor of LAS in previously uninjured athletes.[42,75]

2.7 Sinus Tarsi Syndrome

Residual symptoms of pain and inflammation in the area of the sinus tarsi may indicate the presence of sinus tarsi syndrome. Sinus tarsi syndrome has been described as a distinct clinical entity that involves synovitis of the lateral aspect of the posterior subtalar joint following LAS in which the patient senses instability of the ankle.[76,77] Sinus tarsi syndrome can also develop as a sequelae to inflammatory diseases such as gout or rheumatoid arthritis.[78] O'Conner[79] hypothesised that sinus tarsi syndrome resulted in some cases following LAS because of scarring of the synovium and ligamentous tissue on the floor of the sinus tarsi. Patients present with pain and point tenderness in the sinus tarsi and often complain of FI of the involved ankle. Treatment usually consists of injection of anaesthetic and anti-inflammatory medications directly into the sinus tarsi, in conjunction with rehabilitation. Treatment with a foot orthotic has also been demonstrated to be effective in the treatment of sinus tarsi syndrome following a thorough biomechanical evaluation.[80] If conservative treatment fails, surgical excision of hypertrophied synovial tissue has been shown to be very successful.[76-79]

2.8 Anterolateral Impingement Syndrome

Hypertrophic synovitis can also occur to the talocrural joint capsule and cause residual symptoms following LAS. The most common site of this is in the anterolateral aspect of the talocrural joint capsule, where scar tissue may become impinged between the talus and fibula resulting in bouts of pain and FI. Several accounts of successful surgical intervention for anterolateral impingement syndrome have been reported.[81-85] Early and consistent treatment of LAS with focal compression and cryotherapy around the lateral malleolus has been hypothesised to reduce synovitis following LAS.[19,86]

2.9 Miscellaneous Causes

Several other causes have been reported in association with FI following apparent LAS. These include split lesions of the peroneus brevis tendon,[87] osteoid osteoma of the cuboid bone,[88] fibular osteochondroma,[89] sural nerve entrapment,[90] false aneurysm of the peroneal artery,[91] superior peroneal retinacular laxity[92] and osteochondral lesions of the tibial plafond.[93] While these conditions are rare, they should be considered in cases of FI which do not respond to conservative care.

3. Clinical Applications

3.1 Assessment

The clinician must examine for evidence of FI when evaluating the athlete presenting with an acute or chronic history of LAS. Assessment of balance, joint position sense, reflex responses, cutaneous sensation, strength and range of motion should be integrated into the injury evaluation. Establishing baseline data of such measures can aid in assessment of the gains in restoration of neuromuscular control and functional stability made during rehabilitation.[94]

Assessing balance in single leg stance may be performed either subjectively with the modified Rhomberg test or objectively with a force platform.[34] Joint position sense may be assessed actively or passively and may be performed using an isokinetic dynamometer or a handheld goniometer. Active repositioning tasks require more input for tenomuscular receptors, while passive repositioning tasks isolate joint mechanoreceptors more. [11] Reflex responses of the peroneal muscles to inversion perturbations may be collected using EMG equipment, but caution must be exhibited in testing individuals with acute injuries because the sudden perturbation may compromise healing tissues.[18]

Cutaneous sensation may be assessed using discrimination testing with sharp and dull objects over the sensory distributions of the deep peroneal, superficial peroneal and sural nerves. Strength of the muscles acting on the ankle complex may be assessed using manual muscle testing or using an in-

strumented dynamometer. Range of motion testing should be performed in both the open and closed kinetic chains and recording may be made with a manual or electronic goniometer. Performance of functional strength tests such as single leg hops or agility courses may also be useful assessment modalities of the functional stability of the injured ankle.[95]

3.2 Rehabilitation

Correcting discrepancies between the injured and uninjured limbs in deficient measures of functional stability should be primary goals in the rehabilitation of LAS. Conservative treatment of LAS normally consists of initial cryotherapy and compression, followed by early mobilisation. Rehabilitation emphasising restoration of range of motion, strength, balance and normal gait patterns is standard care. An emphasis on weight bearing exercise is often utilised to promote functional exercise in the closed kinetic chain.[96] Rehabilitation in an attempt to improve neuromuscular control of the injured ankle should address the restoration of the control of volitional contractions of the muscles acting on the ankle, normal reflex responses and normal pattern generated movements of the lower extremity.[94]

Common exercises emphasising neuromuscular control include modified Rhomberg exercises, t-band kicks[97] and balance board exercises. Balance training has been shown to decrease postural control[22,29,32,35,37,98,99] and EMG[52] deficits following LAS. Wester and colleagues[98] demonstrated that injured patients undergoing a 12-week balance and proprioceptive training protocol were more than twice as likely to not experience a recurrent LAS than those who did not perform this training programme following an initial LAS.

