EFFICACY OF BOSU BALL AND NEUROMUSCULAR TRAINING IN REHABILITATION OF LATERAL ANKLE LIGAMENT INJURIES IN MALAYSIAN ATHLETES

DEIVENDRAN KALIRATHINAM

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by

DEIVENDRAN KALIRATHINAM

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LIST OF ABBREVIATIONS

µS	Microsecond
ATFL	Anterior talofibular ligament
AJFAQ	Ankle joint function assessment questionnaire
ANOVA	Analysis of variance
ACSM	American College of Sports Medicine
ANS	Autonomic Nervous System
ADL	Activities of Daily Living
BOSU	Both Sides Up
BBT	BOSU ball training
BMI	Body Mass Index
CFL	Calcaneofibular Ligament
COP	Center of Pressure
COM	Center of Mass
CI	Confidence Interval
CNS	Central Nervous System
CTP	Conventional Training Program
CONSORT	Consolidated Standards of Reporting Trials
DL	Double Leg
DF	Dorsi Flexors
DLS	Double Leg Squat
DNA	Deoxyribonucleic acid
EMG	Electromyography
EV	Evertors

FR	Functional Rehabilitation
FU	Follow – Up
GAG	Glycosaminoglycan
HUSM	Hospital Universiti Sains Malaysia
H_O	Null Hypothesis
H_A	Alternative Hypothesis
Hz	Hertz
INV	Invertors
JPS	Joint Position Sense
Kg	Kilogram
LAS	Lateral Ankle injury
LAI	Lateral Ankle Injury
MHz	Megahertz
mm	Millimeter
MOH	Ministry of Health
MSNT	Majlis Sukan Negeri Terengganu
MVIC	Maximum Voluntary Isometric Contraction
NMTP	Neuromuscular training Program
PF	Plantar Flexors
PL	Peroneous Longus
PB	Peroneous Brevis
PTFL	Posterior Talo Fibular Ligament
PEDro	Physiotherapy Evidence Database
PRISMA	Preferred Reporting Items for Systematic Reviews and Meta-Analyses

pg	Page
RICE	Rest, Ice, Compression and elevation
ROM	Range of Motion
RO	Research Objective
RQ	Research Question
RCT	Randomized Control Trial
SEBT	Star Excursion Balance Test
SL	Single Leg
SPSS	Statistical package for social sciences
Sc	Skin Conductance
SD	Standard Deviation
secs	Seconds
SEMG	Surface Electromyography
SENIAM	Surface Electromyography for Non-invasive Assessment of Muscles
TA	Tibialis Anterior
TTP	Total Time Distance
TENS	Transcutaneous Electrical Nerve Stimulator
USM	Universiti Sains Malaysia
UNISZA	Universiti Sultan Zainal Abidin

KEBERKESANAN LATIHAN BOLA "BOSU" DAN NEUROMUSKULAR DALAM REHABILITASI KECEDERAAN LIGAMEN PERGELANGAN KAKI LATERAL DI KALANGAN ATLET MALAYSIA

ABSTRAK

Kecederaan pergelangan kaki lateral sering dibahaskan adalah merupakan kecederaan sukan yang paling kerap berlaku. Kecederaan pergelangan kaki lateral kerap terjadi di kalangan individu yang aktif secara fizikal disebabkan oleh regangan yang salah pergerakan luar atau pergerakan dalam pada pergelangan kaki yang mana ia menyebabkan sendi menjadi longgar secara patologi dan menyebabkan "sensorimotor" merosot di pergelangan kaki. Kajian ini dilakukan untuk mengkaji peranan latihan konvensional fisioterapi (kumpulan A), latihan bola "BOSU" (kumpulan B), latihan neuromuskular (kumpulan C), dan latihan intervensi gabungan (kumpulan D) dalam memperbaiki keseimbangan dinamik, kekuatan otot, dan propriosepsi di kalangan perserta yang mengalami kecederaan ligamen pergelangan kaki lateral gred II. Lima puluh dua (52) subjek 32 lelaki dan 20 perempuan kekal di dalam kajian ini dan dikira bagi tujuan analisis statistik. Kiraan terulang dua arah ANOVA menunjukkan perbezaan ketara di antara kumpulan. Kesan ketara dapat dilihat selepas penilaian untuk propriosepsi dengan ralat posisi semula aktif dan pasif pada 15 dan 5 darjah pergerakan pergelangan kaki ke dalam bagi kumpulan C dan kumpulan D (p=.000). Begitu juga pada fasa susulan, parameter bagi propriosepsi, keseimbangan dinamik, dan kekuatan pergerakan pergelangan kaki ke dalam menunjukkan perbezaan yang ketara di antara kumpulan A dan kumpulan B (p=.034), kumpulan A dan kumpulan C (p=.036). Kontraksi isomatrik voluntari maksimum (KIVM) bagi otot *peronius longus*, *tibialis anterior*, dan *peroneus brevis* menunjukkan perbezaan yang ketara pada penilaian pertengahan antara kumpulan intervensi. Latihan konvensional memberi kelebihan dalam meningkatkan kekuatan otot tetapi dilihat kurang berkesan dalam meningkatkan propriosepsi, keseimbangan dinamik dan aktiviti fungsi dikalangan peserta, manakala latihan bola **"BOSU"** dilihat lebih baik berbanding latihan konvensional dalam meningkatkan propriosepsi, kekuatan otot, keseimbangan dinamik, dan aktiviti fungsi di kalangan peserta dengan kecederaan pergelangan kaki lateral. Gabungan intervensi latihan bola **"BOSU"** dan neuromuscular dilihat boleh memeka rangsangan reseptor deria pada otot dan tendon dalam meningkatkan propriosepsi dan kontraksi isomatrik voluntari maksimum justeru meningkatkan propriosepsi pergelangan kaki, kekuatan otot, keseimbangan dinamik, dan aktiviti fungsi di kalangan peserta kecederaan ligamen pergelangan kaki lateral gred II. Kesimpulan: Pelbagai kajian dalam menentukan perbezaan kekuatan dan protokol latihan propriosepsi. Sebahagian protokol menunjukkan keberkesanan dalam meningkatkan kestabilan dinamik, kekuatan pergelangan kaki, propriosepsi, atau mengurangkan risiko kecederaan pergelangan kaki, Tambahan lagi, program latihan terbaru (latihan bola **"BOSU"** dan neuromuskular) untuk jangkamasa dua belas minggu boleh memperbaiki tahap keseimbangan dinamik, propriosepsi, dan kekuatan otot semasa simulasi atlet, seterusnya mengurangkan risiko kecederaan di kalangan atlet yang sihat.

