EFFECTS OF VIBRATION FEEDBACK DURING GAIT ON VERTICAL GROUND REACTION FORCE IN CHRONIC ANKLE INSTABILITY PATIENTS

Juliana Leigh Workman

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Approved by:

Erik Wikstrom

Brian Pietrosimone

Kimmery Migel

ABSTRACT

Juliana Leigh Workman: Effects of vibration feedback during gait on vertical ground reaction force in chronic ankle instability patients.
(Under the direction of Erik Wikstrom)

Lateral ankle sprains often lead to long-term impairments including altered gait biomechanics. The current interventions used to treat LAS are ineffective at modifying gait or preventing long-term deficits including PTOA.

Does vibration feedback gait retraining result in a LR change, and is it retained? Do changes in kinetic variables associate with outcomes within CAI domains (perceptual, sensorimotor, mechanical)?

We conducted a repeated measures design with 19 individuals with CAI. Participants completed laboratory and RW sessions. We assessed baseline, post-test, and retention gait kinetics.

We found decreases in vGRF LR after laboratory gait retraining baseline-posttest ($p=0.026$) and posttest-retention ($p=0.016$), but they weren't retained, and no RW differences existed. Positive correlations occurred between LR change and IdFAI ($p=0.019$), LR and plantar cutaneous threshold at 1MTP ($p=0.013$), and phase 1 COP change with eyes open balance ($p=0.035$). An association existed between phase 1 COP change with a cavus foot compared to neutral ($p=0.040$).

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

LIST OF ABBREVIATIONS

AP – Anterior-posterior

CAI – Chronic ankle instability

COP – Center of pressure

COPv – Center of pressure velocity

EC – Eyes closed

EO – Eyes open

FAAM – Foot and Ankle Ability Measure

FSR – Force sensing resistor

GRF – Ground reaction force

IdFAI – Identification of Functional Ankle Instability

JPS – Joint position sense

LAS – Lateral ankle sprain

LR – Loading rate

ML – Medial-lateral

MTP – Metatarsophalangeal

PTOA – Post-traumatic osteoarthritis

ROM – Range of motion

RW – Real world

SWM – Semmes Weinstein monofilaments

vGRF – Vertical ground reaction force

CHAPTER 1

INTRODUCTION

Clinical Significance

Lateral ankle sprains (LAS) are highly prevalent injuries among both athletes and the general population, making up about 15% of all athletic injuries.[1,2] Often, the acute impairments from lateral ankle sprain injuries heal in a short time, and as many as 50% of people don't seek medical attention or injury rehabilitation after a LAS.[3,4] Although about 70% of people return to sports within three days after injury,[5,6] many people may suffer from long-term consequences after an initial lateral ankle sprain, including development into chronic ankle instability (CAI) and/or post-traumatic osteoarthritis (PTOA). Approximately 40% of individuals who sustain a LAS will develop CAI,[7] and LAS may contribute to as many as 80% of all ankle PTOA cases.[1,8] These conditions of CAI and PTOA that can occur after an initial injury contribute to the substantial societal and financial burden of long-term treatment of chronic ankle conditions.[1] With a high incidence of ankle injuries and the contribution of chronic musculoskeletal disorders to global healthcare problems, there is a large portion of the population that may suffer from chronic ankle conditions.

Chronic ankle instability (CAI) is highly prevalent and can lead to many impairments. CAI is a cluster of symptoms that can be defined by sensations of "giving way", recurrent ankle sprains, and persistent disability.[1,2,9–13] The most

commonly reported symptoms by patients with CAI include swelling, pain, weakness, stiffness, and instability.[4,9,10,14] Changes and impairments in individuals with CAI can involve the perceptual, mechanical, and sensorimotor domains. Mechanical changes in individuals with CAI include ligamentous laxity, most frequently of the anterior talofibular ligament.[8,15–17] Despite the ligament disruption, hypomobility of the ankle joint is common with CAI due to arthrokinematic restrictions and positional faults.[5,18–21] Individuals with CAI also display impairments in sensorimotor control, proprioception, balance, and biomechanical alterations.[1,21–23] A biomechanical alteration often associated with CAI includes a gait pattern with a more inverted ankle joint position and a laterally deviated center of pressure (COP) distribution.[12,24–29] An inverted ankle joint position during gait puts the individual in a position close to the typical mechanism of injury for LAS,[1,11,12,30–33] therefore this inversion position and lateral shift in the COP can increase the risk of subsequent injury. Additionally, these common CAI impairments are thought to contribute towards the development of ankle PTOA by contributing to aberrant loading of the talar articular surface[34] which over thousands of steps could facilitate mechanical failure of the talocrural collagen fibers.[35,36] Altered loading is thought to be caused by excessive compressive forces over a reduced contact area.[37] While not an exact match, vertical ground reaction forces (vGRF) and vGRF loading rates can provide insight as to how the talar surface is loaded. Those with CAI have been shown to have elevated peak vGRF and increased vGRF loading rates relative to uninjured controls.[38,39]

A variety of interventions have been traditionally used to treat the impairments from LAS and CAI, but there are no proven treatments to alter gait mechanics or prevent the degeneration of cartilage and subsequent development of PTOA. For example, arthrokinematic restrictions can be improved using anterior-to-posterior mobilizations of the talus.[20,40,41] Similarly, strength training of the evertor muscles in individuals with CAI improves strength,[21,25,28,32,42] and balance training improves postural control in CAI groups.[5,13,24] However, gait alterations are not typically treated in CAI patients and when done, traditional rehabilitation of CAI does not alter this pathologic gait pattern.[43–45] However, novel biofeedback interventions using visual, vibrational, or auditory feedback have been effective to create short-term improvements in foot eversion position and COP location while walking.[27,43,44,46–48] These results suggest that gait retraining with biofeedback could reduce the risk of recurrent injury, common to individuals with CAI. However, there remains no evidence regarding the ability of biofeedback gait retraining to minimize neuromechanical variables (e.g., vGRF) associated with PTOA development at the ankle. Therefore, the purpose of this investigation was to learn how a vibration biofeedback intervention affects vGRF loading rates and if the effects are associated with common CAI impairments.

Research Questions & Hypotheses

1. Does vibration feedback gait retraining result in an immediate change in loading rate in individuals with CAI?

a. If an immediate change in loading rate does occur, is it retained over a brief period of time?

   We hypothesize that loading rate will show an immediate decrease in loading rate and that this decrease will be retained. We hypothesize that there will be larger changes after gait retraining in the laboratory setting versus the real world setting.

2. Do changes in kinetic variables (loading rate change and COP location change) associate with outcomes within the domains (perceptual, sensorimotor, mechanical) of CAI?

   a. Is there an association between kinetic changes and baseline perceptual measures (IdFAI, FAAM-ADL, FAAM-Sport)?

      We hypothesize that no association will exist between kinetic changes and the baseline perceptual measures.

   b. Is there an association between kinetic changes and baseline sensorimotor measures (balance, joint position sense, cutaneous thresholds)?

      We hypothesize that associations will exist between kinetic changes and sensorimotor measures.

   c. Is there an association between kinetic changes and a baseline mechanical measure (foot type)?

      We hypothesize that no association will exist between kinetic changes and foot type.

CHAPTER 2

LITERATURE REVIEW

Epidemiology

Lateral Ankle Sprain

Prevalence Numbers

Lateral ankle sprains (LAS) are very prevalent musculoskeletal injuries, accounting for 15% of all injuries among athletes.[1,2] Approximately 23,000 ankle sprains happen per day in the United States, contributing to an estimated risk of one ankle sprain per 1,000 hours of sports.[1,49] A survey of collegiate athletes identified a history of previous LAS in 65.2% of individuals, demonstrating that athletes are highly susceptible to ankle injuries and therefore at risk for long term consequences.[3] Injuries such as ankle sprains are widely believed to fully recover on their own with time, therefore many people never seek treatment or rehabilitation.[2] However, research indicates that about 40% of people who sustain a LAS suffer from long term consequences of these injuries and develop chronic ankle instability (CAI).[7] A survey by Hiller et al[4] found that three-quarters of the surveyed general community population in Australia reported a history of ankle injury or chronic ankle problems. Similarly, others have determined that about 50% of people do not see a healthcare provider at the time of injury.[1,5,9] This indicates that although lateral ankle sprains are very common, they are often undertreated and result in both short-term and long-term consequences including loss of playing time, laxity, chronic pain,

functional instability, decreases in physical activity and health-related quality of life, and CAI.[1,4,17] LAS and the associated sequela affect many people across the entire lifespan, representing a large overall healthcare burden.[9]

Financial impacts of LAS

An acute LAS has a high direct cost to treat, and there are additional indirect costs accrued from follow-up care and time loss from work or sport. Once the condition has progressed to CAI and/or ankle joint post-traumatic osteoarthritis (PTOA), there are even greater costs to treat.[1] In a study conducted by Knowles et al. in 2007,[50] the mean comprehensive cost for an ankle sprain in a high school athlete in the United States was $2,733, which includes both the direct healthcare costs and the indirect societal costs of time loss.[1] Based on LAS frequency estimates and the approximate cost per injury, it is suggested that the comprehensive cost of LAS in the United States is over $6.2 billion annually.[1,50,51] The societal costs of all LAS sustained during sports participation in the Netherlands over one year was estimated to be about €208 million.[52] These are alarmingly high financial costs for such a prevalent injury. Additionally, since more than half of people do not seek treatment by medical professionals for LAS, these are likely underestimations of the true financial impact of LAS.[1,4,53]

Mechanism of LAS Injury

More than three-quarters of acute ankle sprains are to the lateral ligament complex, which is composed of the anterior talofibular, calcaneofibular, and posterior talofibular ligaments. Approximately 75% of lateral ankle sprain injuries involve the anterior talofibular ligament, the calcaneofibular ligament is involved in 50 to 75% of