The employment of rearfoot orthotics in the treatment of LAS has been previously reported.[25,31] The use of molded foot orthotics has been shown to be advantageous in the treatment of LAS, although the mechanisms by which this works has not been clearly defined.[25,31] Orteza and colleagues[25] found molded neutral orthotics improved balance and led to decreased ankle pain while jogging in patients with acute LAS. Guskiewicz and Perrin[31] found that orthotics reduced the magnitude of postural sway in various balance tasks in patients with acute ankle sprains. The authors theorised that stabilisation of the subtalar joint by the orthotics added stability to the talocrural joint. It should be noted that no specific posting of the orthotics was performed in either of these studies.[25,31]

Clanton[100] has anecdotally suggested that a laterally posted heel wedge be used in the conservative treatment of lateral subtalar instability. Stabilisation of the subtalar joint with orthotics may allow the injured individual to return to the ankle strategy of balance maintenance and may be the mechanism by which orthotics help to improve balance following LAS. Research is needed to validate this hypothesis.

The use of prophylactic ankle taping and bracing has been used extensively in an attempt to prevent LAS. Research has demonstrated that bracing is effective in the prevention of recurrent LAS, but not in preventing initial LAS.[101-103] It is unknown if the prophylactic effect of ankle braces in preventing recurrent LAS is due to the restoration of mechanical stability or the reduction of proprioceptive deficits through stimulation of cutaneous receptors, or both.

The effects of ankle taping and bracing on postural control have been reported by several authors.[23,35,37,104,105] Tropp and colleagues[35] found no differences in postural sway between taped and untaped ankles. Friden and colleagues[23] found diminished frontal plane postural sway with brace application in ankles with FI compared to the unbraced condition. Bennell and Goldie[104] found both taping and bracing to have detrimental effects on balance among 24 healthy individuals. Individuals with MI were shown to use the ankle strategy more than the hip strategy of balancing while wearing an ankle brace compared with not wearing a brace.[37] Kinzey and colleagues[105] found altered COP measurements but no changes in postural sway among 3 different braces in a study of 24 healthy volunteers. There is a need for research to examine the effects

of taping and bracing on balance in an injured population.

3.3 Surgery

The effects of surgical stabilisation of unstable ankles on measures of functional stability have not been extensively examined. Larsen and Lund[49] reported increased magnitude of peroneal EMG activity following surgical stabilisation of unstable ankles. Improved proprioception and neuromuscular control following surgical repair of the knee and glenohumeral joints have been previously reported.[106-108] It is unknown whether functional impairments would dissipate following surgical repair of MI of the talocrural and/or subtalar joints.

4. Conclusion

Functional instability must be considered as a viable cause of residual instability following LAS. FI may exist both in the presence and absence of MI. Clinicians must examine injured athletes for the presence of neuromuscular deficits which may be causing FI. Treatment and rehabilitation goals must be set and achieved to restore and maintain neuromuscular control following LAS. Continued scientific and clinical investigation is needed to aid in the prevention of recurrent LAS due to FI.