EFFICACY OF BOSU BALL AND NEUROMUSCULAR TRAINING IN REHABILITATION OF LATERAL ANKLE LIGAMENT INJURIES IN MALAYSIAN ATHLETES

ABSTRACT

Lateral ankle Sprains are debatably the most common sports injury. Lateral ankle sprains are extremely common among physically active individuals due to sudden and abnormal stretching with excessive inversion or eversion that frequently result in pathologic laxity and sensorimotor deficits about the ankle. The present study was aimed to investigate the role of Conventional physiotherapy training (Group A), BOSU ball training (Group B), Neuromuscular training (Group C), and Combined intervention training (Group D) in improving dynamic balance, muscle strength and proprioception in participants with Grade II lateral ligament injury of the ankle. Fifty-two (52) subjects 32 male, and 20 females remained in the study for the statistical analysis. A two-way repeated measure of ANOVA revealed that there were significant differences among the groups. There was a significant effect observed after post assessment on proprioception at active and passive repositioning error at 15 and 5 degrees of inversion in Group C and Group D (p=.000). At the follow-up phase, the parameters of proprioception, dynamic balance and eversion strength were showed significant differences observed between Group A and Group B (p=.034), Group A and Group C (p=.036). Maximum voluntary isometric contraction (MVIC) of the peroneus longus, tibialis anterior, peroneus brevis muscle, showed significant differences in the mid-term assessment across intervention groups). Conventional training was beneficial in enhancing muscle-strength but was observed less effective in improving proprioception, dynamic balance and functional activities in participants,

while BOSU ball aided training was observed as better than Conventional training in enhancing proprioception, muscle strength, dynamic balance and functional activities in participants with ankle lateral ligament injury. The combined intervention of BOSU ball and Neuromuscular training was observed to sensitise the sensory receptors of the muscle and the tendon in the form of increased proprioception and maximal voluntary isometric contraction thereby causing enhancement in proprioception of ankle joint, the strength of the muscle, dynamic balance and functional activities in participants suffering from grade II lateral ligament injury of the ankle. Conclusions: There have been numerous studies examining the different strength and proprioception training protocols. Some of these protocols have been successful at increasing dynamic stability, ankle strength, proprioception, or decreasing the risk of ankle injuries, Additionally, a new and novel combined training programme (BOSU ball and Neuromuscular training) for a twelve-week period improved the measures of dynamic balance, proprioception and muscle strength during athletes' simulations, thus potentially reducing injury risk in healthy athletes.

CHAPTER 1

INTRODUCTION

1 Background and Scope of The Study

1.1 Epidemiology of Ankle Sprain

Ankle joint is one of the most injured joints in athletes and people participating in sports (Fernandez et al., 2007; Hootman et al., 2007) representing 15% – 20% of all sports injuries (Boruta et al., 1990) and contributing to 22% of visits to the emergency rooms. Approximately 85% of these ankle injuries are due to an inversion injury involving lateral ligament damage (Ekstrand, J., & Tropp, H. 1990). The most common mechanism of injury for ankle inversion sprains is considered to be a combination of forced hyper-inversion and plantar flexion (Nakasa et al., 2006; Renstrom, P.A., & Lynch, S.A 1998). It is estimated that half of the general population has at least one ankle sprain during life (Nyska et al., 2003) and as many as 55% of them do not seek injury treatment from a healthcare professional (Hertel, J. 2002). In the United States alone, approximately 1 in 10,000 people sprain their ankle (Trevino et al., 1994). This figure amounts to an estimated 23,000 – 27,000 ankle sprains per day (Baumhauer et al.,1995; Kannus, P., & Renstrom, P. 1991). The costs associated with treating these many numbers of sprains are staggering, as treatment and rehabilitation of these lateral ankle sprains are estimated to be $2 billion a year (Beynnon et al., 2001). Ankle sprains account for up to one-sixth of all time lost from sports (Garrick, J. G., & Schelkun, P. H. 1997). The average duration of temporary unemployment as a result of a severe ankle sprain was found to be 29 (±33) days (Audenaert et al., 2010). Lateral ankle inversion sprains frequently occur in sports that mostly concern young, physically active individuals, (Balduini, F. C., & Tetzlaff, J. 1982; Holmer, P et al., 1994) constituting between 15%-75% of all sports-related injuries, and mainly occurring in the so-called ‘‘high-risk’’ sports like team handball, basketball, soccer, or volleyball, which are characterized by a high level of jumping and cutting movements (Garrick, J. G., & Schelkun, P. H. 1997; Ekstrand et al.,1983; Maehlum, S., & Daljord, O. A. 1984; Quinn, et al., 2000). Activity limitations may even occur with walking, and up to 72% of people are unable to return to their previous level of activity (Verhagen et al.,1995; Konradsen, L. 2002; Snyder, et al., 2008). Furthermore, an initial ankle sprain leads to high rate of injury recurrence (as high as 80% in high-risk sports) due to alterations in stress distribution causing long-term disability and degeneration (Van Dijk et al., 1996; Hirose et al., 2004; Omori et al., 2004; Valderrabano et al., 2006; Bischof et al., 2010). Recent research has indicated that patients with acute and recurrent ankle joint trauma may show early development of ankle joint osteoarthritis by a decade when compared to patients with primary ankle joint osteoarthritis (Saltzman et al., 2006). Additionally, patients with ankle instability (Arnold et al., 2011) and ankle osteoarthritis (Gage et al., 2003; Knight et al., 2003; Saltzman et al., 2006) have been reported to score either equal or lower self-reported disability scores when compared to patients with other chronic diseases. Therefore, ankle joint sprains and their associated sequelae not only negatively impact an individual’s health and perceived quality of life but also represent a large health care burden.

1.2 Functional Anatomy of the Ankle Joint Complex

The ankle joint complex is a sophisticated musculoskeletal arrangement that allows force transmission between the lower limb and the ground, facilitating stable ambulation and posture (Dawe, E. J., & Davis, J. 2011; Wedmore et al., 2005). The ankle joint complex comprises three major articulations: the talocrural joint, the subtalar joint, and the distal tibiofibular syndesmosis (Hertel, J. 2000). The coordinated movement of these three articulations allows the ankle joint to absorb the body impact forces during various weight-bearing activities and at the same time allows the foot to function as an adjustable shock-absorber on uneven surfaces (Nigg, B. M. 2001). The stability of the ankle joint is mainly provided by the bony congruity of the articular surfaces, the joint capsule as well as ligamentous support, and the musculotendinous structures surrounding the ankle complex (Hertel, J. 2002).

1.2.1 Anatomy and biomechanics of the talocrural joint

The talocrural joint (mortise) is formed by the articulations between the dome of the talus, the medial malleolus, the tibial plafond, and the lateral malleolus (Lundberg et al., 1989; Stiehl, J.B., 1991; Hertel, J. 2002). The talocrural joint is a uniaxial modified hinge joint with the axis of rotation that passes through the medial and lateral malleoli. In the frontal plane, the axis of rotation is slightly anterior as it passes through the tibia and slightly posterior as it passes through the fibula. The oblique axis of rotation at the talocrural joint mainly allows the movement in the sagittal plane (plantarflexion –dorsiflexion), with small amount of transverse (internal/external rotation) and frontal plane motion (inversion-eversion) occurring about the oblique axis of rotation (**Figure 1.1**) (Lundberg et al., 1989). The shape of the talus and the axis of rotation at the talocrural joint allow talus to glide posteriorly and externally rotate about mortise during dorsiflexion and glide anteriorly and internally rotate during plantarflexion (Soavi et al., 2000). The talocrural joint is maximally stable in the closed-pack position of dorsiflexion (Hertel, J. 2002, Louwerens et al.,1995) and injury-prone in the open-pack position (loose) of plantarflexion (Louwerens et al.,1995). Also, the fibula extends further to the lateral malleolus than the tibia does to the medial malleolus, allowing for larger range on inversion than eversion and thus more inversion sprains (Harmon K. G. 2004).