LAS, and the posterior talofibular ligament is involved less than 10% of the time.[8,15–17] Lateral ankle sprains threaten the integrity of these ligaments, causing laxity.[9]

LAS can occur as a result of non-contact or contact injury mechanisms, and are primarily caused by the ankle rapidly inverting and internally rotating.[1,54,55] Several authors[30–32] have reported observations of accidental LAS during research laboratory testing, and the findings of the kinematic patterns differ from what was previously believed to be mechanisms of injury.[1,31] It was previously believed that plantarflexion, inversion, and internal rotation were the kinematic components involved in LAS, however these laboratory-recorded incidences of LAS all observed rapid inversion and internal rotation, with no consistent findings relative to sagittal plane movement (dorsiflexion or plantarflexion).[1,15,30–32,54] A more inverted position of the ankle joint at initial contact with the ground is a vulnerable position for the ankle to sustain a lateral ankle sprain, because the ground reaction force vector is located more medially to the joint axis, creating a large external eversion moment upon loading.[11,33] The foot is forced into inversion, sometimes resulting in hyper-inversion and trauma to the lateral ankle ligaments.[12] About half of ankle injuries are incurred during jump landing, and an additional third of injuries are due to a sharp twist or turn while the foot is planted.[53] LAS most often occur during the transition from non-weight bearing to weight-bearing.[1,15,33,54]

Long Term Sequalae of LAS

Most commonly, initial inflammation causes acute deficits in individuals with lateral ankle sprain, however deficits remaining beyond the acute inflammatory phase create additional mechanisms of dysfunction that lead to the pathway of

chronic ankle instability.[5] At least 40% of LAS patients progress into chronic ankle instability (CAI)[7], a condition which involves recurrent sprains, feelings of instability, and lasting disability for longer than 12 months after the initial injury.[1,2,9] Patients with an acute LAS often exhibit similar deficits within the same four impairment domains (decreased ROM, strength, postural control, and functional activity) that have been identified in patients with chronic ankle instability.[5,9]

A noteworthy impact of LAS is the high recurrence rate, which likely happens because the injured structures are not fully healed prior to return to sport.[5] Approximately 44.4% of LAS are non-time-loss injuries[16] and 70% of patients return to playing sports and participating in activities within three days after an acute LAS.[5,6] It's theorized that this may occur because the initial inflammatory phase lasts approximately 3 days. However, impairments often last even after return-to-sport, and some impairments may last for years or the lifetime after the initial ankle sprain injury. Inadequate treatment of a lateral ankle sprain can lead to the perpetuation of impairments long after the acute event, and the prevalence of this is high because less than half of people seek medical care after experiencing an ankle sprain[1,5]

Chronic Ankle Instability

Chronic ankle instability (CAI) is a condition involving the presence or sensation of "giving way", history of recurrent ankle sprains, and persistent disability post-injury.[1,2,9–13] These symptoms persist for at least 12 months after the initial lateral ankle sprain injury.[5,9,10] Approximately 31.1% of high school athletes and 18.7% of all collegiate athletes surveyed were found to have CAI.[3]

An original model of CAI was developed by Hertel[11] in 2002 that involved both mechanical and functional instability of the ankle joint. This model has been more recently updated to describe many factors that can play into whether an individual develops CAI or recovers fully after an ankle sprain (Figure 1).[19,56] It can be difficult to define cases of CAI in the literature, because there is no single test used to define CAI since it is a cluster of symptoms.[8] A series of inclusion and exclusion criteria for selection of subjects with chronic ankle instability has been recommended by the International Ankle Consortium[9,10] that incorporates contributions from mechanical instability, functional deficiencies, and perceived instability.[11,56]

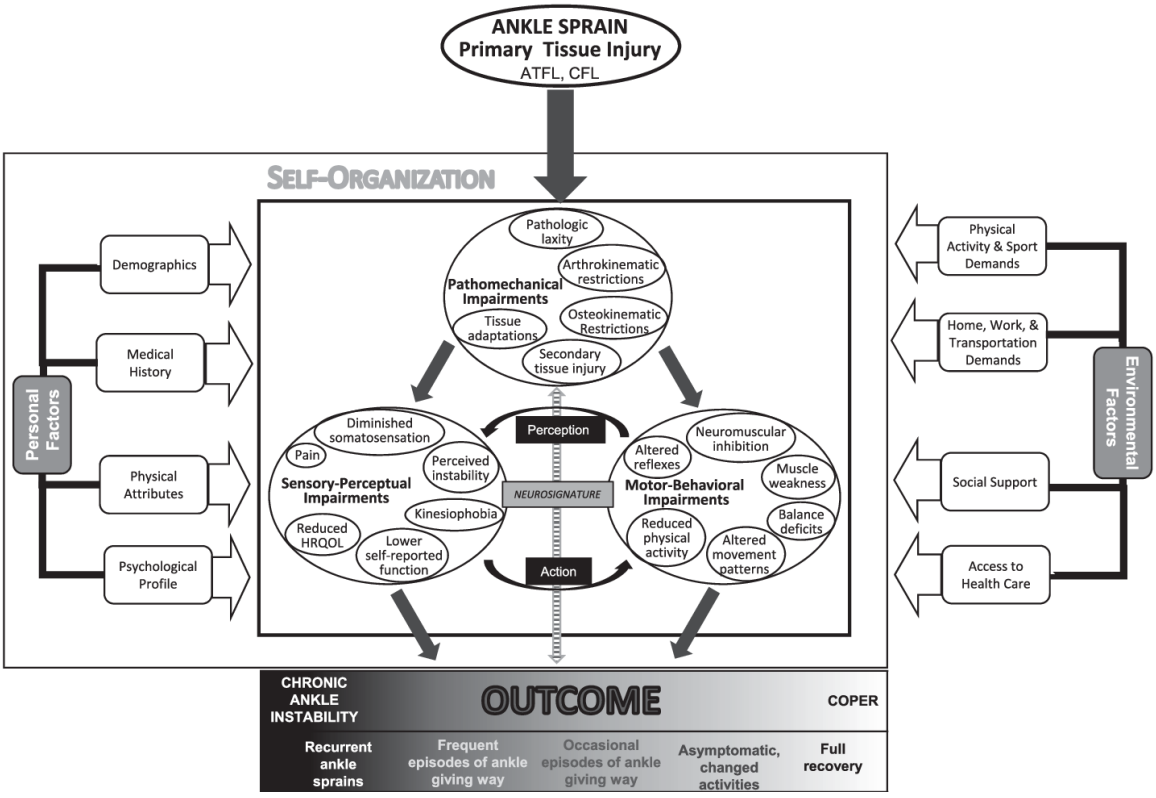

Figure 1: The updated model of CAI. The outcome (i.e. CAI development) cannot be determined until at least 12 months after the initial injury.[19]

CAI to PTOA

Ankle joint posttraumatic osteoarthritis (PTOA) has been linked to CAI. Early degenerative changes, cartilage lesions, and intra-articular pathologies are found in a large percentage of individuals a short time after suffering an acute LAS.[1] Approximately 50% of individuals with end stage ligamentous ankle PTOA report a history of repeated ankle sprains.[8] Individuals who develop PTOA after a LAS or CAI often will show degenerative changes within several months after an acute injury, and they can have substantial deficits at a young age as a result.[1,57] Additionally, altered walking mechanics in people with CAI may create changes in loading at the ankle joint and altered kinematics, which affects the long-term health of the articular cartilage over time.[21,58,59] In other lower extremity joints such as the hip and knee, arthritis is primarily degenerative and seen in older patients. However, 70-80% of ankle arthritis is posttraumatic and is present in younger patients.[60] For example, a study by Wikstrom et al found that people ages 18-35 with CAI had a greater T1ρ relaxation time (CAI: 65.97 ± 10.45 ms, Control: 58.84 ± 7.68 ms; ES = 0.76, 95%CI = 0.02-1.50), which indicates greater degenerative changes of the talar articular cartilage composition.[57] Other research on ankle cartilage has also used T2 relaxation times, a measure of cartilage degeneration, to examine changes during different loading conditions.[57,61] A significant ($P<0.005$) increase in T2 relaxation times in the medial talar cartilage was found in individuals with functional ankle instability compared to ankle sprain copers and healthy controls, indicating that structural modifications are present in this population.[61] Increases in both T1ρ and T2 values demonstrate that early degeneration of the talar cartilage and subsequent

development of PTOA occurs in individuals with CAI.[57,61] This demonstrates the burden on the healthcare system to treat these individuals' impairments early post injury, and emphasizes the importance of strategies to attempt to prevent both CAI and PTOA development.

Impairments/Consequences of CAI

Perceptual (Patient Reported) Outcomes

The chronic symptoms that often occur after LAS and in individuals with CAI are the most limiting factor that affects continued sport participation. Individuals who experienced an ankle sprain and reported they were chronically impaired by the sprain described the sensation of "giving way", weakness, swelling, and pain.[9,14] Of these people, 55% had to limit or modify either activities of daily living or sport activities because of lasting symptoms of their previous ankle sprain.[14].

The most commonly reported deficits associated with CAI are recurrent ankle sprains and episodes of feeling of the ankle joint 'giving way', with residual symptoms including ankle stiffness, pain, swelling, instability, and weakness.[4,10,14] Subjective and perceptual findings from patient-reported outcome instruments are used for evaluation of CAI, in addition to the structural assessment of mechanical ankle instability.[18] There are a variety of clinical assessment tools and patient subjective questionnaires that can be used to evaluate if an individual has CAI.[3,18,62]

IdFAI

The Identification of Functional Ankle Instability (IdFAI) is a self-reported patient outcome used to evaluate if an individual meets the criteria to be included in

a functional ankle instability population.[63,64] The IdFAI has an accuracy of 89.6% and test-retest reliability of 92% and is commonly used to identify participants for CAI research.[63,65] A higher score indicates less functional ability of the involved ankle.