References

1. Smith RW, Reischl SF. Treatment of ankle sprains in young athletes. Am J Sports Med 1986; 14: 465-71
2. Yeung MS, Chan K, So CH, et al. An epidemiological survey on ankle sprain. Br J Sports Med 1994; 28: 112-6
3. Freeman MAR. Instability of the foot after injuries to the lateral ligament of the ankle. J Bone Joint Surg Br 1965; 47: 678-85
4. Freeman MAR, Dean MRE, Hanham IWF. The etiology and prevention of functional instability of the foot. J Bone Joint Surg Br 1965; 47: 669-77
5. Lephart SM, Pincivero DM, Rozzi SL. Proprioception of the ankle and knee. Sports Med 1998; 25: 149-55
6. Garrick JG, Requa RK. The epidemiology of foot and ankle injuries in sports. Clin Sports Med 1988; 7: 29-36
7. Viladot A, Lorenzo JC, Salazar J, et al. The subtalar joint: embryology and morphology. Foot Ankle 1984; 5: 54-66
8. Michelson JD, Hutchins C. Mechanoreceptors in human ankle ligaments. J Bone Joint Surg Br 1995; 77: 219-24
9. Takebayashi T, Yamashita T, Minaki Y, et al. Mechanosensitive afferent units in the lateral ligament of the ankle. J Bone Joint Surg Br 1997; 79: 490-3
10. Norkin CC, Levangie PK. Joint structure and function: a comprehensive analysis. Philadelphia (PA): FA Davis, 1992
11. Lephart SM, Pincivero DM, Giroldo JL, et al. The role of proprioception in the management and rehabilitation of athletic injuries. Am J Sports Med 1997; 25: 130-7
12. Tropp H, Odenrick P, Gillquist J. Stabilometry recordings in functional and mechanical instability of the ankle joint. Int J Sports Med 1985; 6: 180-2
13. De Simoni C, Wetz HH, Zanetti M, et al. Clinical examination and magnetic resonance imaging in the assessment of ankle sprains treated with an orthosis. Foot Ankle Int 1996; 17: 177-82
14. Karlsson J, Andreasson GO. The effect of external ankle support in chronic lateral ankle joint instability: an electromyographic study. Am J Sports Med 1992; 20: 257-61
15. Lentell G, Baas B, Lopez D, et al. The contributions of proprioceptive deficits, muscle function, and anatomic laxity to functional instability of the ankle. J Orthop Sports Phys Ther 1995; 21: 206-15
16. Isakov E, Mizrahi J. Is balance impaired by recurrent sprained ankle? Br J Sports Med 1997; 31: 65-7
17. Bernier JN, Perrin DH, Rijke A. Effect of unilateral functional instability of the ankle on postural sway and inversion and eversion strength. J Athletic Training 1997; 32: 226-32
18. Konradsen L, Olesen S, Hansen HM. Ankle sensorimotor control and eversion strength after acute ankle inversion injuries. Am J Sports Med 1998; 26: 72-7
19. Wilkerson GB, Nitz AJ. Dynamic ankle stability: mechanical and neuromuscular interrelationships. J Sport Rehabil 1994; 3: 43-57
20. Tropp H, Odenrick P. Postural control in single-limb stance. J Orthop Res 1988; 6: 833-9
21. Garn SN, Newton RA. Kinesthetic awareness in subjects with multiple ankle sprains. Phys Ther 1988; 68: 1667-71
22. Gauffin H, Tropp H, Odenrick P. Effect of ankle disk training on postural control in patients with functional instability of the ankle joint. Int J Sports Med 1988; 9: 141-4
23. Friden T, Zatterstrom R, Lindstrand A, et al. A stabilometric technique for evaluation of lower limb instabilities. Am J Sports Med 1989; 17: 118-22
24. Lentell GL, Katzmann LL, Walters MR. The relationship between muscle function and ankle instability. J Orthop Sports Phys Ther 1990; 11: 605-11
25. Orteza LC, Vogelbach WD, Denegar CR. The effect of molded orthotics on balance and pain while jogging following inversion ankle sprain. J Athletic Training 1992; 27: 80-4
26. Cornwall MW, Murrell PM. Postural sway following inversion sprain of the ankle. J Am Podiatric Med Assoc 1991; 81: 243-7
27. Golomer E, Dupui P, Bessou P. Spectral frequency analysis of dynamic balance in healthy and injured athletes. Arch Int Physiol Biomech Biophys 1994; 102: 225-9
28. Leanderson J, Wykman A, Eriksson E. Ankle sprain and postural sway in basketball players. Knee Surg Sports Traumatol Arthrosc 1993; 1: 203-5
29. Goldie PA, Evans OM, Bach TM. Postural control following inversion injuries of the ankle. Arch Phys Med Rehab 1994; 75: 969-75
30. Forkin DM, Koczur C, Battle R, et al. Evaluation of kinesthetic deficits indicative of balance control in gymnasts with unilateral chronic ankle sprains. J Orthop Sports Phys Ther 1996; 23: 245-50
31. Guskiewicz KM, Perrin DH. Effect of orthotics on postural sway following inversion ankle sprain. J Orthop Sports Phys Ther 1996; 23: 326-31

32. Leanderson J, Eriksson E, Nilsson C. Proprioception in classical ballet dancers: a prospective study of the influence of an ankle sprain on proprioception in the ankle joint. Am J Sports Med 1996; 24: 370-4