The stability of the talocrural joint in weight bearing is provided by the congruent articular surfaces, while in non-weight bearing, the ligaments appear to provide the majority of the stability (Stormont et al.,1985). The ligamentous support to the talocrural joint is provided by a joint capsule and several main ligaments, namely the anterior talofibular ligament (ATFL), the calcaneofibular ligament (CFL), and the posterior talofibular ligament (PTFL) on the lateral aspect (**Figure 1.2**) and the deltoid ligament on the medial aspect of the ankle (Hintermann,B (1999); Safran et al.,1999; Golano et al., 2010; Hertel, J. 2002). Research studies have reported that the ligaments on the lateral aspect of the ankle are collectively weaker than the deltoid ligament (Milner, C. E., & Soames, R. W 1998). The ATFL is the most frequently injured ligament at the ankle and is a most observed injury in the emergency room (Bosien et al., 1955; Boruta et al., 1990; Karlsson et al., 1997). The CFL is injured about 50-75% of the time, and PTFL is only injured about 10% of the time (Ferran, & Maffulli 2006). The ATFL is an intracapsular structure and primarily functions to resist anterior displacement and internal rotation of the talus in plantarflexion (Milner, C. E., & Soames, R. W 1998; Golano et al., 2010; Dutton, M 2012). Among the lateral ligaments, the ATFL is the weakest as it exhibits the lowest maximal load and energy to failure values under tensile stress as compared to CFL and PTFL (Attarian et al., 1985). The CFL is an extra-articular structure covered by peroneal tendons and often reinforced by talocalcaneal ligaments (Golano et al., 2010). The CFL restricts excessive supination of both talocrural and subtalar joints (Milner, C. E., & Soames, R. W 1998). The PTFL is the strongest of the lateral ligament complex (Safran et al.,1999) and resists both inversion and internal rotation of the talocrural joint during weight bearing (Stormont et al.,1985; Golano et al., 2010).

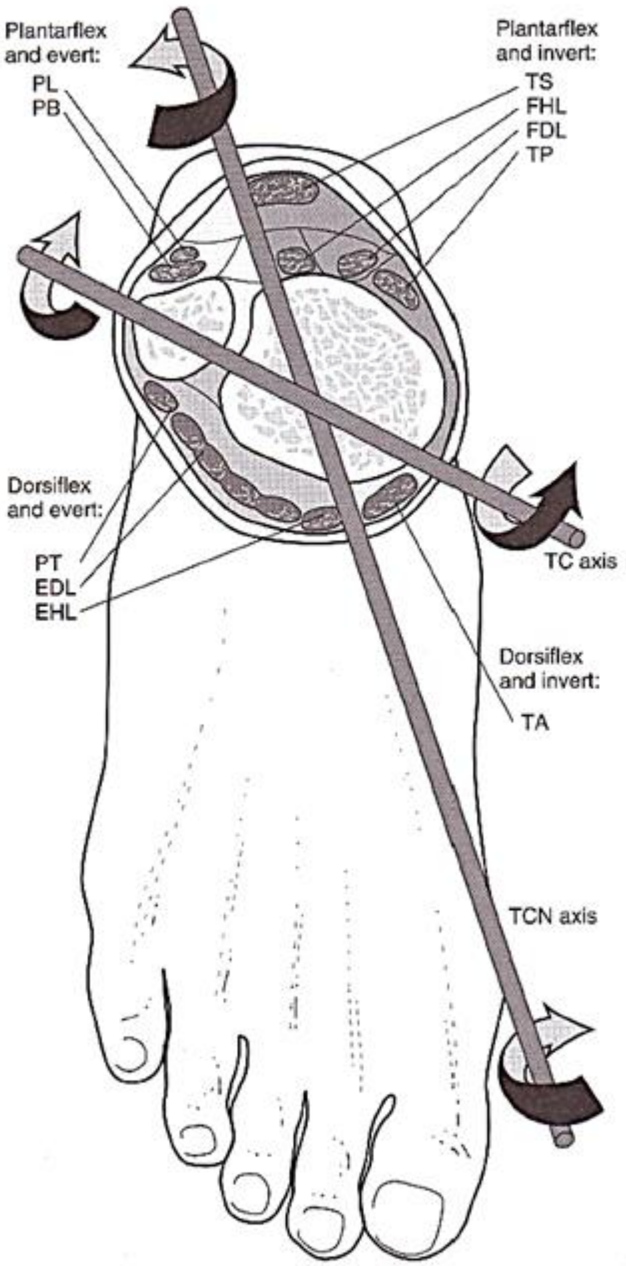

Figure 1.1: Talocrural and talocalcaneonavicular axes of motion. Adapted from Dutton, 2012