FAAM-ADL and FAAM-Sport

The Foot and Ankle Ability Measure (FAAM) has been used for a wide array of ankle and foot disorders, including CAI. It includes two subscales, with 21 items for activities of daily living (ADL) and an additional 8 items for sport, and individuals rate their function on a scale from 0%, complete inability, to 100% of their pre-injury function.[66] The individuals who reported their ankles were “normal” scored higher on both the FAAM-ADL and FAAM-Sport subscales compared to those who reported their ankles were “nearly normal” or “abnormal”.[66,67] A greater difference exists between healthy and CAI groups in the average scores on the sport subscale compared to the ADL, showing that athletes experience less difficulty with performing ADLs compared to sports activities.[67] The FAAM can be used to track an individual’s responses over time, meaning it can show progress and improvements in their function.[18]

Mechanical Changes

Joint Laxity

The anterior talofibular ligament is damaged in about 75% of lateral ankle sprain injuries,[8,15,16] and the calcaneofibular ligament is involved in 50-75% of LAS.[17] A ligament sprain involves stretching or tearing of the collagen fibers that compose the ligament, causing structural damage of the tissue. Disruption of the ligaments that stabilize the ankle creates laxity and instability, as well as clinical symptoms of

pain, swelling, inflammation, and sensorimotor dysfunction. When the ligamentous restraints to excessive motion are damaged, the ankle joint can move into motions beyond the physiologic limit. The term laxity is often used interchangeably with the term mechanical instability, which is movement of the ankle joint beyond the physiologic limit of its range of motion.[12] This can be assessed by performing arthrometry or by using clinical joint integrity tests, such as the anterior drawer and talar tilt tests.[11] These tests evaluate excessive translation of the talus on the tibia.

Alternatively, joint laxity can be assessed with imaging techniques including ultrasound, MRI, and stress radiography.[18,19] Imaging tools like these provide a non-invasive view inside the ankle joint, and changes in the ligamentous integrity and the joint space can be seen and measured either statically and dynamically. The clinician can apply a clinical joint integrity test while using imaging and may visibly see a change in the joint space. When comparing these techniques, MRI established a better true positive identification of CAI (sensitivity 83%, specificity 53%), whereas stress radiography had a better true negative identification (sensitivity 66%, specificity 97%).[18,68] The extent of laxity at the ankle joint is seen inconsistently in CAI patients, which suggests that some of the instability associated with CAI is sensory and perceptual, not mechanical.[19] This joint laxity and resultant instability of the talus within the ankle mortise is what leads to damage to the most superficial layers of ankle cartilage.[60] Cartilage plays a role in joint mechanics and force dissipation, and has an important role in resisting development of osteoarthritis.[60]

Positional Faults

Despite the pathologic laxity involved with disruption of ligaments, individuals with CAI commonly present with hypomobility.[18,19] The lack of motion can be associated with arthrokinematics restrictions and positional faults in the ankle joint complex.[18] When the ankle dorsiflexes, the talus must glide posteriorly. However, in people with CAI, the talus can be translated anteriorly, creating a bony block to achieving full dorsiflexion.[5,19] A positional fault of the fibula may also be present, with the distal end anteriorly translated, which can also contribute to the restricted posterior talar glide.[18]

Range of Motion

Arthrokinematic restrictions, ligamentous laxity, and positional faults may contribute to increases or decreases in range of motion (ROM) in individuals with CAI. The global range of motion of the ankle joint is the same as healthy controls when measured at rest, but ROM during functional activities is altered in individuals with CAI.[5,69] A study by Drewes et al[69] found that while jogging, a CAI group had significantly less dorsiflexion in the midstance phase compared to a control group, which supports prior research that individuals with CAI frequently have functional dorsiflexion hypomobility.[5] In a CAI population, limited range of motion in dorsiflexion may be due to either persistent inflammation or positional faults of the talus and fibula creating bony blocks to full motion.[5,20,21] The weight-bearing lunge test (WBLT) is commonly used to measure dorsiflexion ROM, and a lack of full motion of the ankle joint identified with the WBLT may cause changes in sensorimotor function and gait mechanics, contributing to further dysfunction.[40,70–72]

Foot Type

Foot posture can be categorized into pes cavus, normal, or pes planus. A pes cavus foot is a foot with a high medial longitudinal arch, whereas a pes planus foot has a flatter medial longitudinal arch. The Foot Posture Index (FPI) is used in many studies to measure foot alignment along a scale from supinated to pronated.[73–75] There has been conflicting research about whether a correlation exists between foot type and the incidence of ankle sprains.[76] A pes cavus foot with a high longitudinal arch could cause the center of pressure to be located more laterally, putting an individual's foot in a position where it is more likely to experience excessive inversion and suffer a LAS.[77] Increased calcaneal eversion and increased talar tilt were found to be risk factors for ankle sprains in one study,[78] but other studies found a higher incidence of ankle sprains in individuals with a high arch.[76] More recent research has found no correlation between foot type and the presence of CAI in participants who had a history of ankle sprain.[73,74,79]

Altered joint alignment is commonly present in end-stage symptomatic PTOA. Foot alignment during CAI may contribute to these alignment issues when PTOA develops at a later time. Valderrabano et al.[60] found that in patients with PTOA due to ankle sprains, 52% of ankles had a rearfoot varus malalignment, 27% had a normal alignment, and 21% had a rearfoot valgus malalignment. There were 33 cases examined and 15 of these had CAI. Of the CAI cases, 67% (10 cases) had rearfoot varus malalignment.[60] This research study also found that patients with a history of LAS and/or CAI tended to have cartilage damage on the medial ankle and had developed varus-malalignment ankle osteoarthritis.[60]

Sensorimotor Alterations

Functional instability that contributes to CAI involves adverse changes to the neuromuscular system that provides dynamic support to the ankle, including articular and ligamentous mechanoreceptors in the lateral ankle ligaments.[11] The sensorimotor system uses information from joint, cutaneous, and muscle receptors to control movement, and when damage occurs to any of these mechanoreceptors they are unable to send information to the central nervous system and proprioceptive deficits occur.[12,80] Individuals with CAI demonstrate changes in the spinal-level control during a single leg balance task.[71,81,82] Proprioceptive deficits in both excitability and inhibition result from damage to these mechanoreceptors, and muscle-spindle activity in the peroneus longus and peroneus brevis can also be altered in those with CAI.[11] Balance, proprioception, and reaction time have been used to assess deficits in sensorimotor control.[23] Neuromuscular control deficits cause insufficiencies of the dynamic stabilizers of the ankle that act as a dynamic defense mechanism to preventing excessive inversion of the ankle.[11] Individuals with CAI have been observed to have multiple sensorimotor deficiencies.[18,80]

Deficits in plantar cutaneous sensation have been found in individuals with CAI when compared to uninjured controls and copers.[83,84] Light touch sensation and touch thresholds can be assessed with Semmes-Weinstein monofilaments (SWM).[84] SWMs are applied perpendicular to the skin surface at the test location, and 10 grams of pressure is applied until the SWM bends into a C shape.[84] Decreased cutaneous sensitivity is noted when individuals require a higher SWM threshold, and therefore a larger force applied to achieve the same perception of pressure on the

skin.[83,84] Other sensorimotor impairments present in people with CAI, which may contribute to recurring ankle injuries, includes deficits in both passive and active joint position sense (JPS), theorized to be caused by the damaged mechanoreceptors in the ankle ligaments.[85] Assessments of JPS may have many variations such as the starting foot position, method of repositioning (active or passive), testing ROM, testing velocity, type of comparison (between-groups or between-limbs), and method of data-reduction, however, a meta-analysis by McKeon and McKeon[85] found that none of these variables were more indicative of CAI than the others, and JPS deficits were consistently present across all these variables in people with CAI. People with CAI have subtle impairments in joint position sense and cutaneous sensation, but research has not conclusively determined if these sensorimotor deficits are a cause or a result of CAI.[85]

Strength

Muscle strength changes can occur in patients with CAI, which may influence the ability to dissipate energy and force at the ankle, possibly leading to an even greater ankle inversion motion during subsequent lateral ankle sprain incidents.[32] The ankle evertors, particularly the peroneus brevis and peroneus longus, are sometimes found to have decreased strength.[32] These muscles may not be as active as others in the lower extremities, so improving the ankle evertors' strength may help patients with CAI develop strategies to prevent recurrent episodes of instability.[32,42]

Individuals with greater plantarflexion strength and a smaller dorsiflexion to plantarflexion ratio had a higher incidence of inversion ankle sprain.[86] Individuals

with an elevated eversion to inversion strength ratio had a higher incidence of ankle inversion injury.[12] Additionally, reaction time of the peroneal muscles is delayed in people with CAI ($p<0.001$), and differences in concentric eversion ($p=0.001$) and eccentric eversion ($p=0.008$) peak torque were significant between individuals with CAI compared to healthy controls.[23] Isometric muscular weakness at the ankle is not a major contributing factor to CAI, but muscle strength imbalances can be a predictor for initial injury and can cause continued deficits afterwards.[12,87] Proximal strength deficits have also been identified in this population and may contribute to poor gait biomechanics.[12,26,28,43] Other muscles in the lower extremity can be either overactive or underactive in CAI to compensate for weakness, resulting in abnormal gait patterns.[21,25] Strengthening the entire lower body in addition to specifically targeting the muscles around the ankle can help correct strength deficits and abnormal ratios and could potentially alter abnormal gait patterns developed as a result of CAI, but no evidence currently exists to show the relationship between muscle strengthening and alterations in gait.