33. Perrin PP, Bene MC, Perrin CA, et al. Ankle trauma significantly impairs postural control: a study in basketball players and controls. Int J Sports Med 1997; 18: 387-92

34. Guskiewicz KM, Perrin DH. Research and clinical applications of assessing balance. J Sport Rehabil 1996; 5: 45-63

35. Tropp H, Eckstrand J, Gillquist J. Factors affecting stabiliometry recordings of single limb stance. Am J Sports Med 1984; 12: 185-8

36. Tropp H, Eckstrand J, Gillquist J. Stabilometry in functional instability of the ankle and its value in predicting injury. Med Sci Sports Exer 1984; 16: 64-6

37. Pinstaar A, Brynhildsen J, Tropp H. Postural corrections after standardized perturbations of single leg stance: effect of training and orthotic devices in patients with ankle instability. Br J Sports Med 1996; 30: 151-5

38. DeCarlo MS, Talbot RW. Evaluation of ankle proprioception following injection of the anterior talofibular ligament. J Orthop Sports Phys Ther 1986; 8: 70-6

39. Hertel JN, Guskiewicz KM, Kahler DM, et al. Effect of lateral ankle joint anesthesia on center of balance, postural sway, and joint position sense. J Sport Rehabil 1996; 5: 111-9

40. Glencross D, Thornton E. Position sense following joint injury. J Sports Med Phys Fitness 1981; 21: 23-7

41. Gross MT. Effects of recurrent lateral ankle sprains on active and passive judgement of joint position. Phys Ther 1987; 67: 1505-9

42. Payne KA, Berg K, Latin RW. Ankle injuries and ankle strength, flexibility, and proprioception in college basketball players. J Athletic Training 1997; 32: 221-5

43. Brunt RL, Anderson JC, Huntsman B, et al. Postural responses to lateral perturbation in healthy subjects and ankle sprain patients. Med Sci Sports Exer 1992; 24: 171-6

44. Konradsen L, Voight M, Hojsgaard C. Ankle inversion injuries: the role of the dynamic defense mechanism. Am J Sports Med 1997; 25: 54-8

45. Konradsen L, Ravn JB. Ankle instability caused by prolonged peroneal reaction time. Acta Orthop Scand 1990; 61: 388-90

46. Lofvenberg R, Karrholm J, Sundelin G, et al. Prolonged reaction time in patients with chronic lateral instability of the ankle. Am J Sports Med 1995; 23: 414-7

47. Nawoczenski DA, Cook TM, Saltzman CL. The effect of foot orthotics on three-dimensional kinematics of the leg and rearfoot during running. J Orthop Sports Phys Ther 1995; 21: 317-27

48. Isakov E, Mizrahi J, Solzi P, et al. Response of the peroneal muscles to sudden inversion stress during standing. Int J Sport Biomech 1986; 2: 100-6

49. Larsen E, Lund PM. Peroneal muscle function in chronically unstable ankles: a prospective preoperative and postoperative electromyographic study. Clin Orthop 1991; 272: 219-26

50. Johnson MB, Johnson CL. Electromyographic response of peroneal muscles in surgical and nonsurgical injured ankles during sudden inversion. J Orthop Sports Phys Ther 1993; 18: 497-501

51. Ebig M, Lephart SM, Burdett RG, et al. The effect of sudden inversion stress on EMG activity of the peroneal and tibialis anterior muscles in the chronically unstable ankle. J Orthop Sports Phys Ther 1997; 26: 73-7

52. Sheth P, Yu B, Laskowski ER, et al. Ankle disk training influences reaction times of selected muscles in a simulated ankle sprain. Am J Sports Med 1997; 25: 538-43

53. Lynch SA, Eklund U, Gottlieb D, et al. Electromyographic latency changes in the ankle musculature during inversion. Am J Sports Med 1996; 24: 362-9

54. Hyslop GH. Injuries to the deep and superficial peroneal nerves complicating ankle sprain. Am J Surg 1951; 51: 436-8

55. MacIver DA, Letts RM. Paralysis of the peroneal nerve in association with a plantar flexion inversion injury of the ankle. Med Serv J Can 1966; 22: 285-7

56. Nobel W. Peroneal palsy due to haematoma in the common peroneal nerve sheath after distal torsional fractures and inversion ankle sprains. J Bone Joint Surg Am 1966; 48: 1484-95