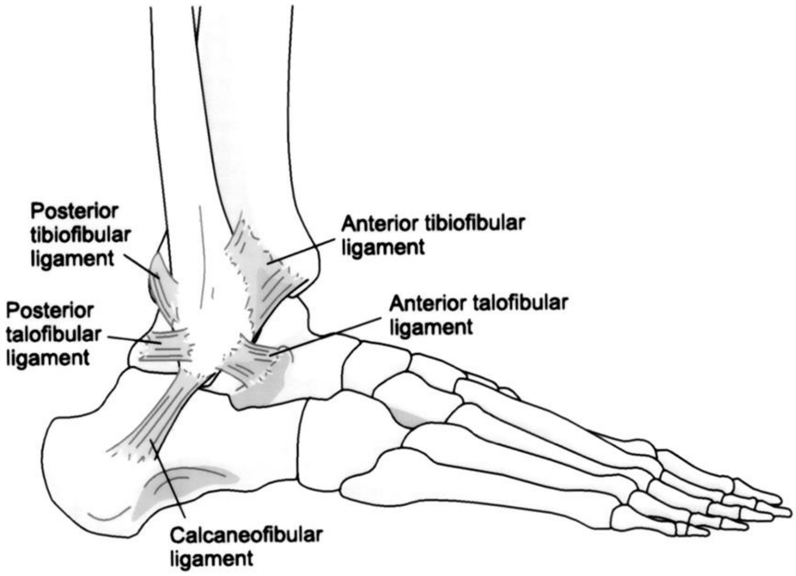

Figure 1.2: Lateral ligaments of the ankle joint. Adapted from Dutton, 2012

1.2.2 Anatomy and biomechanics of the subtalar joint

The subtalar (talocalcaneal) joint is formed by the articulations between the talus and the calcaneus (Stiehl. J.B 1991; Rockar Jr, P. A, 1995; Hertel et al., 1999; Dutton, M. 2012; Hertel, J. 2002; Moore et al., 2013). The subtalar joint is a synovial, bicondylar compound joint consisting of two separates, modified ovoid surfaces with their joint cavities and allows the motion of pronation and supination (Dutton, M 2012; Hertel, J. 2002; Moore et al., 2013). The subtalar joint is divided into two joints; anterior (talocalcaneonavicular) and posterior compartments separated from each other by the sinus tarsi and canalis tarsi (Hertel, J. 2002; Moore et al., 2013; Rockar Jr, P. A, 1995). The anterior subtalar joint is formed from the head of the talus, the anterior-superior-facets, the sustentaculum tali of the calcaneus, and the concave proximal surface of the tarsal navicular (Rockar Jr, P. A, 1995). The posterior subtalar joint is formed between the inferior posterior facet of the talus and the superior posterior facet of the calcaneus (Rockar Jr, P. A, 1995). The anterior and posterior joints share a common axis of rotation with an anterior joint having medial and higher centre of rotation than the posterior joint (Perry, J. 1983). This arrangement of the subtalar joint accentuates its oblique axis of rotation in the sagittal and transverse planes with 42^0 upward tilt and 23^0 medial angulations from the perpendicular axis of the foot (**Figure 1.3)** (Stiehl, J. B. (Ed.) 1991) and produces simultaneous movement in sagittal, frontal, and transverse planes to cause pronation and supination of the foot (Dawe, E. J., & Davis, J. 2011). Pronation primarily incorporates the cardinal plane motions of eversion, external rotation, and dorsiflexion, while supination primarily involves inversion, internal rotation, and plantarflexion during non-weight bearing position (Stiehl, J. B. (Ed.) 1991).

The stability to the subtalar joint is provided by the CFL, the cervical ligament, the interosseous ligament, the lateral talocalcaneal ligament, the tibiotalocalcaneal ligament (ligament of Rouviere), and the extensor retinaculum (Harper, M. C. 1992). Studies have reported greater strain in the cervical ligament following the complete disruption of the CFL (Martin et al.,1998) and subtalar joint injury to occur in as many as 80% of the patients during an initial ankle sprain injury (Meyer et al., 1988). The increased supination moment (associated with excessive inversion and internal rotation of the rearfoot coupled with external rotation of the lower leg) in the closed kinetic chain activities is suggested to be the primary injury mechanism of an ankle sprain (Ekstrand, J., & Tropp, H, 1990; Fuller, E. A. 1999; DiGiovanni, C. W., & Brodsky, A. 2006).

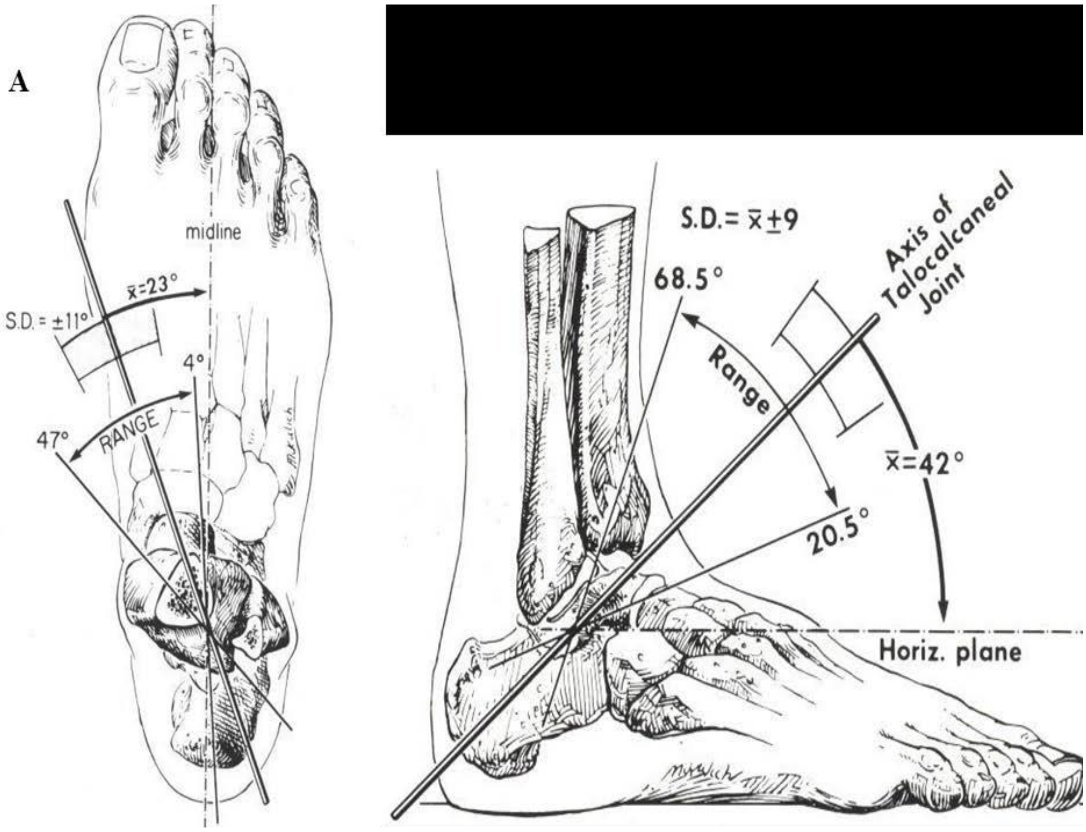

Figure 1.3: Subtalar joint's axis of rotation. Transverse plane (A) and sagittal plane (B). Adapted from Stiehl, 1991.

1.2.3 Anatomy and biomechanics of the distal tibiofibular joint

The distal tibiofibular joint is a formed by the articulations between the concave tibial surface and a convex or plane surface on the medial distal end of the fibula (Dutton, M 2012; Hertel, J. 2002; Lin et al., 2006; Stiehl, J. B. (Ed) 1991). This joint is a fibrous joint (syndesmosis), except for about 1 mm of the inferior portion, which is covered in hyaline cartilage (Dutton, M 2012, Lin et al., 2006). The integrity of the distal tibiofibular joint is critical to provide stability for the talus at the talocrural joint (Dutton, M 2001; Hertel, J. 2002). The syndesmosis allows limited movement between the two bones; however, the accessory gliding motions at this joint are required to maintain standard mechanics of the ankle complex (Hertel, J. 2002; Soavi et al., 2000). The movements at the distal tibiofibular joint consist of involuntary anterior-posterior glide and slight spreading of the mortise of the talocrural joint (Soavi et al., 2000; Lin et al., 2006). Coupled motions occur with the superior tibiofibular joint with fibula gliding superiorly during dorsiflexion and inferiorly during plantarflexion (Soavi et al., 2000). The distal tibiofibular joint is maximally stable in dorsiflexion that results in the greatest talar contact and lowest average pressure (Lin, C. F., Gross, M. T., & Weinhold, P. 2006; Nordin, M., & Frankel, V. H. (Eds.) 2001).