Balance/Neuromuscular Control

A multitude of research has shown that both static and dynamic balance are impaired in CAI groups compared to healthy controls.[22,23,88,89] Individuals with CAI demonstrate an increased amount of time needed to stabilize the ankle during dynamic balance tasks, which likely contributes to the inability to stabilize the ankle during subsequent episodes of instability and then leads to recurring inversion ankle sprains.[23,90] Sensorimotor impairments and impaired postural control can also cause

altered force dissipation during static tasks and during dynamic tasks such as walking, leading to changes in the loading pattern of the ankle joint.[21]

Static postural control can be evaluated with single limb stance, and dynamic postural control can be evaluated with tasks such as the Star Excursion Balance Test.[5,90,91] Both static and dynamic postural control can also be assessed with a force plate to track center of pressure (COP) movements, with greater COP sway indicating either deficits in postural control or the use of alternate strategies to maintain balance.[22] Individuals demonstrate bilateral deficits in postural control after an acute LAS, signifying that central changes to neuromuscular control occur.[22,88,91] Deficits in static and dynamic postural control in people with CAI and after an acute LAS can be due to changes in neural signals and sensorimotor control.[9,71,81] A theory for why some people are LAS-copers while others develop CAI is that postural control deficits occur in all individuals after an acute ankle sprain event, but the copers developed compensatory strategies for postural control while those who developed CAI did not, allowing repeated injuries to occur.[22] It's also possible that impaired balance and postural control could be a causative factor for CAI, not only a result of the proprioceptor damage from the acute injury, and subsequent impairments in balance after injury may further contribute to the functional instability and recurrent injuries present with CAI.[23,56] Deficits in balance and postural control can be greatly prolonged in individuals who aren't treated, lasting up to 6 months post injury.[5,13,24]

Thompson et al[81] found that individuals with CAI have changes in spinal reflex responses of the soleus compared to LAS-coper and healthy individuals. Their data

demonstrate an increase in H-reflex soleus excitability in single leg stance, and a decrease in presynaptic inhibition in both double leg (330% reduction) and single leg stance (160% reduction).[81] Other research has found changes in H-reflex/M-response ratios in both the soleus and the peroneus longus muscles, as well as in the quadriceps and hamstrings.[80] These changes in sensorimotor control in CAI individuals compared to LAS copers and healthy individuals suggest that those with CAI may use alternate mechanisms of motor control in more challenging postures in both muscles that cross the ankle joint and more proximal leg muscles. The gastrocnemius and soleus are the primary muscles that control amount of postural sway and COP displacement in the anterior and posterior directions during stance, so alterations in the excitability and inhibition of the soleus may be an explanation for why some sensorimotor impairments of CAI exist.[81] However, perceptions of instability and pain can explain and predict differences in the soleus spinal reflex excitability in single leg stance and inhibition in both single and double leg stance.[81] This demonstrates that components such as pain and anxiety associated with instability can be contributory factors to why individuals with CAI alter their mechanisms of sensorimotor control.[71,81] The alterations in spinal excitability were not present in the LAS-coper group, only the CAI group, so it's possible that this may contribute to the reason why some people are unable to fully recover from a lateral ankle sprain injury.[81]

Biomechanical Alterations

Within several weeks or months after an acute LAS, most patients return to normal activities, including both athletic participation and daily life, and gait retraining

is not a continued goal of treatment. At 6 months post-LAS, patients walk with increased ankle inversion during push off and demonstrate bilateral increases in knee flexion, a gait pattern also commonly seen in patients with an acute LAS.[5,92,93] This suggests that these gait patterns develop during the acute injury phase, and the altered gait persists for a prolonged time period. Abnormal gait should be evaluated and treated during recovery from an acute LAS to attempt to prevent development of CAI and the later degeneration of cartilage leading to PTOA.[5] In individuals with CAI, a common gait alteration is increased ankle inversion prior to initial contact and a more inverted foot position and laterally deviated center of pressure (COP) during stance.[5,70,92,94]

COP is a kinetic measurement of postural control. As someone walks and moves through the phases of gait, the location on the foot where the greatest amount of pressure is being distributed to the ground changes. Starting with heel strike, as a healthy control moves through the stance phase of gait, the COP starts at the lateral heel and moves medially with the COP trajectory ending at the great toe at push off. Individuals with CAI tend to have a more laterally deviated COP during both single leg balance[24] and the entire stance of gait.[21,25,28] In patients with CAI, increased inversion of the ankle during gait can be a risk factor for recurrent sprain & episodes of instability, because the inverted position puts them closer to the mechanism of injury for an ankle sprain.[5] When the foot is inverted, the axis through which ground-reaction forces act moves laterally, causing the lateral deviation in COP previously mentioned.[12] During gait, postural corrections take place at the subtalar joint of the ankle with the corrective motions of inversion and eversion,

attempting to keep the foot stable.[12,25] If the COP deviates outside of the base of support, an episode of instability or ankle sprain occurs.[25] In people with CAI, greater trajectories of COP tend to occur when postural corrections are made at the hip joint.[12] The hip joint is important for overall postural control, and those with CAI may place a greater demand on the proximal muscles of the leg and hip to help compensate for impairments in static and dynamic balance.[25,43] However, the hip strategy to correct posture in the presence of an unstable ankle creates large shear forces with the ground, which can increase ankle inversion put the ankle at greater risk to give way.[12]

During the swing phase of gait, if the ankle is not appropriately positioned due to sensorimotor deficits, the initial contact can occur in an inverted position and as weight is transferred to the limb an external load is placed on the foot, which will potentially force it further into inversion and increase the likelihood of injury.[12] Walking with a more inverted foot position changes the location and amount of pressure across the talocrural joint, altering joint mechanics. This can be a factor in causing earlier degeneration of the cartilage and post-traumatic osteoarthritis, which then leads to long-term or even lifetime deficits.[57]

The altered COP and ankle position described above is likely due to individuals with CAI demonstrating altered muscle activation while walking. A CAI group had decreased activation of the tibialis anterior, peroneus longus, vastus lateralis, gluteus medius, and gluteus maximus muscles; and increased activation of the medial gastrocnemius relative to a control group.[21] The hip plays an important

role in force generation and dissipation in people with CAI to compensate for altered motor control of the ankle joint.[21]

Because of the connection between CAI and PTOA as well as the alterations in biomechanics that may influence the progression of CAI to PTOA, further research examining the metrics of joint loading is warranted. Several simple kinetic metrics during gait such as peak vertical ground reaction force (vGRF) and vGRF loading rate can be measured. These metrics are important because they have been shown to associate with measures of cartilage health following anterior cruciate ligament injury.[95,96] Individuals with CAI and with ankle osteoarthritis have been shown to have altered loading rates and differences in vGRF, relative to uninjured controls, during both walking[38] and running.[59] A possible contributing factor to this is a stiffer landing pattern; a decrease in functional range of motion of the ankle is common in those with CAI and can lead to landing with a stiffer ankle joint and subsequently a larger peak vGRF.[59] Another possible contribution is that while loading rate (LR) is essential to maintain long-term cartilage health, individuals tend to off-load their injured limb, demonstrating a lower peak vGRF, which associates with worse cartilage health.[95,96] While running, the LR is faster and peak vGRF is higher in those with CAI compared to controls.[59] Increased loading rates and higher ground reaction forces are demonstrated in people with CAI and this places abnormal stresses on the ankle, possible contributing to an increased rate of development of PTOA.[38] Identification of techniques capable of restoring appropriate gait biomechanics, particularly loading metrics, could help to mitigate the degeneration of ankle cartilage and slow the progression from CAI to PTOA.

Interventions

Numerous treatment strategies have been developed for those with CAI. Most are successful at treating impairments with a variety of short-term outcomes. However, the long-term impact of common treatment strategies remains unknown.

Traditional Treatment Strategies

A common measure taken to provide additional support to the ankle after an acute LAS and in patients with CAI is taping the ankle or using an ankle brace. It has been found that the application of an ankle brace provides proprioceptive information to the cutaneous receptors, improving ankle joint-position sense and improving static balance.[12,84] Taping or bracing the ankle can prevent excessive inversion of the foot during the swing phase of gait, prior to foot contact.[12] It can also protect against excessive inversion motion during the weight-bearing phases of gait, helping to avoid the vulnerable position of the ankle while the tape is in place. Ankle taping and bracing have both been shown to reduce the risk of ankle sprains in those with previous ankle injury.[97,98]

Anterior-to-posterior joint mobilizations of the talus on the tibia, a common manual therapy technique, can help improve dorsiflexion, improve the sense of stability, and restore arthrokinematic deficits in both CAI patients and acute LAS patients.[20,55] Additionally, this approach has improved patient reported outcomes and postural control.[40,41,71] However, limited evidence exists to suggest that ankle joint mobilizations can restore gait biomechanics. Strength training after LAS has been shown to improve balance, strength, and patient-reported outcomes.[99] Additionally, a correlation between decreased muscle strength and slower walking

speed was found in patients four weeks after experiencing an ankle sprain.[100] However, evidence does not exist to show a relationship between strength training and alterations in gait biomechanics. Similarly, rehabilitation can restore postural control to pre-injury levels within two weeks of supervised training.[5,13,24] Proprioceptive and balance preventative programs implemented for the rehabilitation of LAS have been effective in reducing ankle sprain recurrence in those with CAI.[8,12,49,52] Cumulatively, the available treatment strategies for CAI address and improve sensorimotor deficits, but do not alter gait mechanics and therefore a novel treatment approach is needed.