57. Sidey JD. Weak ankles: a study of common peroneal nerve entrapment neuropathy. BMJ 1969; 3: 623-9

58. Meals RA. Peroneal nerve palsy complicating ankle sprain: report of two cases and review of the literature. J Bone Joint Surg Am 1977; 59: 966-8

59. Streib EW. Traction injury of the peroneal nerve caused by minor athletic trauma: electromyographic studies. Arch Neurol 1983; 40: 62-3

60. Connolly TJ, Fitzgibbons TC, Weber LE. Injury to the peroneal nerve after ankle sprain: a case report. Nebr Med J 1990; 75: 6-7

61. Stoff MD, Greene AF. Common peroneal nerve palsy following inversion ankle injury: a report of 2 cases. Phys Ther 1982; 62: 1463-4

62. Nitz AJ, Dobner JJ, Kersey D. Nerve injury and grades II and III ankle sprains. Am J Sports Med 1985; 13: 177-82

63. Kleinrensink GJ, Stoeckart R, Meulstee J, et al. Lowered motor conduction velocity of the peroneal nerve after inversion trauma. Med Sci Sports Exer 1994; 26: 877-83

64. Bullock-Saxton JE. Local sensation changes and altered hip muscle function following severe ankle sprain. Phys Ther 1994; 74: 23-34

65. Bosien WR, Staples OS, Russell SW. Residual disability following acute ankle sprains. J Bone Joint Surg Am 1955; 37: 1237-43

66. Tropp H. Pronator weakness in functional instability of the ankle joint. Int J Sports Med 1986; 7: 291-4

67. Bush KW. Predicting ankle sprain. J Manual Manipulative Ther 1996; 4: 54-8

68. Wilkerson GB, Pinerola JJ, Caturano RW. Invertor vs. evertor peak torque and power deficiencies associated with lateral ankle ligament injury. J Orthop Sports Phys Ther 1997; 26: 78-86

69. Soderberg GL, Cook TM, Rider SC, et al. Electromyographic activity of selected leg musculature in subjects with normal and chronically sprained ankles performing on a BAPS board. Phys Ther 1991; 72: 514-22

70. Ryan L. Mechanical stability, muscle strength, and proprioception in the functionally unstable ankle. Aust J Physiother 1994; 40: 41-7

71. Ashton-Miller JA, Ottaviani RA, Hutchinson C, et al. What best protects the inverted weightbearing ankle against further inversion? Evertor muscle strength compares favorably with shoe height, athletic tape, and three orthoses. Am J Sports Med 1996; 24: 800-9

72. Kaminski TW, Perrin DH, Mattacola CG, et al. The reliability and validity of ankle inversion and eversion torque measurements from the Kin Com II isokinetic dynamometer. J Sport Rehabil 1995; 4: 210-8

73. Wiesler ER, Hunter DM, Martin DF, et al. Ankle flexibility and injury patterns in dancers. Am J Sports Med 1996; 24: 754-7

74. Wilson RW, Gieck JH, Gansneder BM, et al. Reliability and responsiveness of disablement measures following acute ankle

sprains among athletes. J Orthop Sports Phys Ther 1998; 27: 348-55

75. Baumhauer JF, Alosa DM, Renstrom PAFH, et al. A prospective study of ankle injury risk factors. Am J Sports Med 1995; 23: 564-70

76. Meyer JM, Lagier R. Post-traumatic sinus tarsi syndrome: an anatomical and radiological study. Acta Orthop Scand 1977; 48: 121-8

77. Taillard W, Meyer JM, Garcia J, et al. The sinus tarsi syndrome. Int Orthop 1981; 5: 117-30

78. Lowy A, Schilero J, Kanat IO. Sinus tarsi syndrome: a postoperative analysis. J Foot Surg 1985; 24: 108-12

79. O'Conner D. Sinus tarsi syndrome: a clinical entity. J Bone Joint Surg Am 1958; 18: 231-3

80. Shear MS, Baitch SP, Shear DB. Sinus tarsi syndrome: the importance of biomechanically-based evaluation and treatment. Arch Phys Med Rehabil 1993; 74: 777-81

81. McCarroll JR, Schrader JW, Shelbourne KD, et al. Meniscoid lesions of the ankle in soccer players. Am J Sports Med 1987; 15: 255-7

82. Martin DF, Curl WW, Baker CL. Arthroscopic treatment of chronic synovitis of the ankle. Arthroscopy 1989; 5: 110-4

83. Thein R, Eichenblat M. Arthroscopic treatment of sports-related synovitis of the ankle. Am J Sports Med 1992; 20: 496-8