The stability of the distal tibiofibular joint is provided by four ligaments, collectively known as the syndesmotic ligaments. These include the inferior interosseous ligament (primary stabiliser), the anterior inferior tibiofibular ligament, the posterior inferior tibiofibular ligament, and the inferior transverse ligament (Dutton, M 2012), Lin et al.,2006). The ligaments of the distal tibiofibular joint are thought to be more commonly injured than the ATFL (Vaes, P. H., & Duquet, et al., 1998). Injury to the ankle syndesmosis often occurs as a result of forced external rotation of the foot or during internal rotation of the tibia on the planted foot (Hockenbury, R. T., & Sammarco, G. J. 2001, Lin et al.,2006). The injury to the syndesmotic ligaments of the distal tibiofibular joint results in high (syndesmotic) ankle sprain (Miller et al., 1995; Hertel, J 2002).

1.2.4 Muscles of the lower leg

Musculotendinous units that cross the ankle joint complex afford adequate protection to the joint by generating stiffness during various activities (Hertel, J. 2002); Grüneberg et al., 2003). The extrinsic muscles of the lower leg can be divided into anterior, posterior superficial, posterior deep, and lateral compartments (Moore, K.L et al., 2013; Dutton, M. 2012).

The anterior compartment of the leg contains the dorsiflexors (extensors) of the foot. The muscles of the anterior compartment include tibialis anterior (dorsiflexion and inversion of ankle), extensor digitorium longus (extends lateral four digits and dorsiflexes ankle), extensor hallucis longus (extends great toe and dorsiflexes ankle), and peroneus tertius (dorsiflexes ankle and aids in foot inversion) (Dutton, M 2012), Moore et al., 2013). These muscles are active during walking, helping with clearing the forefoot off the ground by contacting concentrically during the swing phase and lowering the forefoot to the ground by contracting eccentrically after heel strike during the stance phase (Rockar Jr, P. A. 1995). The deep peroneal nerve innervates all muscles of the anterior compartment and supplied by the anterior tibial artery (Moore et al., 2013).

The superficial posterior compartment of the leg contains the calf muscles that plantarflex the foot, necessary for walking in an upright bipedal stance, running, and jumping via push off (Moore et al., 2013). The muscles of the superficial posterior compartment include the gastrocnemius (plantarflexes ankle and flexes leg at the knee joint), soleus (plantarflexes ankle independent of knee position), and the plantaris muscle (plantarflexes ankle) (Dutton, M 2012), Moore et al., 2013). All muscles of both the superficial and deep posterior compartments are innervated by the tibial nerve and supplied by the posterior tibial artery and the fibular artery (Moore et al., 2013).

The deep posterior compartment of the leg contains the flexors of the foot that provide dynamic stability to the lateral ankle complex by contracting eccentrically during forced supination of the rearfoot (Moore et al., 2013). The muscles of the posterior deep compartment include the tibialis posterior (plantarflexes ankle and inverts foot), flexor digitorium longus (flexes lateral four digits, plantarflexes ankle, and supports longitudinal arch of the foot), and flexor hallucis longus (flexes great toe at all joints, weak plantarflexor, and supports medial longitudinal arch of the foot) (Moore et al., 2013). All muscles of the deep posterior compartment are innervated by the tibial nerve and supplied by the posterior tibial artery and the fibular artery (Moore et al., 2013).

The lateral compartment of the leg contains the evertors of the foot that are integral to the control of supination of the rearfoot and help protect against lateral ankle sprains (Ashton-Miller et al., 1996, Moore et al., 2013). The muscles of the lateral compartment include the peroneus longus (everts foot and weakly plantarflexes ankle) and peroneus brevis muscle (everts foot and weakly plantarflexes ankle) (Moore et al., 2013). All muscles of the lateral compartment are innervated by the superficial peroneal nerve and supplied by the perforating branches of the anterior tibial artery superiorly and the perforating branches of the peroneal artery inferiorly (Moore et al., 2013).

1.3 Mechanism of Injury for Lateral Ankle Sprain

Ankle sprains commonly occur in the so-called ‘‘high-risk’’ sports like team handball, basketball, soccer, or volleyball, which are characterized by a high level of jumping and cutting movements (Garrick, J. G., & Schelkun, P. H. 1997; Maehlum, S., & Daljord, O. A. 1984; Ekstrand, J & Tropp, H. 1990; Quinn et al., 2000). The most common mechanism for a lateral ankle sprain is the forced inversion or supination of the foot complex during landing on an unstable or uneven surface (Almquist, G.1974; Kannus, P. E.., & Renstrom, P. 1991; Baumhauer et al., 1995; Wolfe, M. W. 2001). Excessive inversion and supination of the ankle joint are limited by the lateral joint capsule, the lateral ligament complex of the talocrural joint, and the ligaments supporting the subtalar, and distal and proximal tibiofibular joints. If the supporting structures are overloaded (strained) beyond their tensile strength, disruption in their fibrous integrity occurs leading to dysfunction of one or more joints in the ankle complex (Bahr et al., 1994, Ekstrand, J., & Tropp, H. 1990). This injury mechanism may also lead to lesions (overstretching) of the sensory nerves (branches of the sural and superficial peroneal nerves) or the peroneus tendons (van den Hoogenband et al., 1984).

A lateral ankle sprain occurs when there is ankle inversion accompanied with an internal twisting of the foot or when there is plantarflexion with an adducted and inverted subtalar joint (Safran, et al., 1999; Vitale, T. D., & Fallat, L. M. 1988). External rotation of the lower leg concerning the ankle joint soon after the initial contact of the rearfoot can also cause a lateral ankle sprain (Hertel, J. 2002). Stormont and coworkers (1985) suggested that joint stability is established by bony congruency during weight bearing. They observed that most of the ankle sprains occurred during the systematic loading and unloading, but not while the ankle joint was already loaded. Konradsen et al., (1997) reported that before landing, the body must rely on ligamentous and musculotendinous sources of stability rather than the bony congruency. Since the ligamentous and musculotendinous structures are not as stable as bony structures, lateral ankle sprains frequently occur during landing. The ATFL is reported to be most often injured when landing during plantarflexion; however, when the landing is done during dorsiflexion, the calcaneofibular ligament is often injured (Bennett, W.F., 1994). Andersen et al., (2004) in their video analysis of the ankle sprain injury mechanisms in football players, identified two primary mechanisms: (1) Landing with the ankle in a vulnerable inverted position due to laterally directed force on the medial aspect of the leg by an opponent, either before or at a foot strike; (2)

Forced plantarflexion due to landing on the opponent's foot when attempting to shoot or clear the ball.