Gait Retraining

Gait training to correct motor patterns and improve postural control can help prevent the mechanism of recurrent injury in individuals with CAI, who tend exhibit this aberrant gait pattern.[43] Previous research has shown that traditional rehabilitation after an ankle sprain, involving restoring range of motion, strength, and postural control, does not cause a change in pathologic gait patterns.[43–45] Gait retraining in individuals with CAI using a feedback device, an elastic resistance band on the lower leg providing a medial force, was able to cause a significant medial shift in the location of COP ($p<0.005$) and increased muscle activity of the peroneus longus ($p<0.05$) during the stance phase over five sessions.[43] An auditory biofeedback device during gait retraining also was effective to alter the center of pressure more medially and improve the inverted foot position commonly found in CAI patients, however these were acute changes and may not persist during gait without the device.[46] Torp et al[27] found that the use of a laser pointer to provide

visual feedback during gait retraining created a significant medial shift in COP during the first 80% of the stance phase ($p<0.002$). With visual feedback, participants with CAI walked with less peak pressure on the lateral midfoot and lateral forefoot, creating a greater peak pressure at the great toe, and demonstrating a medially shifted COP trajectory.[27]

Haptic feedback, or vibration, may be more effective for gait retraining than elastic devices, visual feedback, or auditory feedback. Individuals with CAI have decreased plantar cutaneous sensation and decreased proprioception, contributing to their sensorimotor dysfunction, so haptic feedback may stimulate their somatosensory system and create motor changes in ways that other biofeedback devices cannot.[1,101] Changes in gait mechanics in a laboratory setting have been demonstrated with visual[27] and auditory[46] feedback, and haptic feedback has been exhibited to create a medial shift in COP location in people with CAI during both laboratory and real world gait retraining.[102] However, it remains unknown if gait retraining has an effect on variables (e.g. vGRF LR) associated with cartilage loading while walking and/or the subsequent degeneration in this population.

Summary

LAS are a prevalent musculoskeletal injury that often progresses to CAI and PTOA. This progression is believed to be facilitated by altered biomechanical patterns during gait. While numerous treatment strategies exist to address sensorimotor impairments observed in those with CAI, only gait retraining has been effective at altering gait biomechanics in those with CAI. However, it remains

unknown if gait retraining also improves gait loading characteristics in this population in the hopes of mitigating altered cartilage loading.

CHAPTER 3

METHODS

Design

A repeated measures design was used to determine the impact of vibration feedback on vGRF loading rate in individuals with CAI during independent sessions of laboratory training and real world training. Variables remained consistent between the real world and laboratory training sessions. The independent variable was time, measured at baseline, post-test, and retention, and the dependent variable was loading rate, calculated as the peak vGRF divided by the time from initial contact to peak vGRF.[38] Other variables measured included perceptual, sensorimotor, and mechanical outcomes, as well as COP location change, measured in millimeters.

Participants

Nineteen individuals with chronic ankle instability volunteered from a university setting to participate in this study. The university's Institutional Review Board approved this study and written informed consent was collected from all participants prior to study enrollment. Participants were between 18 and 45 years of age, had at least one significant ankle sprain more than one year prior to study enrollment, and had repeated episodes of "giving way".[9] Participants demonstrated self-reported limitations in function including a score ≥ 11 on the IdFAI questionnaire,[63] $\leq 90\%$ on the FAAM-ADL, and $\leq 80\%$ on the FAAM-S.[9,67] Individuals were excluded from this study if they had a history of lower extremity

surgeries, fractures in the lower extremity requiring realignment, or acute injuries requiring at least one missed day of physical activity within the 3 months prior to enrollment.[9] These criteria for inclusion and exclusion are in agreement with the guidelines established by the International Ankle Consortium.[9] If a participant reported limitations in both ankles (n=1), the patient identified their least stable ankle and that was used as the involved limb. The effect sizes from prior research on feedback devices in individuals with CAI has ranged from 0.2-3.04.[27,46,93] The sample size for this study was originally calculated with an estimated effect size of 0.3,[27,46] power of 0.8, and an alpha =0.05 to detect differences in kinetic variables. The power analysis indicated that at least 20 participants were needed to detect statistical differences over time.[102] This effect size estimate was a fairly conservative estimate, as this is an exploratory gait retraining technique in the CAI population.

Procedures

A convenience sample of a participants were recruited from a large university setting via fliers, word of mouth, and mass campus emails. The target population of this study was individuals with self-perceived CAI, and eligibility was determined through an initial online (Qualtrics) screening survey. The primary recruiter and principal investigator (PI) of this study was a third-year doctoral candidate. Following completion of the online screening protocol, a member of the research team followed up with potential participants via email to schedule the enrollment and first data collection session. Data was collected in the gait biomechanics laboratory in the MOTION Science Institute on the instrumented treadmill by the research team, who were trained in the research techniques prior to participant recruitment. A member of

the research team chaperoned the participant in the real world portion of data collection.

First, participants completed 5 walking trials between two timing gates (Dashr 2.0, Dashr Motion Performance Systems, Lincoln, NE) to determine their self-selected comfortable walking speed. The average from these 5 trials was used as the treadmill speed for the laboratory training and data collection portions of this research study. An instrumented split-belt treadmill (Bertec Corporation, Columbus, Ohio) was used to capture kinetic measurements. Kinetic data (i.e. vGRF) was collected at sampling rate of 1200 Hz, using two force plates embedded in the treadmill.[103]

Prior to the first gait retraining session, participants completed testing of joint position sense, monofilament plantar cutaneous sensation threshold at the head of the 1st and 5th metatarsals, and a postural sway assessment with eyes open and closed.

Participants then had the vibration feedback tool attached in their shoe and to their lower leg on the involved side. The vibration feedback tool was custom made and secured to the shoe and lower leg (See Figure 2). A force sensing resistor (FSR) (Model 402, Interlink Electronics, Inc, Camarillo, CA) was secured in the shoe with tape underneath the fifth metatarsal head, and the electronics and battery were housed in a custom enclosure attached to the shoelaces. The 200 Hz vibration motor with a displacement of <1 mm was attached to the lateral malleolus with an elastic strap. Pressure applied to the FSR under the lateral foot turned on the vibration motor which delivered a vibration stimulus to the lateral malleolus, notifying

the participant of an incorrect foot position. Each study participant had an individual threshold determined for the amount of pressure on the FSR before the stimulus was applied. The vibration stimulus at the lateral malleolus was received when pressure under the lateral border of the foot exceeded the threshold, encouraging a medial COP shift. During the gait cycle, the pressure typically fell below the threshold as the individual approached the swing phase. The intent of the feedback was altering the subsequent stance. To determine the FSR threshold the lowest electrical resistance was set so that standing on the involved limb triggered the vibration stimulus but standing on two limbs did not. This technique was based on previous calibration techniques for gait feedback devices but was modified based on pilot testing.[46] Study participants then walked on level ground with standard instructions “walk so you do not get the vibration” to test the calibration of the device, adapted from Donovan et al.[46] These standard instructions were utilized to avoid influencing the movement strategy selected by participants. The vibration feedback device was calibrated when the participants were able to walk with minimal vibration, and if not, the device was recalibrated with a decreased FSR threshold.

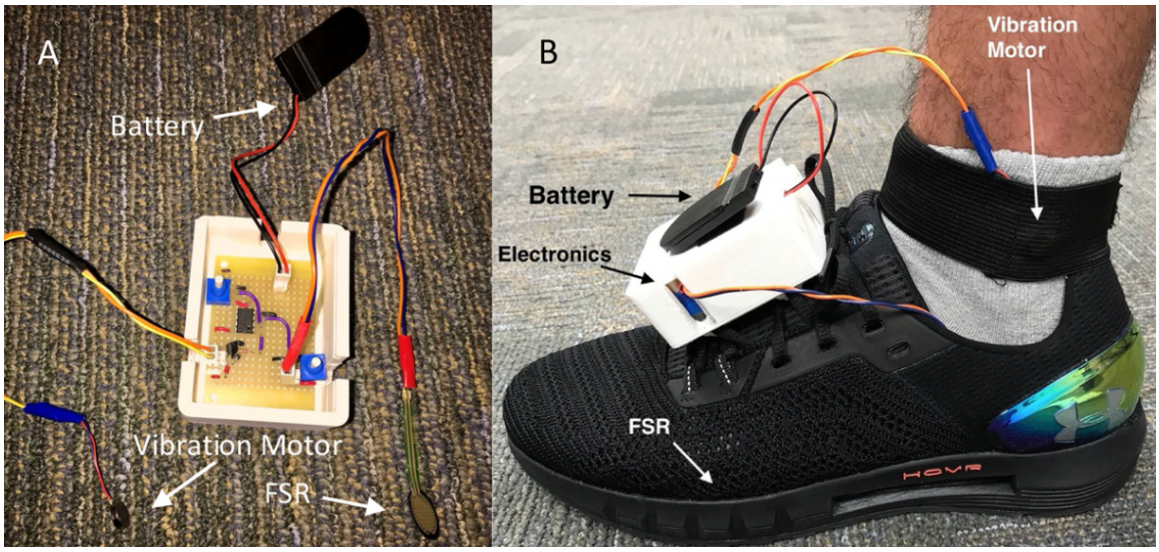

Figure 2: A: Feedback tool and its components. B: Placement of the feedback device on the participant's shoe.[102]

The baseline assessments began with the participant walking on the treadmill for 2 minutes without feedback. Baseline data was collected in the second minute to allow participants to adapt to any perception of weight from the feedback tool.[104] After the baseline assessment, participants completed one of two training sessions. The first type of training session involved laboratory training while walking on an instrumented treadmill with vibration feedback for 10 min. The second type of training session was real world (RW) training where the participant walked with vibration feedback for a one mile loop on a brick sidewalk, with supervision. The RW training session was timed with a stopwatch, and speed was quantified by dividing the known distance of 1 mile by the time needed to cover that distance.