84. Meislin RJ, Rose DJ, Parisien JS, et al. Arthroscopic treatment of synovial impingement of the ankle. Am J Sports Med 1993; 21: 186-9

85. Liu SH, Nuccion SL, Finerman G. Diagnosis of anterolateral ankle impingement: comparison between magnetic resonance imaging and clinical examination. Am J Sports Med 1997; 25: 389-93

86. Wilkerson GB. Treatment of inversion ankle sprains through synchronous application of focal compression and cold. Athletic Training 1991; 26: 220-37

87. Bonnin M, Tavernier T, Bouysset M. Split lesions of the peroneus brevis tendon in chronic ankle laxity. Am J Sports Med 1997; 25: 699-703

88. Trettin DM, Browne JE. Osteoid osteoma of the tarsal cuboid presenting with recurrent ankle sprains in an adolescent: a case report. Foot Ankle Int 1995; 16: 30-3

89. Montella BJ, O'Farrell DA, Furr WS, et al. Fibular osteochondroma presenting as chronic ankle sprain. Foot Ankle Int 1995; 16: 207-9

90. Toy BJ. Conservative treatment of bilateral sural nerve entrapment in an ice hockey player. J Athletic Training 1996; 31: 68-70

91. Bandy WD, Strong L, Roberts T, et al. False aneurysm: a complication following an inversion ankle sprain. A case report. J Orthop Sports Phys Ther 1996; 23: 272-9

92. Geppert MJ, Sobel M, Bohne HO. Lateral ankle instability as a cause of superior peroneal retinacular laxity: an anatomical and biomechanical study in cadaveric feet. Foot Ankle 1993; 14: 330-4

93. Taga I, Shino K, Inoue M, et al. Articular cartilage lesion in ankle with lateral ligament injury: an arthroscopic study. Am J Sports Med 1993; 21: 120-7

94. Hertel J, Denegar CR. A rehabilitation paradigm for restoring neuromuscular control following athletic injury. Athletic Ther Today 1998; 3 (5): 12-6

95. Worrell TW, Booher LD, Hench KM. Closed kinetic chain assessment following inversion ankle sprain. J Sport Rehabil 1994; 3: 197-203

96. Bunton EE, Pitney WA, Kane AW, et al. The role of limb torque, muscle action, and proprioception during closed kinetic chain rehabilitation of the lower extremity. J Athletic Training 1993; 28: 10-20

97. Tomaszewski D. T-band kicks ankle proprioception program. Athletic Training 1991; 26: 216-9

98. Wester JU, Jespersen SM, Nielsen KD, et al. Wobble board training after partial sprains of the lateral ligament of the ankle: a prospective randomized study. J Orthop Sports Phys Ther 1996; 23: 332-6

99. Bernier JN, Perrin DH. Effect of coordination training on proprioception of the functionally unstable ankle. J Orthop Sports Phys Ther 1998; 27; 264-75

100. Clanton TO. Instability of the subtalar joint. Orthop Clin North Am 1989; 20: 583-92

101. Surve I, Schwellnus MP, Noakes T, et al. A fivefold reduction in the incidence of recurrent ankle sprains in soccer players using the Sport-Stirrup orthosis. Am J Sports Med 1994; 22: 601-6

102. Sharpe SR, Knapik J, Jones B. Ankle braces effectively reduce recurrence of ankle sprains in female soccer players. J Athletic Training 1997; 32: 21-4

103. Tropp H, Askling C, Gillquist J. Prevention of ankle sprains. Am J Sports Med 1985; 13: 259-62

104. Bennell KL, Goldie PA. The differential effects of external ankle support on postural control. J Orthop Sports Phys Ther 1994; 20: 287-95

105. Kinzey SJ, Ingersoll CD, Knight KL. The effects of selected ankle appliances on postural control. J Athletic Training 1997; 32: 300-3

106. Warner JJ, Lephart S, Fu FH. Role of proprioception in pathoetiology of shoulder instability. Clin Orthop 1996; 330: 35-9

107. Barrett DS, Cobb AG, Bentley G. Joint proprioception in normal, osteoarthritic, and replaced knees. J Bone Joint Surg Br 1991; 73: 53-6

108. Harter RA, Osternig LR, Singer KM. Knee joint proprioception following anterior cruciate ligament reconstruction. J Sport Rehabil 1992; 1: 103-10

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