Fuller, E.A (1999) described that most ankle sprains are caused by increased supination moment at the subtalar joint, which occurs as a result of the position and the magnitude of the vertically projected ground reaction force at initial foot contact. If the center of pressure lies medial to the subtalar joint axis, a greater supination moment from the vertical ground reaction force can be achieved when compared to a foot that has a center of pressure lie lateral to the subtalar joint axis (**Figure 1.4**). The increased supination moment may result in sudden explosive ankle supination (excessive inversion and internal rotation of the rearfoot) during closed kinetic chain activities, and if the movement is beyond physiologic limits, a lateral ankle sprain may occur. In another study, Stiehl and Inman (1991) reported significant variability in the subtalar joint axis alignment across individuals and suggested that a foot with a laterally deviated subtalar joint axis would have a greater area on the medial side of the joint axis. This lateral deviation would increase the likelihood of medial placement of the center of pressure about the subtalar joint axis and thus more extended supination arm. If the magnitude of the supination moment exceeds the counterbalancing pronation moment, excessive inversion and internal rotation of the rearfoot may occur, leading to lateral ligament injuries (Fuller, E.A 1999).

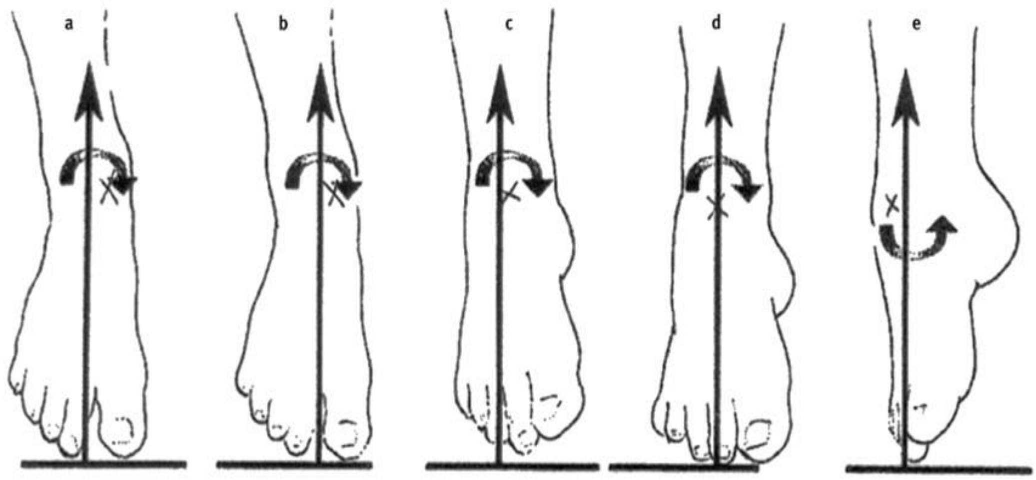

Figure 1.4: Diagram showing the lateral drift of the subtalar joint axis from (a) neutral to (e) plantarflexion and inversion, increasing the risk of injury. Adapted from Tropp, 2002

In a computational forward dynamic stimulation study, Wright and colleagues (2000) reported that increased plantarflexion at initial contact might increase the likelihood of encountering a lateral ankle sprain. Some studies have also suggested a strong association between limited ankle joint dorsiflexion and lower extremity overuse injuries (Johanson et al., 2006; Lynch et al., 1996).

Using a biomechanical model, Konradsen, L., & Magnusson, P. (2000) suggested a connection between a defect in ankle position sense and an increased risk of recurrent lateral ankle sprains. They reported that in a healthy individual, an inversion error greater than 7 degrees would drop the lateral border of the foot by 5 mm and engage the ground during the late swing phase. For a rotational error of approximately 8 degrees, it was calculated that placement error would occur for once for every 1000,000 steps before heel strike (Konradsen et al., 1998). Foot contact at the later stage of the swing phase may result in tripping, causing possible sprain of the ankle joint. Angle replication errors are usually increased after an initial sprain, so theoretically for an injured patient who has 100% greater replication error, a small difference in angle replication errors may increase the placement error to once for every 1000 steps before heel strike (Konradsen, L. 2002).

Another aetiology that has been proposed for a lateral ankle sprain is the delayed reaction time of the peroneal muscles during a rapid inversion event (Isakov, E et al., 1986; Konradsen et al., 1997; Vaes et al., 2002; Fong et al., 2007). Numerous research groups have reported peroneal muscles reaction time to be 50 ms or more (Dufek, J. S., & Bates, B. T. 1991; Konradsen, L., & Ravn, J. B 1991; Hopper et al., 1998; Konradsen et al., 1998; Fernandes et al., 2000; Vaes et al., 2002;Hopkins et al., 2007) which is not quick enough to oppose the ankle supination motion that is initiated around 40 ms when landing from a jump (Ashton-miller et al.,1996). It has been proposed that if the peroneal muscles are to protect against an unexpected inversion of the foot, preparatory pre-activation of the peroneal muscles before the foot contact is necessary (Konradsen et al., 1997). Additionally, researchers have suggested that the peroneal muscles may not be strong enough to withstand a body-weight load acting with a lever arm longer than 3 to 4 cm and if shear force is added, torque around the ankle increases (Tropp, H. 2002). Ashton-Miller and colleagues (1996) further reported that a force of one body weight located more than 3.4 cm medial to the midline of the near-maximally inverted foot would result in forced inversion injury despite maximal evertor muscle force.

1.4 Incidence and Risk Factors for Ankle Sprains

The successful rehabilitation of a lateral ankle sprain is often tricky because of unknown risk factors that lead to high injury recurrence rate (Safran et al., 1999;

Willems et al., 2002). Several studies have tried to identify the incidence (Doherty et al., 2014) and risk factors associated with ankle sprains (Fong et al., 2007; Beynnon et al., 2002; de Noronha, et al., 2013; Fousekis et al., 2012; Hiller et al., 2008; McHugh, et al., 2006; Willems, et al., 2005) but a review of literature reveals conflicting results.

The ankle is one of the most injured joints in the body. Fong et al., (2007) in their systemic review on ankle injury and ankle sprain found ankle to be most commonly injured body site in 24 of 70 included sports an ankle sprain to be a major ankle injury in 33 of 43 sports. Ankle ligament sprains are reported to be the most common injury for college athletes in the United States (Hootman et al., 2007). Recently in a meta-analysis of 181 prospective epidemiological studies, Dohert et al., (2014) found lateral ankle sprains to be the most common type of ankle sprain. They noted a higher incidence of ankle sprain in females compared with males (13.6 vs 6.94 per 1,000 exposures), in children compared with adolescents (2.85 vs 1.94 per 1,000 exposures) and adolescents compared with adults (1.94 vs 0.72 per 1,000 exposures). The sports category with the highest incidence of ankle sprain was indoor/court sports, with a cumulative incidence rate of 7 per 1,000 exposures or 1.37 per 1,000 athlete exposures and 4.9 per 1,000 hours.