Verbal instruction provided to participants was consistent across training sessions. The order in which the sessions were completed was counterbalanced to avoid a training effect, and the two sessions were separated by at least 48 hours to allow for sufficient wash out time. After the training session was completed, an

immediate posttest assessment was collected in the same manner as the baseline assessment, with participants walking on the instrumented treadmill for 2 minutes without feedback but with the device still attached to their shoe. The participants were provided with instructions to “walk normally,” and the posttest data was collected in the second minute. After a 5 minute rest break, participants completed a 2 minute walk on the treadmill without vibration feedback to assess for retention. Each data collection session lasted approximately one hour.

Outcome Measures

Kinetic

The force plates within the treadmill belts provided data to determine the kinetic variables (i.e., heel strike peak vGRF, COP location change) during walking gait. Visual 3D v7 (C-Motion, Germantown, MD) was used to distinguish the key stance phase events (i.e., steps) and calculate the COP location within the lab space. Synchronized marker trajectories were sampled at 120 Hz and used to calculate the COP location relative to the lateral border of the foot for each time point. For all complete steps of the involved limb, the stance phase was averaged and used for analysis. The stance phase was defined by a heel strike followed by toe off. Heel strike occurred when the vGRF increased above 20 N, and toe off occurred when the vGRF fell below 20 N. Loading rate of the vGRF was defined as the slope of the line between heel strike and the first peak of the vGRF divided by the time between heel strike and the first peak vGRF and was normalized to body weight.[38] The COP data were filtered with a second order Butterworth filter with a cut-off frequency of 10 Hz.[105] The COP location was then determined by subtracting

the COP location within the lab space from the position of the 5th metatarsal head trajectory within the lab space to calculate the distance of the COP from the lateral border of the foot for each timepoint. The stance phase was then divided into 10 subphases and the data within each subphase was averaged to become one point of representation.[27] The COP change was calculated by subtracting the baseline COP location from the posttest COP location. A medial shift of the COP was identified by a positive COP change value. For this investigation, the change in COP location was only calculated for the first two subphases of gait as they represent heel strike and loading response gait events, which is when peak vGRF occurs.

Perceptual

The Identification of Functional Ankle Instability (IdFAI) was developed based on the Ankle Instability Instrument (AII)[65] and the Cumberland Ankle Instability Tool (CAIT),[106] and has an accuracy of 89.6%.[63] It is a short questionnaire that takes less than 5 minutes to complete, on average.[63] A score of 11 or higher indicates the individuals who are likely to have functional ankle instability (FAI), and a score of 10 or lower indicates individuals who are unlikely to have FAI.[63] However, this questionnaire does not identify or rule out any other ankle conditions, and the score does not indicate the severity of FAI. We examined questionnaire data from our online survey, and used a minimum score of 11 on the IdFAI as one component of our inclusion criteria.[9]

The Foot and Ankle Ability Measures (FAAM) is an evaluative instrument which includes two sets of questions, separated into activities of daily living (ADL) and sports (S) related activities subscales. The FAAM-ADL includes 21 questions

and has a test retest reliability of 0.89, and the FAAM-S subscale has 8 items and a test retest reliability of 0.87.[66] This patient-reported outcome tool is used for determining functional deficits due to any musculoskeletal disorder of the leg, ankle, or foot. Changes in physical function over time is when the FAAM questionnaires are most useful.[66,67] Results of ≤ 90% on the FAAM-ADL, and ≤ 80% on the FAAM-S were used to determine if participants were included.[9,66,67]

Sensorimotor

Sensorimotor data (i.e., joint position sense, plantar cutaneous sensation, postural sway) was collected prior to the first gait retraining session. To measure participants' joint position sense, three trials were completed and averaged. For each trial their foot was passively positioned at the target position which was defined as half of their inversion active range of motion. They remained in that position for 2-3 seconds, and then were asked to relax. Participants were then instructed to move their foot back to the same position. The difference (error) in the degrees of ROM from the target position to the actively replicated position was recorded. JPS was measured with a goniometer with participants laying supine to prevent them from using vision to replicate the foot position.

Cutaneous sensation was measured on the plantar surface of the foot with Semmes Weinstein monofilaments (SWM). A Semmes Weinstein monofilament (SWM) set consists of a series of prenumbered single nylon filaments of decreasing diameters. The tools are prenumbered by the amount of force that needs to be applied through the tool to create 10 grams of pressure and therefore make the monofilament bend into a "c" shape. Decreased cutaneous sensitivity is noted when

individuals require a higher SWM threshold, and therefore a larger force applied, before they perceive the sensation.[83,84] The researcher tested the cutaneous receptors on the bottom of the foot at the 1st and 5th metatarsal heads by lightly touching the filament to the skin for 1-2 seconds until the SWM bent into the “c” shape, and the participant was asked to verbally indicate if they could feel the pressure from the monofilament. The researcher used a 4-2-1[107,108] stepwise procedure to find the smallest diameter filament that the participant could reliably sense. The 4-2-1 algorithm indicated that if the participant was able to accurately sense the largest diameter filament by indicating “yes”, the researcher would move down 4 diameter sizes with each subsequent trial until the participant was no longer able to sense the pressure from the monofilament. Once that filament was reached, the researcher selected the monofilament 2 sizes larger than the current monofilament to assess. If sensation was confirmed the researcher selected a filament one size smaller, or if there was a lack of apparent sensation the researcher selected a filament one size larger for the subsequent test. Single increments of monofilament changes continued until the researcher identified the smallest diameter monofilament that the participant could sense. The final monofilament was assessed 3 times to ensure accurate identification by the participant.

Balance was assessed with participants on the treadmill, standing on their involved leg for 3 sets of 10 seconds with their eyes open, followed by their eyes closed. The force plates within the treadmill recorded the excursion of their COP. Postural sway was quantified by the COP velocity (cm/sec), calculated from the duration of the trial (seconds) and the sway excursion (cm). 95% confidence ellipses

were calculated as a spatial measure to quantify COP. The average COP velocity and 95% ellipse from the three trials were calculated for each visual condition.

Mechanical

Participants' foot type was recorded during the baseline assessment. The foot was classified as pes planus, pes cavus, or neutral by a single researcher with 10 years of clinical experience in foot and ankle rehabilitation.

Statistical Analysis

To achieve Aim 1, 1-way repeated measures ANOVAs were used to compare vGRF loading rate over time (baseline, post-test, retention) for the laboratory and RW conditions separately. Statistical significance was determined based on an apriori alpha level of 0.05. Post hoc testing was completed as needed by using Wilcoxon Signed-Ranks tests. Hedges g effect sizes were calculated for pairwise comparisons and interpreted as large if g was above 0.80, moderate between 0.50-0.79, small between 0.20-0.49, and trivial if less than 0.20. Medians and ranges were calculated for vGRF LR at each time point. To achieve Aim 2, bivariate correlations were conducted between the kinetic variables and each of the measures within domains of sensorimotor, perceptual, and mechanical outcomes. Multiple regressions with dummy coding were used to compare foot type and kinetic variables. For this study, effect sizes (β values) above 0.80 were interpreted as strong associations, between 0.50-0.79 were considered moderate, 0.20-0.49 were small, and below 0.20 were trivial.[109] Associations were considered to be significant if $p \leq 0.05$.

CHAPTER 4

RESULTS

Q1: Does vibration feedback gait retraining result in an immediate change in loading rate in individuals with CAI?

1a: If an immediate change in loading rate does occur, is it retained over a brief (5 minute) period of time?

The data were not normally distributed, so a non-parametric Friedman's repeated measures ANOVA was used, and significant differences were noted among time points in the laboratory setting ($\chi^2(2) = 7.126$, $p=0.028$). More specifically, a statistically significant decrease in vGRF LR was found between baseline and posttest ($p=0.026$, $W=0.765$) with a small Hedge's g effect size ($g=0.29$). The laboratory based vGRF LR significantly increased between posttest and retention ($p=0.016$, $W=-0.824$) with a small effect size ($g=0.42$). LR also increased from the posttest to retention but the difference was not significant ($p=0.864$, $W=-0.059$) and had a small effect size ($g=0.21$). Medians and ranges for vGRF LR over the different time points can be found in Table 1.

A repeated measures ANOVA illustrated no significant differences in RW vGRF LR among baseline, posttest, and retention time points ($F(2,17)= 2.18$, $p=0.142$). Hedge's g effect sizes were small for vGRF LR changes in the RW session for baseline to posttest ($g=0.36$) and baseline to retention ($g=0.35$), and the effect size was trivial for posttest to retention ($g=0.11$).

	vGRF Loading Rate	Median	Range
Laboratory Training	Baseline	6.803	9.000
	Post-test	6.760	6.617
	Retention	6.951	6.650
Real World Training	Baseline	6.888	6.787
	Post-test	6.747	7.017
	Retention	6.735	6.502

Table 1: Medians and ranges for vGRF loading rate (N/BW/s)

Q2: Do changes in kinetic variables (LR change and COP location change) associate with outcomes within the domains (perceptual, sensorimotor, mechanical) of CAI?

As statistically significant changes in vGRF LR were only present within the laboratory training session and not the RW setting, associations were only examined using laboratory training data (Table 2).

	Variable	Mean	Median	SD	Range
Kinetic	Loading Rate change (N/BW/s)	-0.919	-0.104	2.793	12.470
	COP location change (mm)	3.335	3.156	3.977	21.910
Perceptual	IdFAI	21.470	22.000	4.454	14.000
	FAAM-ADL	82.280%	83.333%	7.410%	26.190%
	FAAM-S	65.572%	67.857%	9.366%	32.140%
Sensorimotor	Balance (EO in AP direction) (cm/s)	2.703	2.712	0.755	3.120
	Balance (EO ML direction) (cm/s)	2.737	2.554	0.752	3.256
	Balance (EC AP direction) (cm/s)	5.725	5.505	1.209	3.525
	Balance (EC ML) (cm/s)	6.039	5.604	1.878	6.946
	JPS (JPS error) (degrees of ROM)	4.007	2.333	3.636	13.663
	Cutaneous threshold (1MTP)	3.718	3.610	0.404	1.910
	Cutaneous (5MTP)	3.827	3.840	0.325	1.480
Mechanical		**Number**		**Percentage of sample**	
	Pes planus	7		35.6%	
	Neutral	10		52.9%	
	Pes cavus	2		11.5%	

Table 2: Descriptive data for variables included in Question 2 analysis. COP: center of pressure, IdFAI: Identification of Functional Ankle Instability, FAAM-ADL: Foot and Ankle Ability Measure Activities of Daily Living, FAAM-S: Foot and Ankle Ability Measure Sport, EO: eyes open, EC: eyes closed, AP: anteroposterior, ML: mediolateral, JPS: joint position sense, MTP: metatarsophalangeal joint.

Q2a: Is there an association between kinetic changes and baseline perceptual measures (IdFAI, FAAM-ADL, FAAM-Sport)?

Kinetics & Perceptual

To identify if there were associations between the change in vGRF loading rate and perceptual outcomes, Spearman's correlations were conducted because the data were not normally distributed. A statistically significant moderate positive correlation was found between LR change and the IdFAI (r_s=0.563, p=0.019). Individuals with a higher score on the IdFAI demonstrated a greater decrease in LR. No relationship was present between LR change and the FAAM-ADL (r_s=0.262, p=0.310), or the FAAM-S (r_s=0.322, p=0.207). To identify if an association existed between the perceptual outcomes and changes in the COP location at both phase 1 (initial contact) and phase 2 (loading response) of gait, Spearman's correlations were also conducted. No significant relationships were present between the IdFAI and COP change in phase 1 (r_s=0.095, p=0.727) or COP change in phase 2 (r_s=-0.170, p=0.529). There were no significant relationships between the FAAM-ADL and COP change in phase 1 (r_s=0.325, p=0.219) or phase 2 (r_s=0.261, p=0.329), and no significant relationships were found between the FAAM-S and COP change in either phase 1 (r_s=0.377, p=0.150) or phase 2 (r_s=0.338, p=0.200).

Q2b: Is there an association between kinetic changes and baseline sensorimotor measures (balance, joint position sense, cutaneous thresholds)?

Kinetics & Balance

There were no significant associations between LR change and COPv in the AP direction (r_s=-0.127, p=0.626) or COPv in the ML direction (r_s=-0.29, p=0.911) during a single leg eyes open stance task. There were no significant relationships between LR change and COPv in the AP (r_s=0.235, p=0.363) or ML direction (r_s=-0.005, p=0.985) during a single leg eyes closed task. There were no significant associations between LR change and the COP 95% ellipses for either eyes open or eyes closed single leg stance tasks (EO: r_s=-0.007, p=0.978, EC: r_s=-0.007, p=0.978).

Nonparametric Spearman's correlations were used to examine if a correlation existed between the change in COP in phase 1 and balance outcomes because the data was not normally distributed. No significant associations existed between COP change in phase 1 (initial contact) and COPv in the AP direction (r_s=0.068, p=0.803) or COPv in the ML direction (r_s=0.267, p=0.264) for single leg eyes open stance. There was no significant association between COP change in phase 1 and COPv in the AP (r_s=0.003, p=0.991) or ML direction (r_s=-0.032, p=0.905) for single leg eyes closed balance. There was a significant moderate positive association between COP change in phase 1 and the COP 95% ellipses with eyes open (r_s=0.529, p=0.035). This indicates that people with a greater change in COP at initial contact had a larger spatial area and therefore worse balance. The association between COP

change in phase 1 and the COP 95% ellipse with eyes closed was not significant (r_s=0.032, p=0.905).

To assess the relationship between balance outcomes and changes in COP in phase 2 of gait (the loading response, where peak vGRF occurs), nonparametric Spearman's correlations were used. No significant associations existed between COP change in phase 2 and COPv in the AP direction (r_s=0.068, p=0.803) or COPv in the ML direction (r_s=0.259, p=0.333) during single leg eyes open stance. There was no significant association between COP change in phase 2 and COPv in the AP (r_s=-0.179, p=0.506) or ML direction (r_s=-0.103, p=0.704) during single leg eyes closed balance. There was no significant association between COP change in phase 2 and the COP 95% ellipse with eyes open (r_s=0.406, p=0.119) or the COP 95% ellipse with eyes closed (r_s=-0.021, p=0.940).

	Balance outcome	Spearman's correlation coefficient (r_s)	Significance (p-value)
Loading Rate	COPv AP EO	-0.127	0.626
	COPv ML EO	-0.290	0.911
	COP 95% ellipse EO	-0.007	0.978
	COPv AP EC	0.235	0.363
	COPv ML EC	-0.005	0.985
	COP 95% ellipse EC	-0.007	0.978
COP change phase 1	COPv AP EO	0.068	0.803
	COPv ML EO	0.267	0.264
	COP 95% ellipse EO	0.529	*0.035*
	COPv AP EC	0.003	0.991
	COPv ML EC	-0.032	0.905
	COP 95% ellipse EC	0.032	0.905
COP change phase 2	COPv AP EO	0.068	0.803
	COPv ML EO	0.259	0.333
	COP 95% ellipse EO	0.406	0.119
	COPv AP EC	-0.179	0.506
	COPv ML EC	-0.103	0.704
	COP 95% ellipse EC	-0.021	0.940

Table 3: Correlation coefficients and significance for kinetic measures and balance outcomes. COP: center of pressure, COPv: center of pressure velocity, ML: mediolateral, AP: anteroposterior, EO: eyes open, EC: eyes closed.

Kinetics & Joint Position Sense

Pearson's correlations were utilized to examine the relationship between LR change and joint position sense and no significant relationship was identified (r_p=0.107, p=0.682). No relationship was found between JPS and COP change in phase 1 (r_s=-0.190, p=0.481), or between JPS and COP change in phase 2 (r_s=-0.190, p=0.481).

Kinetics & Plantar Cutaneous Threshold

Bivariate Pearson's correlations were conducted and identified a significant association between LR change and cutaneous threshold at the 1st metatarsophalangeal (MTP) joint (p=0.013). A moderate positive correlation (r_p=0.587) was found, signifying that people with greater changes in LR had higher cutaneous thresholds at the 1st MTP at baseline. No significant association was found between LR change and cutaneous threshold at the 5th MTP (r_p=0.031, p=0.907).

Spearman's correlations were used to examine the relationship between COP changes in phase 1 and cutaneous thresholds and no significant association was found at the 1st MTP (r_s=-0.040, p=0.882) or at the 5th MTP (r_s=-0.187, p=0.488). No significant associations existed between COP changes in phase 2 and cutaneous threshold at the 1st MTP (r_s=-0.195, p=0.469) or the 5th MTP (r_s=-0.399, p=0.126).

Q2c: Is there an association between kinetic changes and a baseline mechanical measure (foot type)?

Kinetics & Mechanical (Foot Type)

Multiple regressions with dummy coding were used to examine associations between kinetic variables and the categorical predictor of foot type. Pearson's correlations found no significant associations between LR change and a planus foot compared to a neutral foot (r_p=-0.373, p=0.070). There were no significant associations between LR change and a cavus foot compared to a neutral foot

(r_p=0.091, p=0.364), or a cavus foot compared to a planus foot (r_p=-0.270, p=0.148). (See Table 3 for 95% confidence intervals).

Multiple regressions were used to examine relationships between COP change in phase 1 and foot type. A statistically significant association was identified between COP change in phase 1 and a cavus foot compared to a neutral foot (β=-9.368, p=0.040, 95% CI [-18.257, -0.480]), indicating that people with a cavus foot had less change in COP at initial contact compared to those with a neutral foot. There were no significant associations found between COP change in phase 1 and a planus foot compared to neutral. There were no significant associations found between COP change in phase 1 and a planus foot compared to cavus (See Table 4). There were no significant associations present between COP change in phase 2 (loading response) and any foot type. No significant associations existed for COP change in phase 2 and planus compared to neutral, cavus compared to neutral, or planus compared to cavus (See Table 4).

	Condition	β coefficient	Lower bound	Upper bound	Significance
Loading Rate Change	Planus vs neutral	-2.189	-5.407	1.029	0.167
	Cavus vs neutral	-0.088	-4.861	4.685	0.969
	Planus vs cavus	-2.101	-7.086	2.885	0.381
Center of Pressure change Phase 1	Planus vs neutral	-2.635	-8.977	3.707	0.386
	Cavus vs neutral	-9.368	-18.257	-0.480	*0.040*
	Planus vs cavus	6.733	-2.780	16.246	0.150
Center of Pressure change Phase 2	Planus vs neutral	-2.174	-8.116	3.768	0.443
	Cavus vs neutral	-6.248	-14.576	2.080	0.129
	Planus vs cavus	4.074	-4.839	12.987	0.341

Table 4: 95% confidence intervals for kinetic variables and foot type.

CHAPTER 5

DISCUSSION

The purpose of this investigation was to examine the effects of a vibration biofeedback intervention, aimed at improving COP location, on vGRF loading rates, and if the kinetic effects of this intervention are associated with common CAI impairments in the mechanical, sensorimotor, or perceptual domains. We found a significant decrease in LR from baseline to posttest after the vibration feedback gait retraining in the laboratory setting. However, we did not see this decrease retained. Our hypothesis that LR would decrease and would be retained was partially supported by our results for the laboratory setting. Our hypothesis of finding greater changes in the laboratory setting compared to RW was supported because a no change was found after the RW session.