Risk factors for an ankle sprain injury are commonly classified as intrinsic (those from within the body) and extrinsic (those from outside the body) (Williams, J. G. P. 1971). Various studies have investigated anthropometrical characteristics, foot type and size, ankle and foot laxity, the range of motion, history of previous ankle sprain, functional motor performances, ankle joint position sense, isokinetic ankle muscle strength, lower leg alignment, balance and postural control, and muscle reaction time with conflicting results. Of all the variables studied, the literature has consistently indicated the history of previous ankle sprain as the most significant predictor of an ankle sprain (Hertel, J. 2002). Barker et al. (1997) reported that a previous sprain history, a foot size with increased width, an increased ankle eversion to inversion strength, plantarflexion strength and the ratio between dorsiflexion and plantarflexion strength, and limb dominance could increase the ankle sprain injury risk. The foot type, an indication of ankle instability, and high general joint laxity were not identified to be risk factors. They also suggested that among external risk factors, increased exercise intensity can lead to increased injury risk whereas the use of orthosis in players with previous sprain history could help in decreasing the risk for an ankle sprain injury. Beynnon and colleagues (2002) found little agreement in the literature and reported that gender, generalised joint laxity and anatomical foot type were not risked factors for ankle sprain injury. In contrast to this finding, Morrison and Kaminski (2007) noted that increased foot width, cavovarus deformity, and increased calcaneal eversion range of motion could increase chances of sustaining a lateral ankle sprain injury.

Willems et al. (2005) investigated the intrinsic risk factors separately for males and females. The intrinsic risk factors for males included slower running speed, reduced cardiorespiratory endurance, decreased balance, reduced dorsiflexion muscle strength, decreased dorsiflexion range, less coordination ability, and the faster reaction of the tibialis anterior and gastrocnemius muscles. For females, they concluded that a reduced passive joint inversion position sense, a higher extension range of motion at the first metatarsophalangeal joint, and a decreased coordination of postural control were the major risk factors. Some recent studies have also identified reduced ankle dorsiflexion range (de Noronh et al., 2013), posteriorly positioned fibula (Eren et al., 2003), decreased single leg balance (Trojian, T. H., & McKeag, D. B. 2006), being overweight (Tyler et al., 2006), and no stretching before exercise (McKay et al., 2001) as other major intrinsic factors for ankle sprains.

1.5 Mechanical Ankle Instability

Mechanical instability of the ankle is generally considered to be present when the ankle joint motion is beyond the normal expected physiological or accessory range of motion (Delahunt et al., 2010; Hertel, J. 2002; Karlsson, J., & Lansinger, O. 1992). Mechanical ankle instability has been defined as excessive inversion laxity of the rear foot or excessive anterior laxity of the talocrural joint as assessed by using instrumented (arthrometry or stress radiography) or manual stress testing (Delahunt et al., 2010). Hertel, J. 2002) believes that anatomical changes following an initial ankle sprain lead to insufficiencies in the joint stability and function, which predisposes the ankle to recurrent episodes of instability. The changes associated with mechanical ankle instability may include pathological ligament laxity (Eren et al., 2003, Hubbard, T. J., & Hertel, J. 2008, Liu W. S Siegler & Techner 2001) impaired arthrokinematics (Hubbard, T. J., & Hertel, J. 2008; Hubbard et al., 2007; Wikstrom et al., 2010), and synovial and degenerative changes (Digiovanni et al., 2004; Hintermann, et al., 2002).

1.5.1 Ligament laxity

Ankle joint stability is provided by both active and passive components. While the active stability is derived from active or reflex mediated muscle contraction, the passive stability of the ankle joint complex is provided by the static ligamentous restraints, congruency of the articular surfaces and other connective tissues (Hertel, J. 2002), Liu, W., Siegler, S., & Techner, L. 2001). The lateral side of the ankle complex is stabilised primarily by three ligaments – anterior talofibular ligament (ATFL), calcaneofibular ligament (CFL), and posterior talofibular ligament (PTFL) (Golano et al., 2010, Hertel, J. 2002). The function of ATFL is to restraint plantarflexion and inversion motion and since most of the lateral ankle sprains occur during a jump or placing a foot in a plantarflexed and inverted position (Wolfe, M. W. 2001), the ATFL is the most commonly injured ligament during a lateral ankle sprain (Karlsson, J., & Lansinger, O. 1992. Renstrom, P.A., & Lynch, S.A, 1998) reported that an isolated tear of ATFL occurs in approximately 80% of all lateral ankle sprains and combined tear of ATFL and CFL occur in other 20%. Injury to one or more of the lateral ligaments and less than the optimal healing of the injured tissues often results in residual talocrural and subtalar joint laxity (Hertel, J. 2002).

Several studies have investigated talocrural joint laxity in patients with CAI (Cordova et al., 2010), however conflicting results have been reported in the literature. While some authors reported an increased mechanical laxity (Croy et al., 2012; Hubbard, T.J 2008; Hubbard, J et al., 2005) others did not report an increase in laxity (Liu, W et al., 2001) , On average, more studies have reported greater laxity being present in unstable ankles than in those without symptoms of ankle instability; however, it has also become clear that hypomobility may be as much of a concern as hypermobility (Hubbard et al., 2007). Similarly, some authors have reported hypermobility at the subtalar joint Hertel, J. (2002) while others reported of hypomobility at the subtalar joint after a lateral ankle sprain (Denegar, C. R., & Miller III, S. J. 2002). The inconclusive evidence reported in the literature could be due to the use of varied assessment methods in quantifying and diagnosing the talocrural and subtalar joint laxity. Some studies have also questioned the reliability and validity of the methods and tests used to measure mechanical joint laxity (Tohyama et al., 2003). These findings could also suggest that mechanical instability is not present in all patients with CAI and the observed residual symptoms may result from impaired neuromuscular control.

Clinical assessment of the mechanical ankle joint laxity typically involves manual examination techniques such as the anterior drawer, talar tilt, and inversion-eversion stress tests (Kerkhoffs et al., 2002). However, the subjectivity in differentiating the degree of lateral ligament stability make manual stress tests inaccurate for diagnosing specific ligament involvement (Fujii et al., 2000). Also, researchers have questioned the reliability and usefulness of stress radiographs despite its use in numerous ankle-ligament injury studies (Harper 1992). Siegler, S et al., 1996 described a six-degrees-of-freedom instrumented linkage for measuring the flexibility characteristics of the ankle joint complex in vivo. Since then ankle arthrometer has been used in many studies for the diagnosis of mechanical ankle instability. An ankle arthrometer is a reliable and valid diagnostic tool (Hubbard et al., 2004) and provides an objective assessment of the load-displacement characteristics of the joint within physiological range at a lower cost (Kerkhoffs et al., 2002). However, one of the disadvantages of the arthrometry test is the inability to control involuntary muscle contractions that may affect the measurement outcome (Kerkhoffs et al., 2002). Another issue is the fixation of the arthrometer across the joint. Stable fixation of the arthrometer is required to minimise soft-tissue motion, but too tight fixation can result in pain and will be intolerable to the patient. On the other hand, if the fixation is too loose, correct bone-to-bone motion cannot be measured due to excessive soft-tissue motion (Lapointe et al.,1997).