For Aim 2, we found several significant associations. A moderate positive correlation between LR change and the IdFAI indicates that individuals with lower functional abilities had a larger change in LR, contradicting our hypothesis that patient-reported outcomes would associate with kinetic changes. We found moderate positive associations between kinetic variables and measures of sensorimotor function (e.g. COP 95% ellipses for eyes open balance, cutaneous threshold at the 1st MTP joint). These associations suggest that those with worse sensorimotor function had larger changes in the kinetic variables following the intervention. Our hypotheses were partially supported. We also identified an

association between COP change in phase 1 and a cavus foot compared to neutral, suggesting that people with a cavus foot had less change in COP contrary to our hypothesis.

While this investigation is preliminary in nature, we believe the results are valid as our sample demonstrates consistency in key metrics with the existing literature. For example, Blackburn et al[110] found significantly higher instantaneous LR in ACLR limbs compared to contralateral limbs during walking, but found no difference in peak vGRF. Given what the literature shows about ACLR and PTOA, this provides evidence to support our investigation into LR as a key variable that plays a role in PTOA development. Additionally, the CAI metrics of our sample are similar to those reported in the literature. For example, our sample had a mean IdFAI score of 21.47 $\pm$ 4.39 while Torp et al[27] reported a mean of 21.2 $\pm$ 3.7, and Donovan et al[46] reported a mean of 23.6 $\pm$ 5.3.

Kinetic Variables

Migel et al[102] was the first to investigate biomechanical gait alterations after gait retraining in the real world setting, and this study is the first to examine LR, so we are unable to compare our findings with much other relevant literature. Previous research found that individuals with CAI had a higher loading rate compared to a control group, signifying that these individuals have less ability to dampen vGRF.[38,39,59] Although our investigation did not use a control group to compare directly between the typical gait patterns of CAI and healthy individuals, we found that when our intervention was utilized to alter the lateral COP location during gait typical of the CAI population, loading rate decreased. Several prior studies have investigated kinetic changes with different types of biofeedback. Torp et al[27]

examined COP location and peak pressure changes with laser guided visual feedback and found a medial shift in COP. Donovan et al[46] examined peak pressure changes with auditory feedback and found higher pressures in the medial foot and decreased peak pressure under the lateral foot. Migel et al[102] found that vibration haptic feedback was effective at creating a medial shift in COP location. Because we also found that haptic feedback decreases LR, it could be insightful to replicate the methods of these other studies[27,46] but examine LR change as a primary outcome to see if these other gait retraining biofeedback tools can also decrease LR.

From this same research study data collection, Migel et al[102] examined the change in COP location. Immediate medial shifts (improvements) in COP location over the first 90% of the stance phase were found after the laboratory gait retraining session, and following the real world session, COP location changes occurred during the first 70% of the stance phase.[102] These changes were retained for at least 5 minutes in both lab and RW.[102] In contrast, from our data examination, vGRF LR changes were only present immediately after the laboratory training and not after the RW training, and these changes were not retained. Our vibration feedback tool was designed specifically to alter COP location, not LR, so it is reasonable that we would find more meaningful changes in COP than LR. A reduced vGRF LR is important for mitigating PTOA development so reducing vGRF as a secondary benefit from this intervention is positive. Larger changes in vGRF LR are likely achievable using a variety of tools that are specifically designed to altered LR during gait.

No prior research exists to examine the ability of gait retraining with biofeedback to minimize vGRF LR in CAI patients. However, in an article published

using the same methods examining different outcomes, Migel et al[102] found significant medial shifts in COP that were also retained for at least 5 minutes. It is likely that multiple training sessions over time could create a change in LR, since COP location was able to be changed after only one training session. Future investigations should include multiple sessions of both laboratory and real world training to elucidate the full potential effects of vibration feedback on gait biomechanics because we saw benefits in the laboratory setting but did not examine multiple training sessions for either setting.

However, a study conducted by Chan et al[111] to examine the effects of midfoot strike gait retraining in healthy people used multiple sessions over time, and no change in LR was found. The participants completed eight gait retraining sessions over two weeks, with sessions gradually increasing from 15 to 30 minutes long, and feedback was gradually removed over the last 4 training sessions.[111] The study's goal was to shift runners from a rearfoot strike to midfoot strike pattern using visual feedback, and participants ran on a treadmill with a real-time visual feedback diagram showing their foot strike patterns.[111] This gait retraining intervention did not find any differences in vGRF LR, and they also did not find a difference in promoting a midfoot strike versus rearfoot strike after the visual feedback was removed.[111] Although this study differed from ours in the purpose, population, methods, and intervention and they did not find a significant difference in LR, their multi-session visual feedback gait retraining protocol suggests an even larger training volume may be needed to permanently change LR in those with CAI.

Associations

This is the first study of its kind to investigate the relationships between baseline outcomes in three domains of CAI (sensorimotor, perceptual, and mechanical) and kinetic changes following a gait retraining intervention therefore we cannot compare our findings to results from other studies. The associations found suggest that individuals with CAI who have lower functional abilities represented by a higher IdFAI score and worse balance (a greater COP 95% ellipse) are more likely to have a large positive response (i.e., large medial COP shift) to the laboratory vibration training intervention. This is important because a more inverted position of the ankle when initial contact with the ground occurs is a highly vulnerable position for the ankle to roll into inversion and sustain an LAS.[11,33] While future research is needed, the results suggest that athletic trainers and other rehabilitation specialists can identify higher-risk individuals and implement prevention strategies, specifically gait retraining, to attempt to reduce the risk of recurrent ankle sprains in those with CAI.

Although we only saw associations between COP location change in phase 1 and the baseline outcomes of eyes open balance and a cavus foot compared to neutral foot, it could still be meaningful that these individuals with a cavus foot are less able to alter COP location, potentially identifying them as non-responders to the intervention, and it could be meaningful that the individuals with higher COP 95% ellipse are more likely to alter their COP location which may help them to prevent this common mechanism of LAS. Individuals with a cavus foot (higher longitudinal arch) may tend to walk with a more laterally located COP than those with a neutral or planus foot, so they may be more susceptible to repeated episodes of inversion

ankle sprains.[76,77] Our results indicate that a cavus foot type could be a predictor of individuals who are less able to create changes in their gait with this intervention, helping to identify potential “responder” and “non-responder” groups to target with either gait retraining or alternative interventions. Further research is needed to identify if these baseline variables can predict the magnitude of change following a prolonged intervention.

Limitations

There were limitations present with this investigation. This study does not consider kinematics or muscle activity that could explain the change or lack thereof in vGRF LR. Additionally, we only implemented one session of gait training for approximately 10-15 minutes, limiting the time in which the participants were able to learn and adapt their gait to the feedback. Throughout the gait retraining, shoe type was not standardized beyond instructions for individuals to wear athletic shoes. Because the participants wore their own shoes, the type and amount of cushion in shoes varied, which has an impact on LR. In the laboratory setting, the gait retraining task utilized a standardized surface of the instrumented treadmill, so it was anticipated that training benefits would be more consistent than from the real world gait retraining session. The variation of sidewalks as walking surfaces, other external feedback including other pedestrians, and numerous other uncontrollable variables in the RW setting may have made it difficult to consistently make gains from the RW training session. Participants may have created meaningful changes in kinetic variables during the RW training session, but because we weren’t collecting data during training and the posttest assessment was conducted several minutes later and not in the RW setting, changes may not have translated to the posttest data

collection. Additionally, we had a small sample size for this study, which may limit the clinical significance of our data. Because of the inclusion criteria for our study to involve only individuals with CAI, the perceptual outcome measures also had small ranges, which was a limiting factor in the correlations.

Clinical Implications and Future Research

We only saw small decreases in LR after the gait retraining intervention. However, small reductions with each step can result in large cumulative reductions when the number of steps an individual takes over the course of a day, week, year, etc. is considered. These small changes in LR could be meaningful to decrease joint loading. Individuals who develop PTOA after a LAS often show degenerative cartilage changes within several months after the acute injury, which suggests that if we can identify and alter gait mechanics soon after an injury, there is a potential for the individual to avoid cartilaginous degeneration and avoid the development of PTOA.[21,57] After gait retraining in the RW setting, we did not find significant changes in LR, but it's possible that changes existed but were too small to be significant. RW gait retraining could still be effective in creating small alterations in joint loading that have a positive effect of decreasing cartilage loading over many steps and prevent PTOA.

Further research into this vibration feedback gait retraining intervention is needed to consistently identify the characteristics of individuals who will respond to the biofeedback intervention and make kinetic adaptations. Additionally, using multiple sessions of vibration feedback gait retraining may be able to identify changes in vGRF LR in the RW setting that were not found in this study, and may also show larger changes in LR following the laboratory training session. Examining

the associations between kinetic variables and the baseline measures with both CAI patients and healthy controls could help to identify more meaningful relationships between these variables. Future research utilizing both a CAI group and a control group of healthy individuals, as well as a larger sample size, would help further support these results and could also identify if these baseline outcome measures could be used to predict people who are more likely to respond to the intervention and create the desired biomechanical changes.

Conclusion

This study is the first to examine the relationships between baseline outcome measures and kinetic changes after vibration feedback gait retraining in the real world and laboratory settings for individuals with CAI. We found that gait retraining can improve gait mechanics in this population after laboratory training but not RW training, and that improvements in these gait mechanics are associated with the baseline outcomes of worse functional abilities, worse balance, and a cavus foot type. Our results provide preliminary support for further use of this vibration feedback tool in the CAI population to alter LR during gait and provide insight into characteristics of individuals who may respond and create changes with gait retraining.

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