Researchers have often relied on the quantity of motion and the amount of resistance at the extreme of passive physiological motion to determine the flexibility characteristics of the ankle joint. Increased joint flexibility or the decrease in passive stiffness has been suggested to represent mechanical laxity indicating a weakness in passive joint restraints (Pope, M. H., & Panjabi, M. 1985). Previous in vivo studies have indicated that there is higher reliability in assessing the amount of resistance at the extreme of passive physiological motion than assessing the range of motion (Siegler et al.,1994). These results indicate that ligament laxity can be indirectly evaluated through the measurement of the passive joint stiffness (a measure of resistance to stretch). The average load-displacement characteristics (moment relative to angular displacement) can be used to demonstrate the neutral zone and non-linear behaviour of the passive resistance with increasing range of motion. In this study, we will be Assessing the ligamentous laxity or instability in the ankle by performing Anterior drawer test; this test primarily assesses the strength of the Anterior Talofibular Ligament.

1.5.2 Arthrokinematic impairments

Many in vitro studies have found a significant increase in the ankle joint laxity following sectioning of the lateral collateral ligaments (Kjaersgaard-Anderson, P et al.,1991). In agreement with the in vitro studies, in vivo studies have indicated the presence of ankle joint hypermobility and increased accessory motion following an acute lateral ankle sprain (Hubbard, T. J., & Hertel, J. 2006). The increased accessory motion at the joint leads to enlargement of the neutral zone of a joint (Panjabi, M.M 1992), which further strains the injured ligaments. The early loading and frequent straining may lead to subsequent effusion from the soft tissue damage (Hetherington, B. (1996)). Delayed collagen fibers healing, and alterations in the crimp pattern of the ligaments (ligament elongation) during the healing process that may result in the change of talocrural joint's axis of rotation to become more anterior or posterior in the frontal plane (Hubbard, T. J. 2008). Some studies have also indicated fibular positional faults following a lateral ankle sprain (Hubbard, T. J., & Hertel, J. 2006; Mavi et al., 2002; Scranton et al., 2000; Wikstrom et al., 2010). If the axis of rotation shifts anteriorly, posterior gliding of the talus may be limited resulting in decreased dorsiflexion range of motion and thus joint hypomobility (Denegar, C. R., & Miller III, S. J. 2002). Alternatively, joint hypomobility can result from the scar tissue formation disrupting the fibrous structure of the ligament following an injury (Wikstrom et al., 2010). Additionally, Hertel, J. (2000) believe that injury to ligamentous structures can also lead to alteration of the mechanoreceptors, joint capsule, Golgi tendon organs and muscle spindles. The altered input may affect the output in both the injured and healthy ankle (Lephart et al.,1997) leading to changes in the neuromuscular system, altered muscle recruitment patterns and joint arthrokinematics.

1.6 Functional Ankle Instability

Past studies have suggested that the mechanical instability alone cannot independently explain the ongoing residual symptoms in patients after an initial ankle sprain (Delahunt, E. 2007). Researchers agree that some pathological process distinct from the MAI is present in these patients, and the phenomenon of recurrent, persistent symptoms in the absence of aberrant mechanical laxity has been termed as functional ankle instability (FAI) (Delahunt et al., 2010).Wilkerson et al., (1997) described this enigmatic nature of FAI, in which no relationship between the method of initial treatment and the prolonged residual symptoms is apparent. In contrast to MAI, there is no universally accepted definition or inclusion criteria for FAI (Konradsen, L. 2002).

Frequent/recurrent ankle sprain and episodes of, or the reporting of, feelings of ankle giving way are the most commonly described symptoms to define FAI in research and clinical literature (Delahunt et al., 2010). Recently, attempts have been made to devise operational definitions related to ankle joint sprain and its subsequent sequelae, as well as provide standards for patient/participant selection criteria focused on CAI (Delahunt et al., 2010; Gribble et al., 2013).

FAI was first described by Freeman et al., in (1965) as a condition of a recurrent ankle sprain and ongoing episodes of ankle giving way. Freeman proposed that FAI can be attributed to damage to the afferent receptors of the joint after a sprain injury. Such damage can result in proprioceptive deficits that consequently lead to an increased incidence of the ankle giving way into hyper-supination because of inadequate peroneal muscle response to the aberrant ankle positioning. This theory was challenged in the 1990s by the reports that failed to consistently show deficits in measures of proprioception or postural control after anaesthetizing the lateral ankle ligaments (Riemann, B et al., 2004). Since then several causal factors of FAI have been suggested to explain why this condition develops in a high percentage of patients. The functional insufficiencies have been attributed to impaired proprioception (Matsusaka et al., 2001; Refshauge et al., 2003; Robbins et al., 1995; Santos, M. J., & Liu, W 2007; You et al., 2004) strength deficits (Munn et al., 2003; Pontaga, I. 2004) , altered neuromuscular control (Van Cingel et al., 2006). However, there have been conflicting results reported in the literature regarding the role of each suggested etiological factor of FAI, and none of those factors can explain why and how the ‘recurrent sense of ankle giving way’ occurs.

1.6.1 Impaired proprioception

The term proprioception was first used by Sherrington in 1910 (Burke, R.E 2007), who coined the term from the Latin *(re)ceptus* (the art of receiving) and *propius* (one's own). Proprioception is generally defined as the ability to assess a respective limb's position without the assistance of vision. (Lephart et al., 1992) Defined proprioception as a "specialised variation of the sensory modality that encompasses the sensation of joint movement (kinesthesia) and joint position (joint position sense)". Proprioception system is governed by both central and peripheral mechanisms that are mainly obtained from mechanoreceptors upon detection of joint displacements or perturbations (Ashton-Miller et al., 2001). Michelson, J. D., & Hutchins, C. 1995) believe that two levels of proprioception exist: conscious (voluntary) and unconscious (reflexive). The conscious aspect of proprioception is thought to be involved in controlling activities of daily living and during sports, whereas unconscious aspect is thought to be involved in maintaining joint stability and postural stability through complex responsive neural muscle activation (Riemann, B. L., & Lephart, S. M. 2002).

Freeman et al., (1965) hypothesised that following a lateral ankle sprain, the ligamentous tissue might heal effectively, but mechanoreceptor disruption within the lateral ligaments and talocrural joint capsule can lead to articular partial deafferentation, causing impaired proprioception. This impairment may affect the stabilisation of the foot, leaving it functionally unstable, i.e. with a tendency to give way that may further lead to the faulty ankle joint positioning and increase in the probability of injuring the joint. Since then, proprioceptive deficiency following ankle sprain has been the focus of many studies that tried to identify the cause of FAI, but